Second Beijing Forum
on Parkinson's disease and Movement Disorders
September 14-17, 2012

Introduction to
Basal Ganglia
Anatomy, Physiology, Physiopathology

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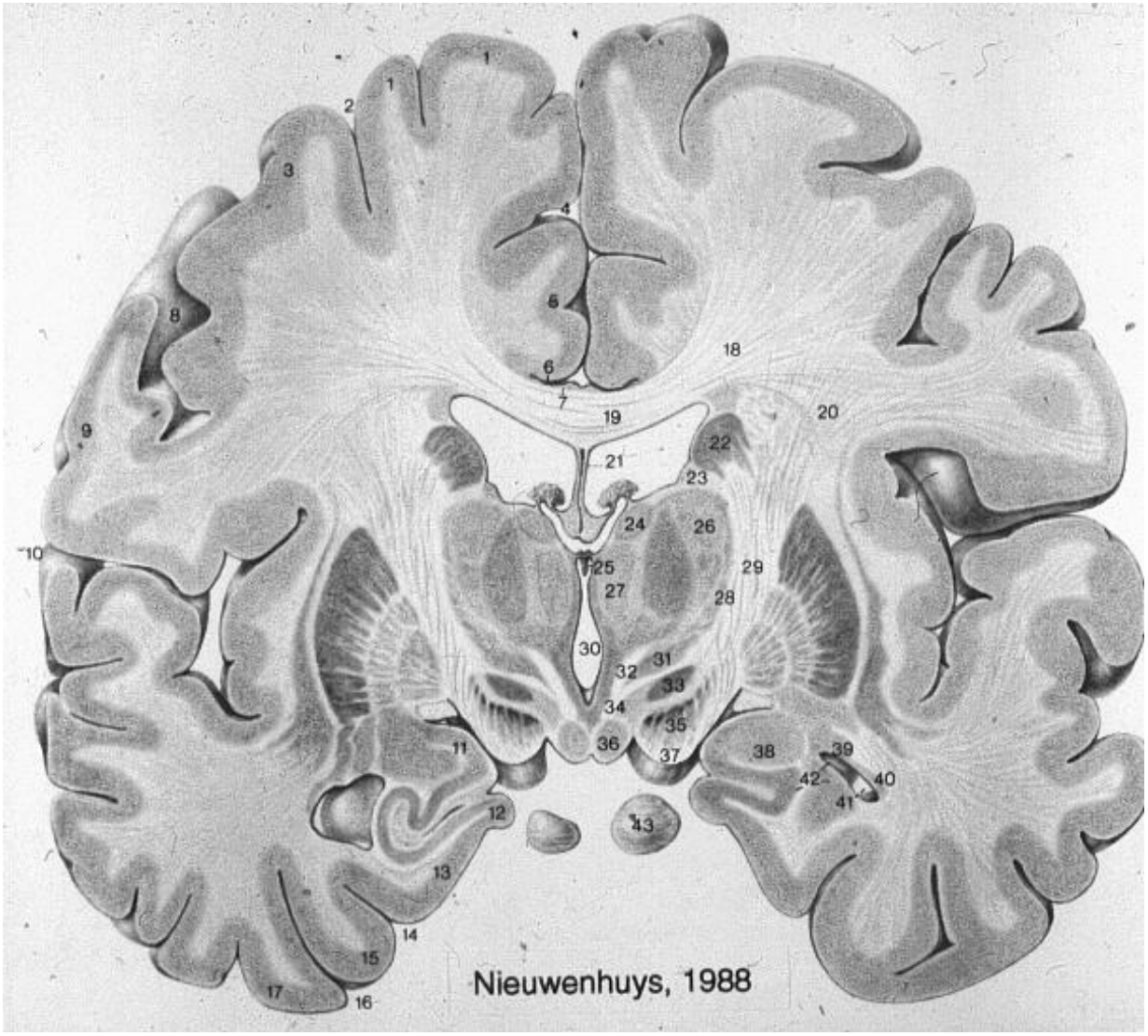
Sorbonne Université,

Paris, France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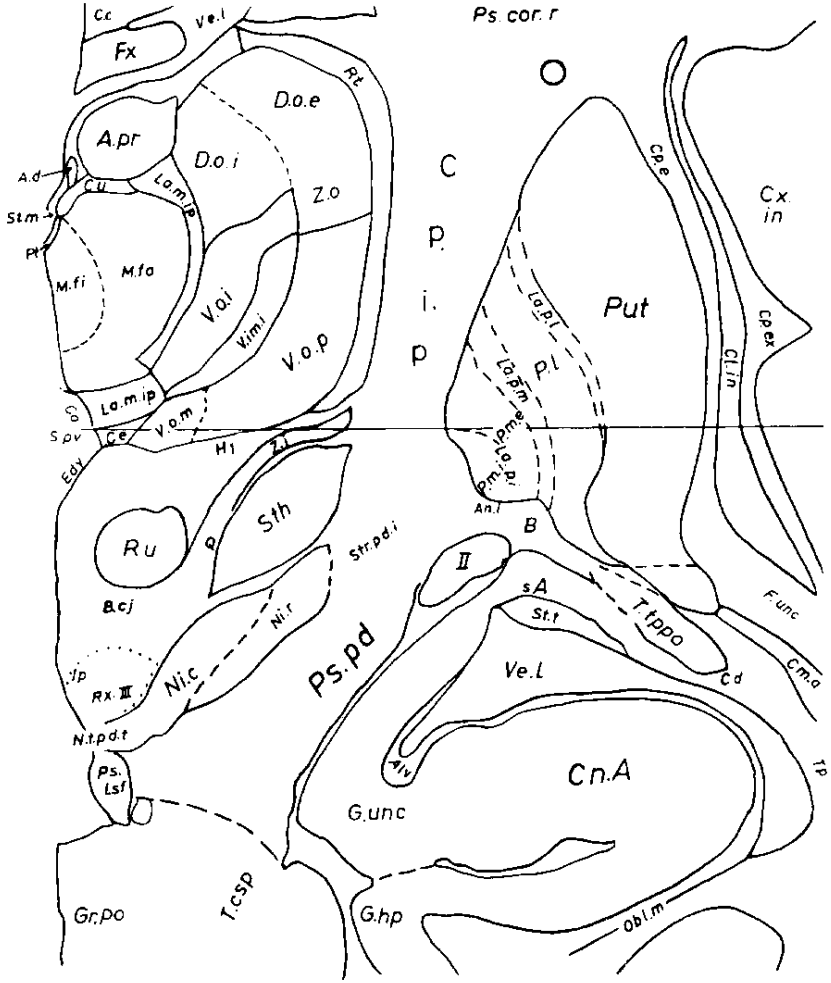
Nieuwenhuys, 1988

Swfp30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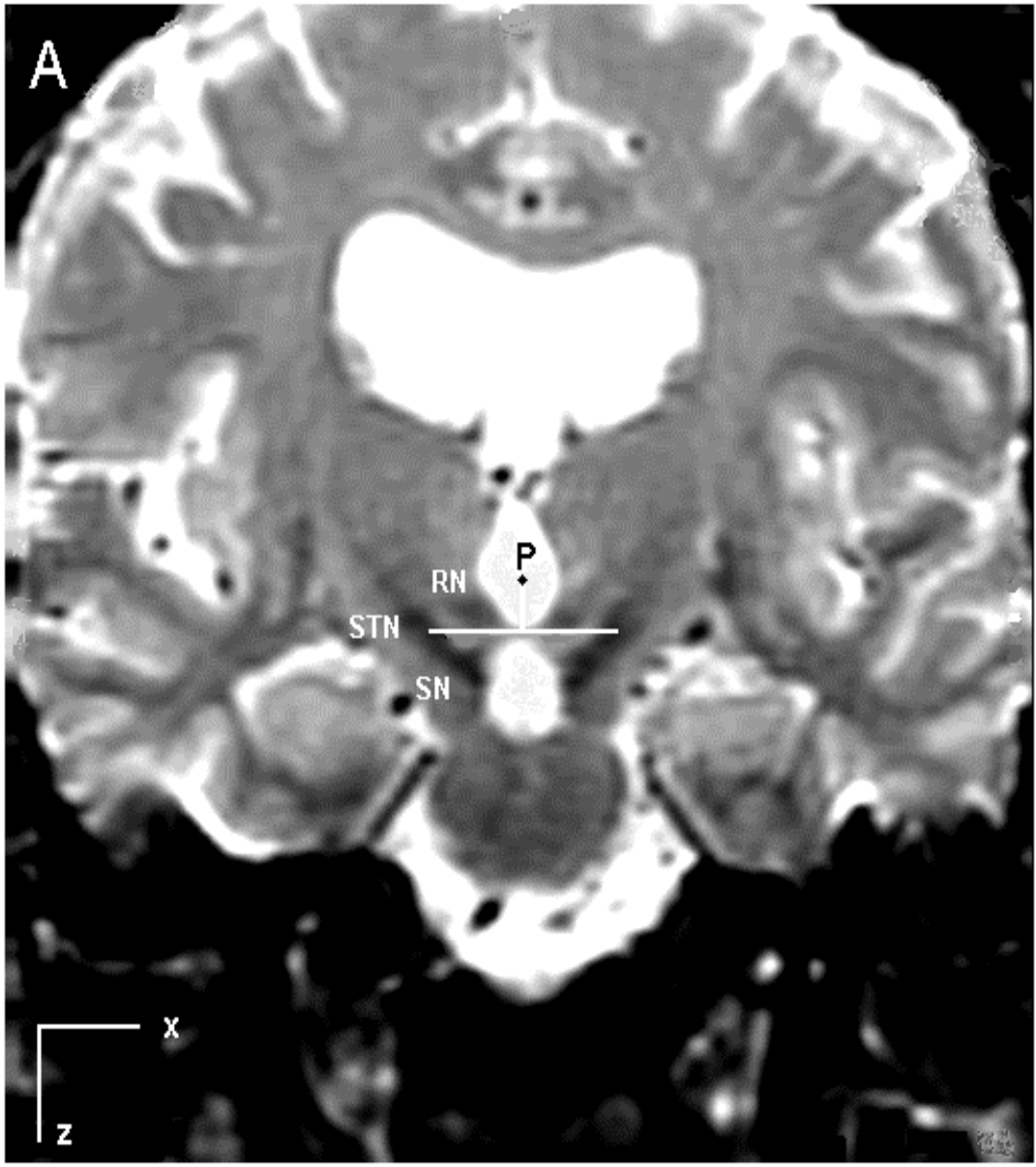
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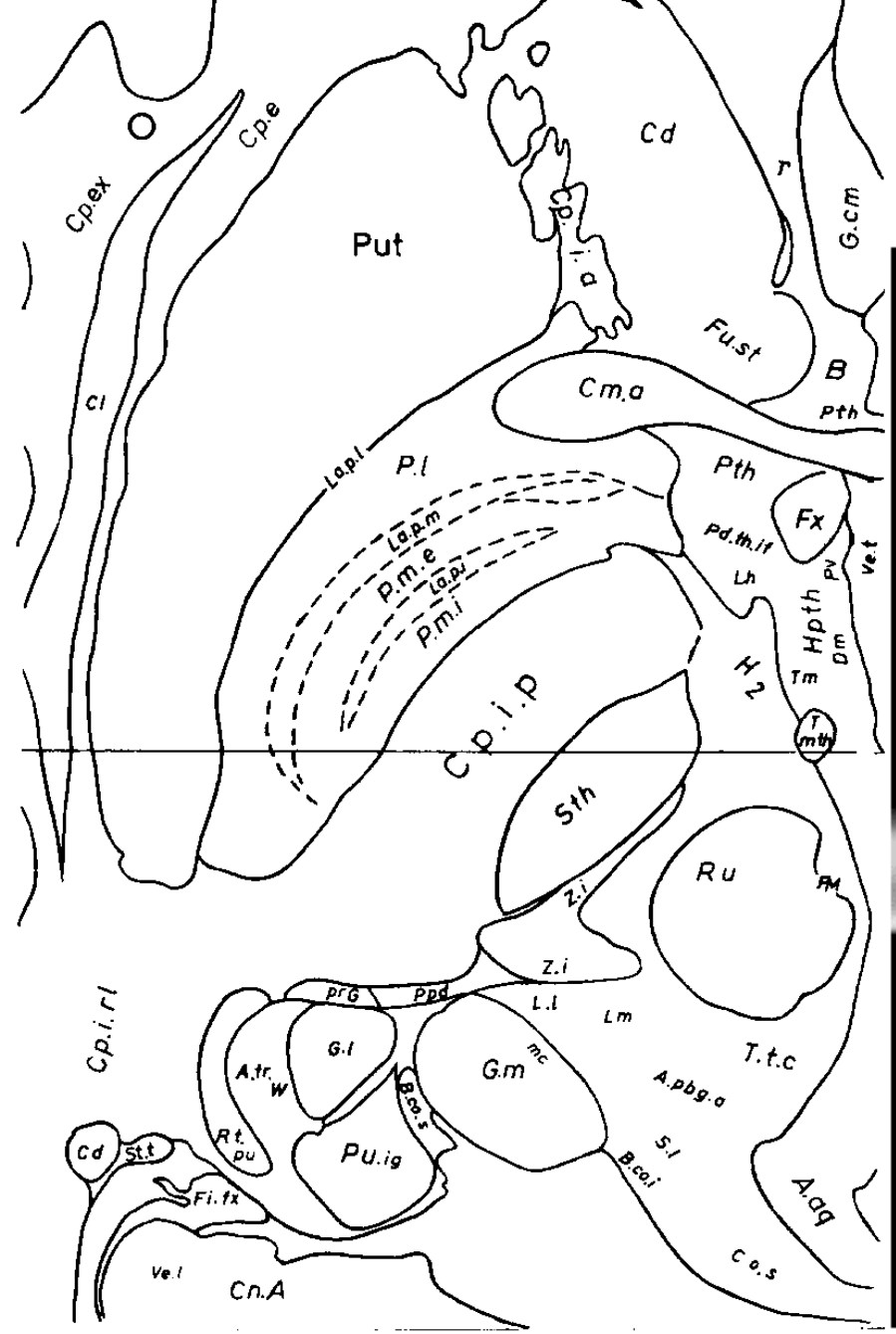
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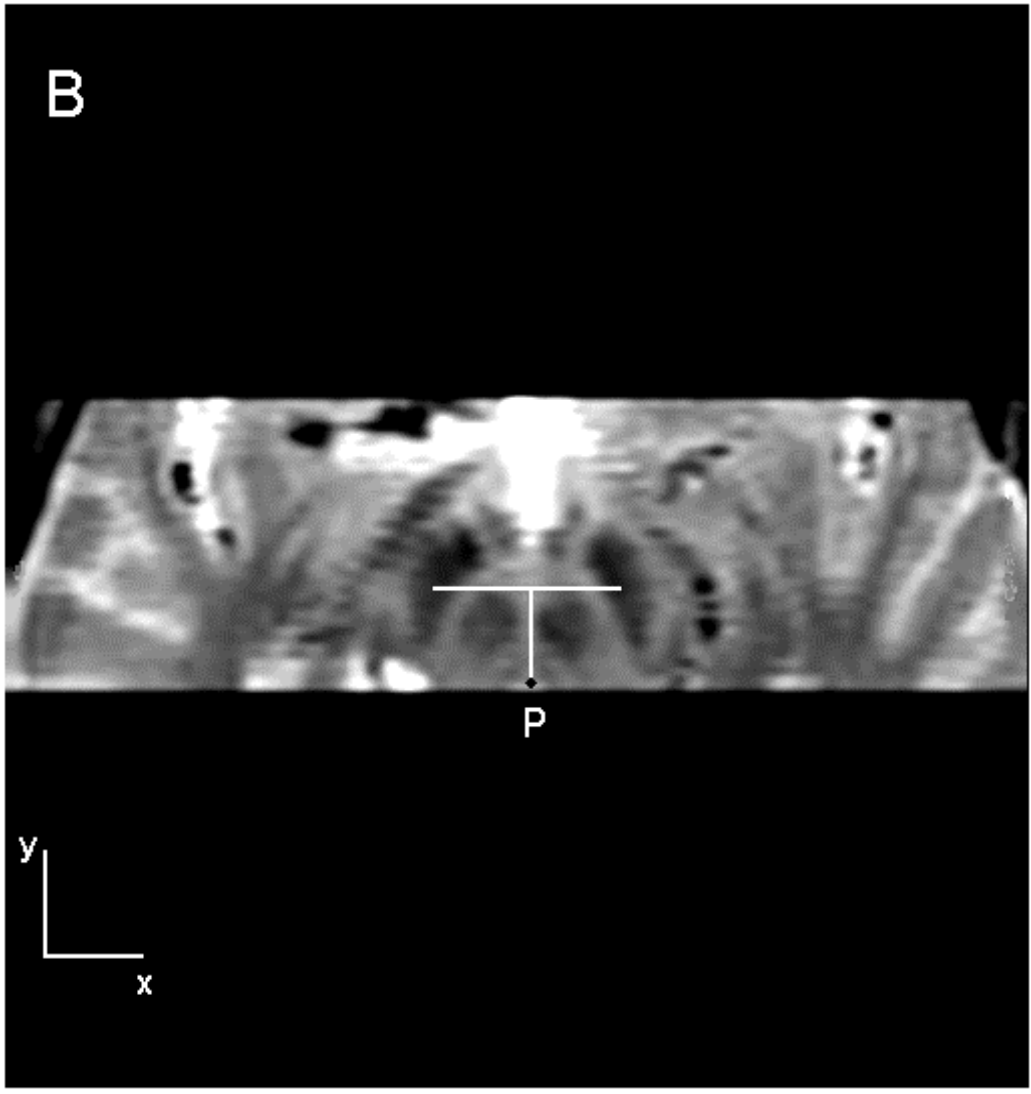
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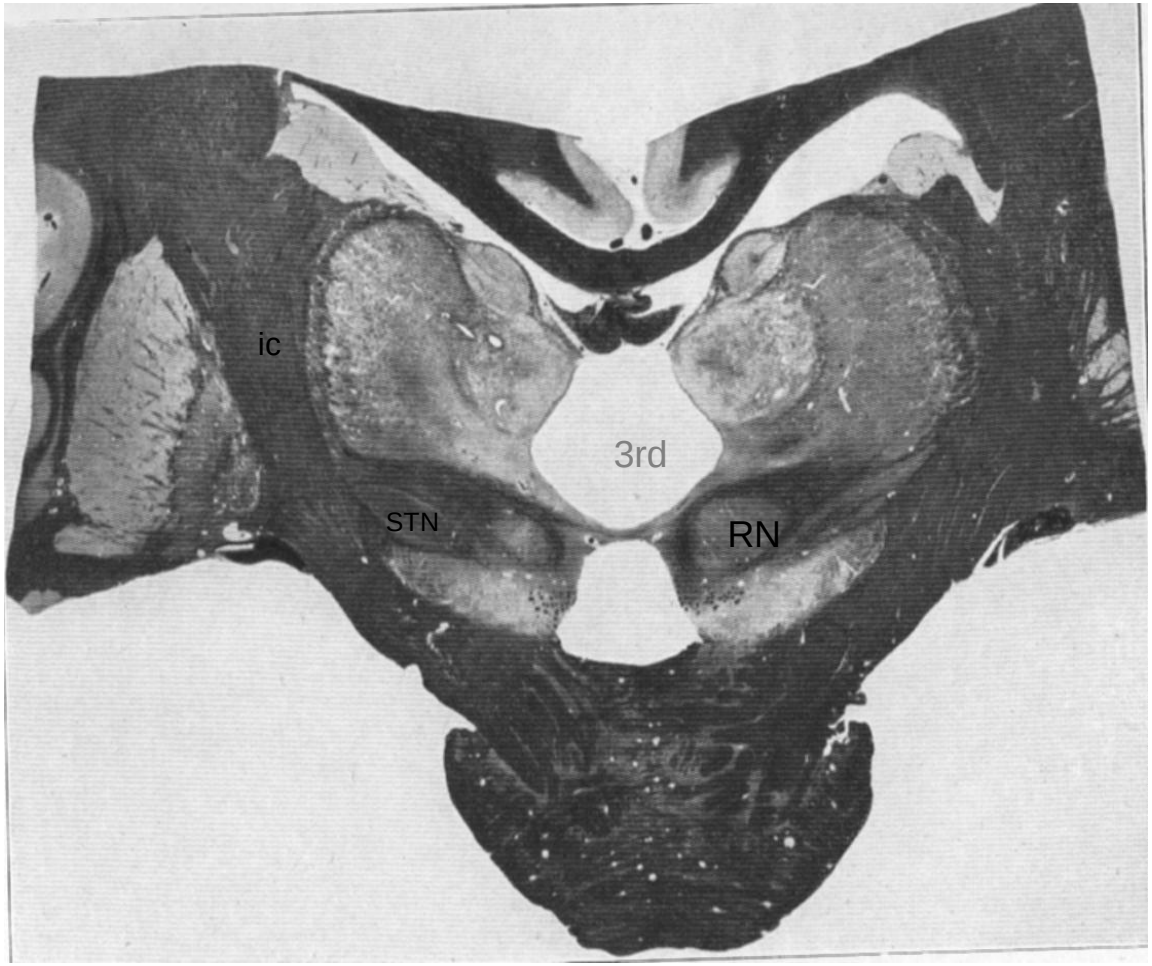


Fig 71.- Coupe frontale passant par le tiers antérieur du noyau rouge (Foix et Nicolesco, Masson 1925)

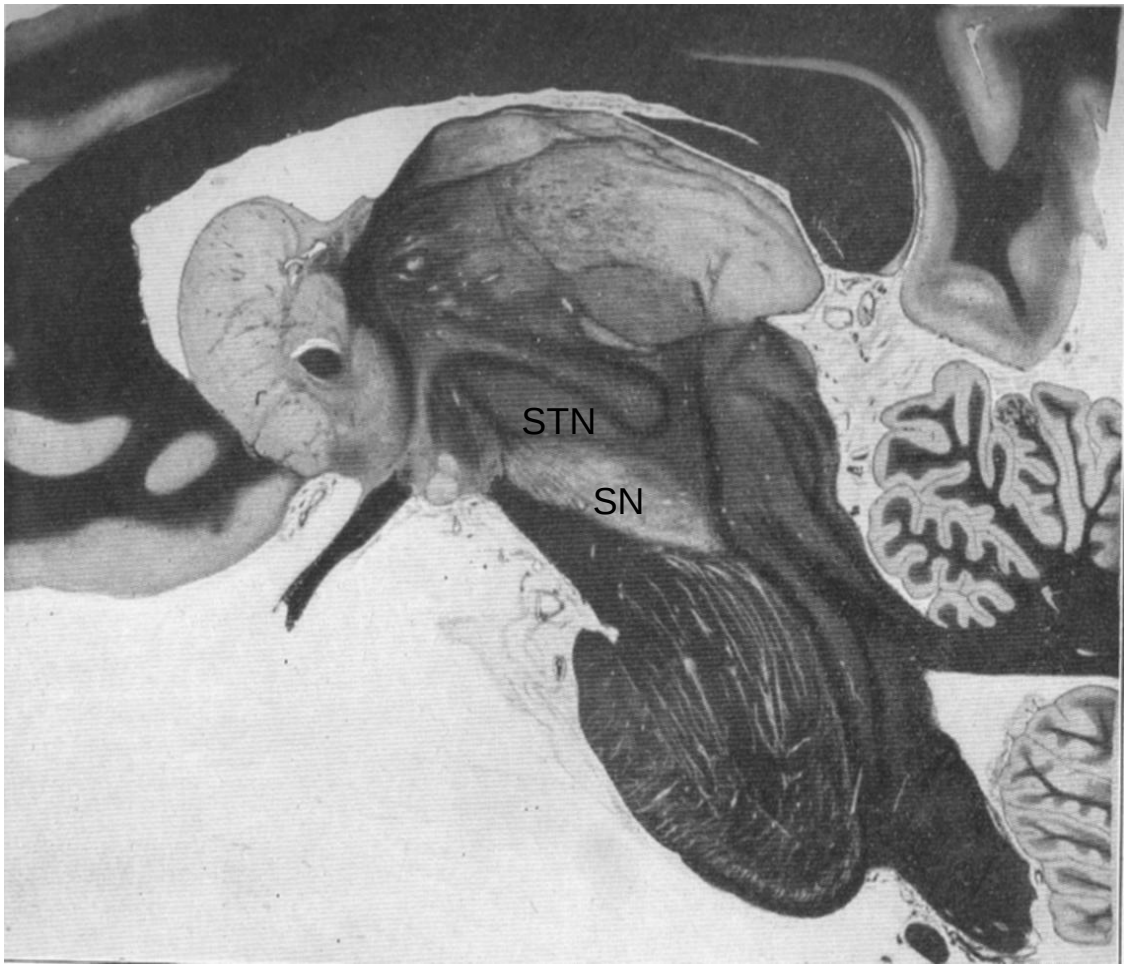
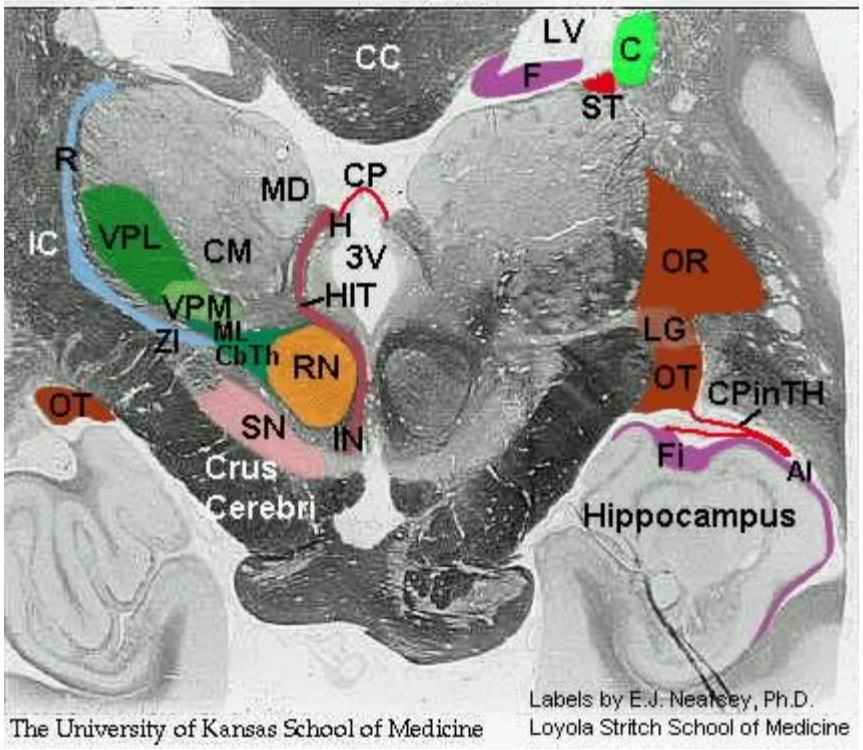


Fig 135.- Coupe Sagittale région sous optique, colliculus du noyau caudé; locus niger, centre médian de Luys (Foix et Nicolesco, Masson 1925).

Slide 9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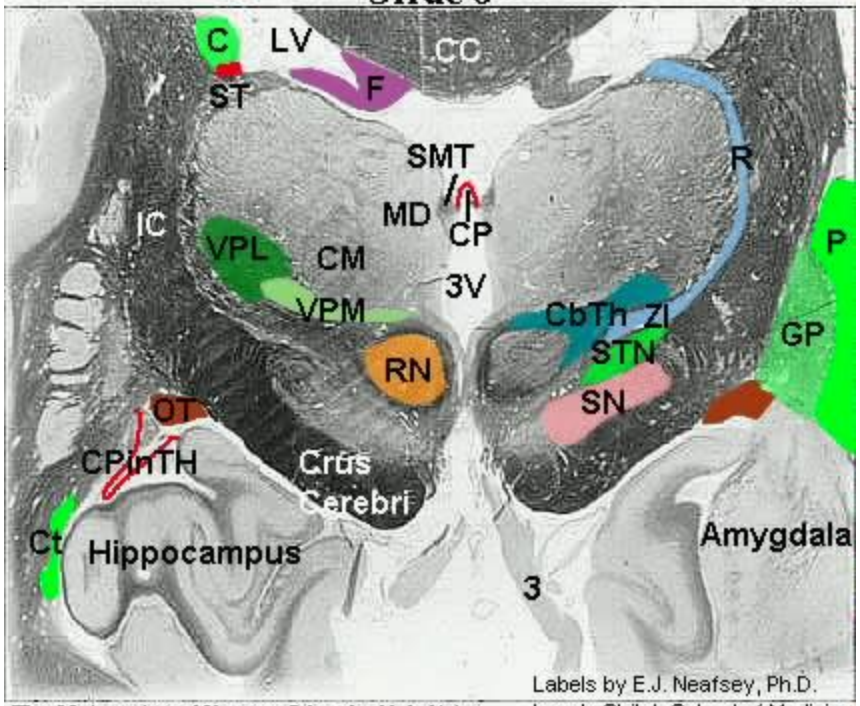
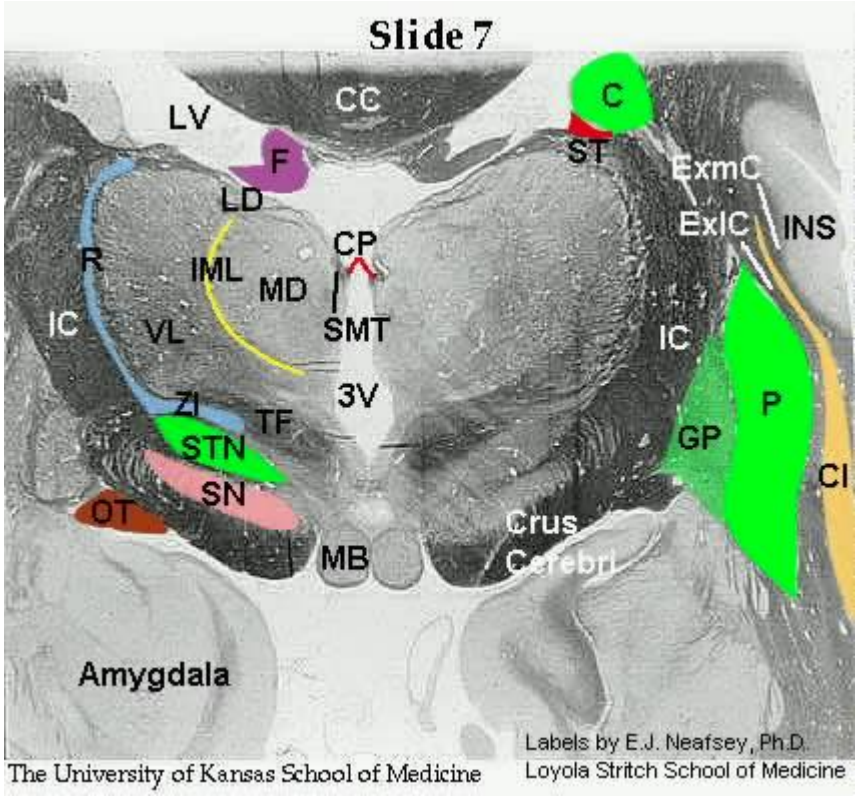
Cerebri
Hippocampus
Labels by E.J. Neafsey, Ph.D.
Loyola Stritch School of Medicine
The University of Kansas School of Medicine

Slide 8



The University of Kansas School of Medicine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Labels by E.J. Neafsey, Ph.D.
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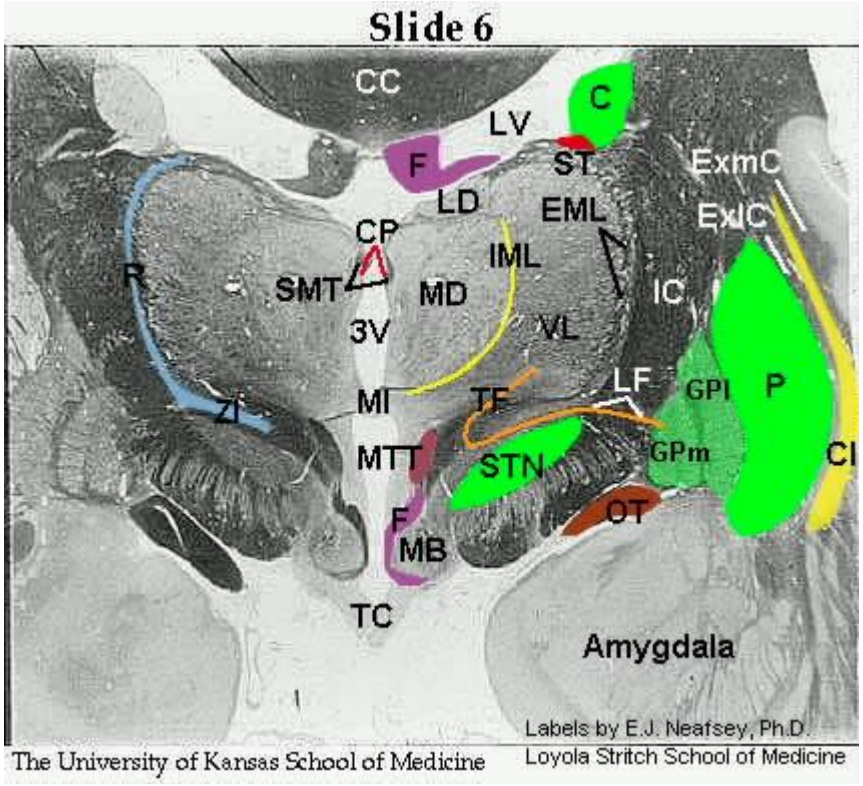
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Labels by E.J. Neafsey, Ph.D.
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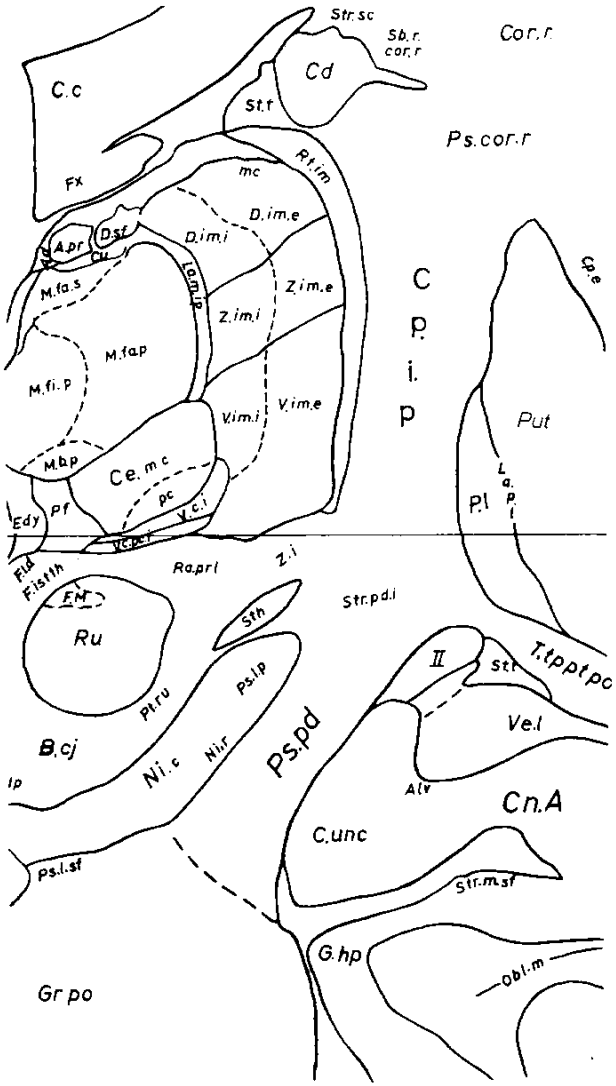
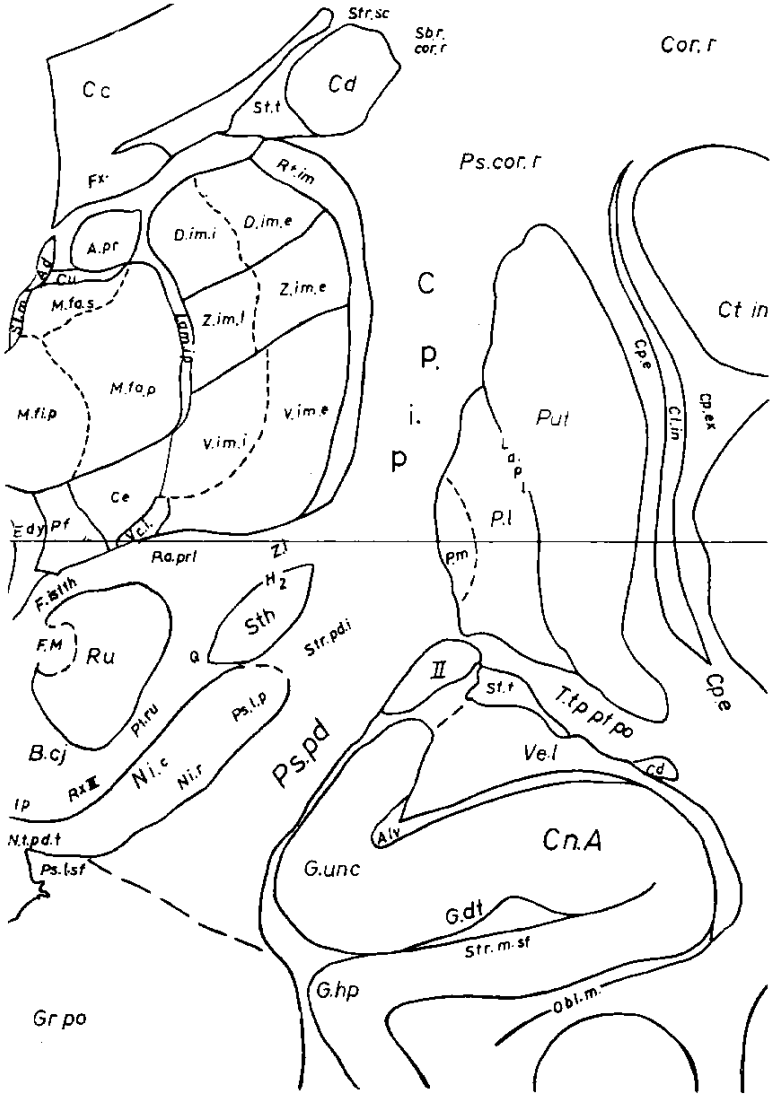
Schaltenbrand
& Wahren atlas



Frontal view
AC-PC plane

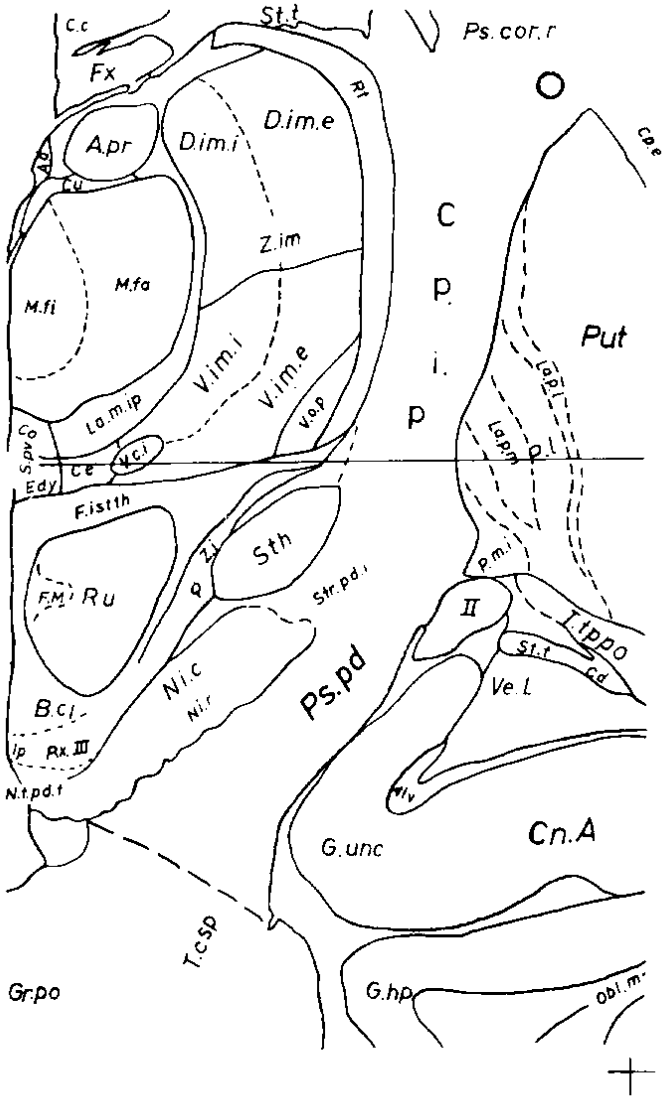
Swfp70

Schaltenbrand
& Wahren atlas



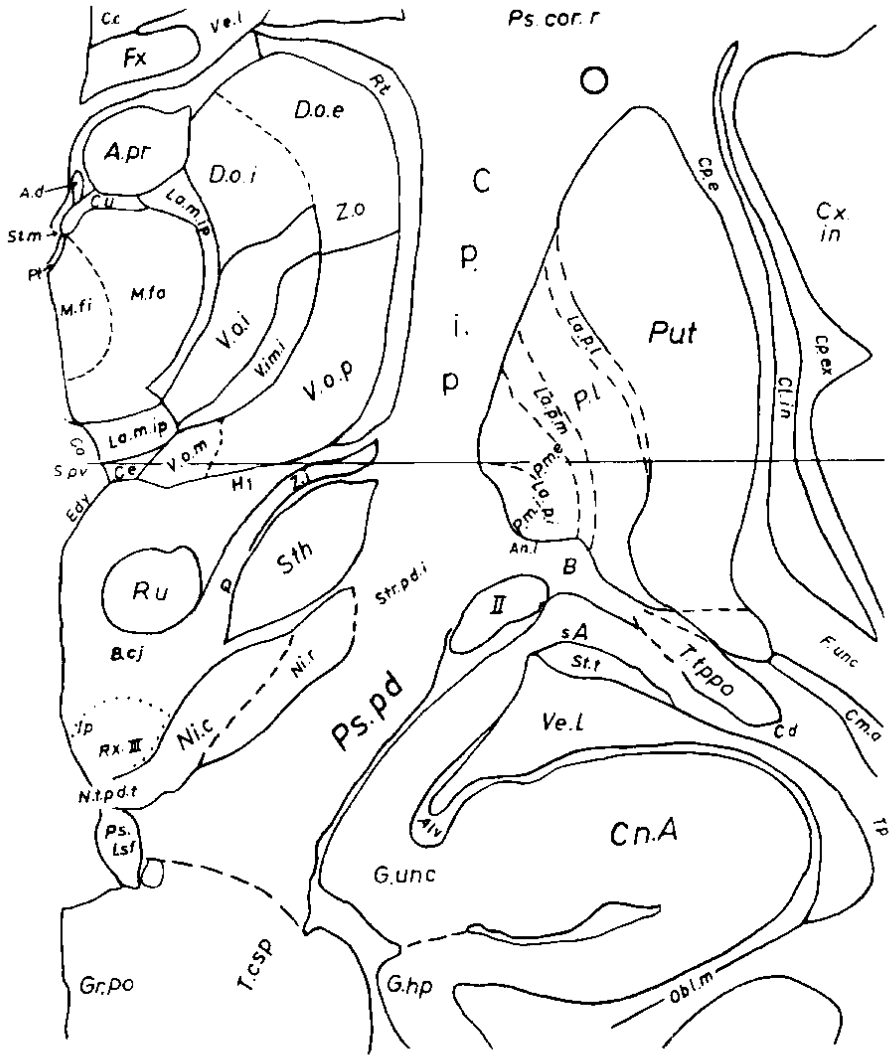
Frontal view
AC-PC plane

Swfp50



Frontal view
AC-PC plane

Swfp40



Frontal view
AC-PC plane

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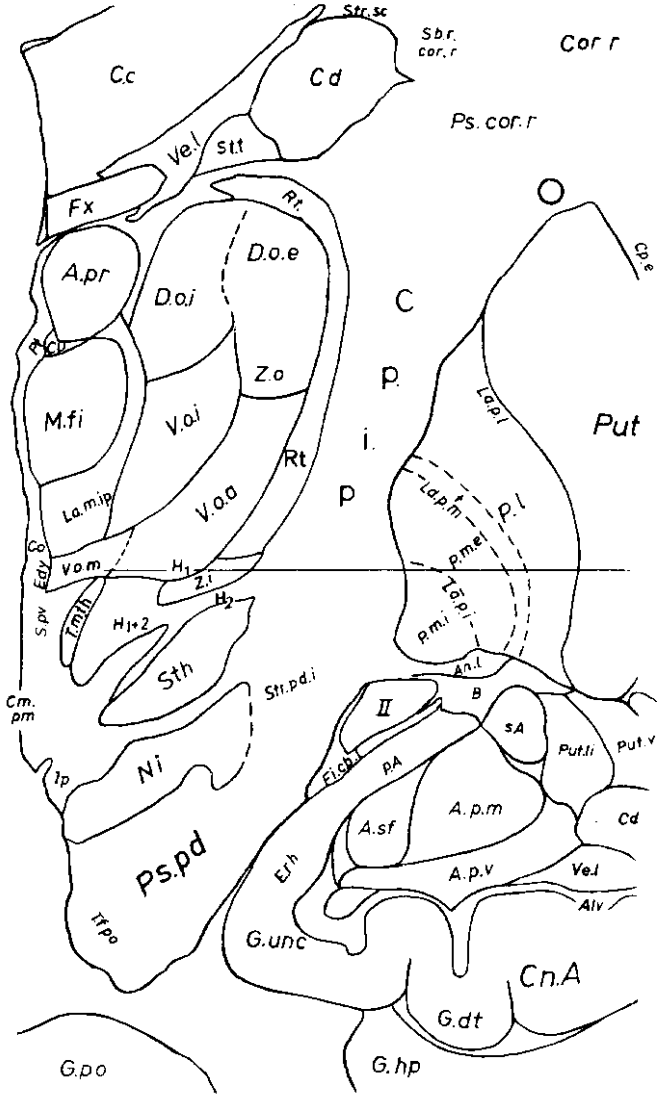

Frontal view
AC-PC plane

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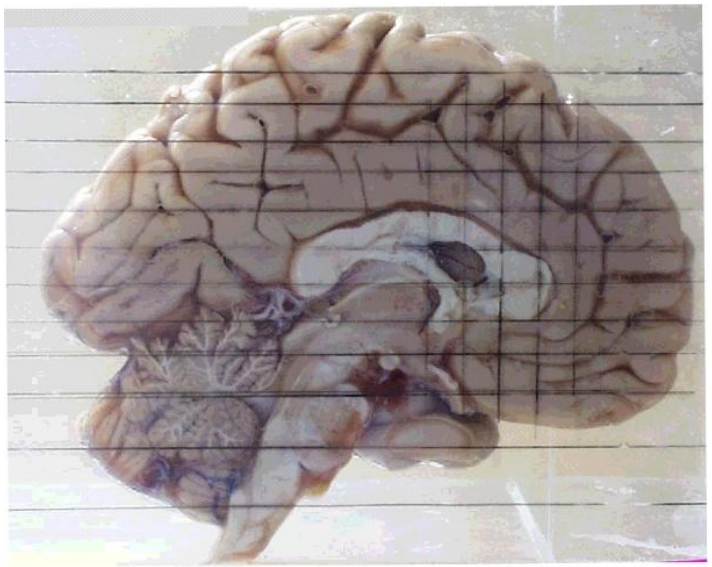

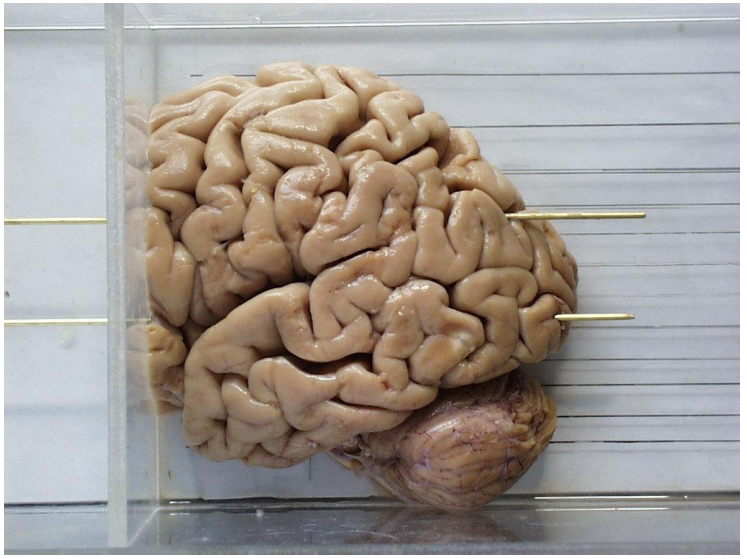

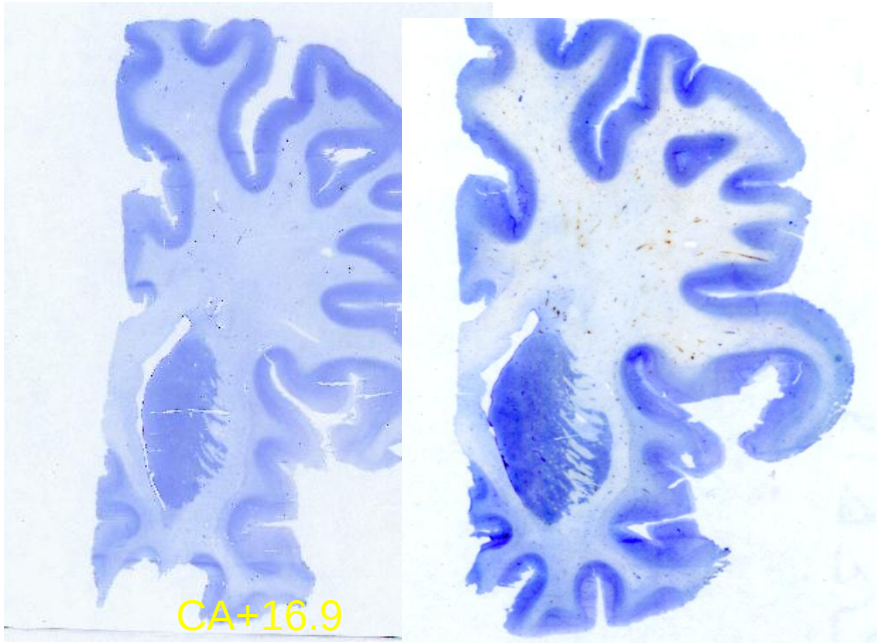
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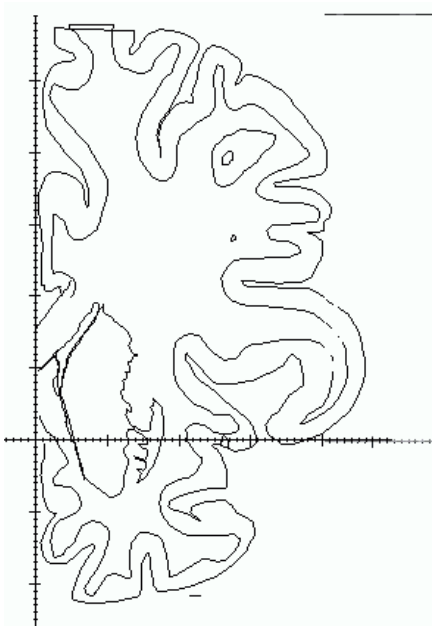
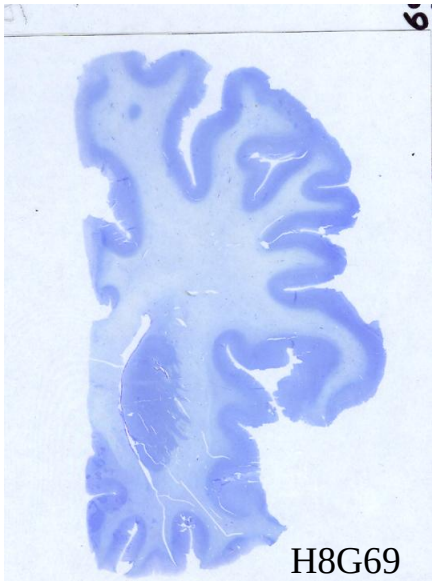
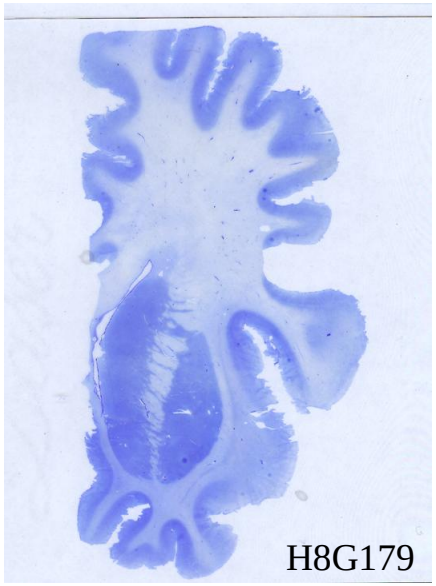


Functional


H8G69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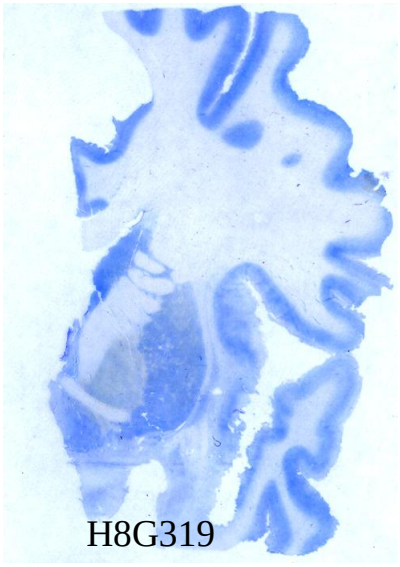
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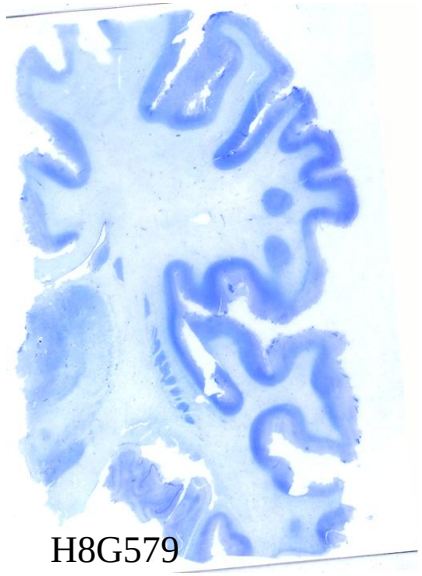
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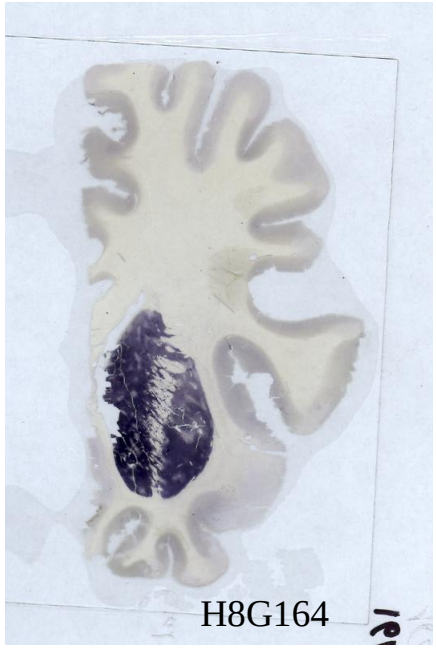
H8G54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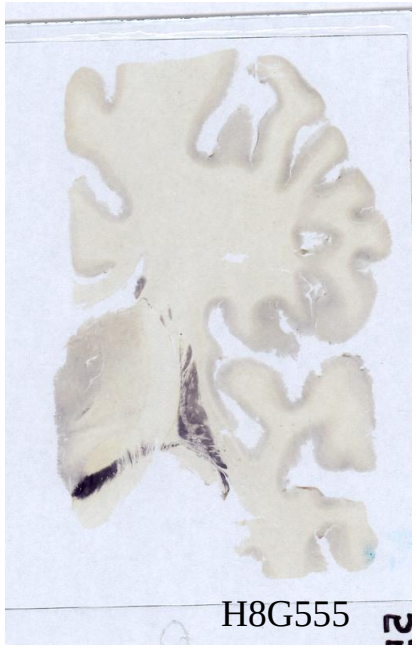
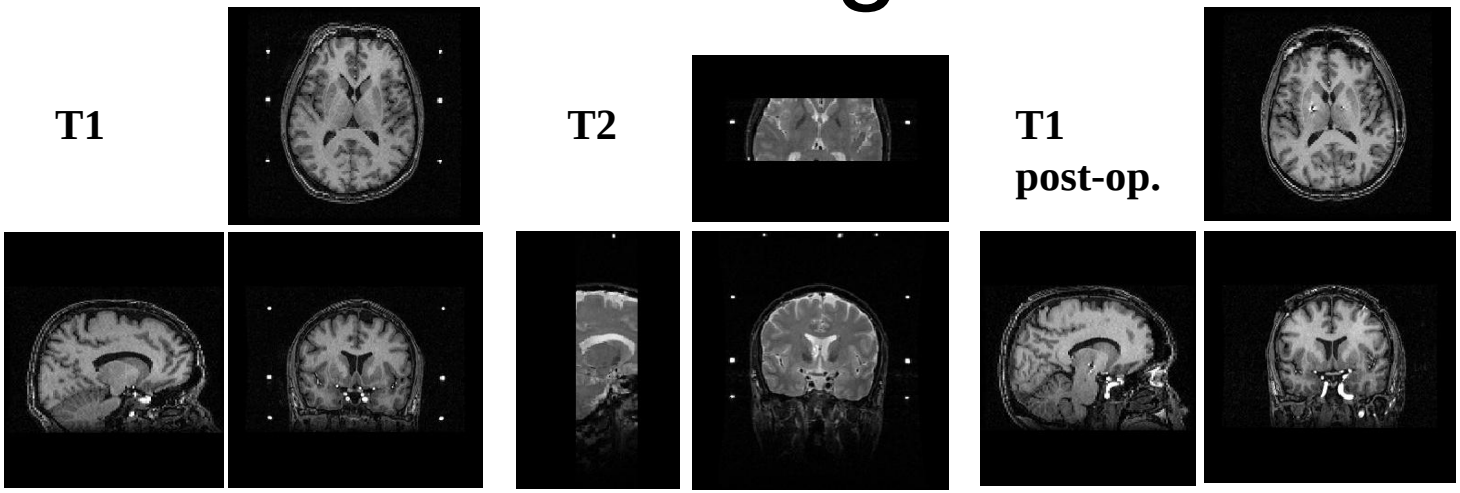
H8G345


H8G555

ATLAS/MRI Registration

Patient

T1

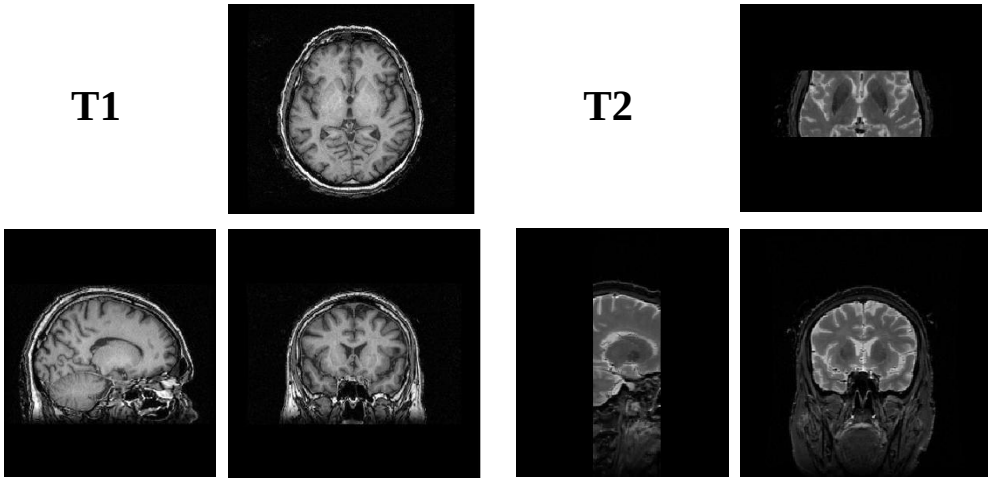


T2

T1 post-op.

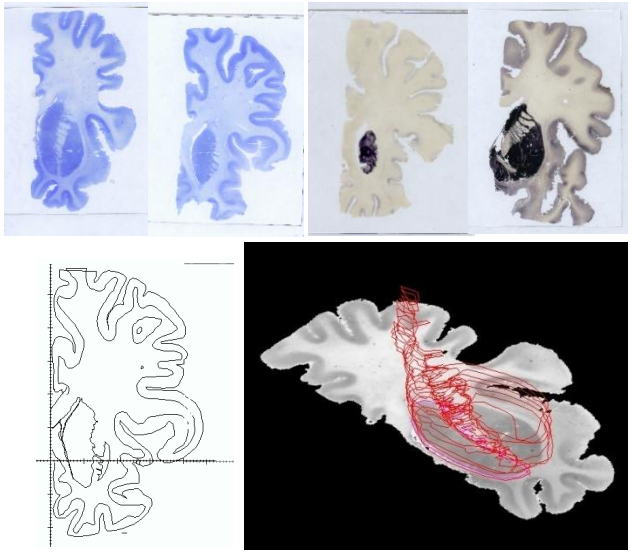
Atlas

T1



T2

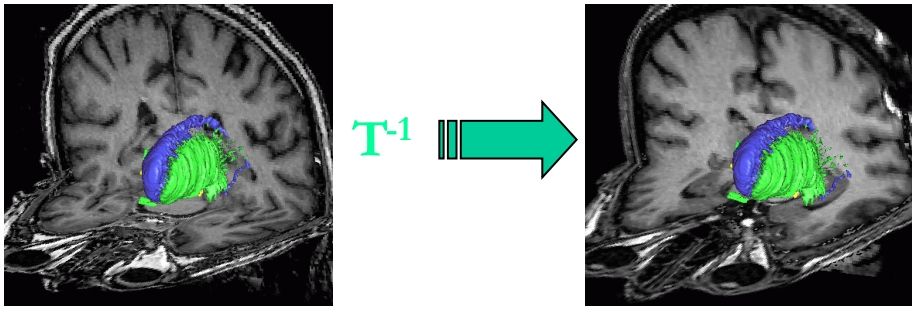
Histology



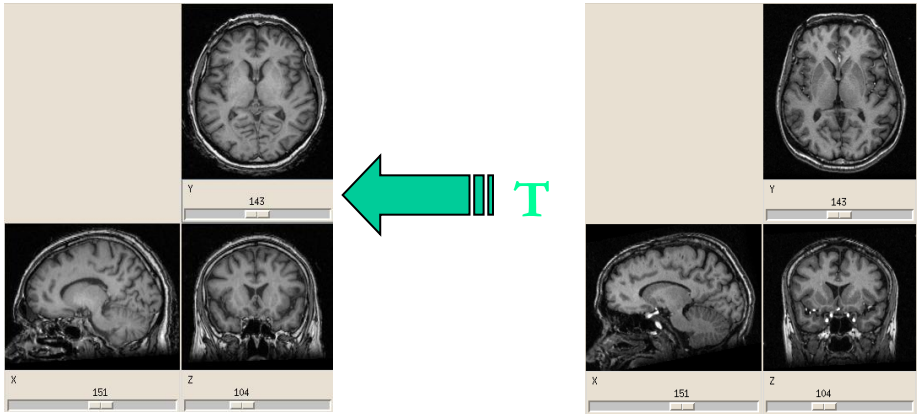
Atlas : deformation

Atlas

Patient

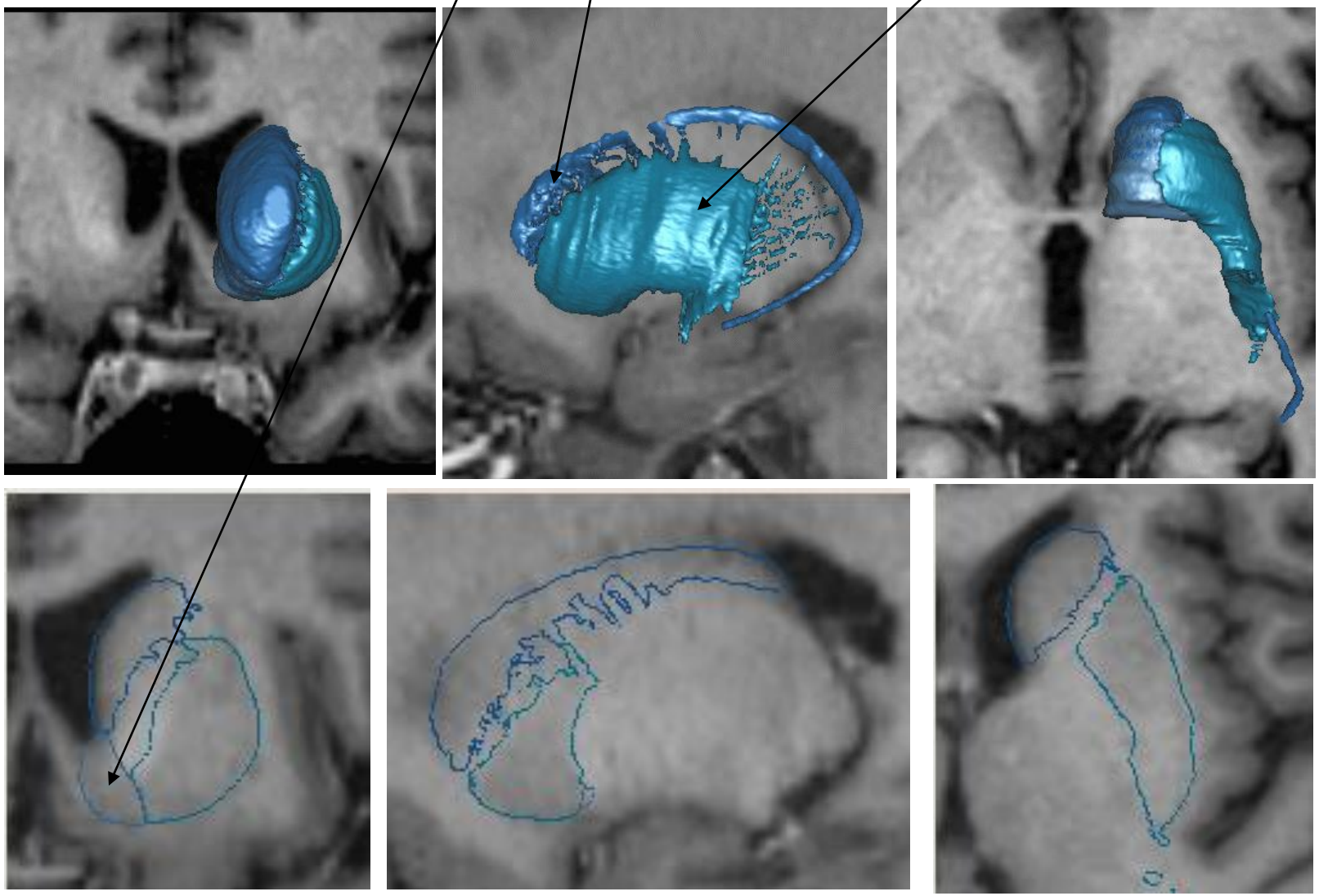


Report atlas structures
on the patient

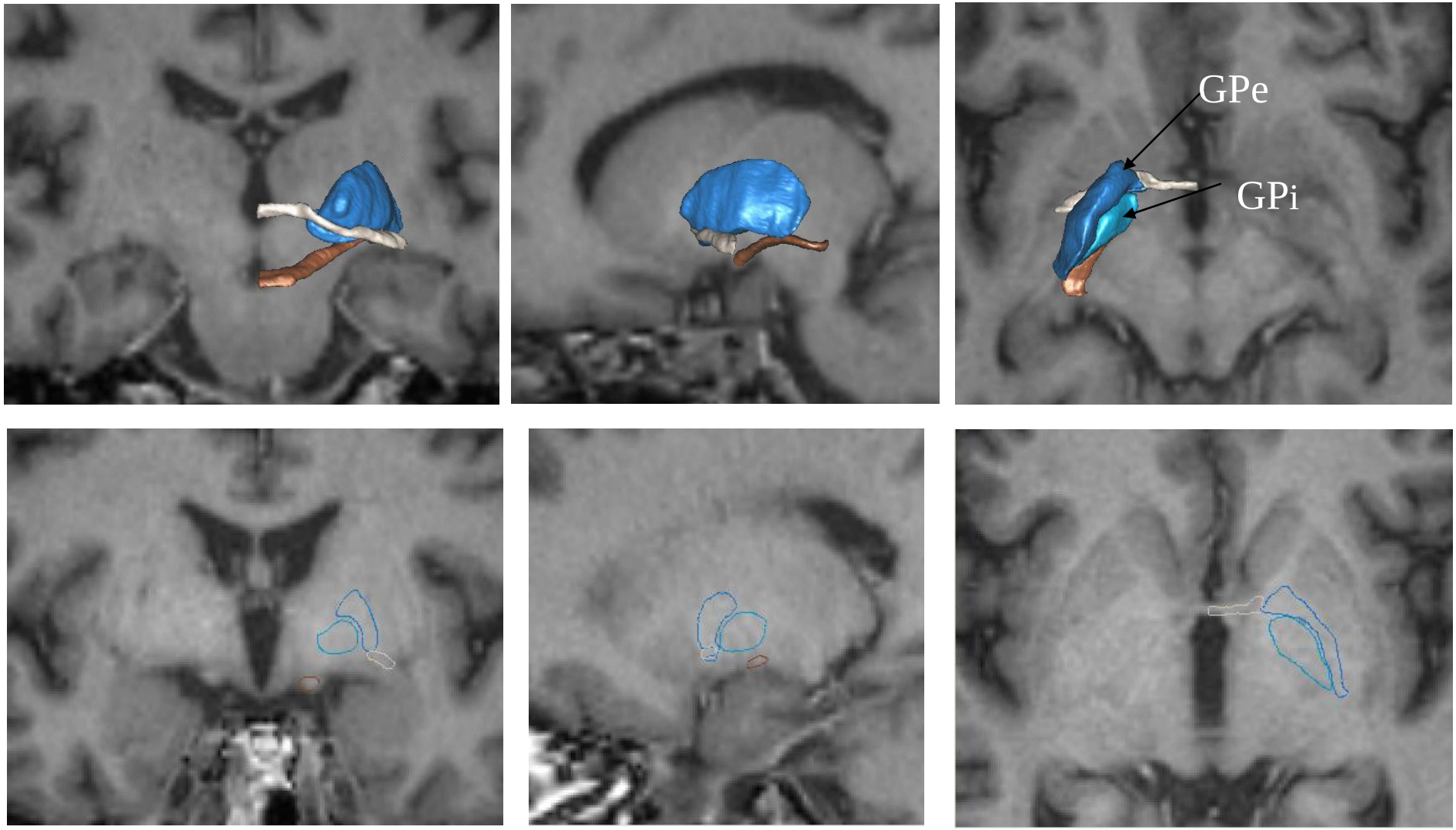


Patient MRIs deformed in atlas space
(spatial normalisation)

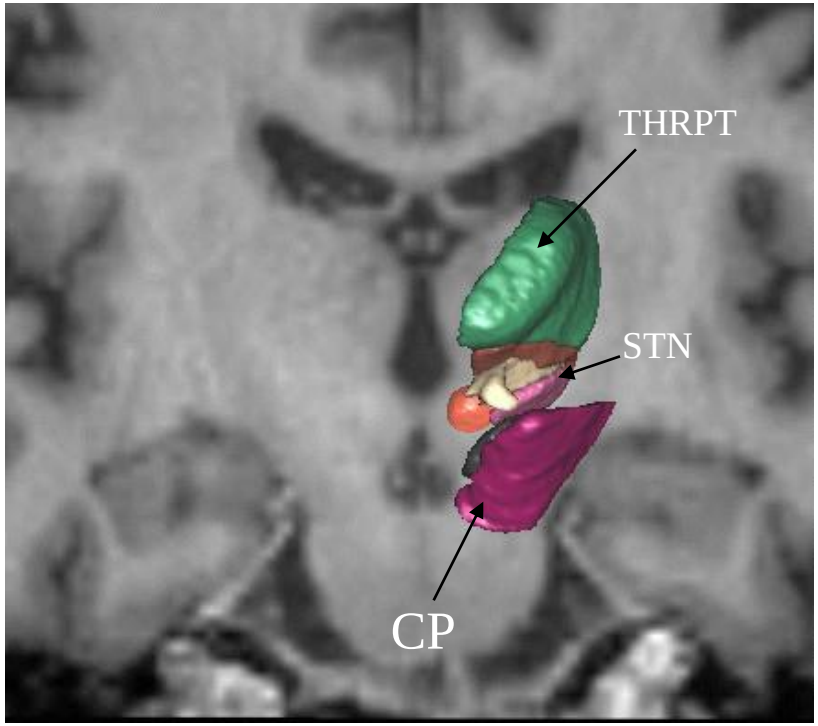
Accumbens, caudate nucleus, putamen

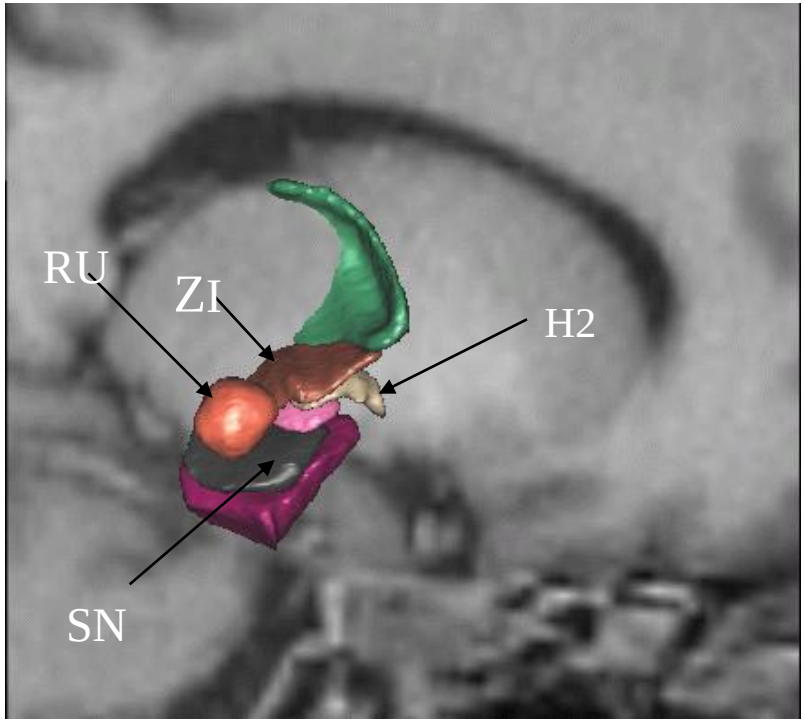


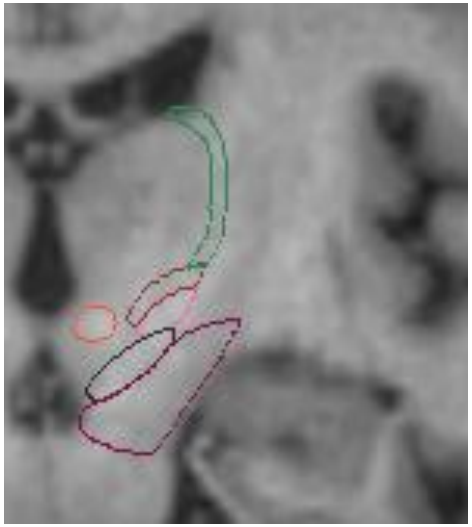
Globus pallidus

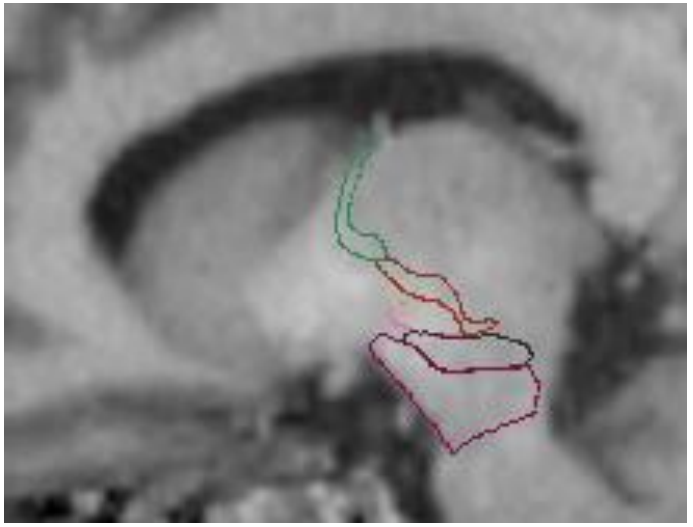


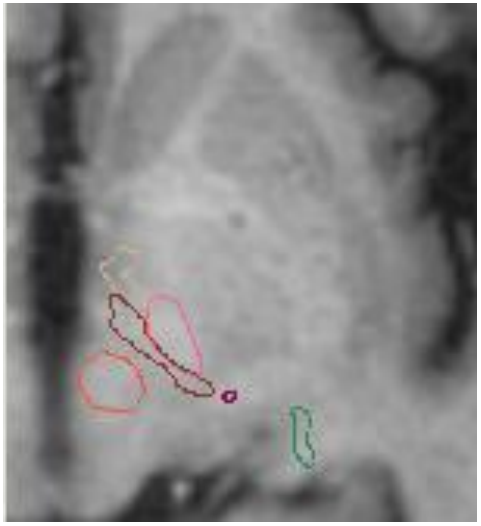
Sub Thalamic Nucleus, Substantia Nigra, red nucleus RU



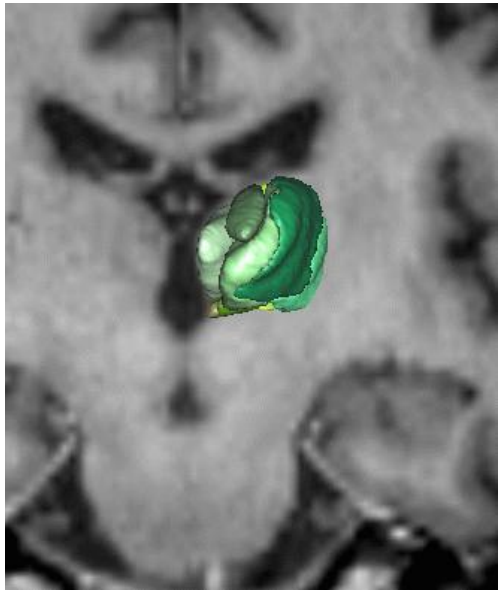


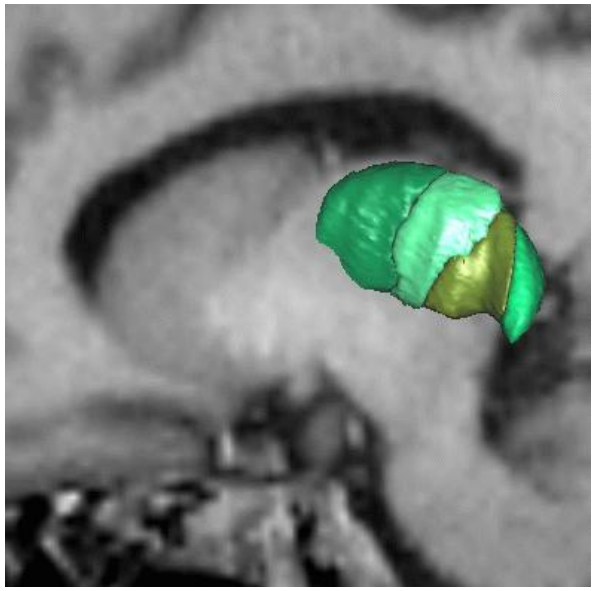


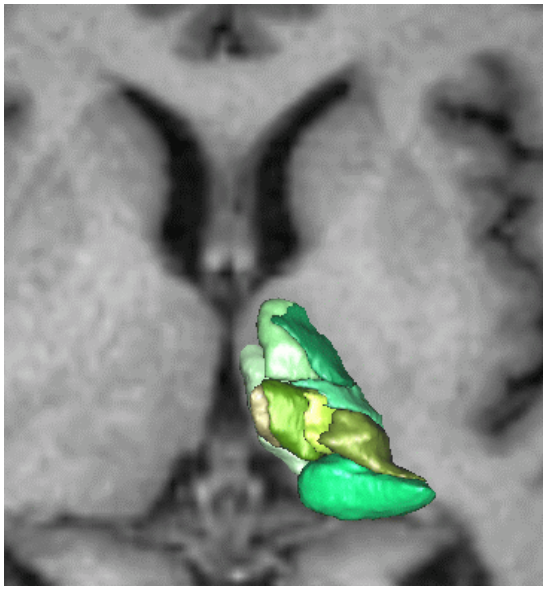


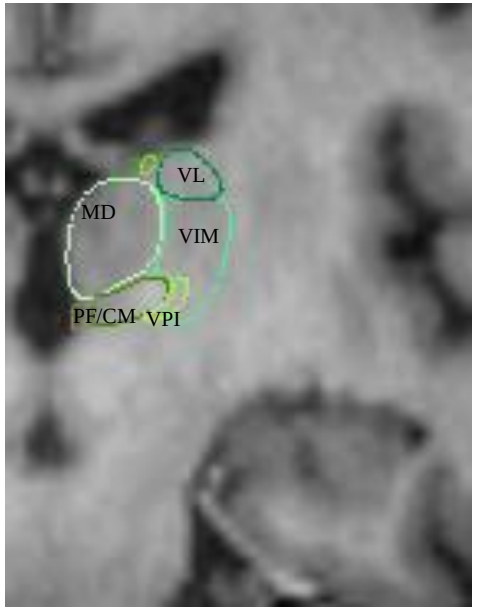


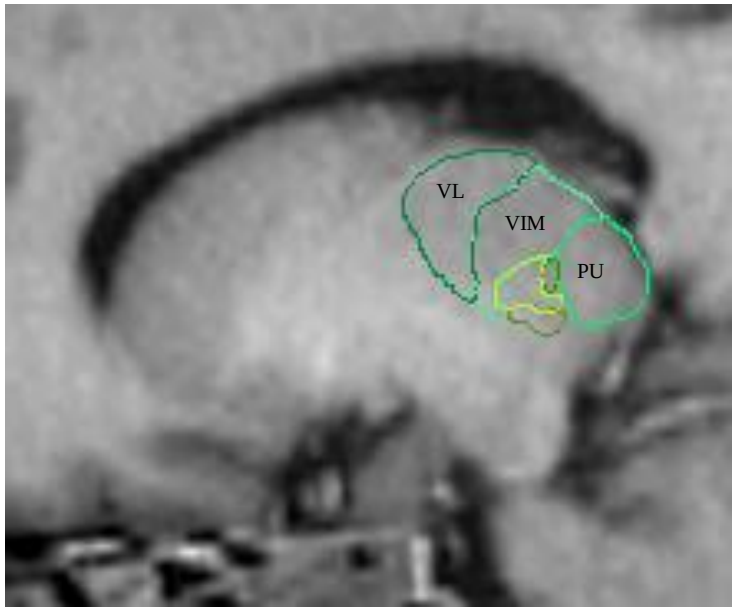
Thalamus

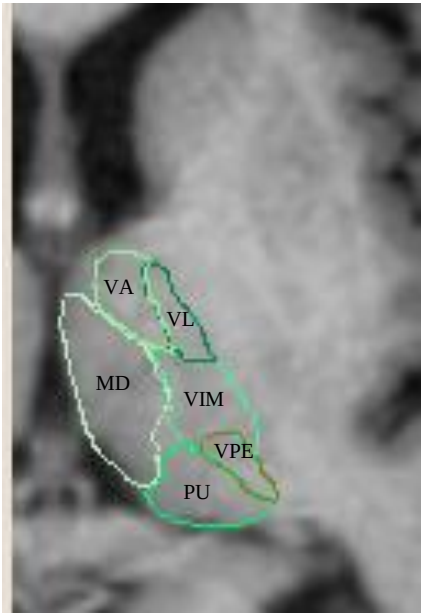




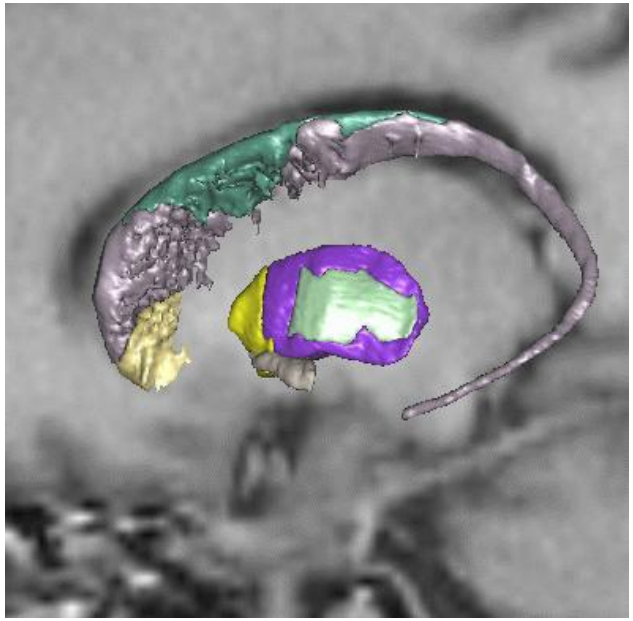


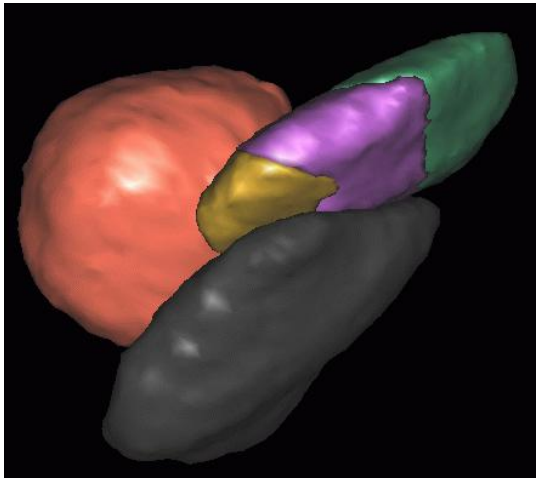


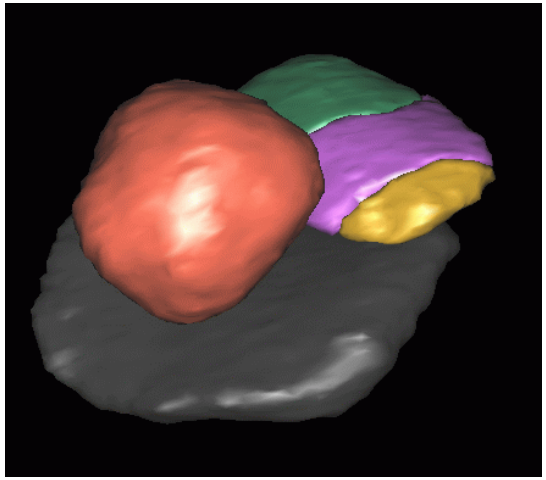


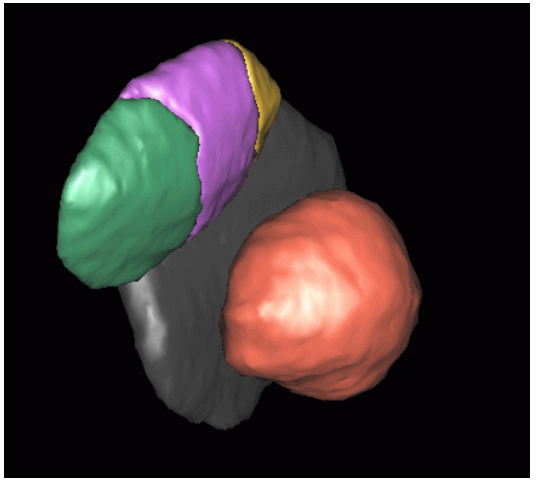


Functional territories



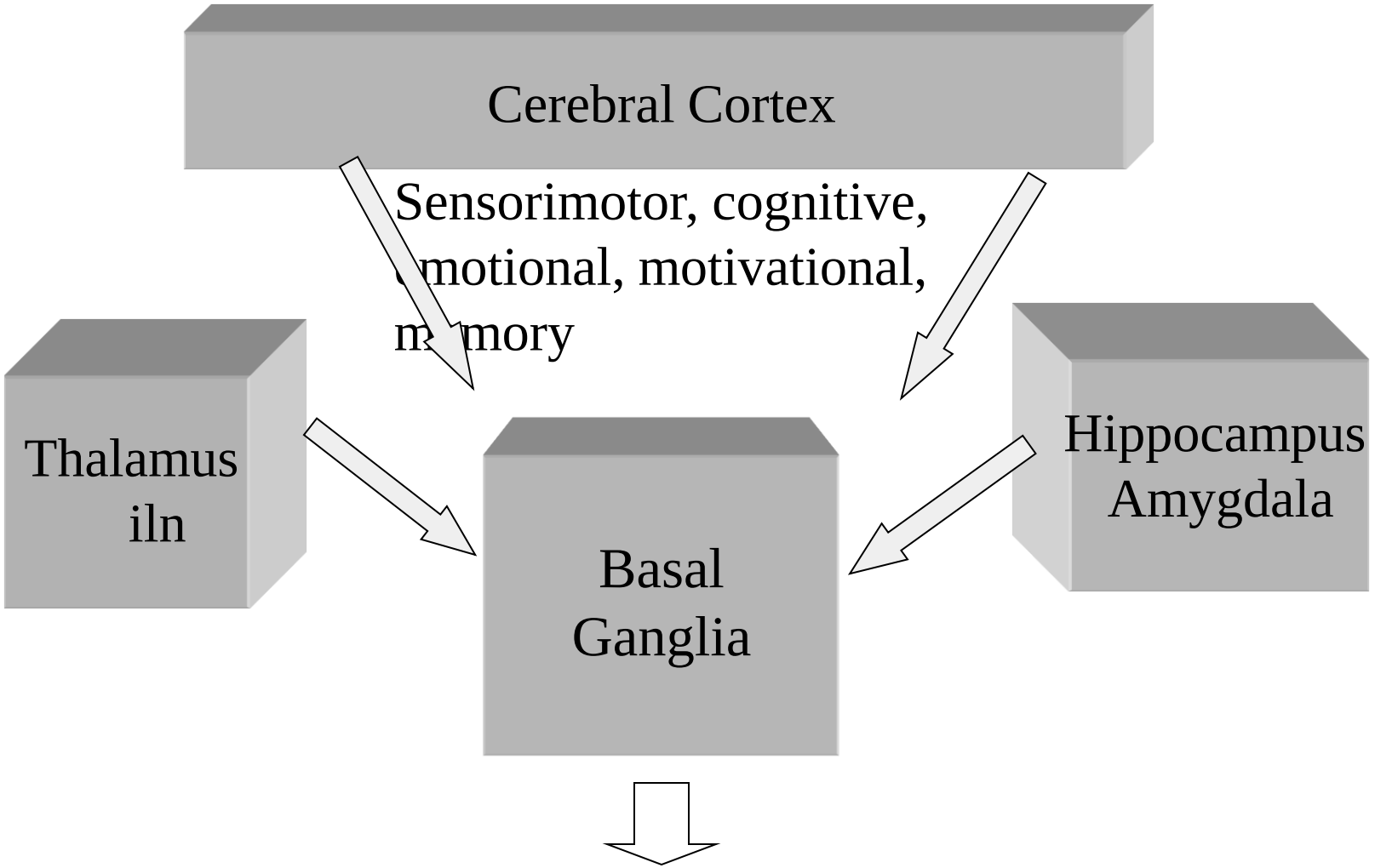




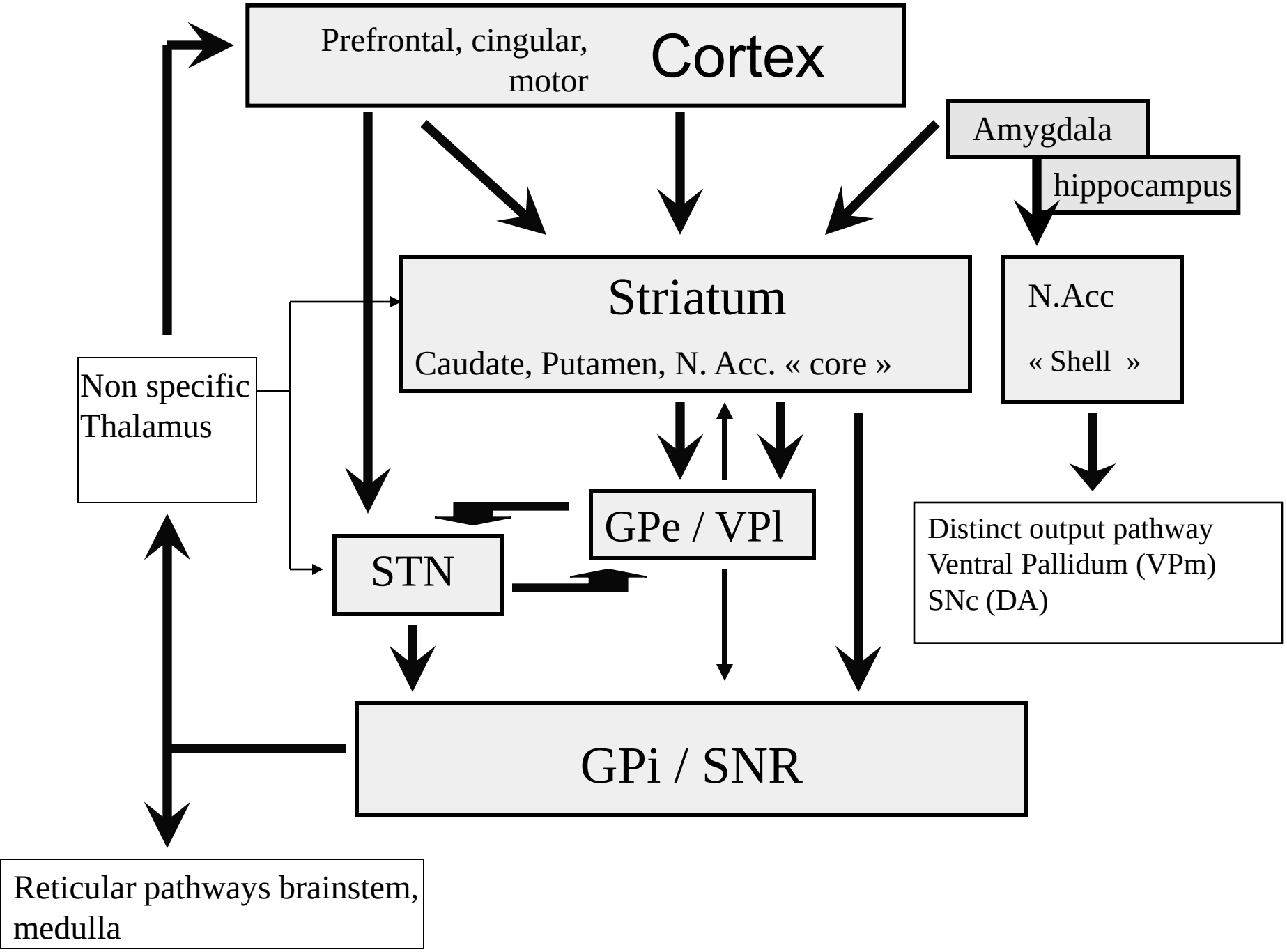


Functions of basal ganglia

from J.M. DENIAU



Environmental contextual analysis and organisation of a contextually adaptated behavior


Prefrontal, cingular, motor
Cortex
Amygdala
hippocampus
Striatum
Caudate, Putamen, N. Acc. « core »
N.Acc
« Shell »
Non specific Thalamus
GPe / VPl
STN
Distinct output pathway
Ventral Pallidum (VPm)
SNc (DA)
GPi / SNR
Reticular pathways brainstem, medulla

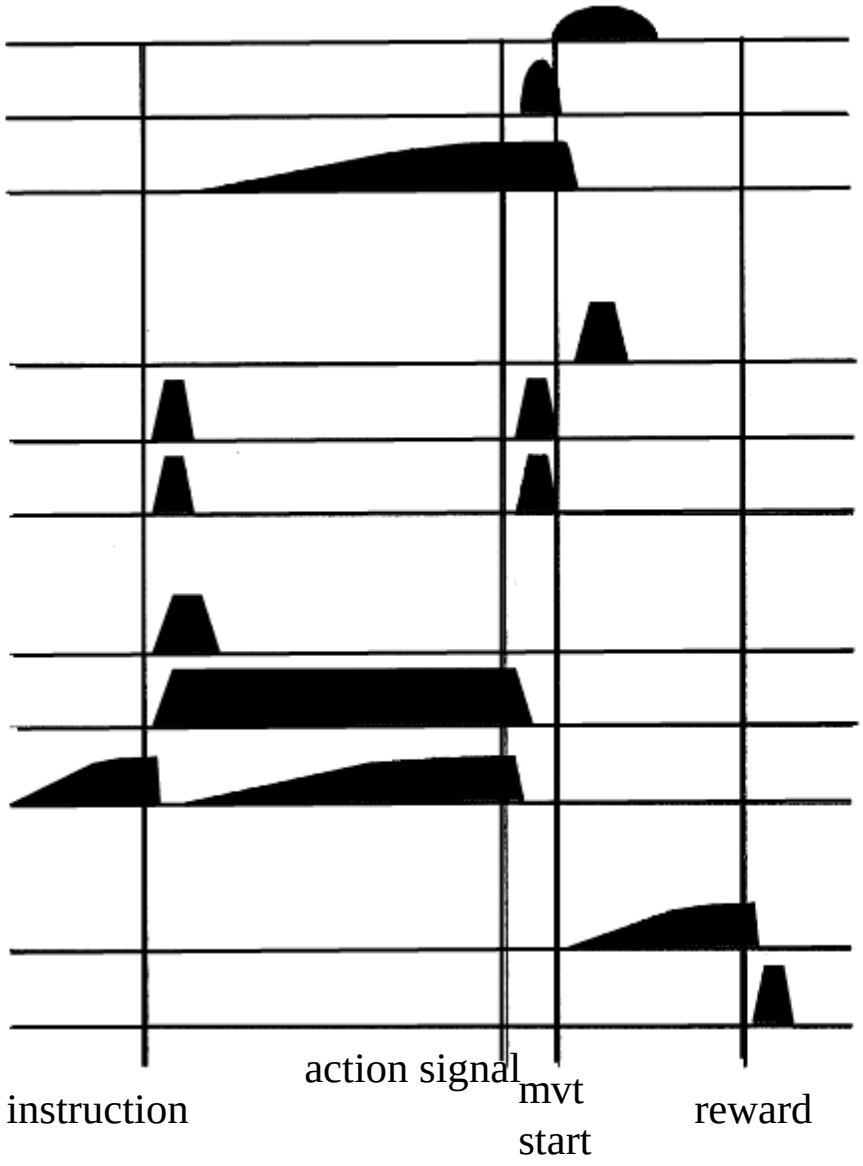
Striatum is active along all key phases of behavior organisation

Movement execution
Movement initiation
Movement preparation

Somatosensoy response
Visual response
Auditory response

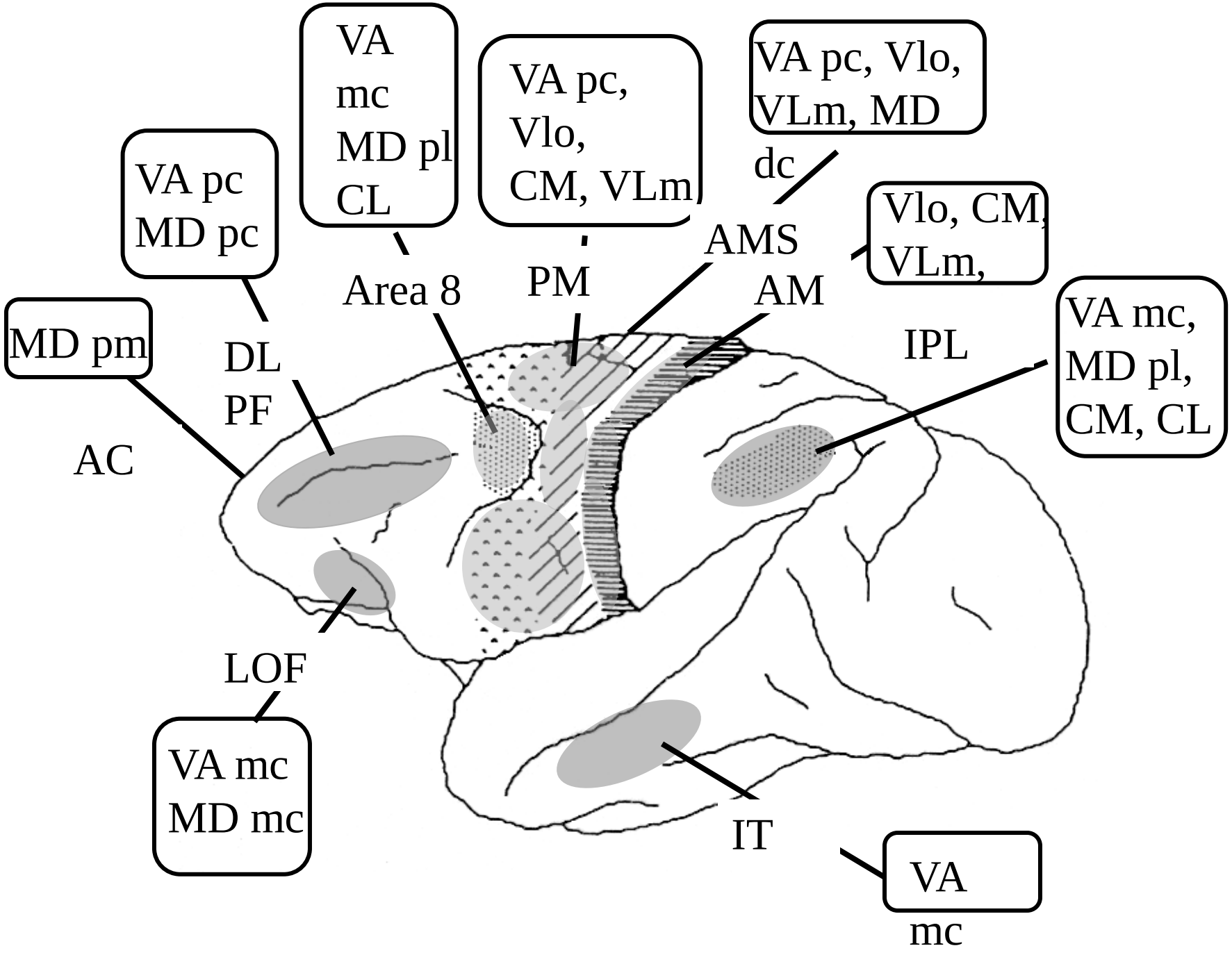
Short term memory
Work memory
Prediction, waiting

Waiting for a reward
Reward



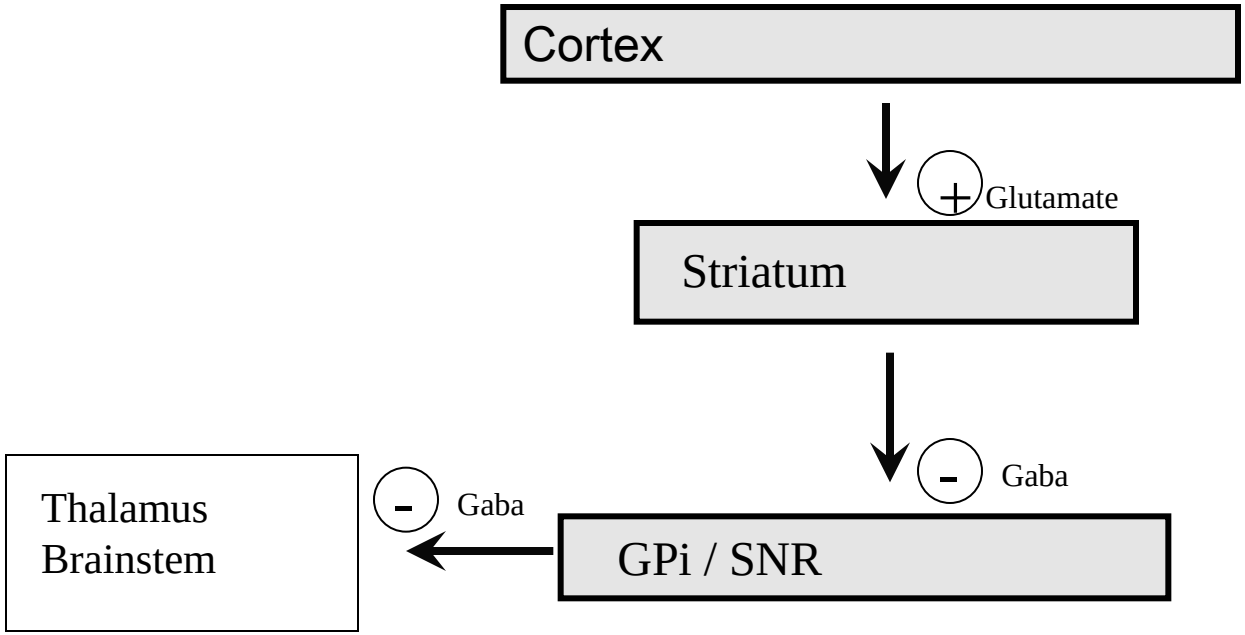
From W. Schultz

Basal ganglia
modulate
prefrontal cortical areas, premotor, motor
temporal and parietal cortex
via thalamic projections of
Substantia Nigra and Globus Pallidus

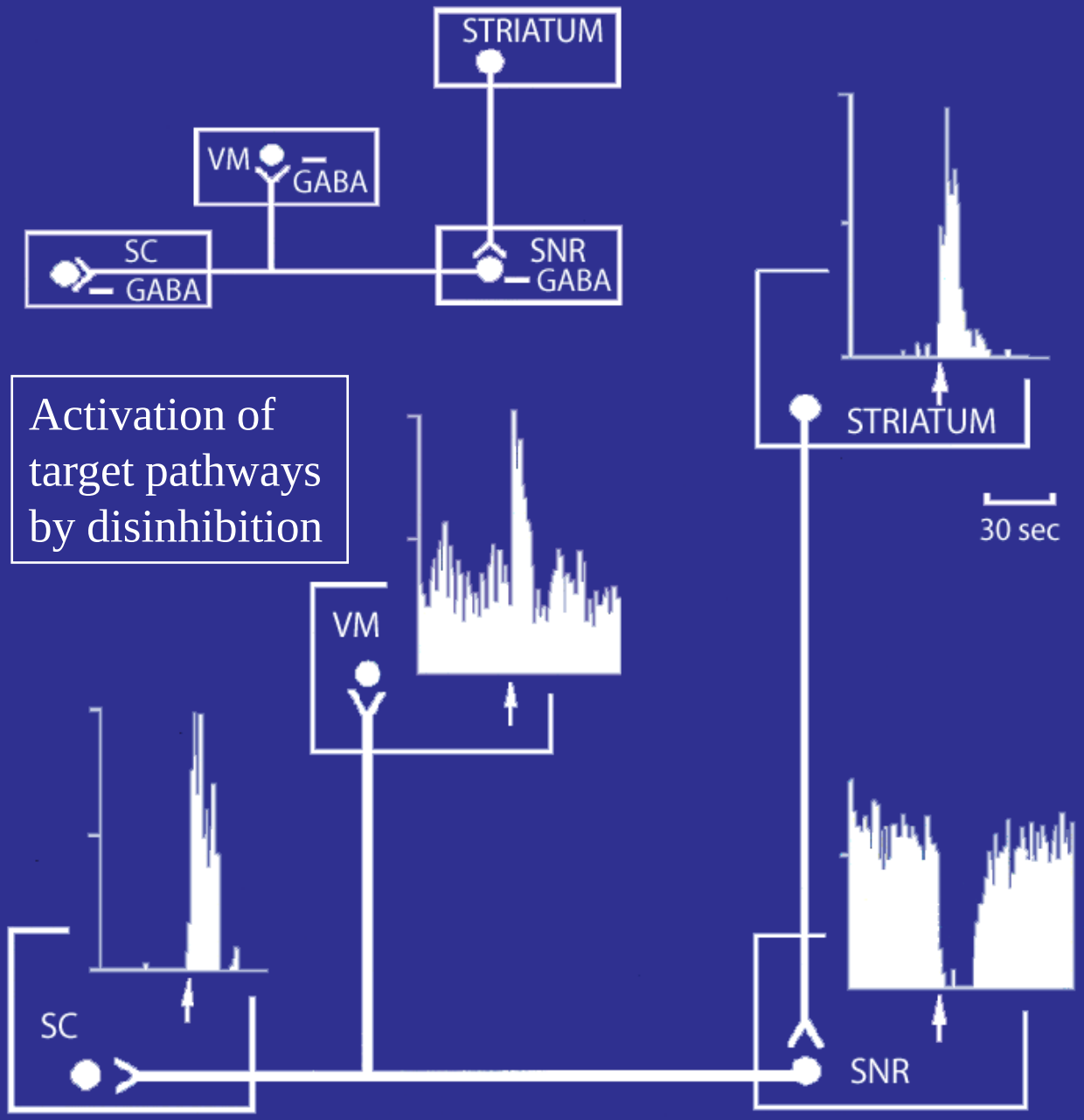

VA
mc
MD pl
CL
VA pc,
Vlo,
CM, VLm
VA pc, Vlo,
VLm, MD
dc
VA pc
MD pc
Vlo, CM,
VLm,
AMS
AM
Area 8
PM
MD pm
DL
PF
IPL
VA mc,
MD pl,
CM, CL
AC
LOF
VA mc
MD mc
IT
VA
mc

Informations originating from cortical areas are transmitted to output structures of basal ganglia through three
main pathways:

- **A direct trans-striatal circuit,**
- **An indirect trans-striatal circuit,**
- **A direct trans-subthalamic circuit**



Direct trans-striatal circuit activates target pathways of basal ganglia via a disinhibition mechanism

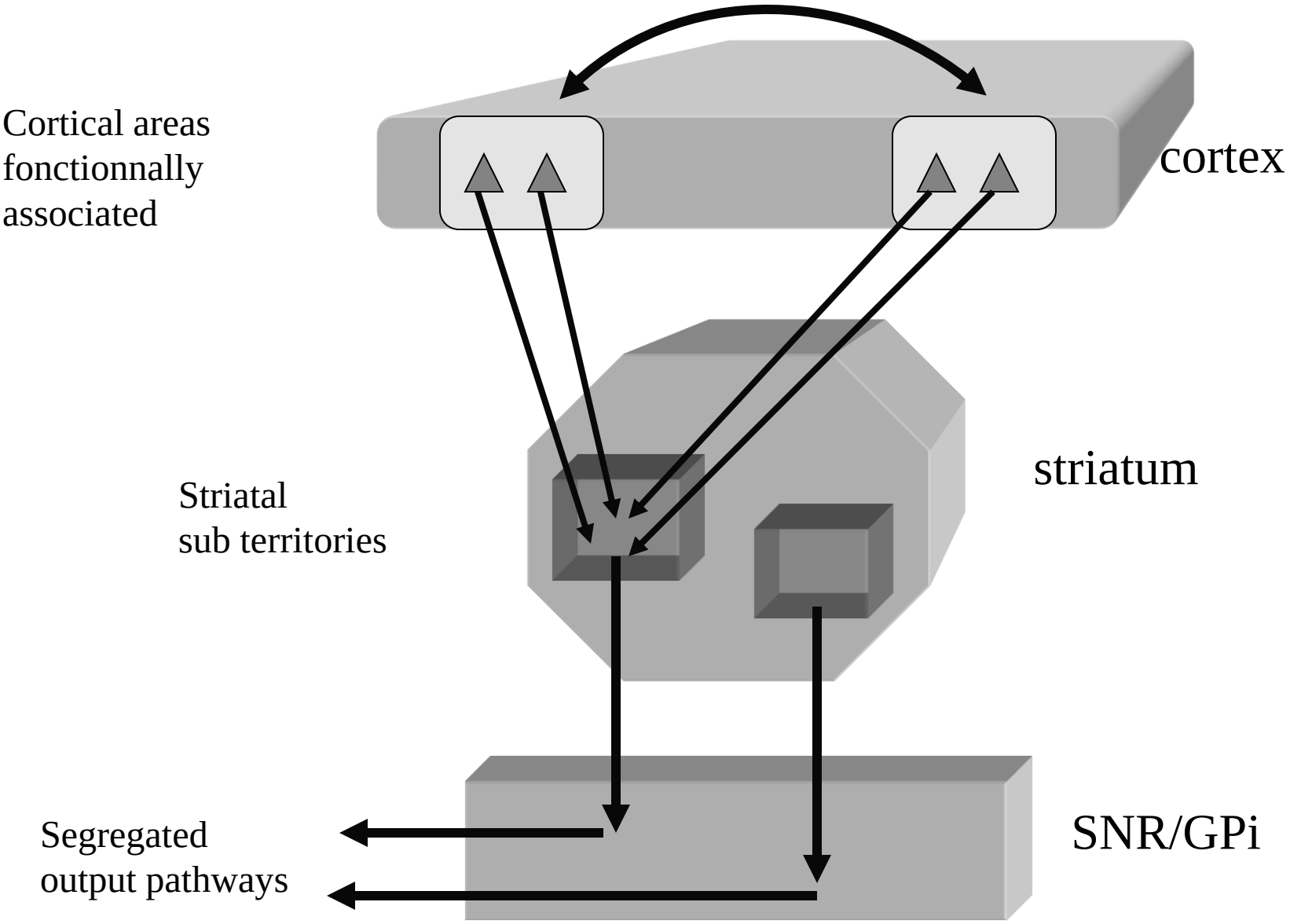


Activation of target pathways by disinhibition

Striatal activation

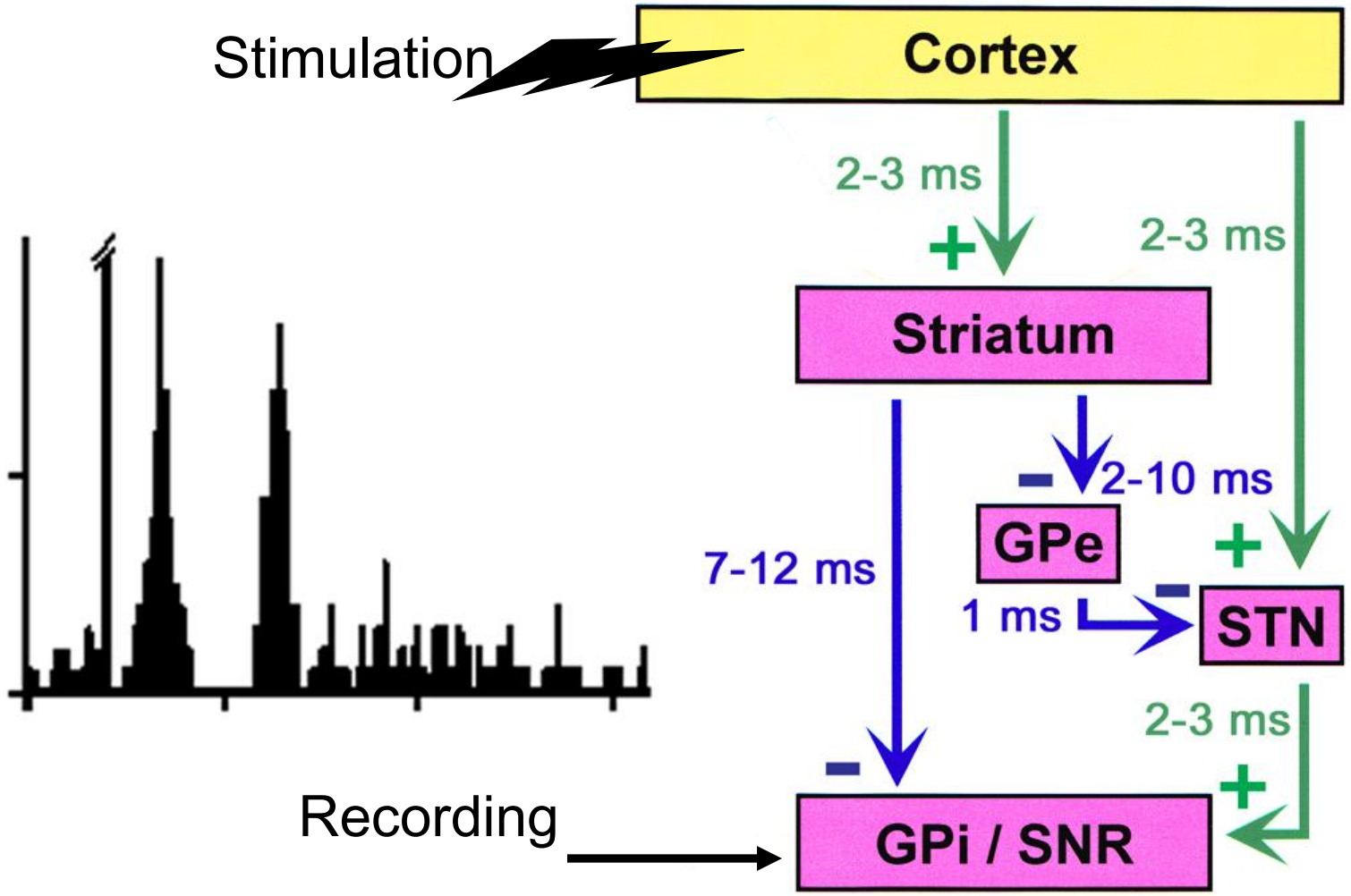
Output neurons inhibition

Modular parallel architecture and convergence of direct trans-striatal pathway



Trans-subthalamic pathways perform a temporal and spatial configuration of the striatal disinhibiting signal

Cortical stimulation evoques a triphasic response (excitation-inhibition-excitation) in SNR neurones. Early excitation results from the activation of direct trans-subthalamic pathway, inhibition results from the activation of direct trans-striatal circuit and late activation from indirect striato-pallido-subthalamo-nigral pathway.



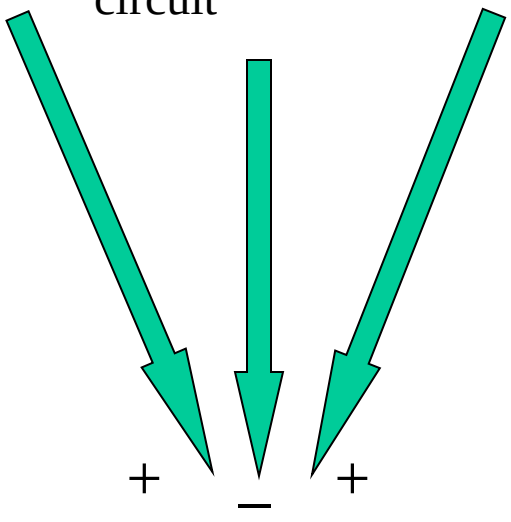
Temporal calibration within a chanel

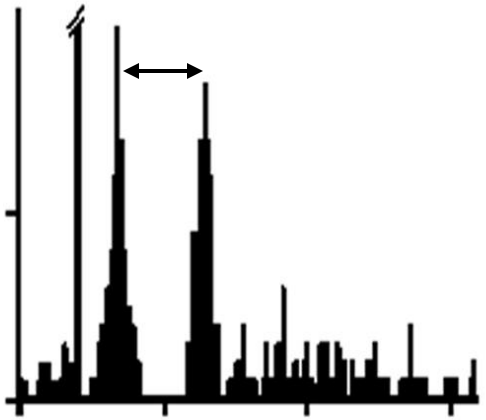
Functionnally associated cortical areas

Direct striatal circuit

Indirect circuit

Direct subthalamic circuit

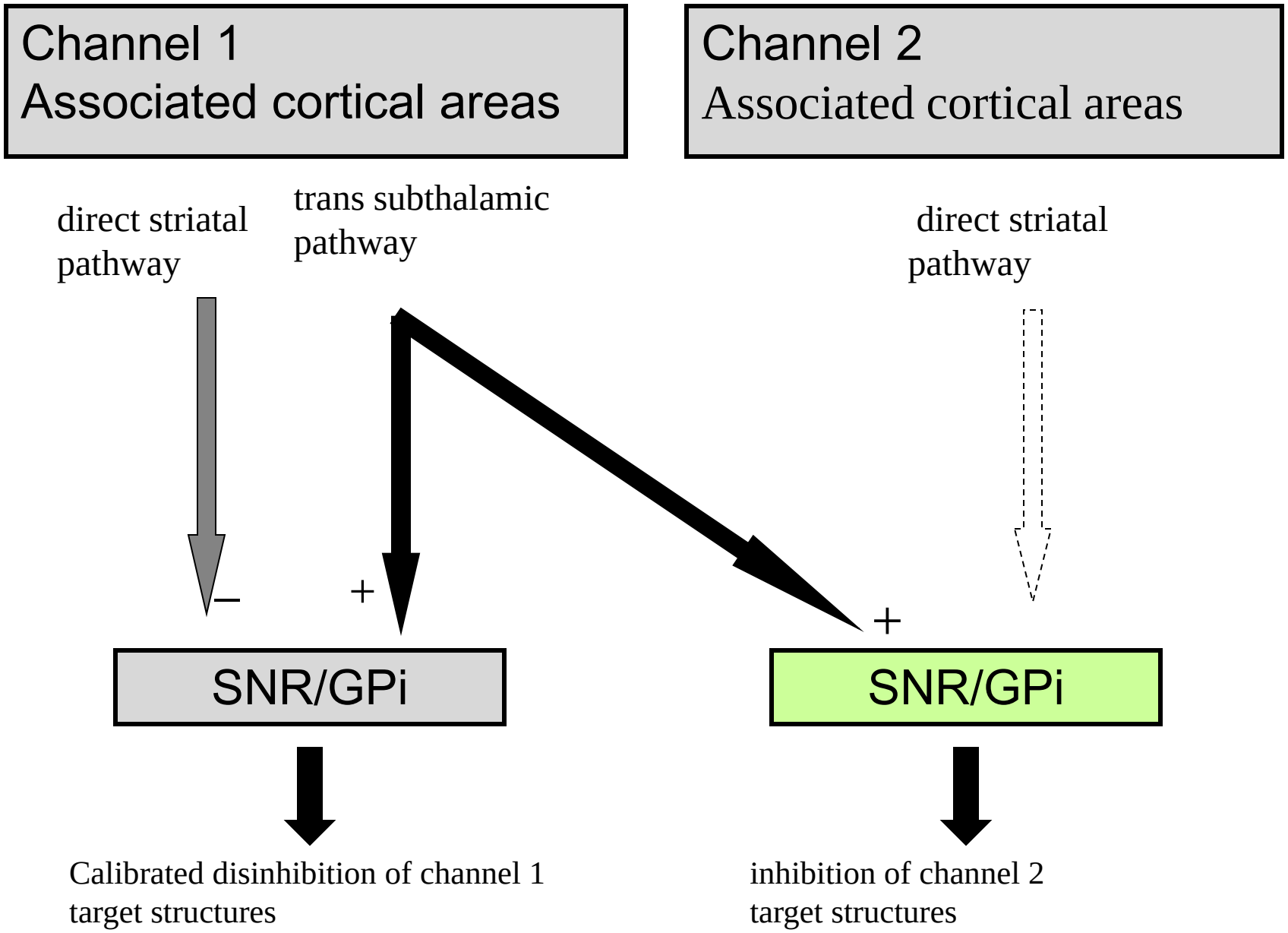




SNR/GPi

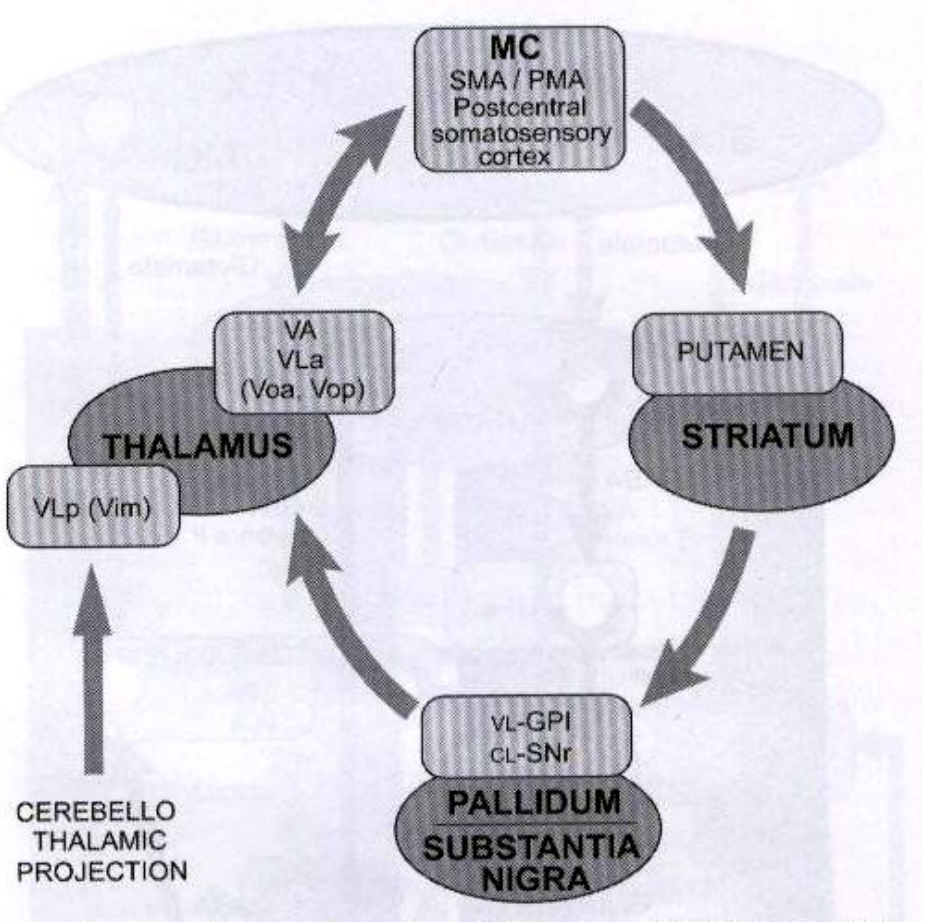
Calibration of disinhibiting striatal signal duration

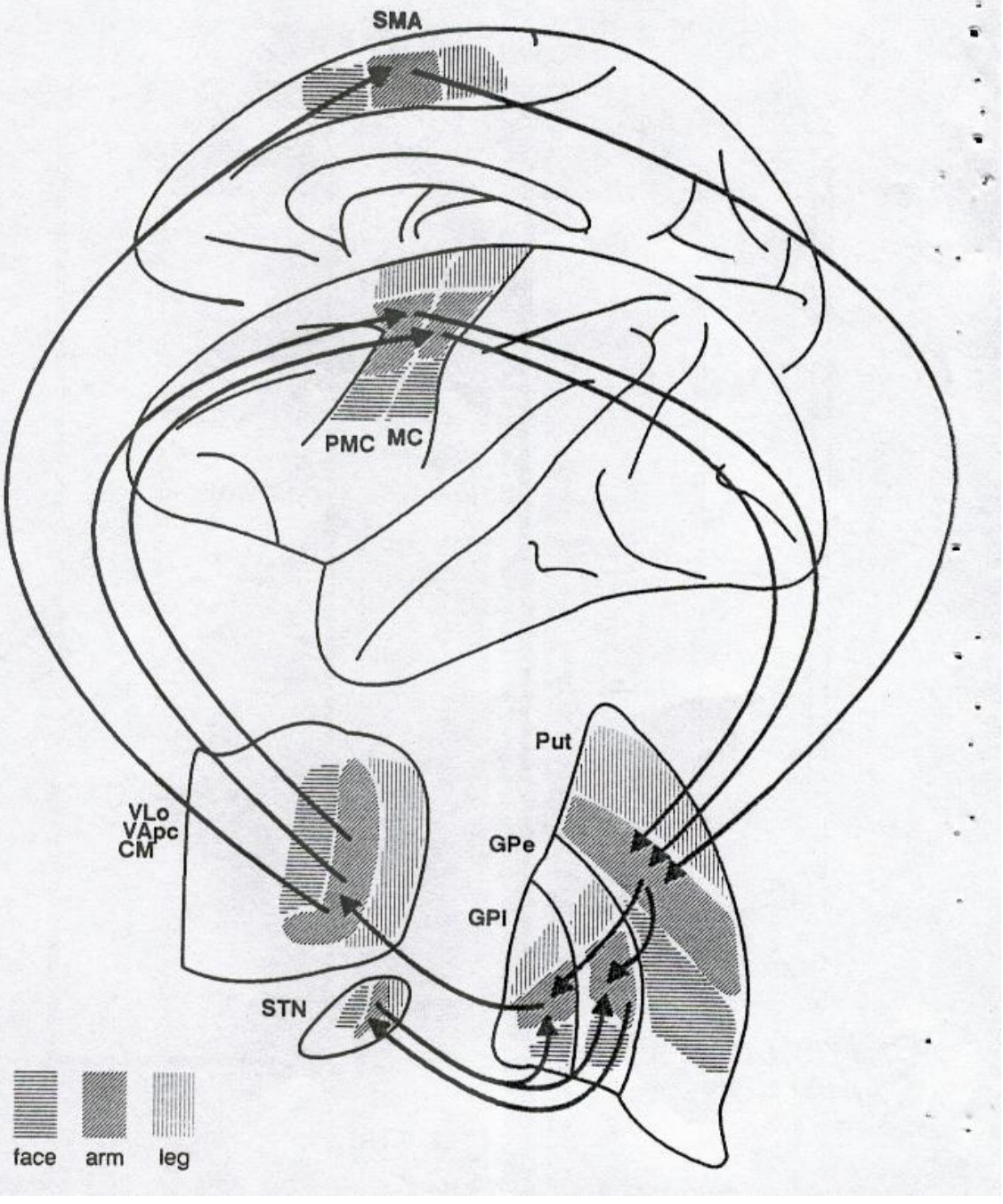
Spatial selection : interaction between channels

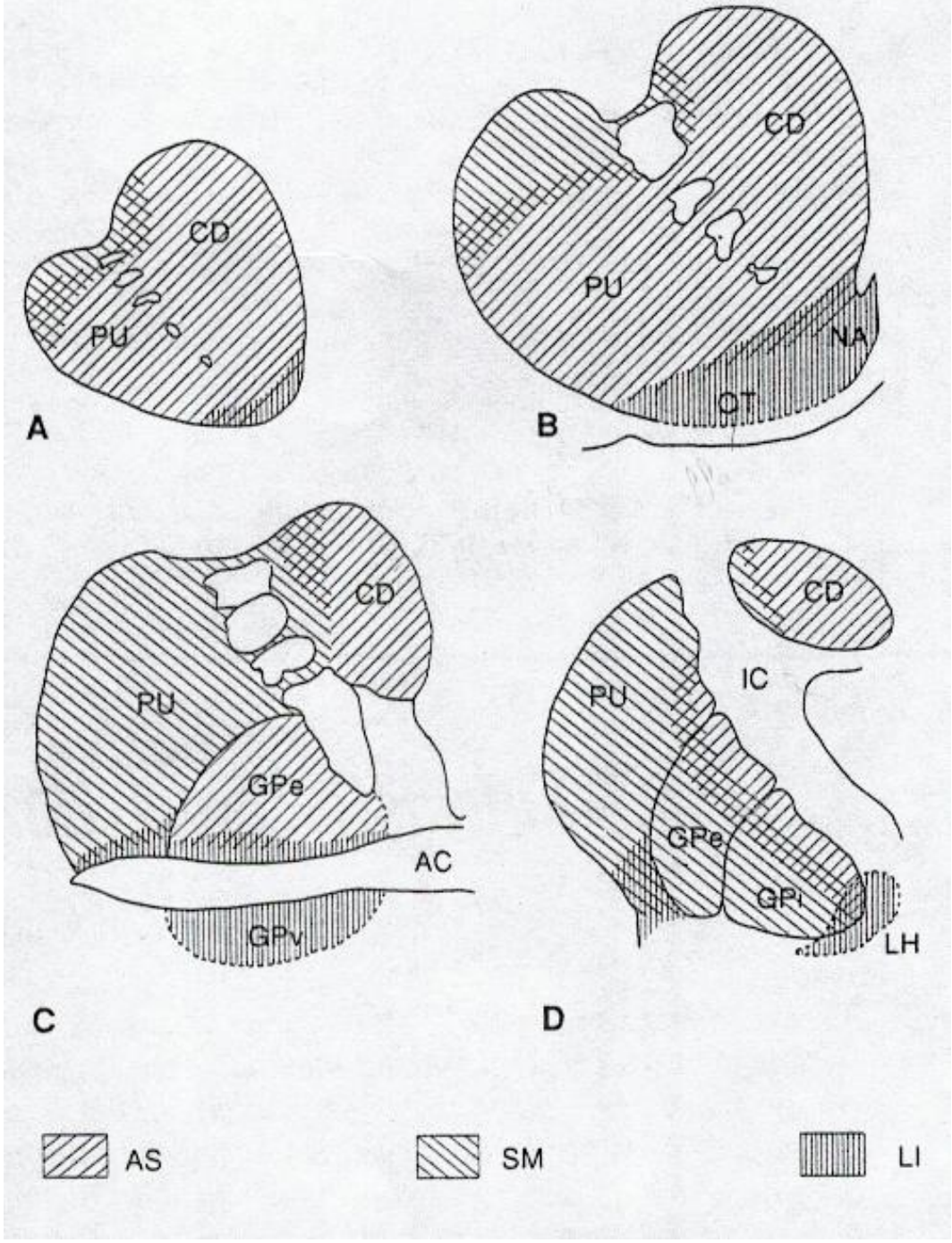


cortico-striato-pallido-thalamo-cortical loop circuits

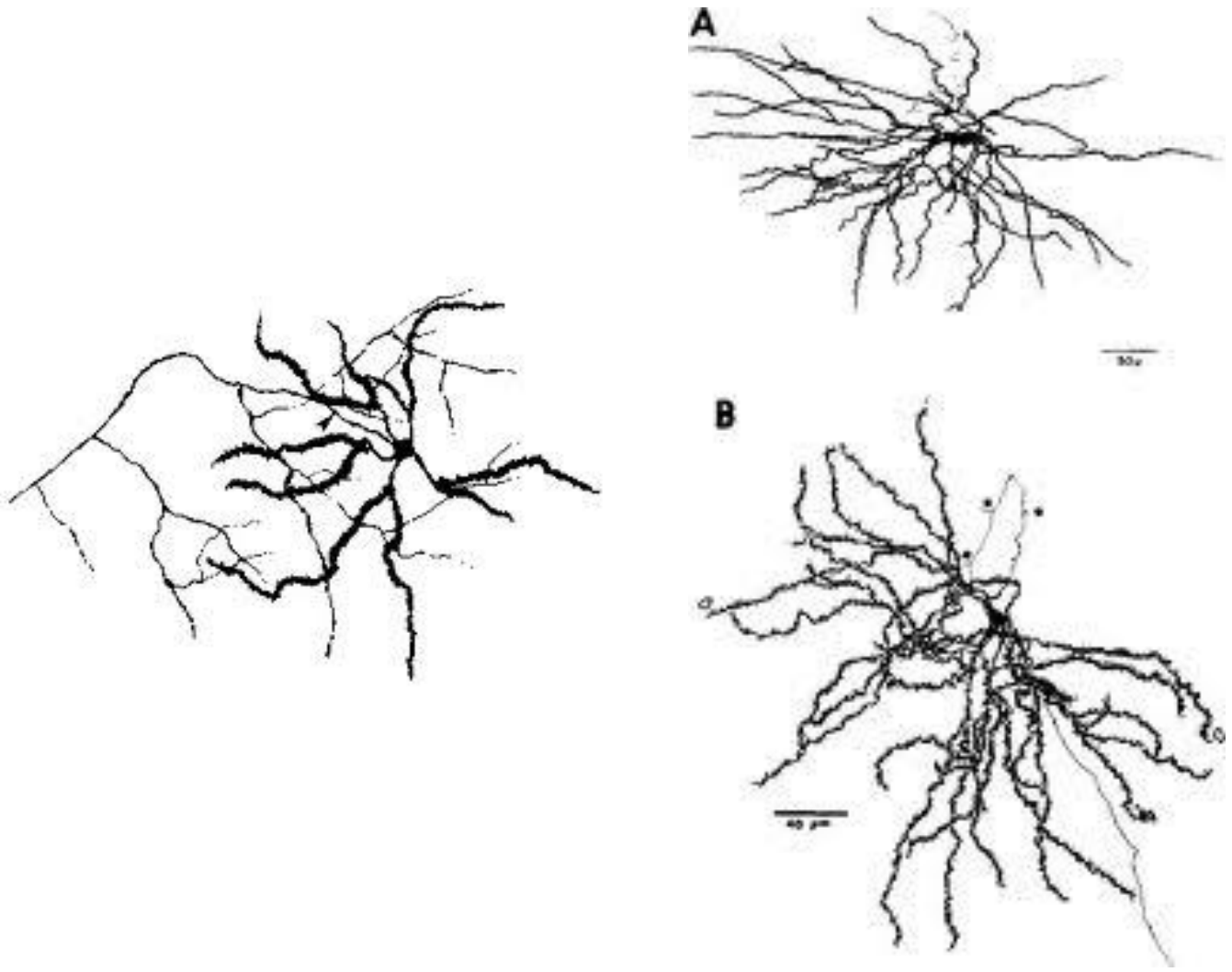
Alexander & Delong




SMA
PMC
MC
Put
VLo
VApc
CM
GPe
GPI
STN
face
arm
leg

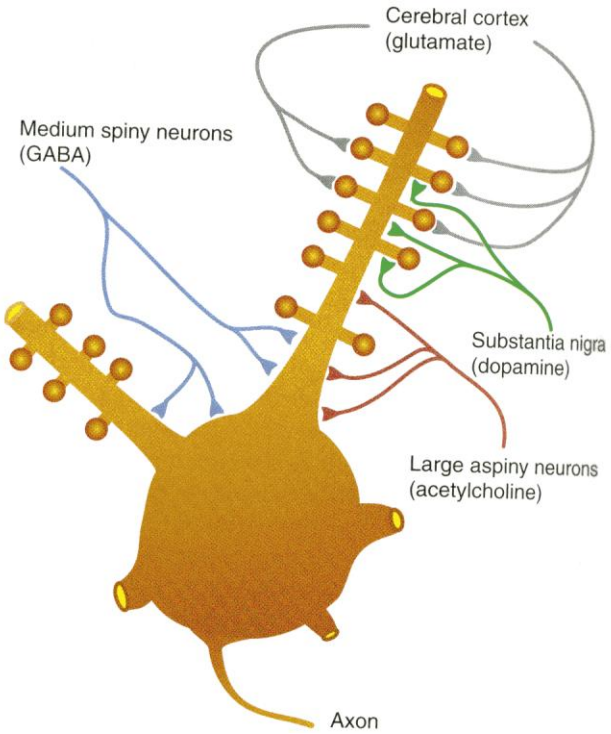

CD
PU
A
CD
PU
NA
OT
B
PU
CD
GPe
AC
GPv
C
CD
IC
PU
GPe
GPi
LH
D
AS
SM
LI

Striatal medium spiny neuron



Striatal output neurones afferents

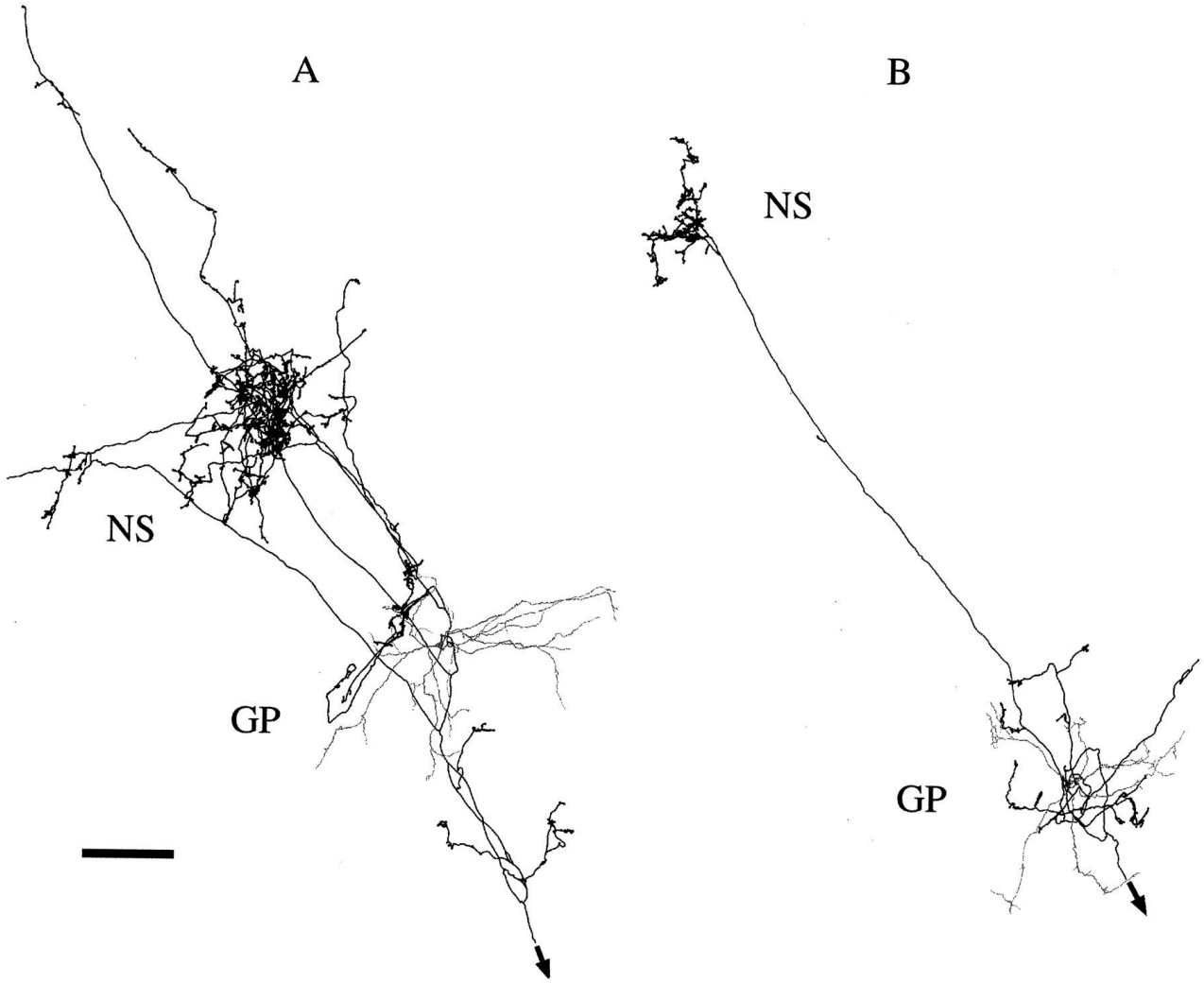
dopamine modulates messages from cerebral cortex



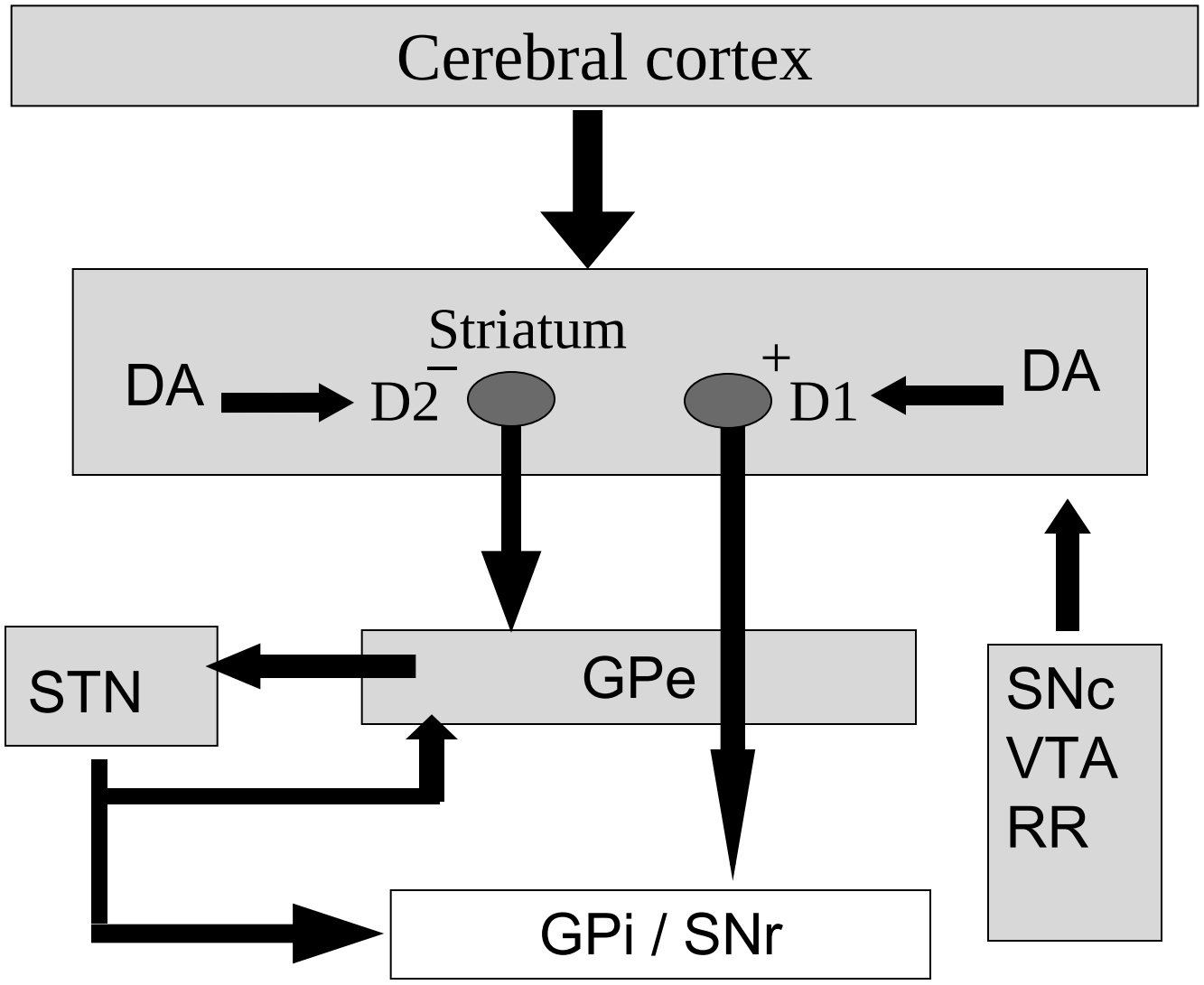
Medium spiny neurons

Adapted from Squire, 2003

Globus Pallidus neurons



Physiopathological models are based on the hypothesis of a distinct neuronal origine of direct and indirect trans-striatal pathways and on a differential control by dopamine on these two sub populations of striatal neurones.



Hypokinetic disorders

- Parkinson’s disease
- neuroleptics parkinsonian syndrom
- MPTP monkey parkinsonism model

Parkinson’s disease

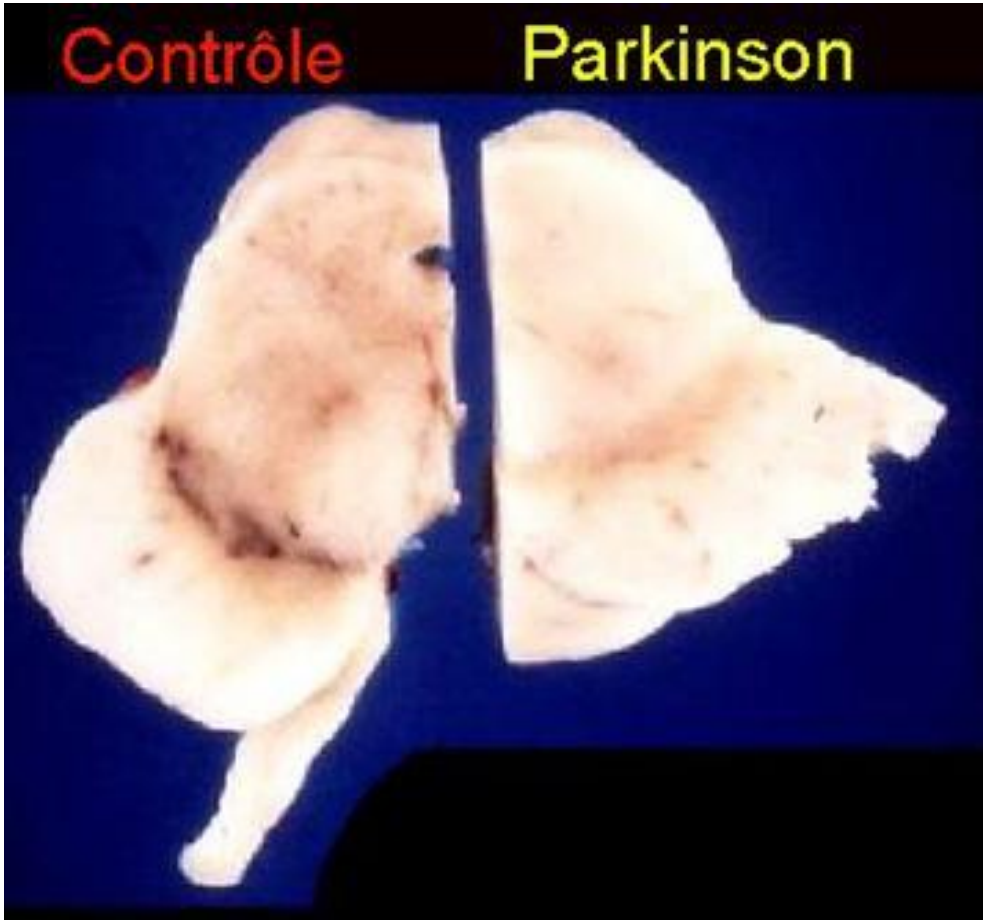
PHYSIOPATHOLOGY

• Death of Substantia Nigra dopaminergic neurons.

• Less dopamine in target structure : Striatum (caudate nucleus and pallidum);

• When 50-60 % DA neurons have died, PD clinical signs begin.

Loss of dopaminergic neurons, DA



- Human mesencephalon
- Parkinson’s disease : DA neurons contain neuromelanine (black pigment)

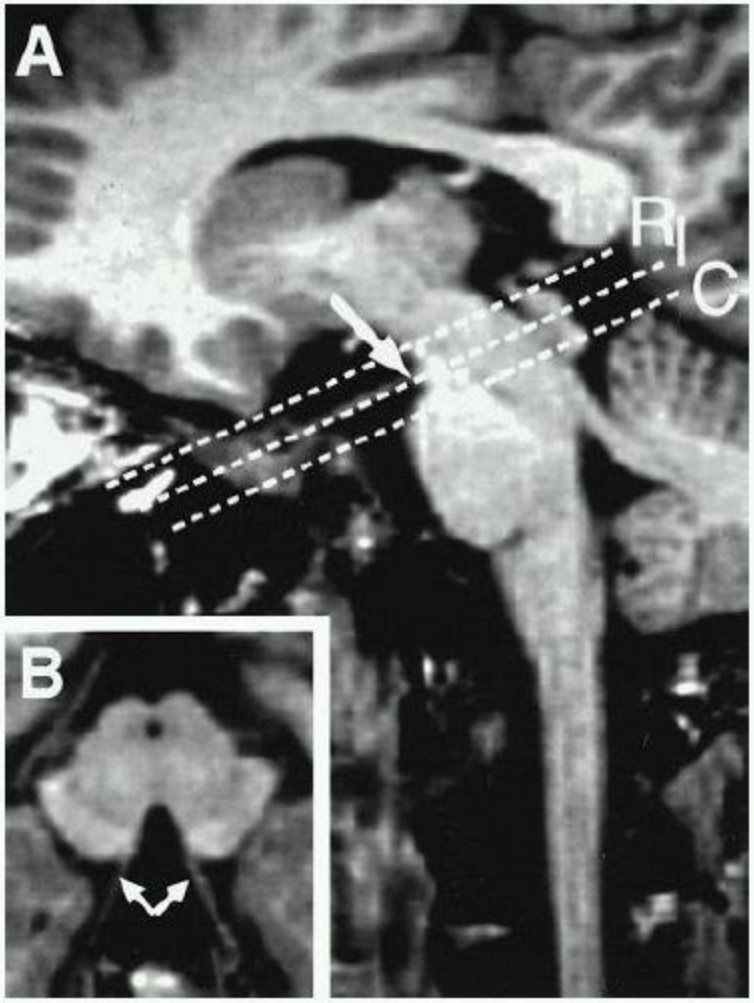
Dopaminergic loss location

Possible role of calbindin afferents

Calbindin : calcium binding protein
neuroprotective effect by inhibiting free radical formation ?

- *Ventral DA* neurons have less calbindin => less protected => more neuronal loss.

- *Dorsal DA* neurons have numerous calbindin afferents.



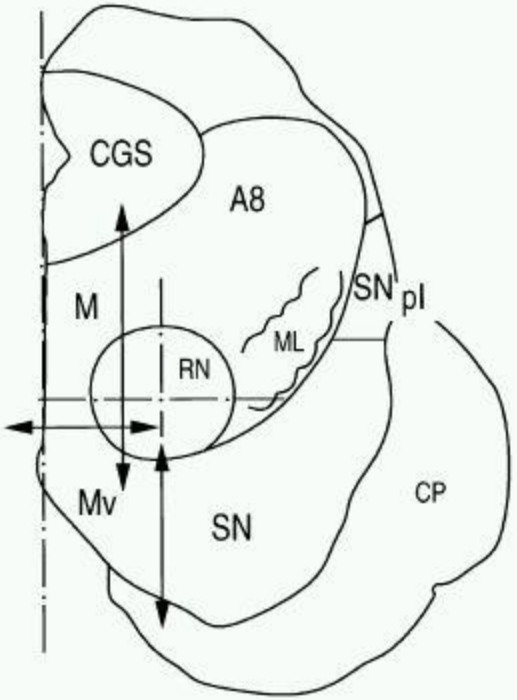


Fig. 2 Subdivision of dopamine-containing neurons of the midbrain into six dopaminergic groups. CGS = central grey substance; CP = cerebral peduncle; M = medial group; Mv = medioventral group; A8 = dopaminergic group A8; SN = substantia nigra; SNpl = substantia nigra pars lateralis; RN = red nucleus; ML = medial lemniscus. Based on work by Hirsch and colleagues (Hirsch *et al.*, 1988).

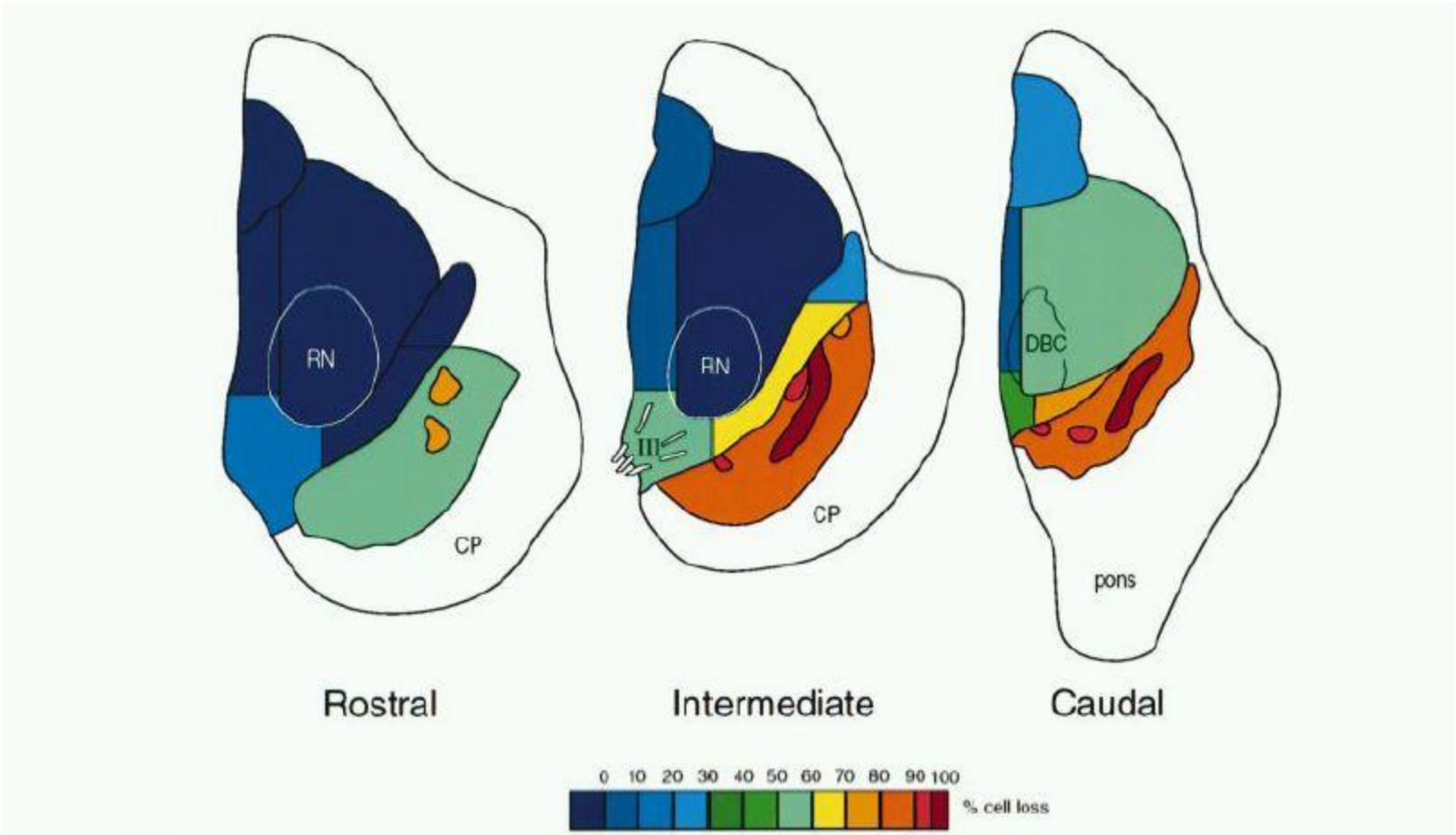
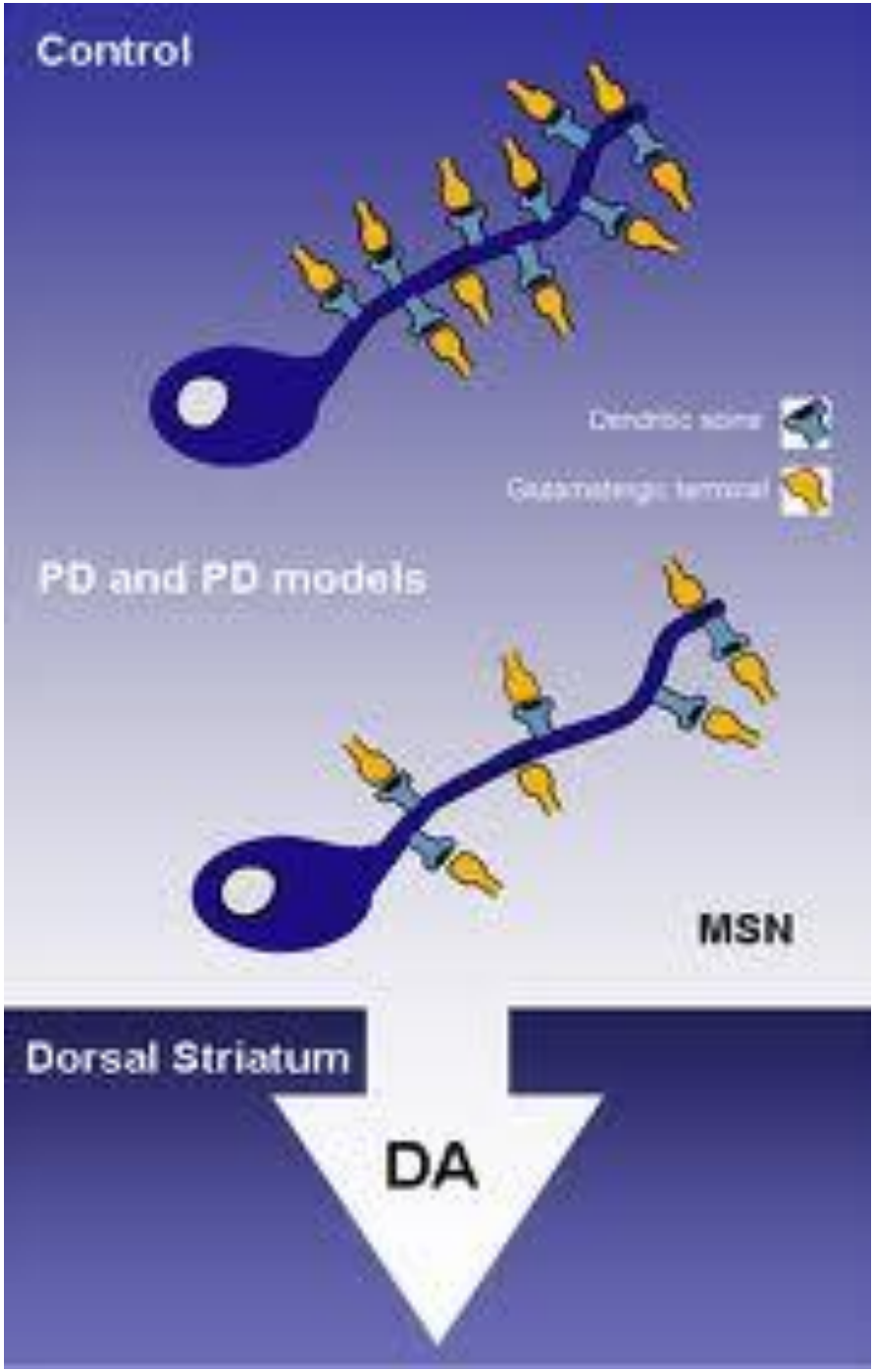
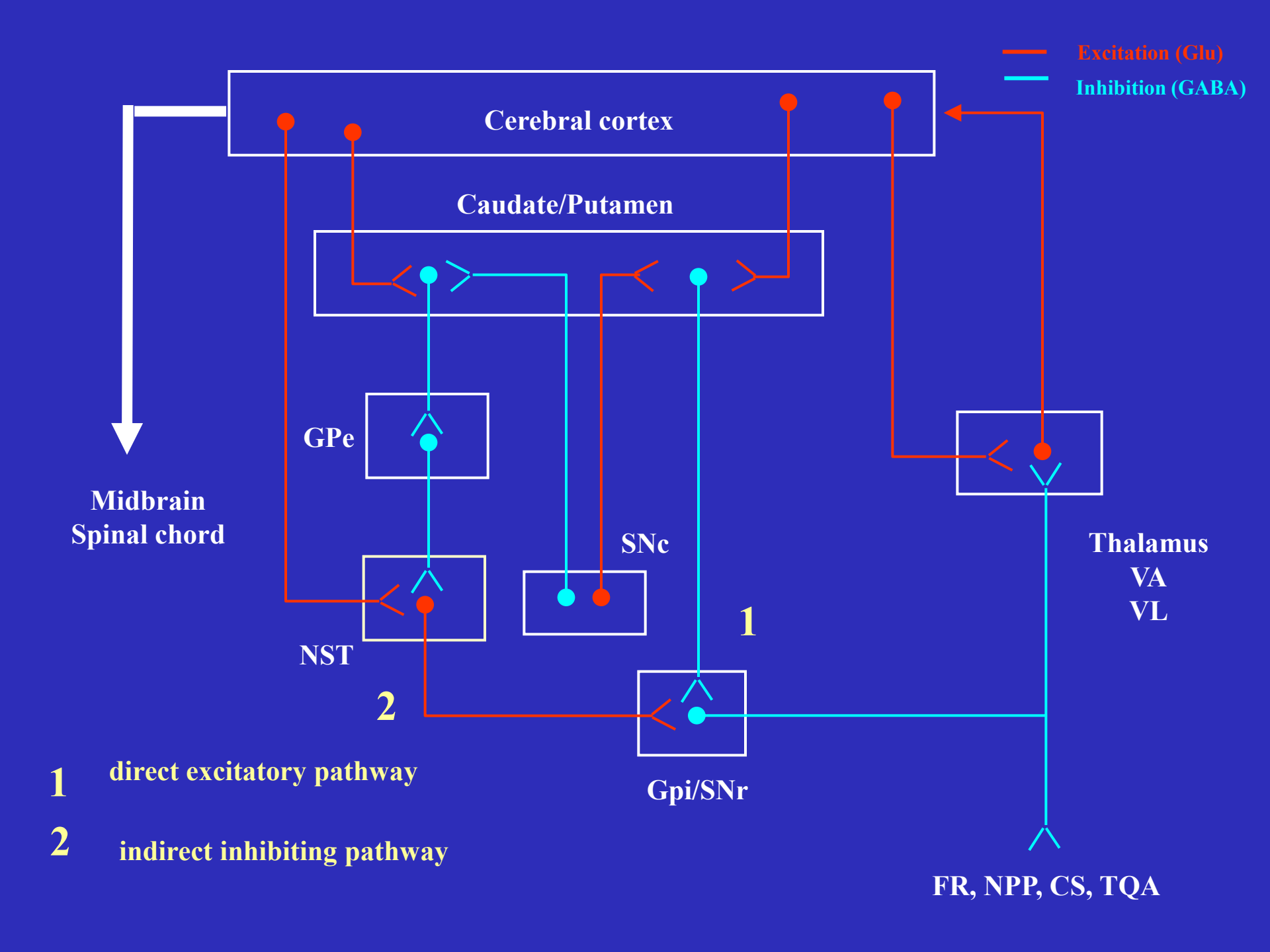


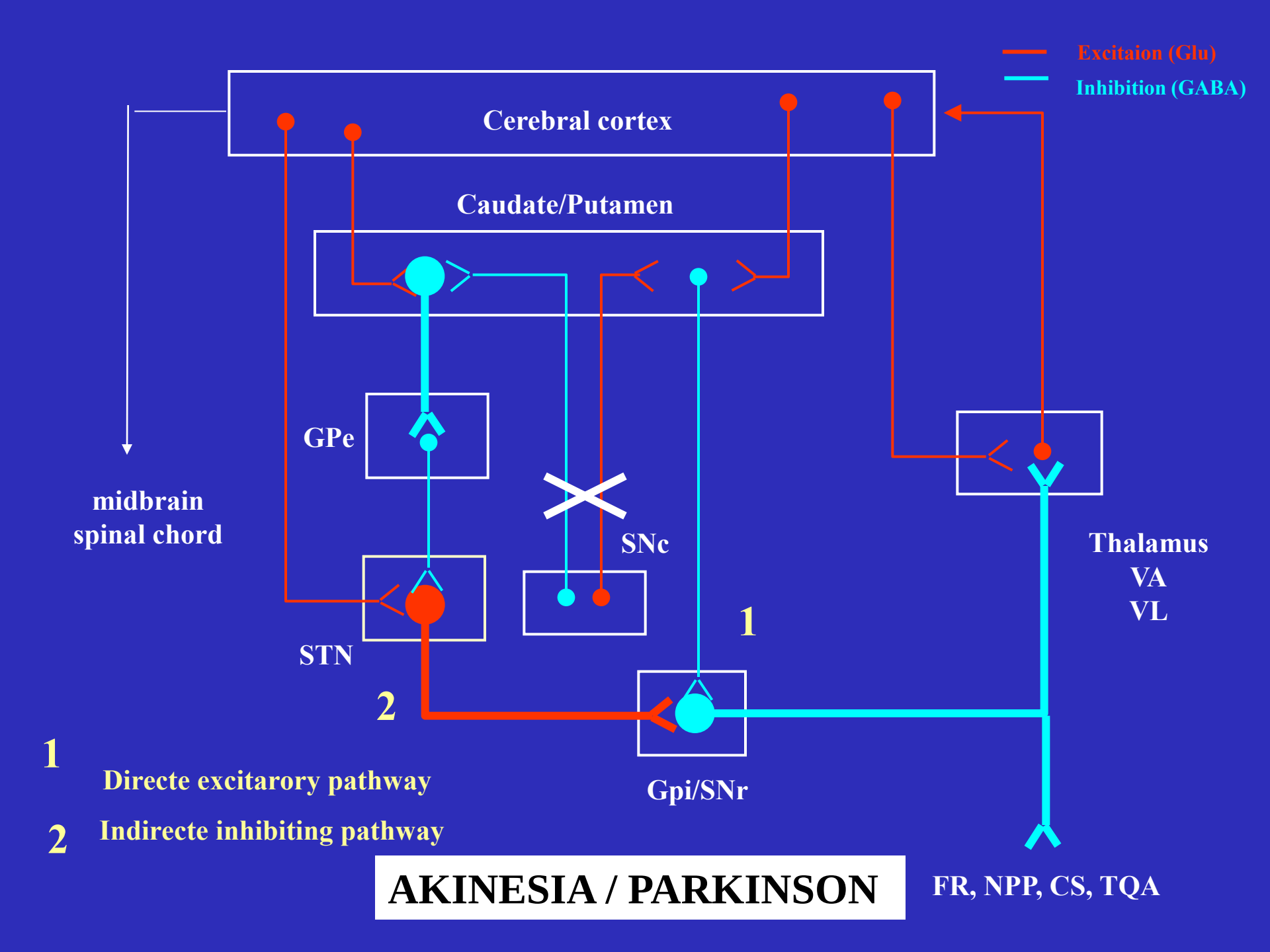
Fig. 2 Regional and intranigral loss of dopamine-containing neurons in Parkinson's disease. The colorimetric scale indicates the estimated amount of cell loss (least = blue; most = red). Cell losses in the different groups of the midbrain were calculated by comparing mean numbers of TH-positive neurons in each group of five parkinsonian midbrains (disease duration = 7–32 years) with corresponding means for five control midbrains. A8 = dopaminergic cell group A8; CGS = central grey substance; CP = cerebral peduncle; DBC = decussation of brachium cunjunctivum; M = medial group; Mv = medioventral group; N = nigrosome; RN = red nucleus; SNpd = substantia nigra pars dorsalis; SNpl = substantia nigra pars lateralis; III = exiting fibres of the third cranial nerve.


Control
Dendritic spine
Glutamatergic terminal
PD and PD models
MSN
Dorsal Striatum
DA


Excitation (Glu)
Inhibition (GABA)
Cerebral cortex
Caudate/Putamen
GPe
Midbrain
Spinal chord
SNc
Thalamus
VA
VL
NST
1
2
Gpi/SNr
1 direct excitatory pathway
2 indirect inhibiting pathway
FR, NPP, CS, TQA

Parkinson’s disease

- Resting tremor (4-6 Hz) : trembling in the hands, arms, legs, jaw and face
- Rigidity: stiffness of the limbs and trunk
- Bradykinesia: slowness of movements
- Akinesia: difficulty in initiating movement
- Postural instability: impaired balance
- and depression…



1 Directe excitarory pathway

2 Indirecte inhibiting pathway

AKINESIA / PARKINSON

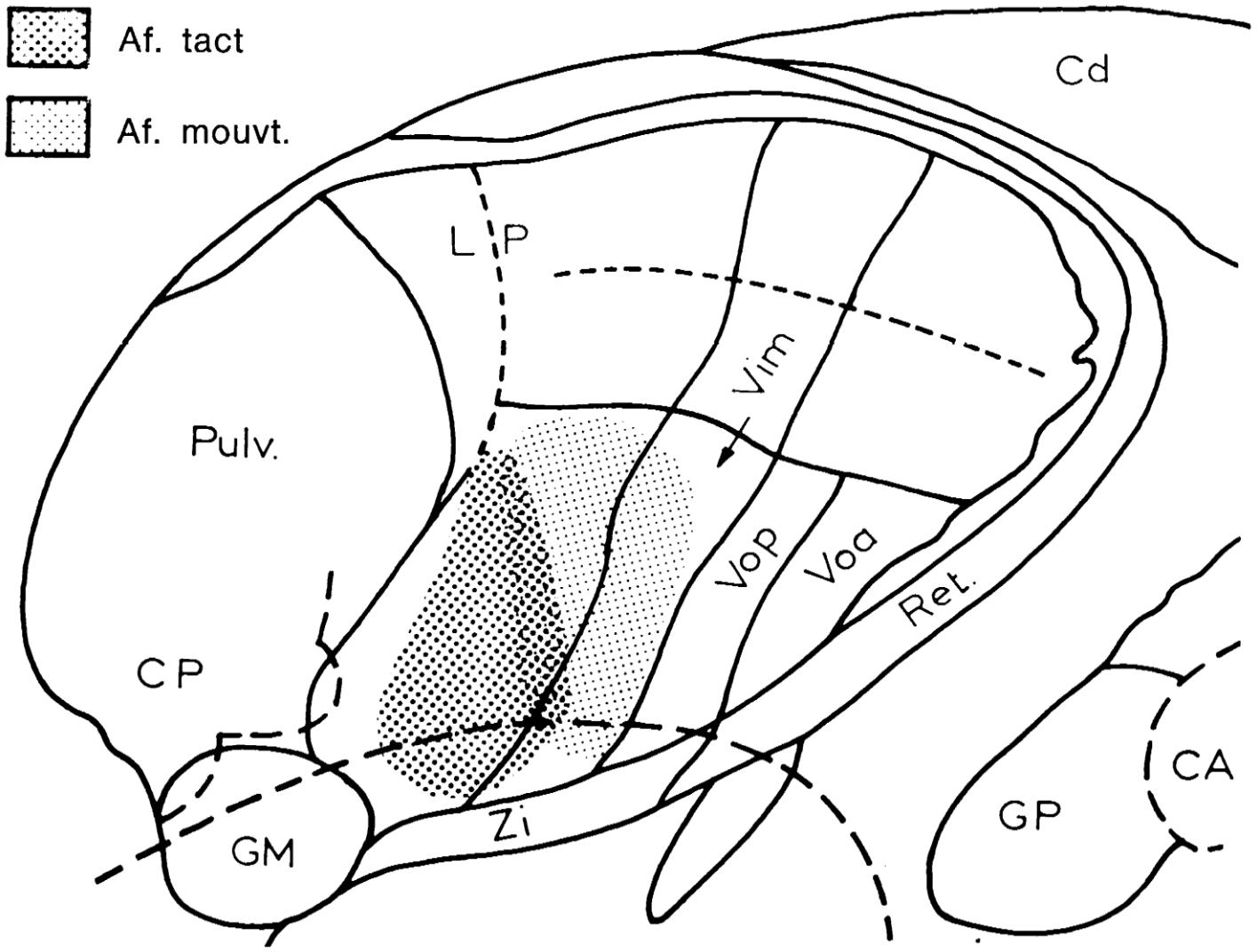
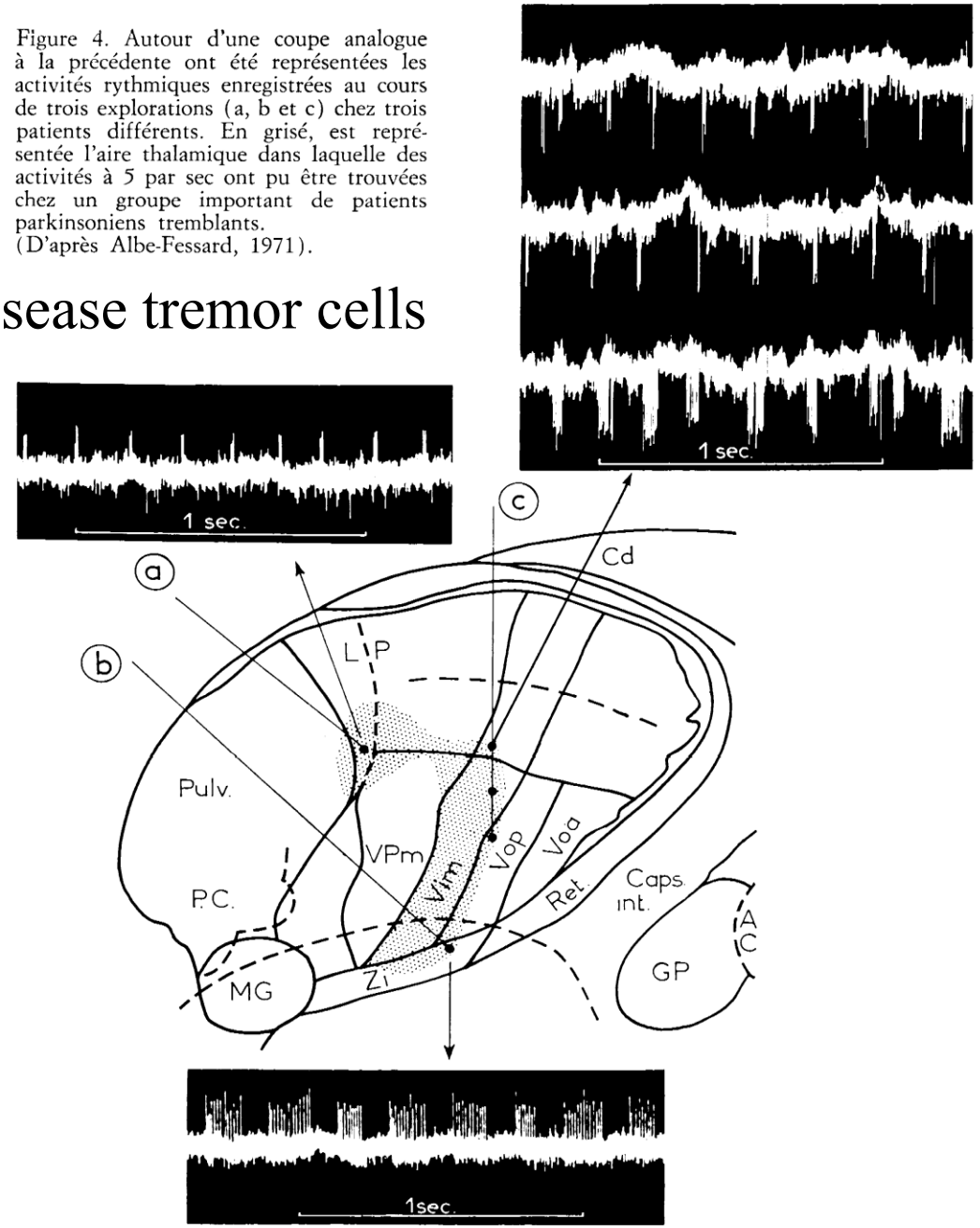


Figure 3. Coupe du thalamus humain dans un plan sagittal se trouvant à 15 mm de la ligne

D. Albe – Fessard, 1967

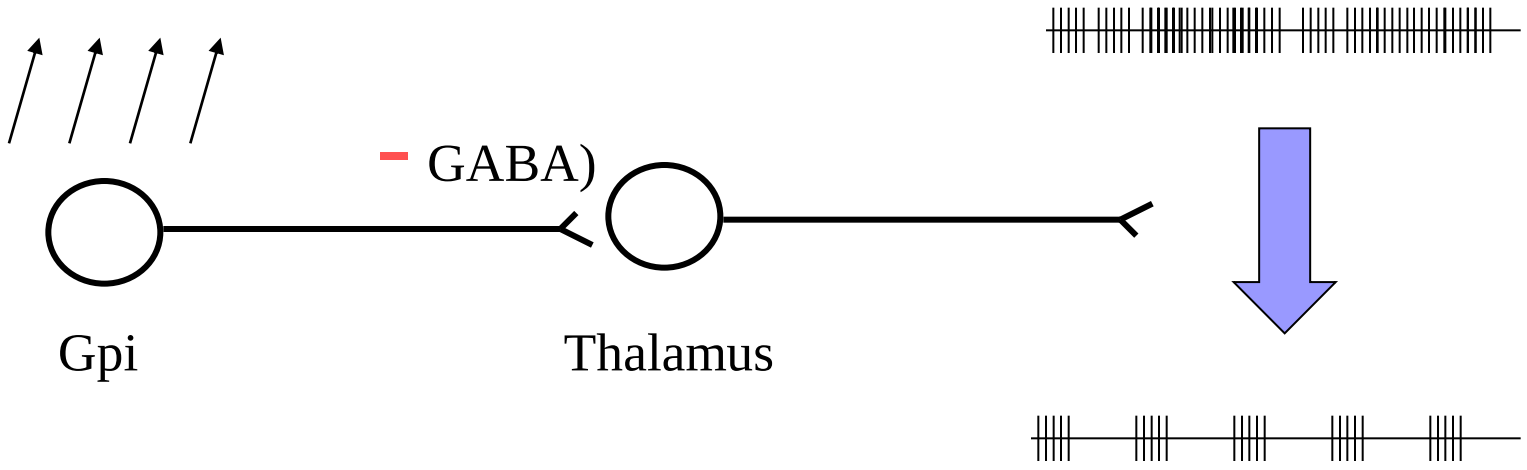
Figure 4. Autour d'une coupe analogue à la précédente ont été représentées les activités rythmiques enregistrées au cours de trois explorations (a, b et c) chez trois patients différents. En grisé, est représentée l'aire thalamique dans laquelle des activités à 5 par sec ont pu être trouvées chez un groupe important de patients parkinsoniens tremblants.
(D'après Albe-Fessard, 1971).

Parkinson's disease tremor cells

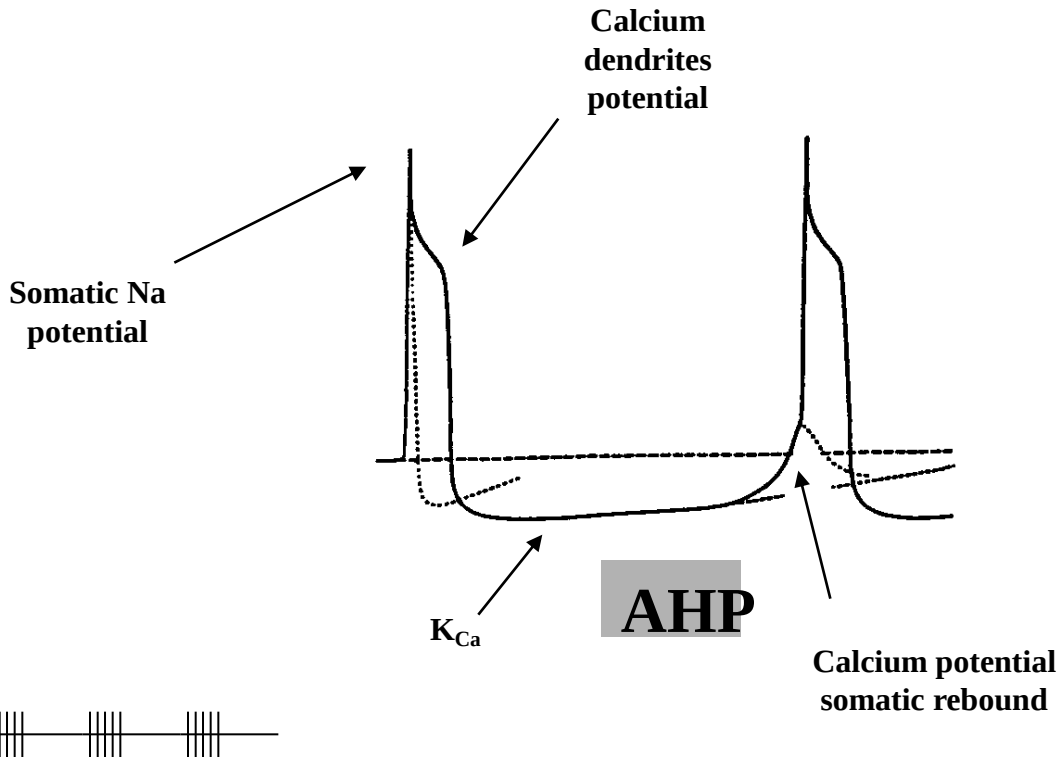


rythmic activity induced by hyperpolarisation of thalamic neurons

- In Parkinson's disease, Gpi is hyperactive
- Gpi neurotransmitter GABA creates post synaptic hyperpolarisation
- hyperpolarized thalamic neurones change from tonic to rythmic activity

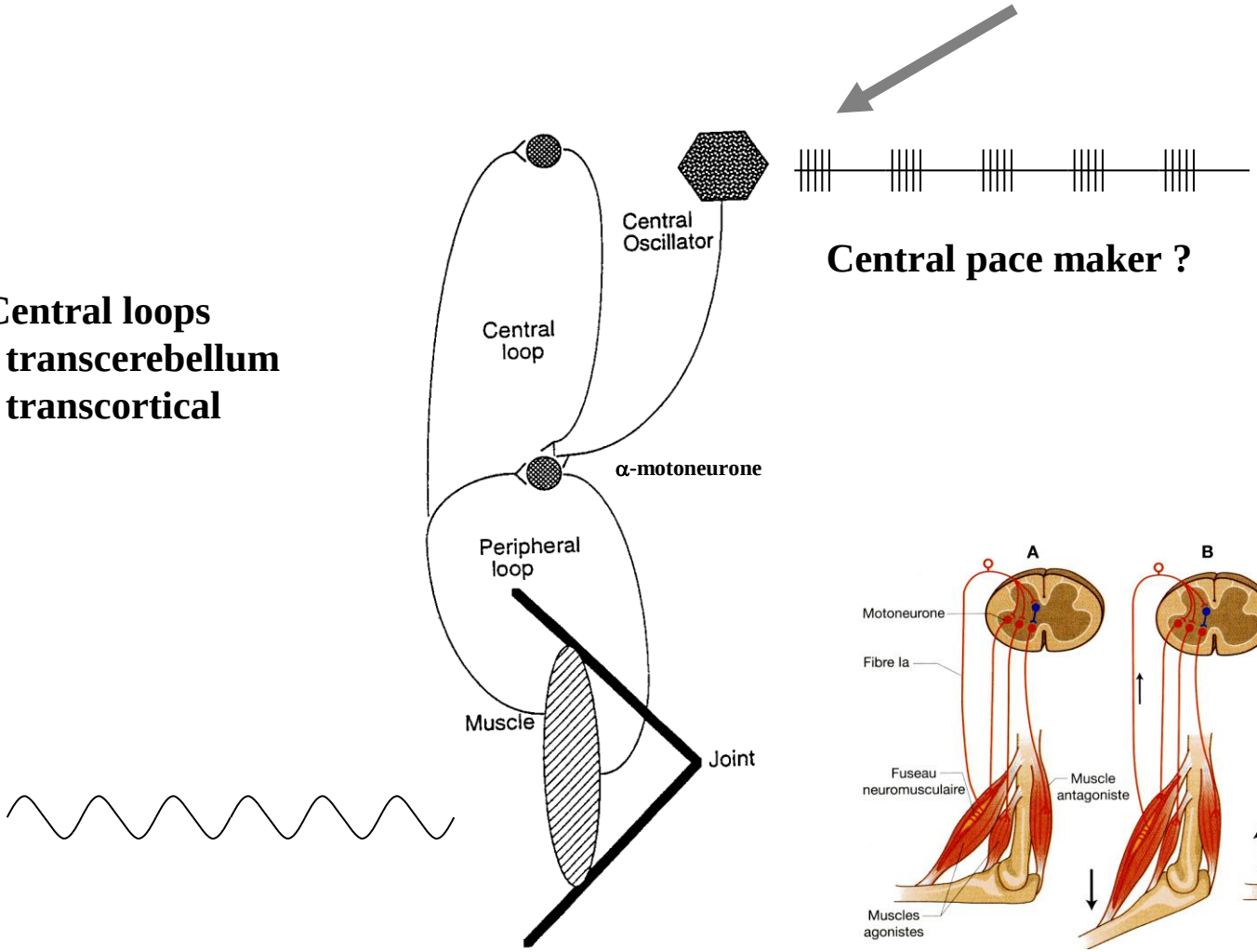


Neuronal pace-maker



Phenomenon facilitated by hyperpolarisation

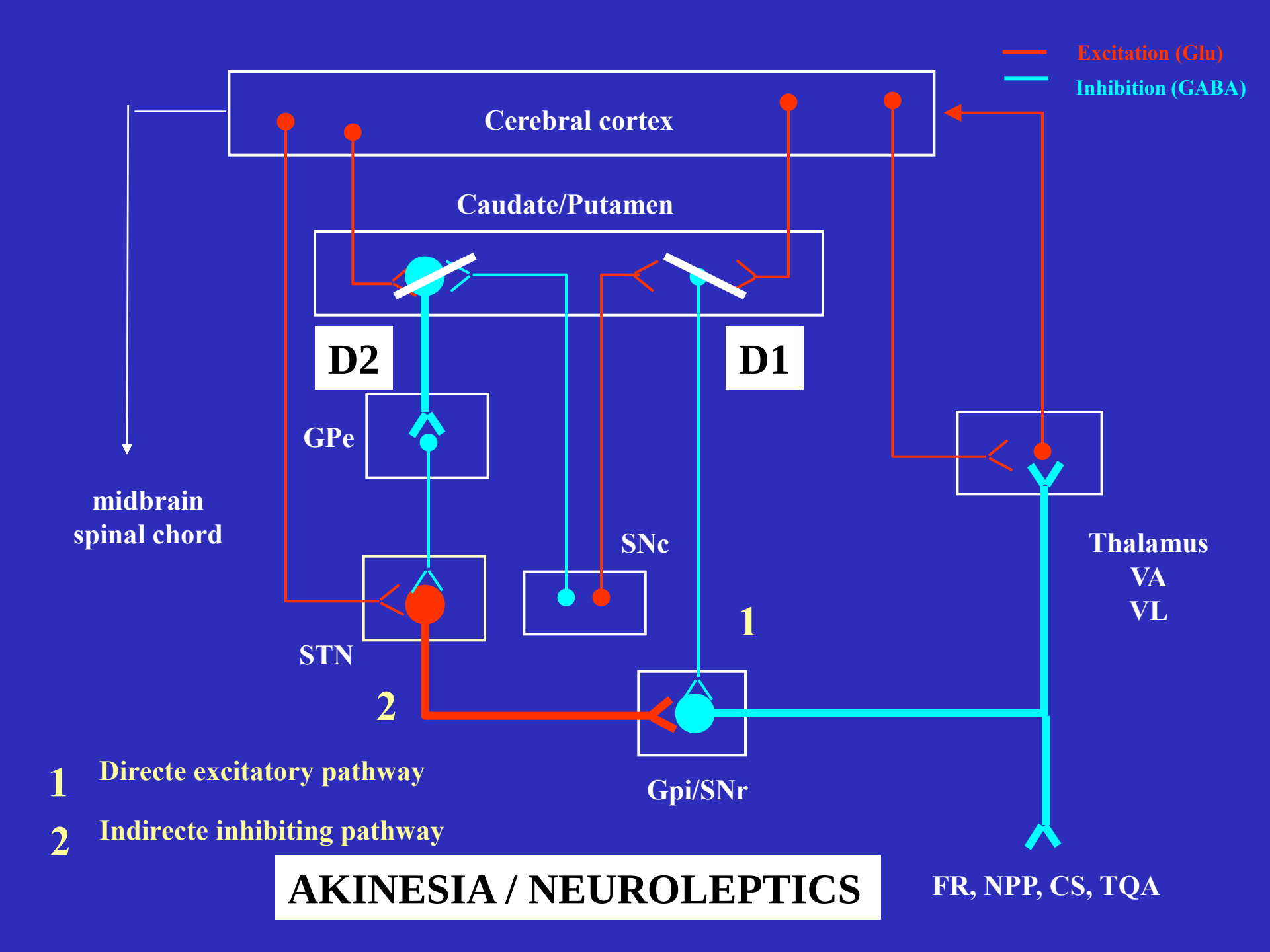
Tremor



Central pace maker ?

Central loops
- transcerebellum
- transcortical

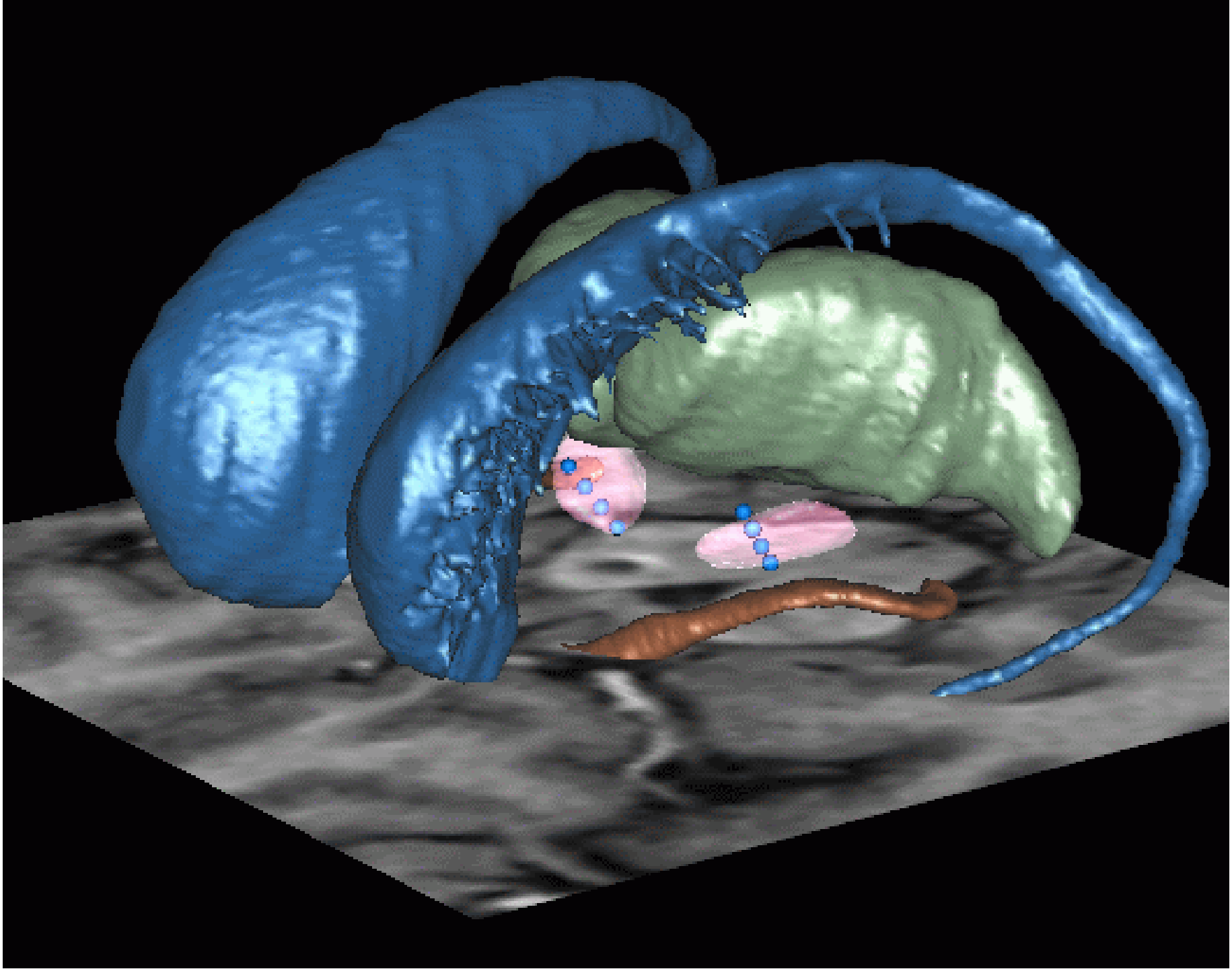
Peripheral reflex loop



1 Directe excitatory pathway

2 Indirecte inhibiting pathway

AKINESIA / NEUROLEPTICS



Parkinson’s disease

Vidéo projection

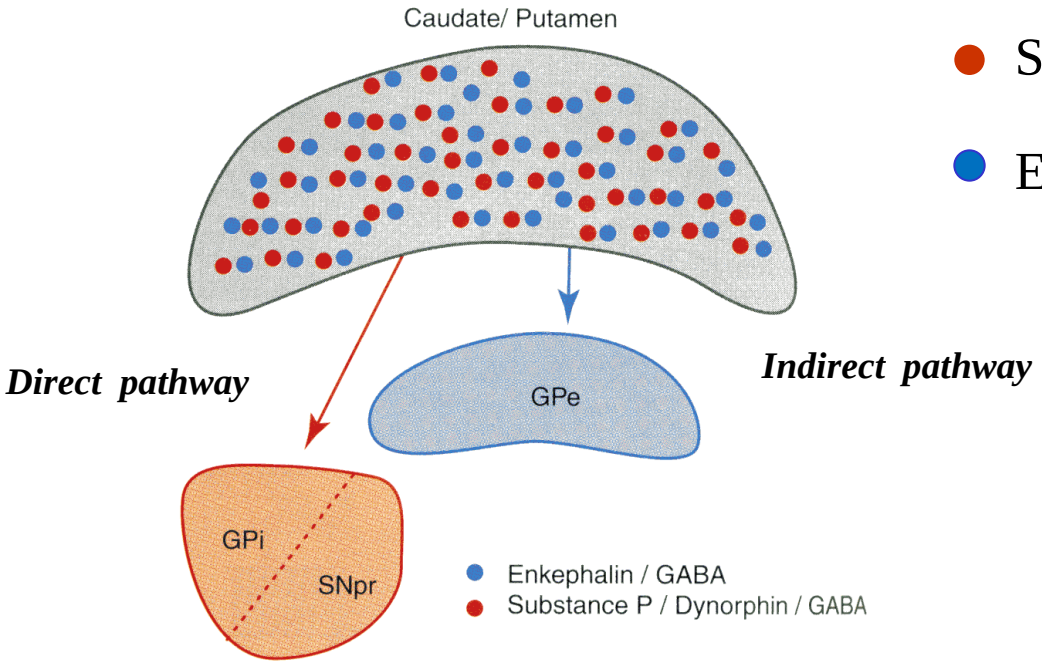
Hyperkinétic disorders

- Huntingtons’ chorea
- Ballism
- L-dopa induced dyskinesia
- Neuroleptic dyskinesia
- Gilles de la Tourette syndrome
- Obsessive Compulsive Disorders

Chorea

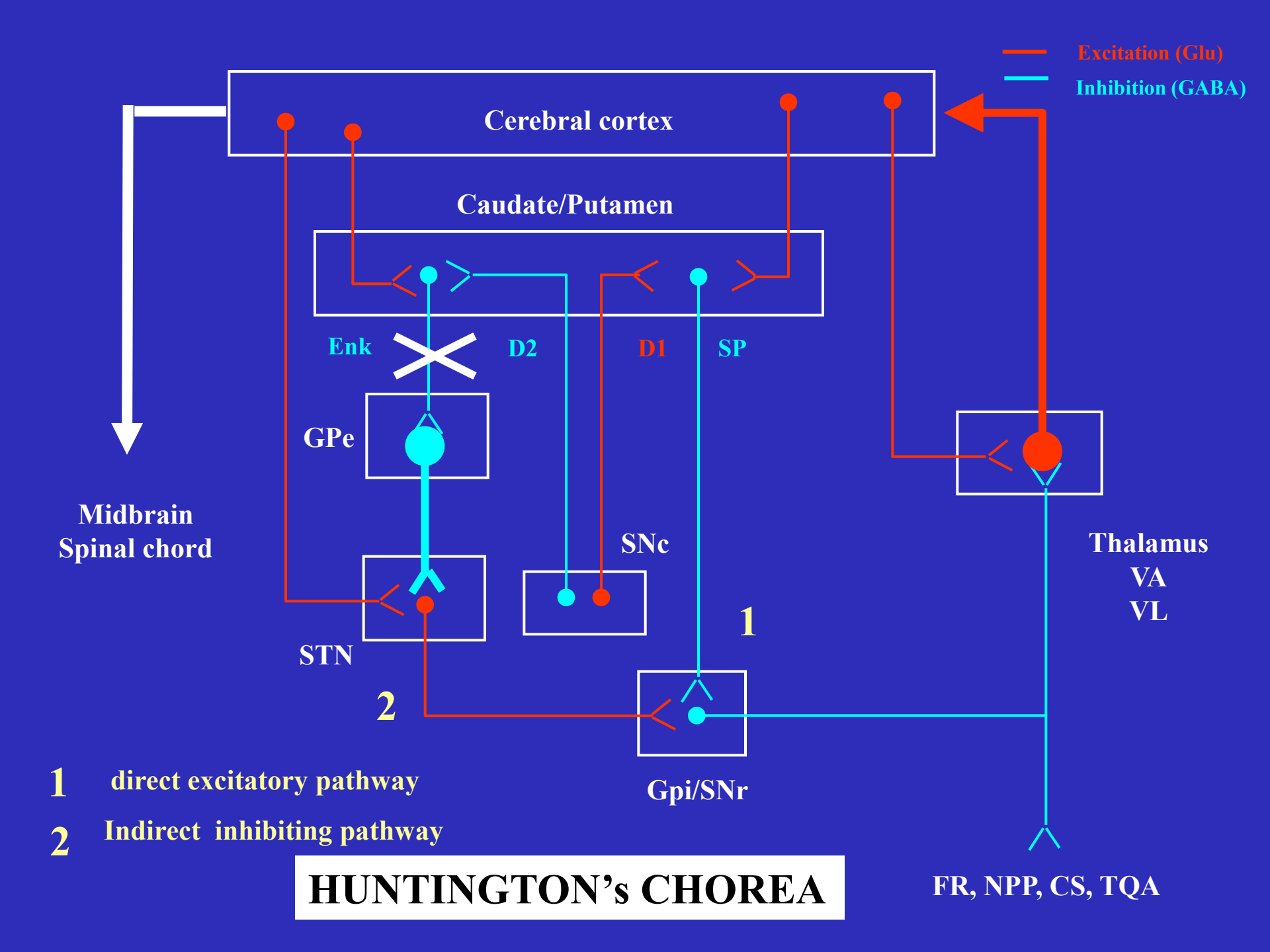
- Huntington’s chorea
  - abnormal hungtingtin gene coding for protein : cell death (apoptosis)
  - Loss of enkephalinergic « medium spiny neurons »
  - Progressively neurodegenerative
  - Hereditary
  - Chorea, depression, cognitive troubles
- Other causes

Two sub-population of striatal « medium spiny neurons »



- Substance P / Dynorphine, D1 recept
- Enkephalines, D2 receptors

From Squire, 2003

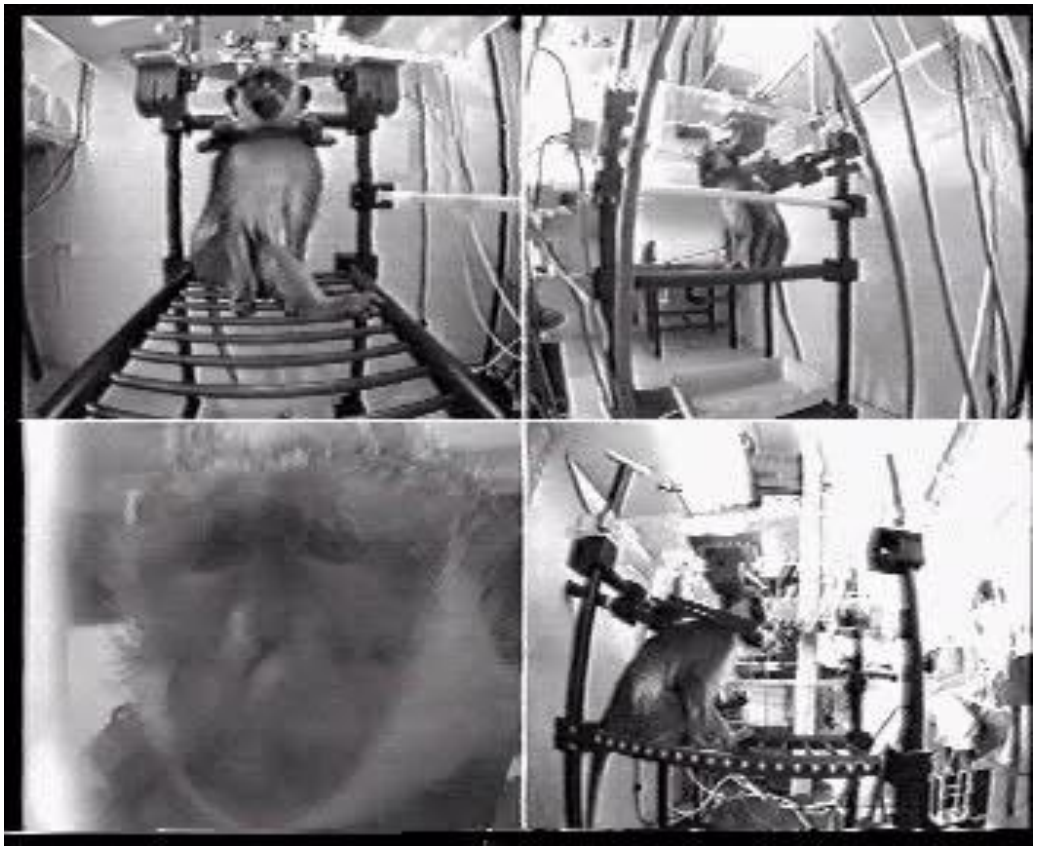


HUNTINGTON's CHOREA

Ballism

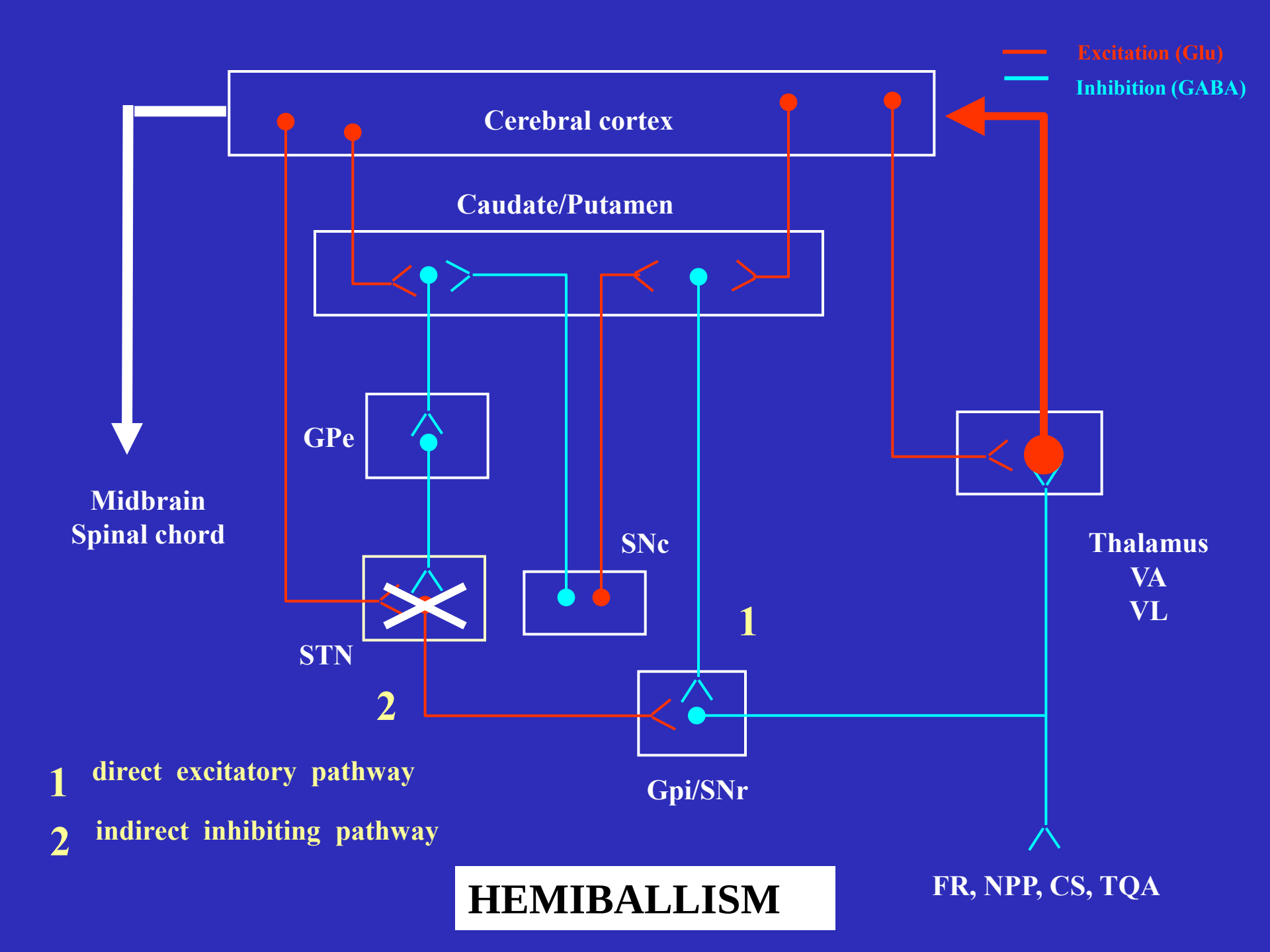
(violent and large amplitude movements)

Focal lesion of right STN
(cerebral toxoplasmosis)



bicucculine STN perfusion
(L Tremblay & D Grabli, U679, Paris, France)

Bicucculine: GABA-A antagonist
Depolarisation bloc with high dosage



1 direct excitatory pathway

2 indirect inhibiting pathway

HEMIBALLISM

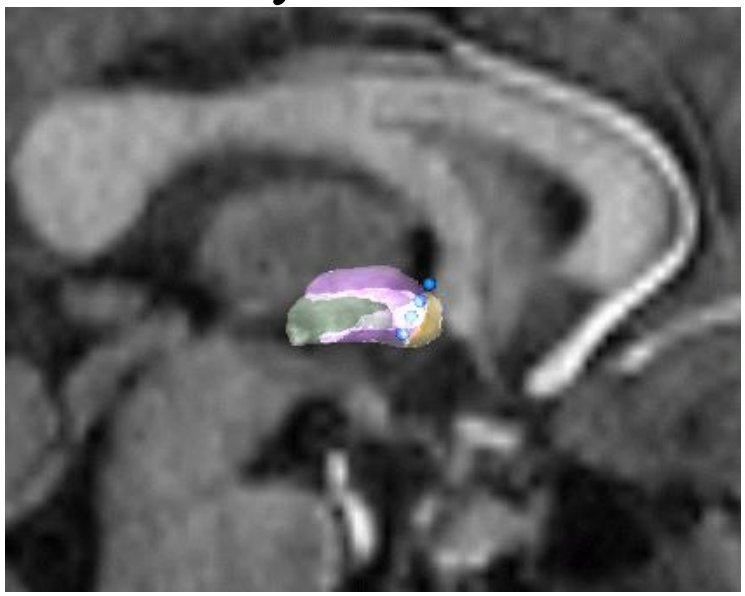
Gilles de la Tourette syndrome
(Tourette's disease)

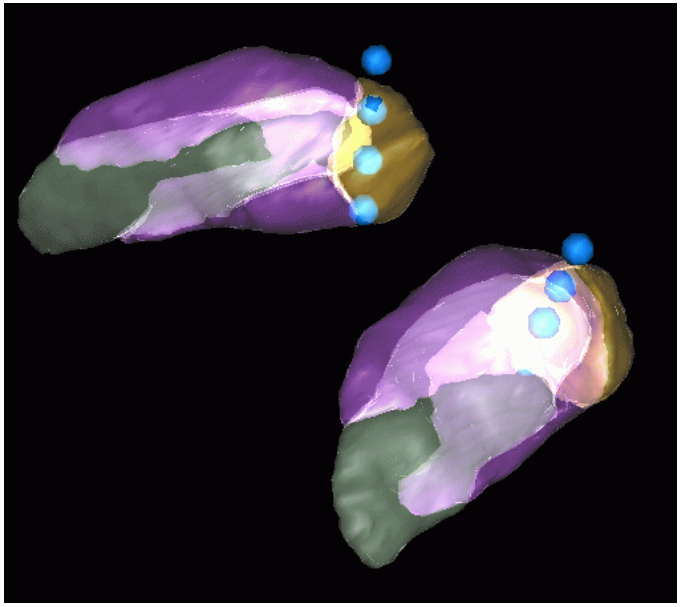
- Multiple physical motor tics
- Vocal tics, simple or complexe (coprolalia).
- Sometime associated with Obsessive Compulsive Disorders (OCD)

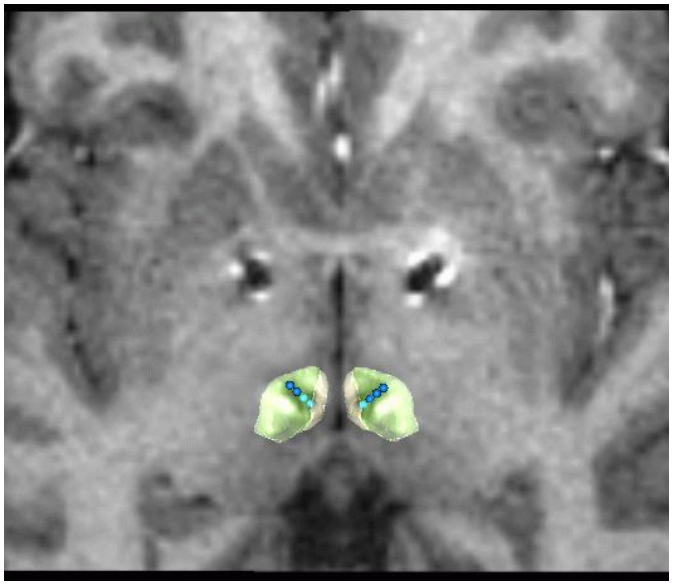
« Urge to move » and rebound after volontary control

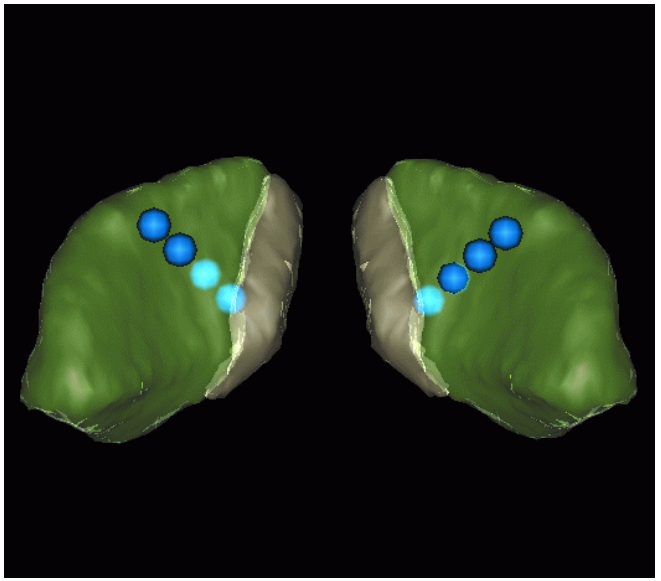
Case reports

Tourette's syndrome and DBS – Houeto et al. 2005 JNNP



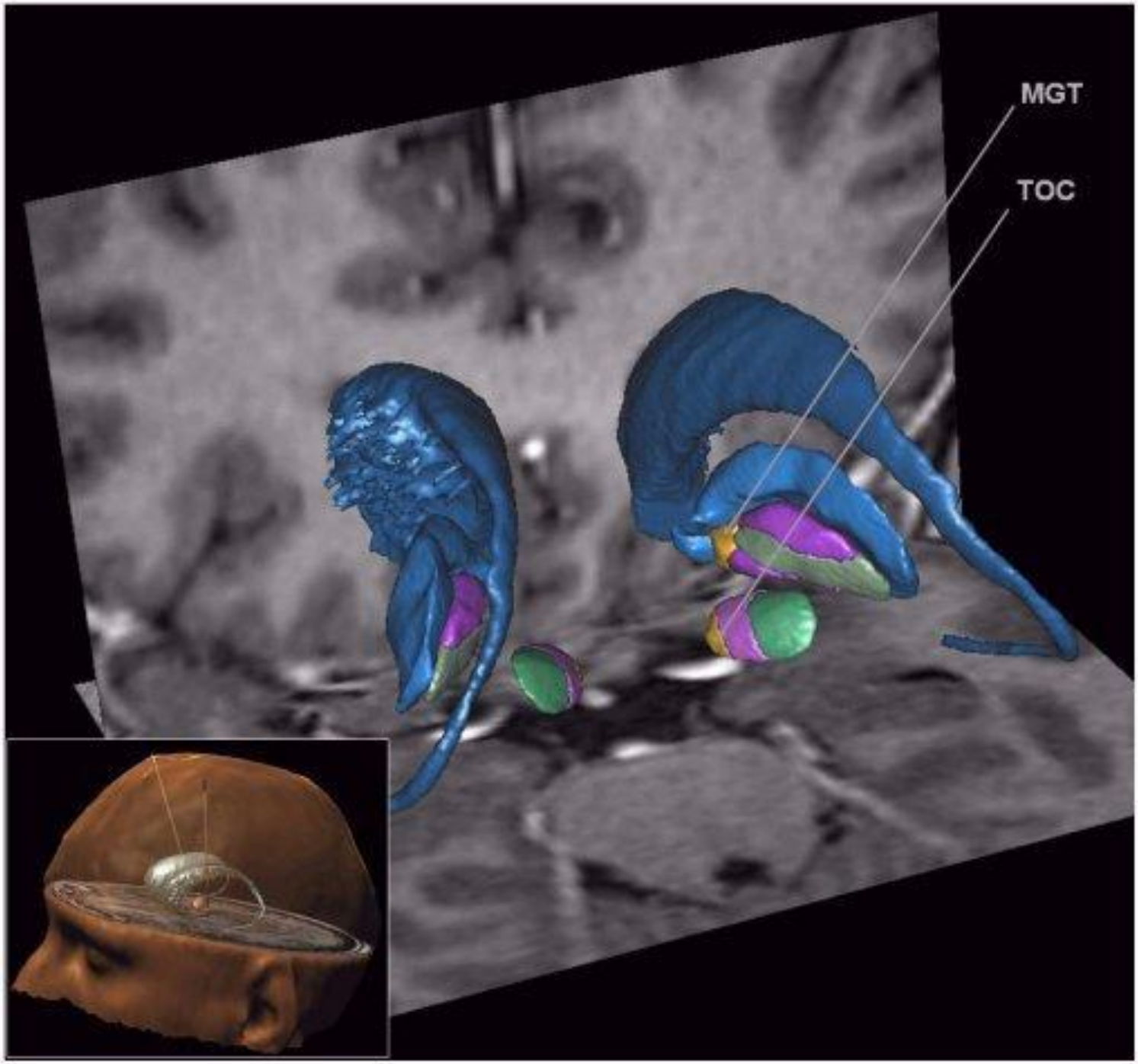




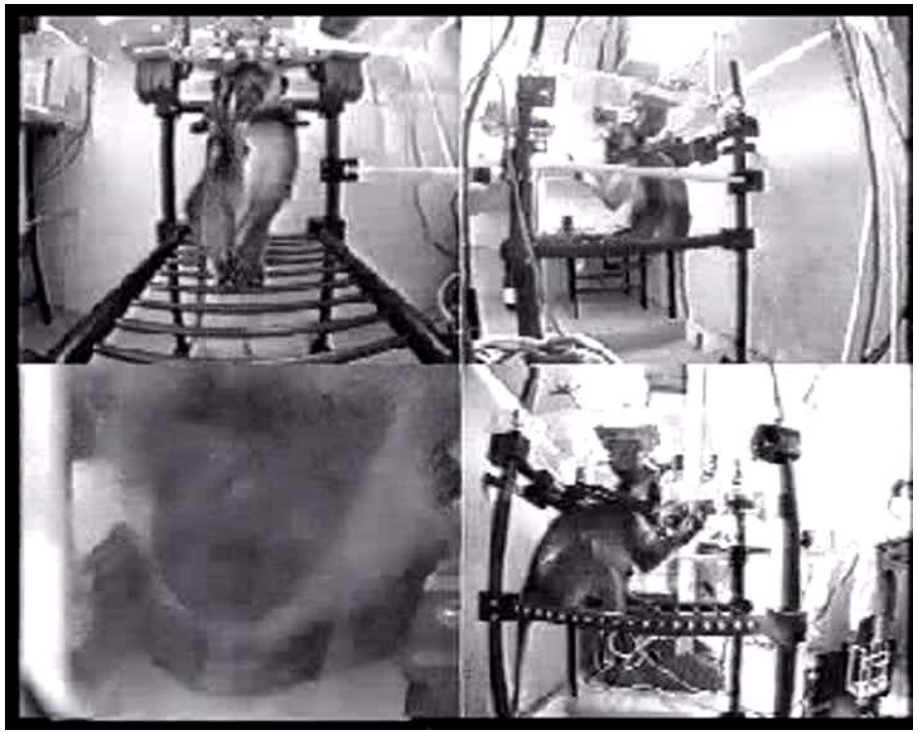


Gilles de la Tourette syndrome
(Tourette's disease)

Vidéo projection

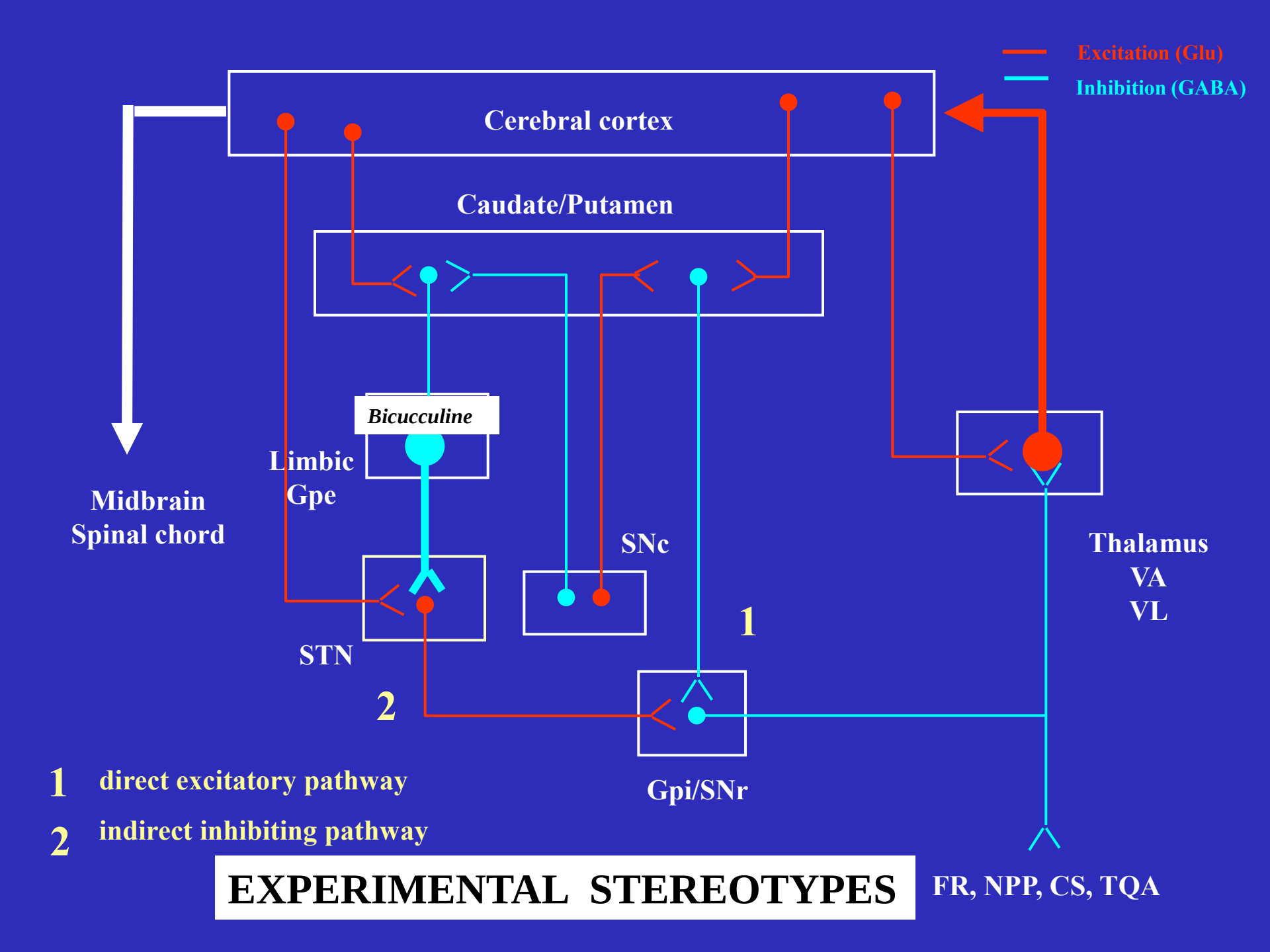

MGT
TOC

Bicucculine injected into « limbic » GPe



- stereotypical movements
- Licking
- « Touching »

Bicucculine: GABA-A post synaptic receptor antagonist Inducing GABA-A inhibition (moderate dosage)



EXPERIMENTAL STEREOTYPES

FR, NPP, CS, TQA

Micro Electrode Recording & stimulation during neurosurgery

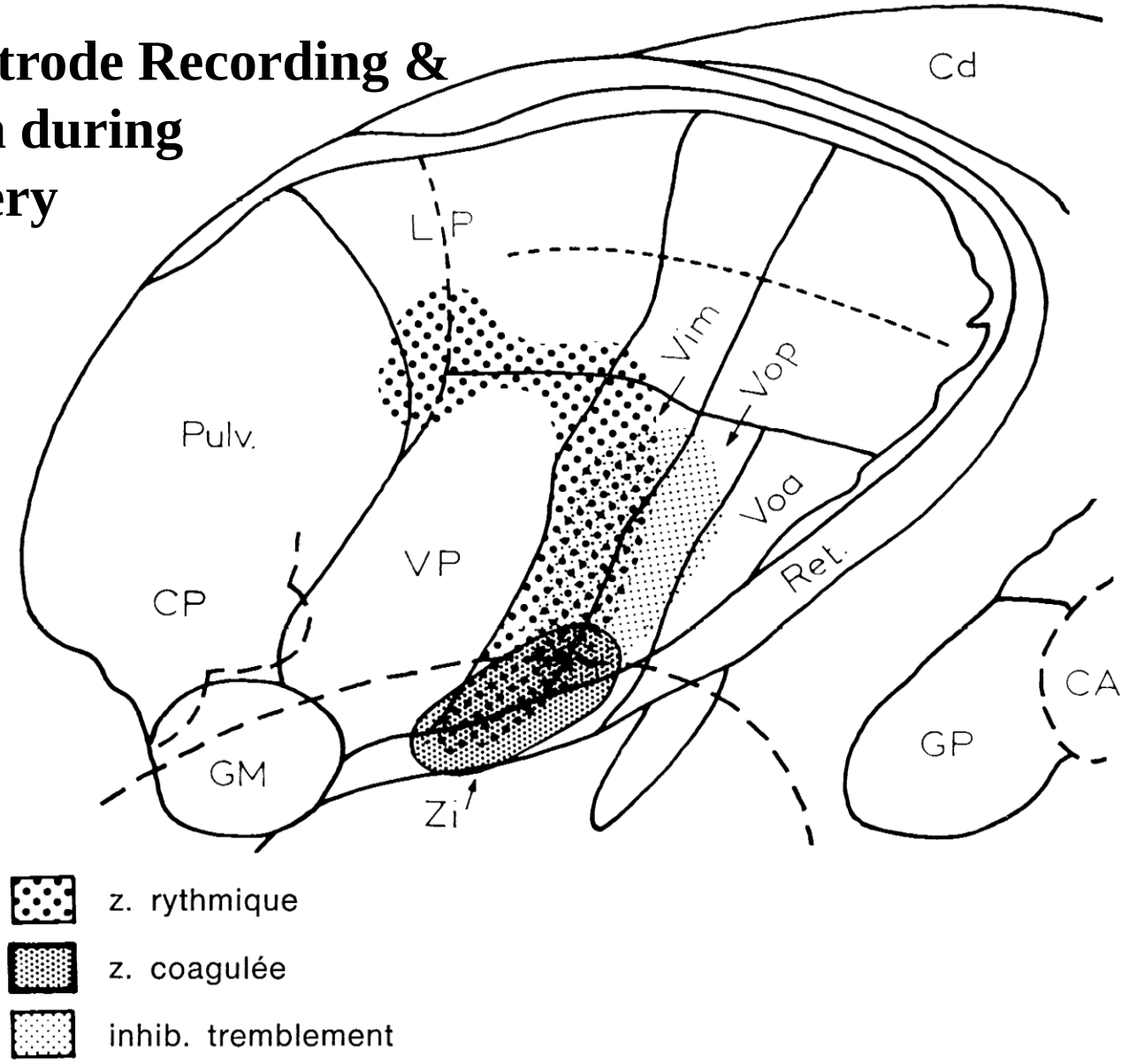


Figure 7. Sur une coupe sagittale du thalamus sont schématisées, en avant du noyau ventral postérieur, trois zones : la zone dans laquelle sont trouvées des activités rythmiques; celle dont la stimulation inhibe le tremblement; la zone où les lésions thérapeutiques sont le plus souvent réalisées.

Rev. Neurol. (Paris), 1989, *145*, 4, 320-323.

1989, the year of DBS birth in Grenoble, France :
A.L. Benabid & P. Pollak
high frequency chronic stimulation of thalamus Vim for the treatment of Parkinson's disease tremor

TRAITEMENT DU TREMBLEMENT PARKINSONIEN PAR STIMULATION CHRONIQUE DU NOYAU VENTRAL INTERMÉDAIRE DU THALAMUS

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RÉSUMÉ

La thalamotomie stéréotaxique du noyau Ventral Intermédiaire (Vim) peut améliorer le tremblement parkinsonien sévère résistant aux médicaments. Afin de diminuer le nombre d'échecs et les effets indésirables, une stimulation chronique à haute fréquence (130 Hz) du noyau Vim a été effectuée chez 4 patients dont deux avaient déjà subi une thalamotomie de l'autre côté. Le tremblement a été supprimé chez tous les patients, au prix de paresthésies légères, avec un recul allant de 2 à 14 mois. Les effets aussi bien bénéfiques que secondaires ont été immédiatement supprimés à chaque arrêt de la stimulation. Ces résultats préliminaires sont encourageants, mais il convient d'apprécier la persistance de l'efficacité au long cours sur un plus grand nombre de cas.

Treatment of parkinsonian tremor by chronic Vim-thalamic stimulation.

A.L. BENABID, P. POLLAK, M. HOMMEL, J.M. GAIO, J. DE ROUGEMONT, J. PERRET. *Rev. Neurol. (Paris)*, 1989, *145* : 4, 320-323.

SUMMARY

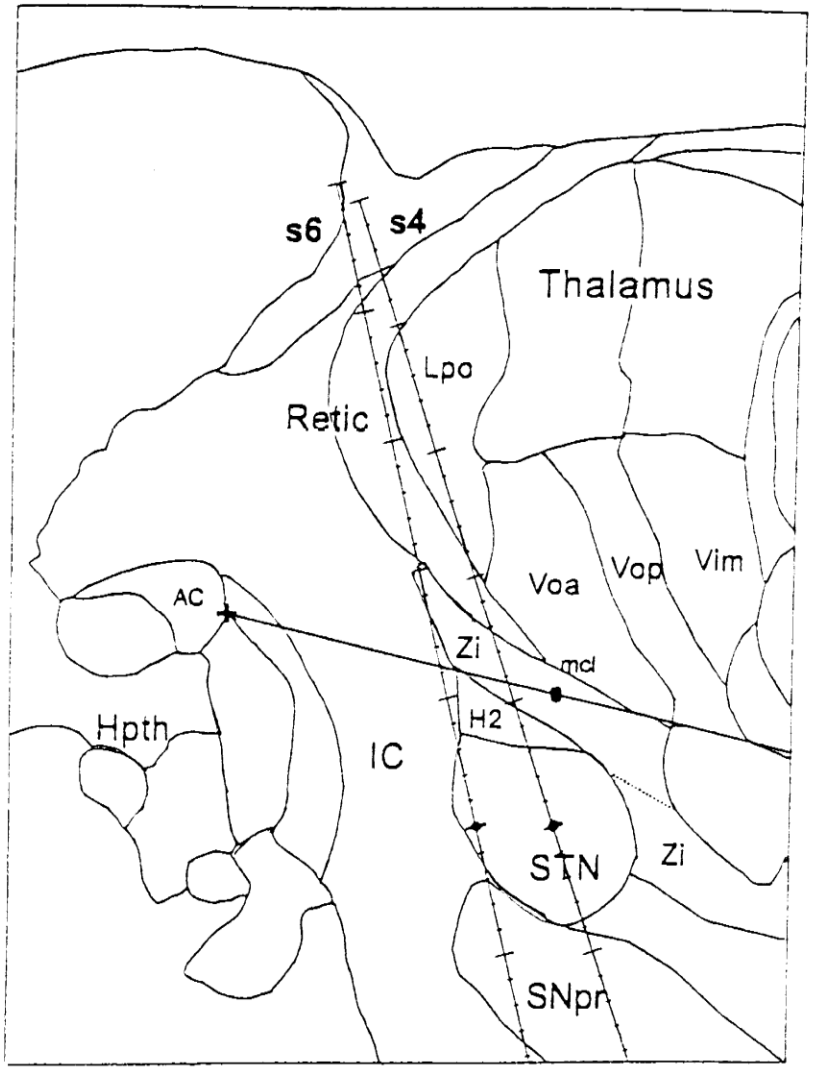


Fig 1. Sagittal section of Schaltenbrand and Wahren stereotactic atlas at 10.5 mm from the midline showing the location of the electrode trajectories targeting the subthalamic nucleus.

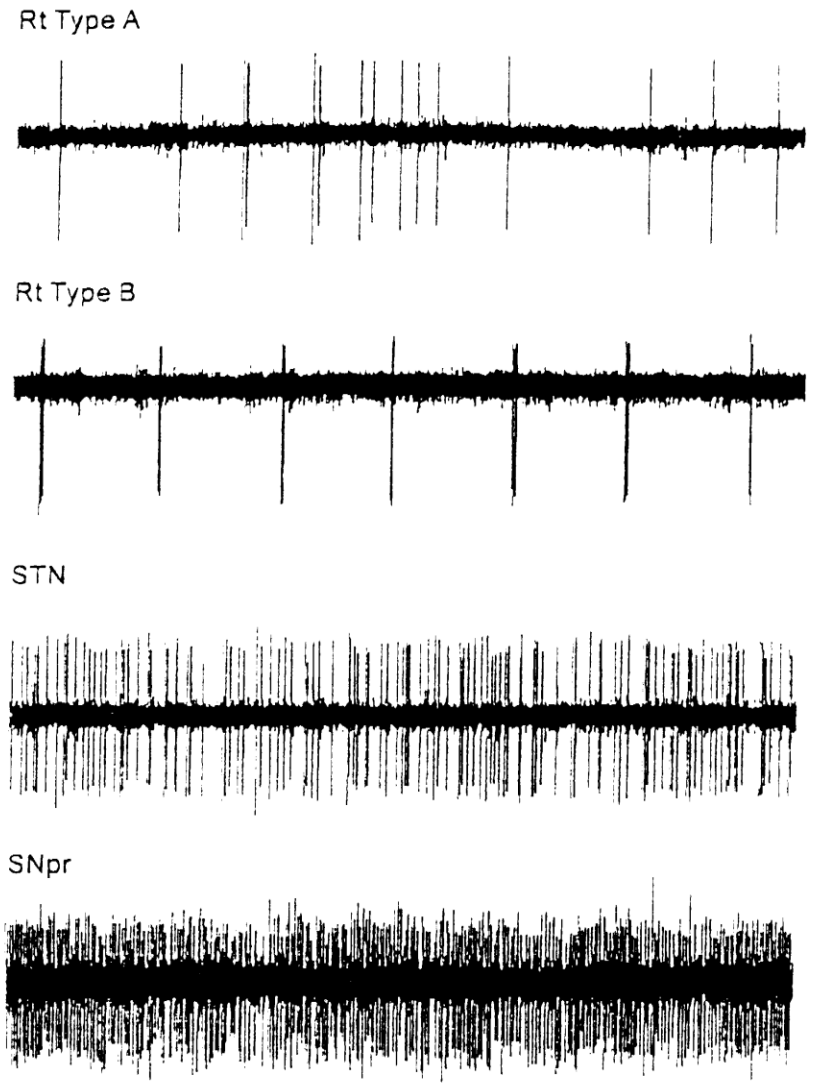
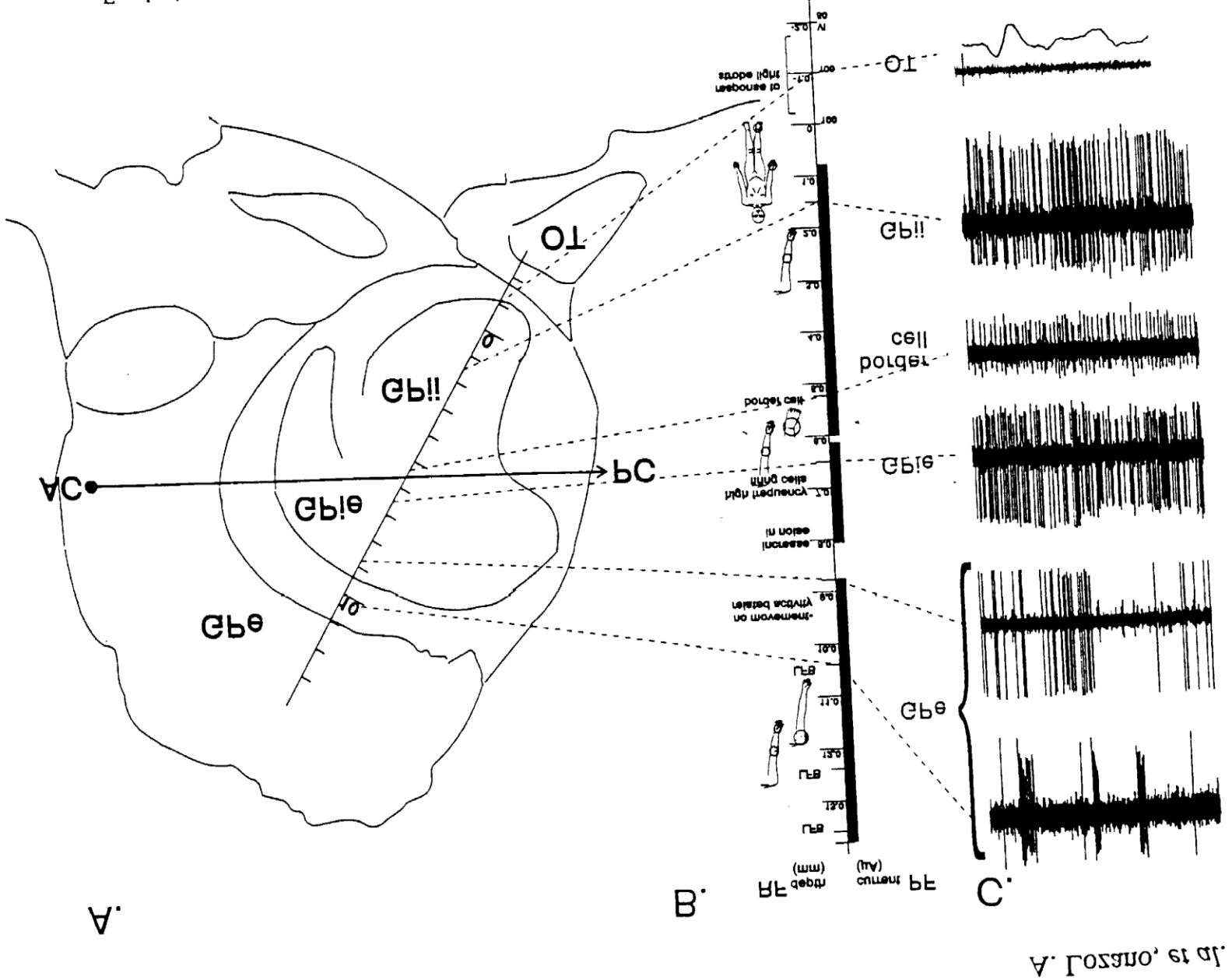


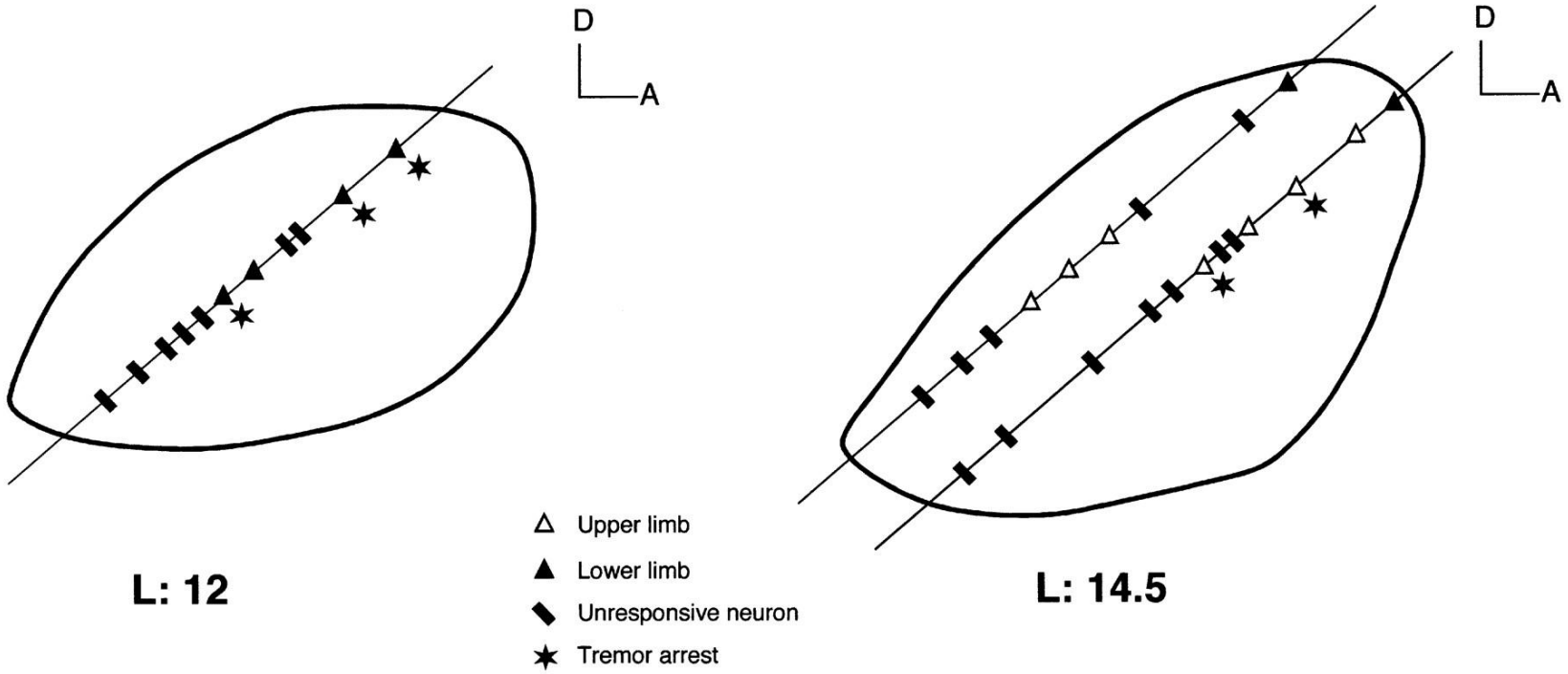
Fig 2. Spontaneous ongoing discharge of typical neurons recorded in trajectories targeting the subthalamic nucleus. (Top

Hutchinson et al., Annals of Neurol., 1998, 622-627



Lozano et al, J. Neurosurg. 84:194-202, 1996

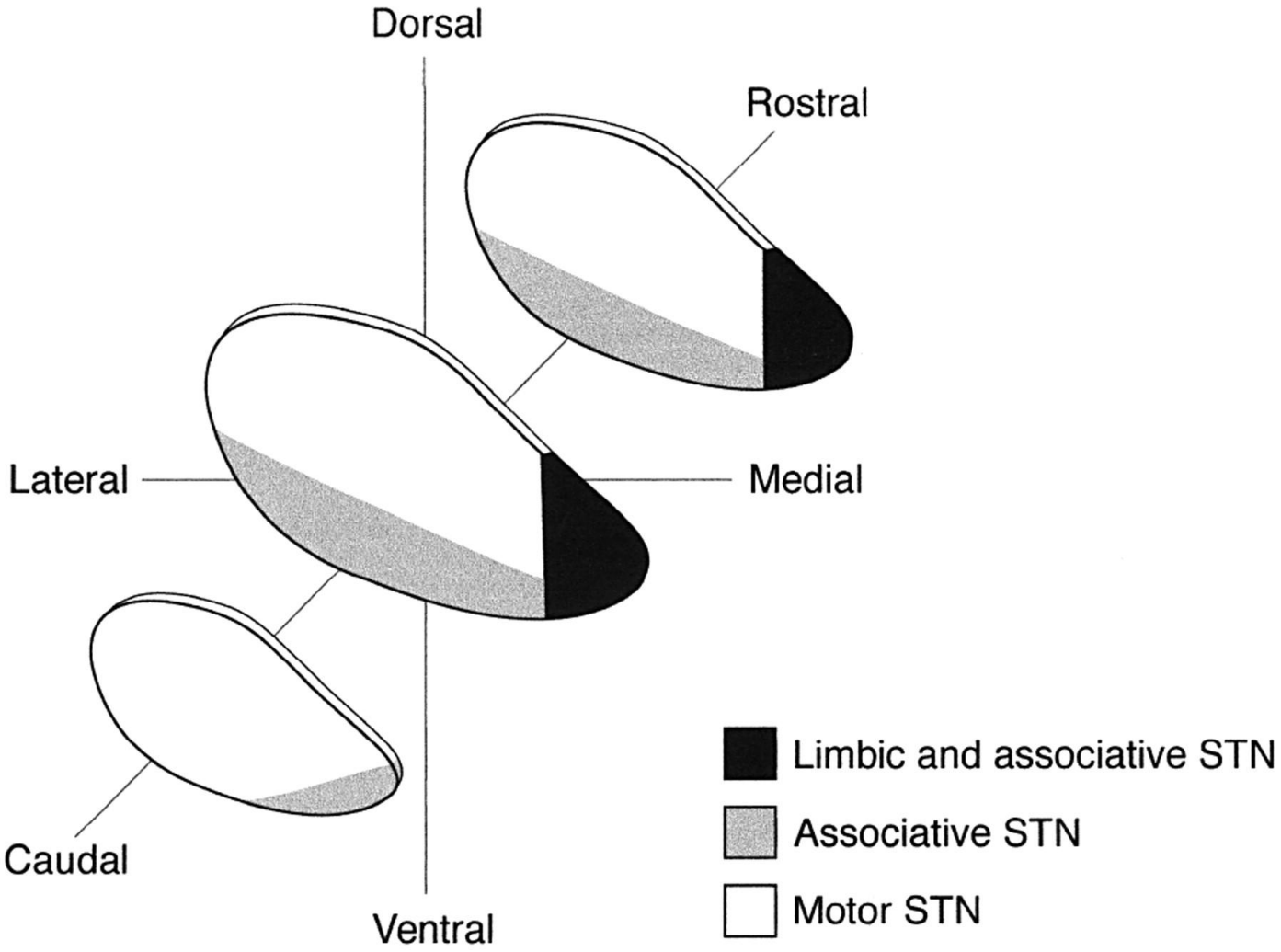
Somatotopic arrangement of STN neurones responding to passive or active movements in a patient with Parkinson's disease.



Rodriguez-Oroz M C et al. Brain 2001;124:1777-1790

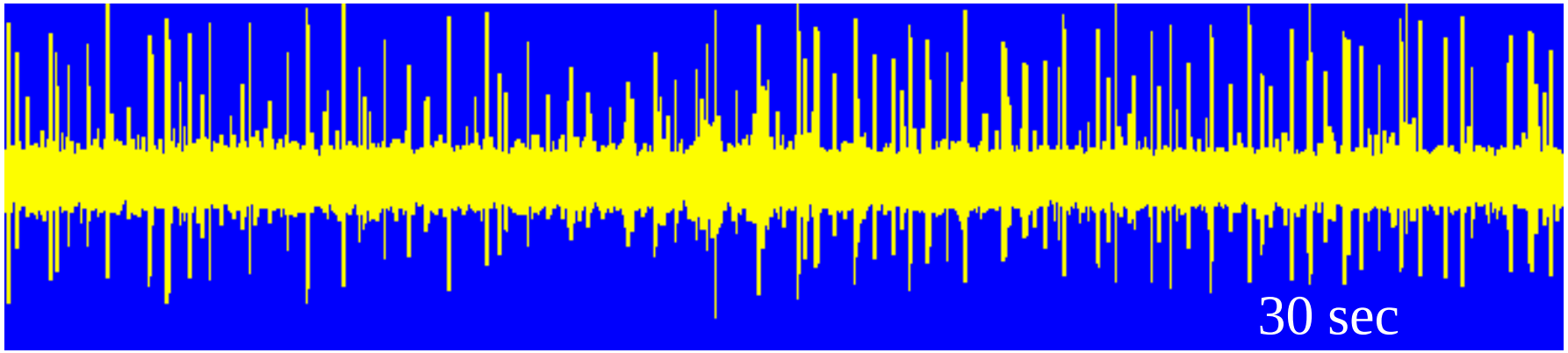
BRAIN A JOURNAL OF NEUROLOGY

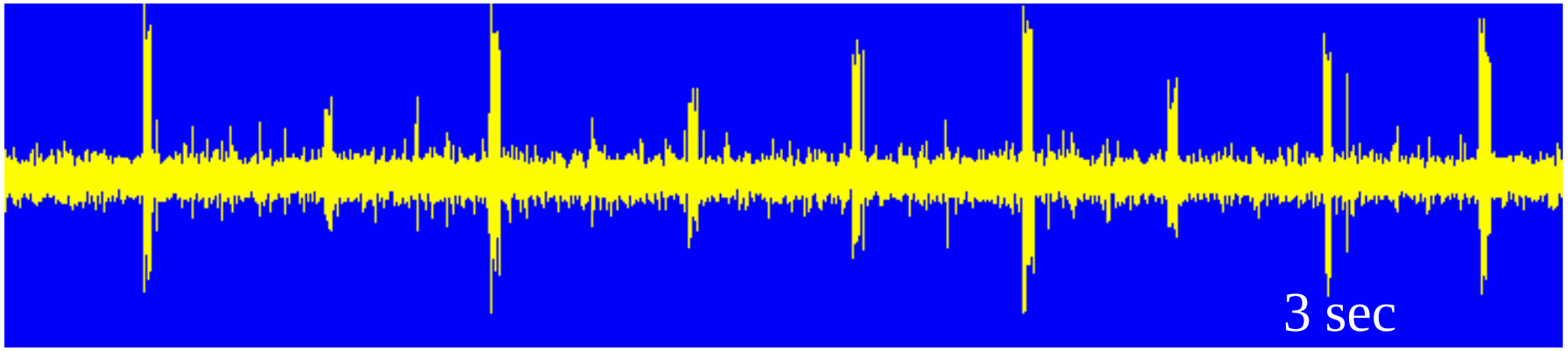
Fig. 2 Schematic representation of the intrinsic organization of the subthalamic nucleus (STN) according to the tripartite functional subdivision of the basal ganglia.

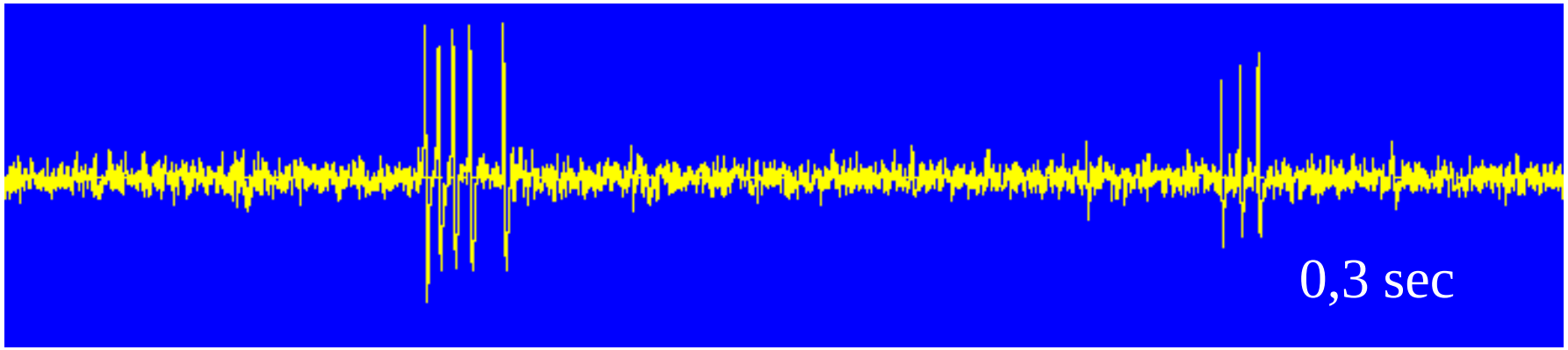


Hamani C et al. Brain 2004;127:4-20

BRAIN A JOURNAL OF NEUROLOGY

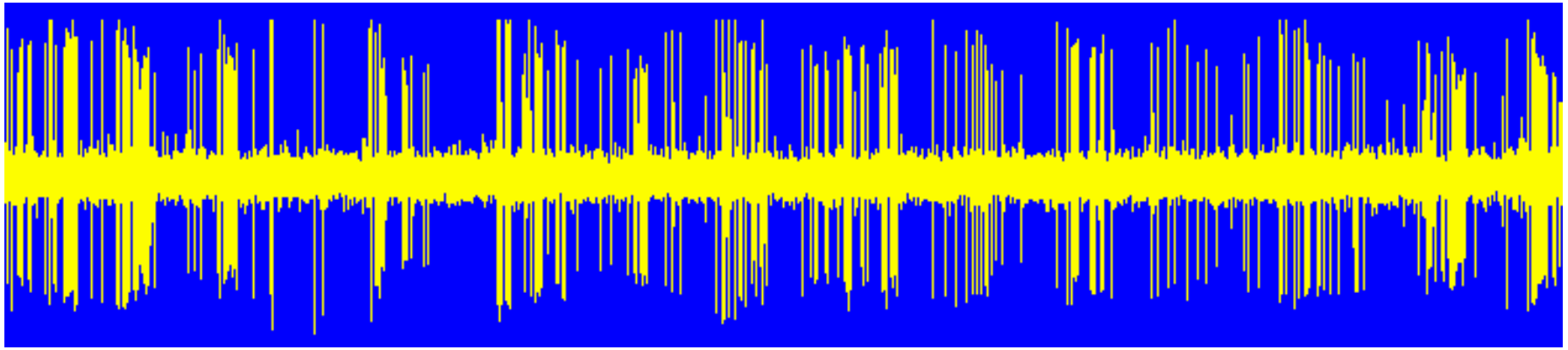






microelectrode réticular thalamic nucleus recording

Parkinson : Subthalamic Nucleus - STN



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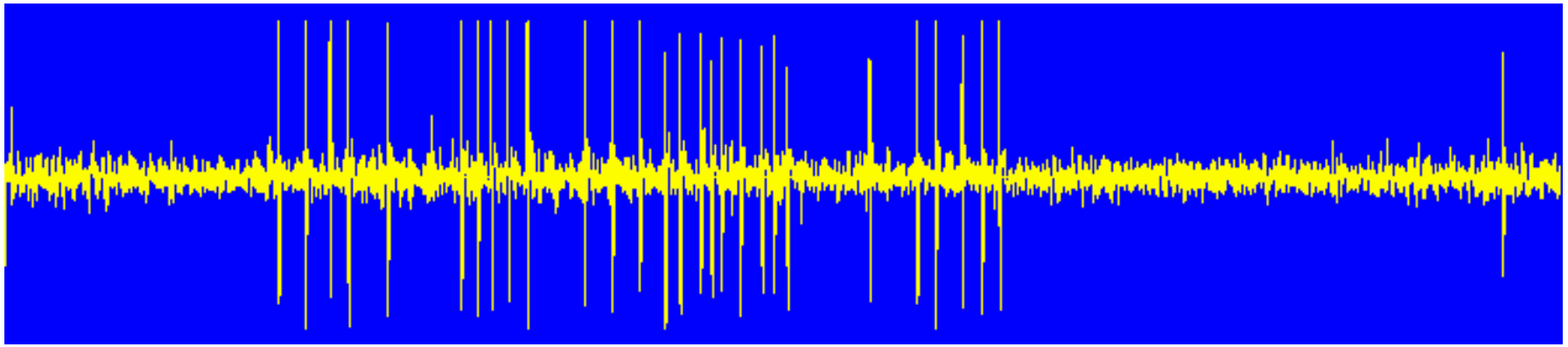

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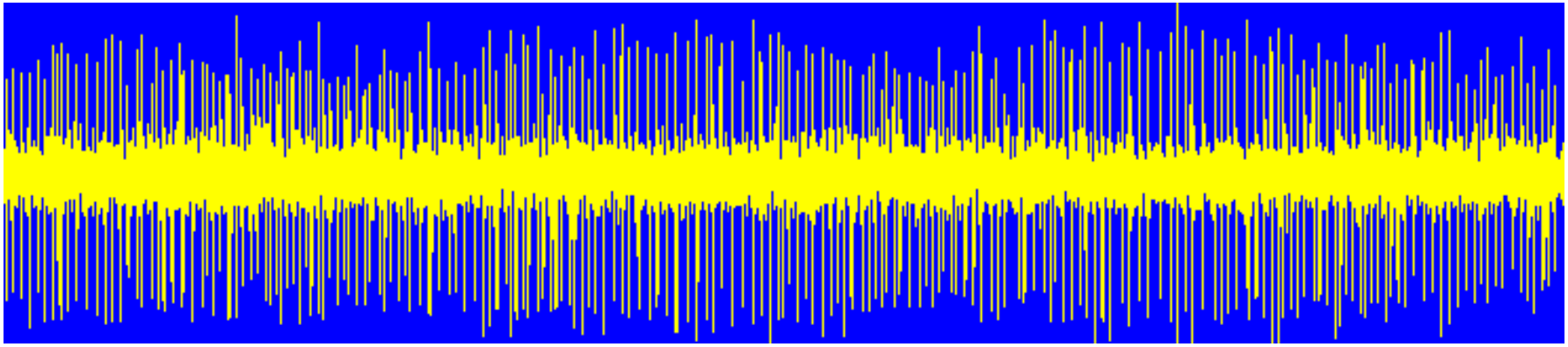
Microelectrode single cell recording

Parkinson : Substantia Nigra recording



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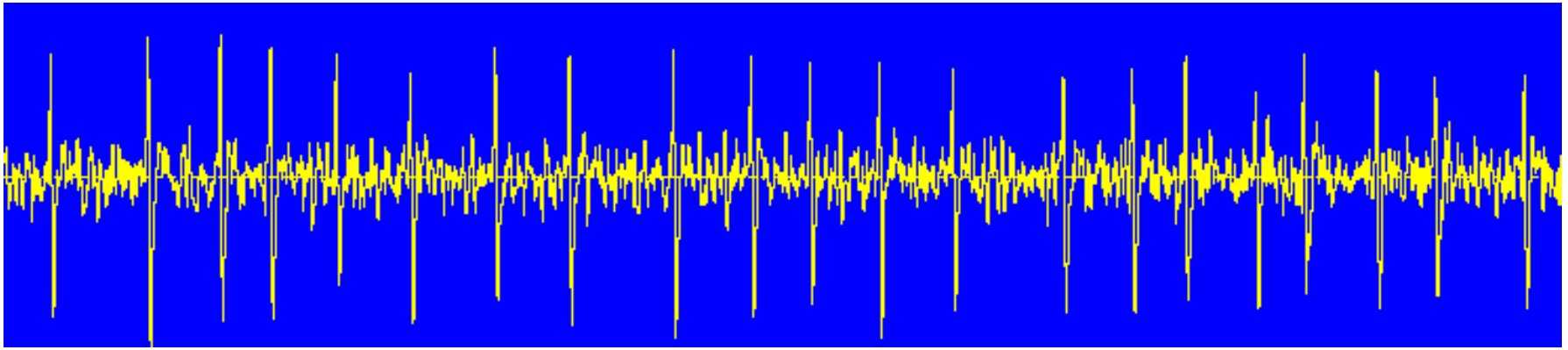


0,2 sec

DBS HFS of GPi improves Levo-DOPA Induced Dyskinesia and Dystonia

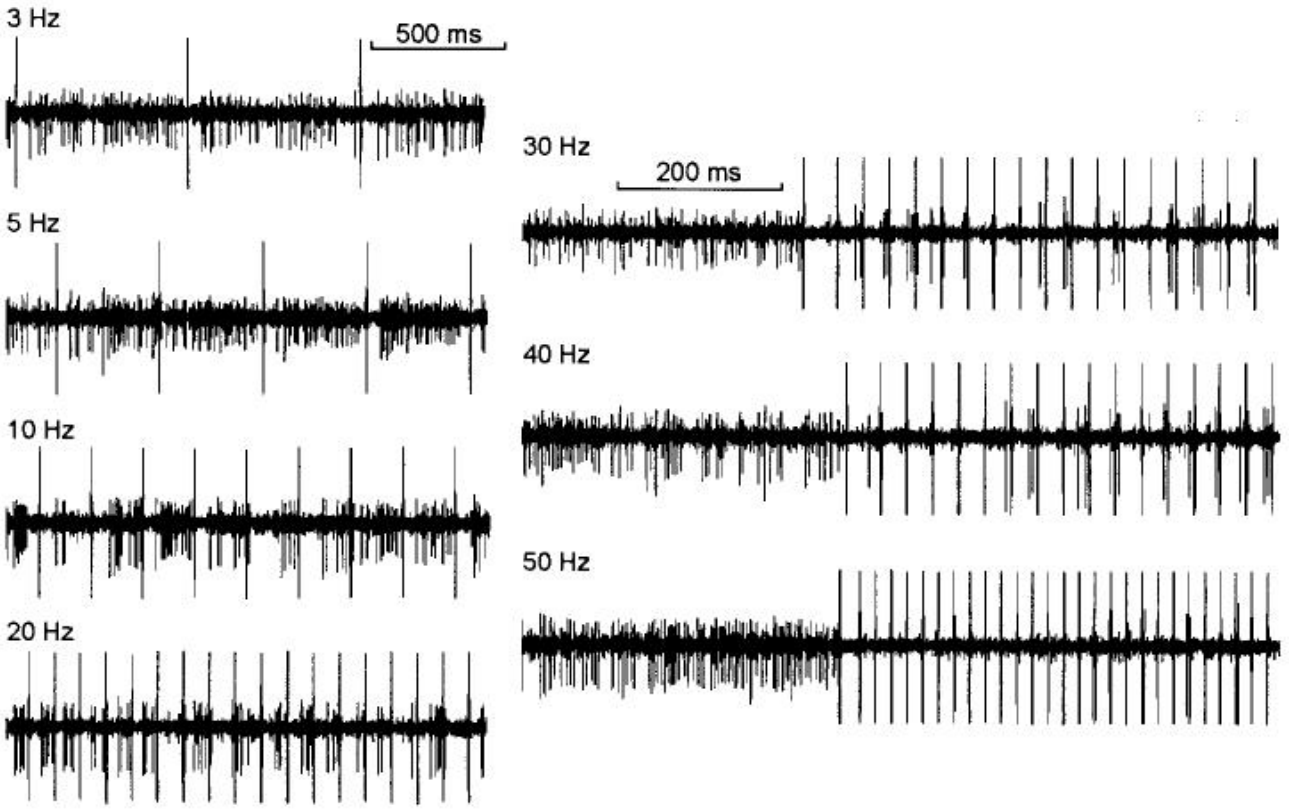
Possible mechanisms :

DBS may induce GABA release in GPi

(Dostrovksy et al, J. Neurophysiol 2000)

HFS may introduce more regular pattern in GPi output cancelling abnormal movements

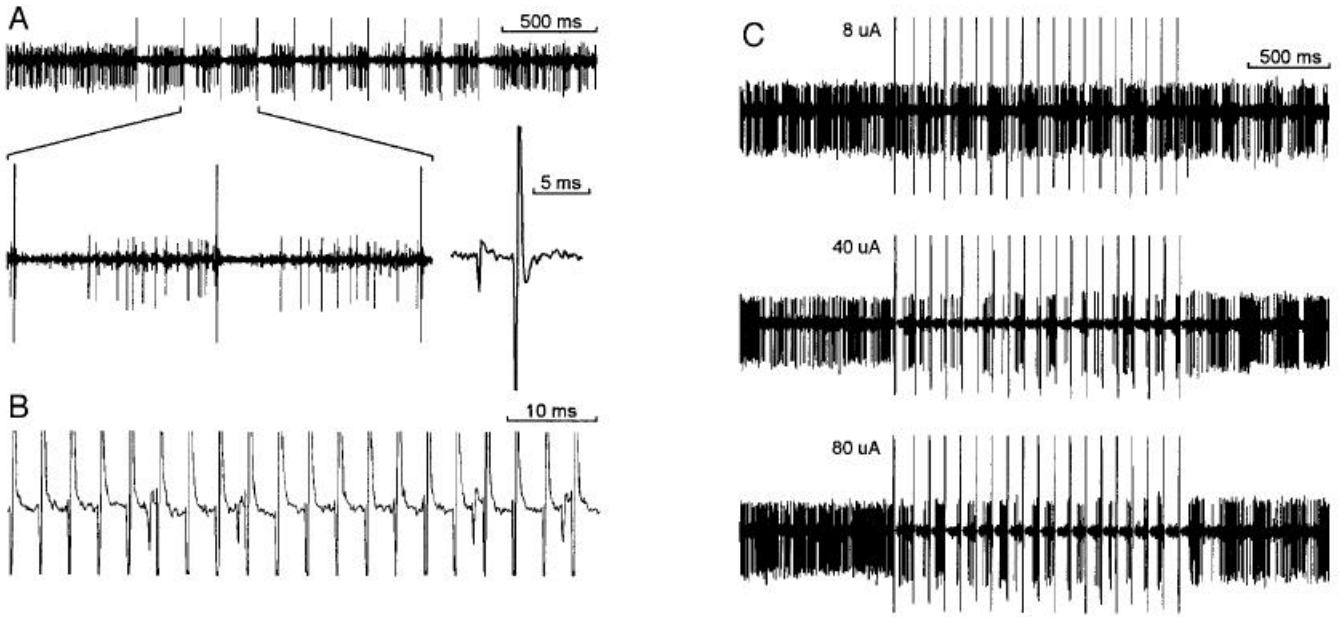
Effects of Pallidal HFS



Effect of increasing stimulation frequency

Dostrovsky JO, Levy R, Wu JP, Hutchison WD, Tasker RR, and Lozano AM. Microstimulation-induced inhibition of neuronal firing in human globus pallidus. *J Neurophysiol* 84: 570–574, 2000.

Effects of Pallidal HFS



Dostrovsky JO, Levy R, Wu JP, Hutchison WD, Tasker RR, and Lozano AM. Microstimulation-induced inhibition of neuronal firing in human globus pallidus. *J Neurophysiol* 84: 570–574, 2000.

Inhibition by release of GABA neurotransmitter ?

ORIGINAL CONTRIBUTION

Effects of High-Frequency Stimulation on Subthalamic Neuronal Activity in Parkinsonian Patients

Marie-Laure Welter, MD; Jean-Luc Houeto, MD; Anne-Marie Bonnet, MD; Paul-Boulos Bejjani, MD; Valérie Mesnage, MD; Didier Dormont, MD; Soledad Navarro, MD; Philippe Cornu, MD, PhD; Yves Agid, MD, PhD; Bernard Pidoux, MD, PhD

Background: High-frequency stimulation of the subthalamic nucleus (STN) is a neurosurgical alternative to medical treatment in levodopa-responsive forms of Parkinson disease. The mechanism of action of STN stimulation remains controversial, although an inhibition of overactive STN neurons has been postulated.

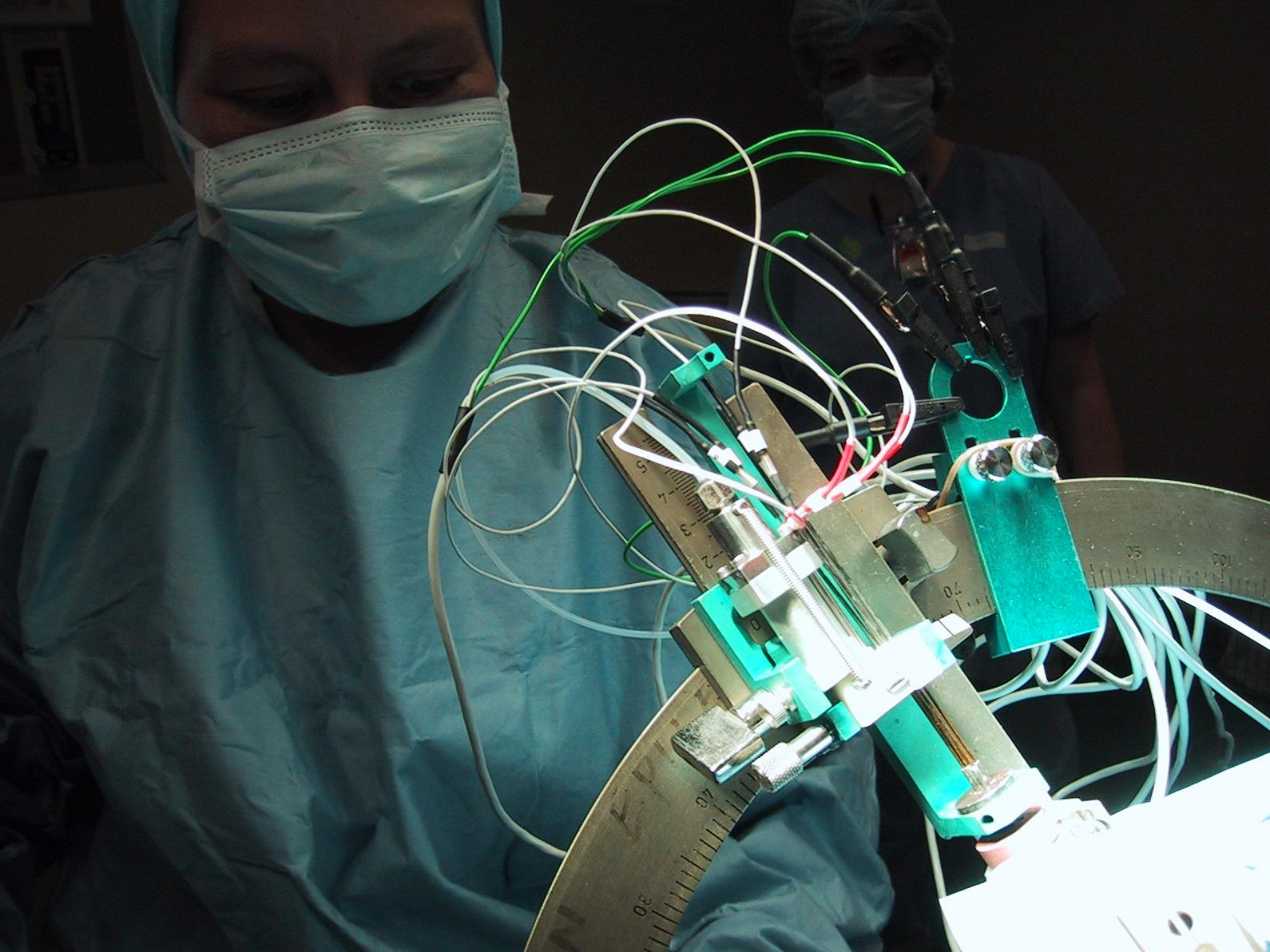
Objective: To determine the effects of high-frequency STN stimulation on the neuronal activity of STN neurons in Parkinson disease patients.

Patients: Single-unit recordings of the neuronal activity of the STN were obtained before, during, and after the application of intra-STN electrical stimulation in 15 Parkinson disease patients. Changes in firing frequency and pattern were analyzed using various combinations of stimulus frequency (range, 14-140 Hz).

Results: Stimulation at a frequency greater than 40 Hz applied within the STN significantly decreased the firing frequency and increased the burst-like activity in the firing pattern of STN neurons. An aftereffect was observed in cells that had been totally inhibited during high-frequency stimulation.

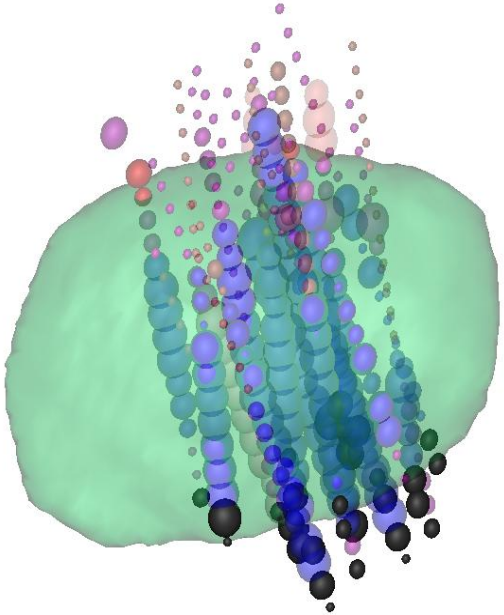
Conclusion: The beneficial effects of high-frequency stimulation result from a change in the firing pattern of cellular discharge and a blockade of the spontaneous overactivity of STN neurons.

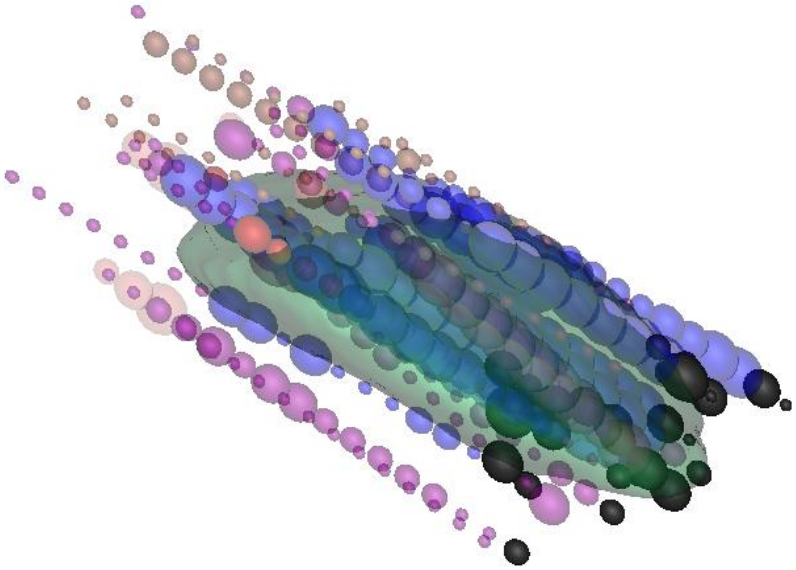
Arch Neurol. 2004;61:89-96

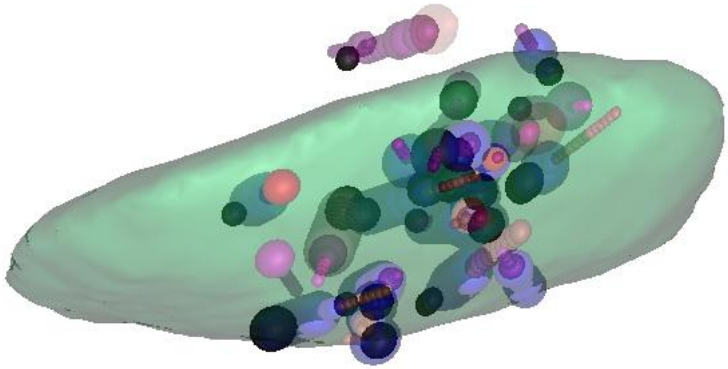



Ry u°g
2 = 121
= float

STN electrophysiology merged into 3D normalized Atlas





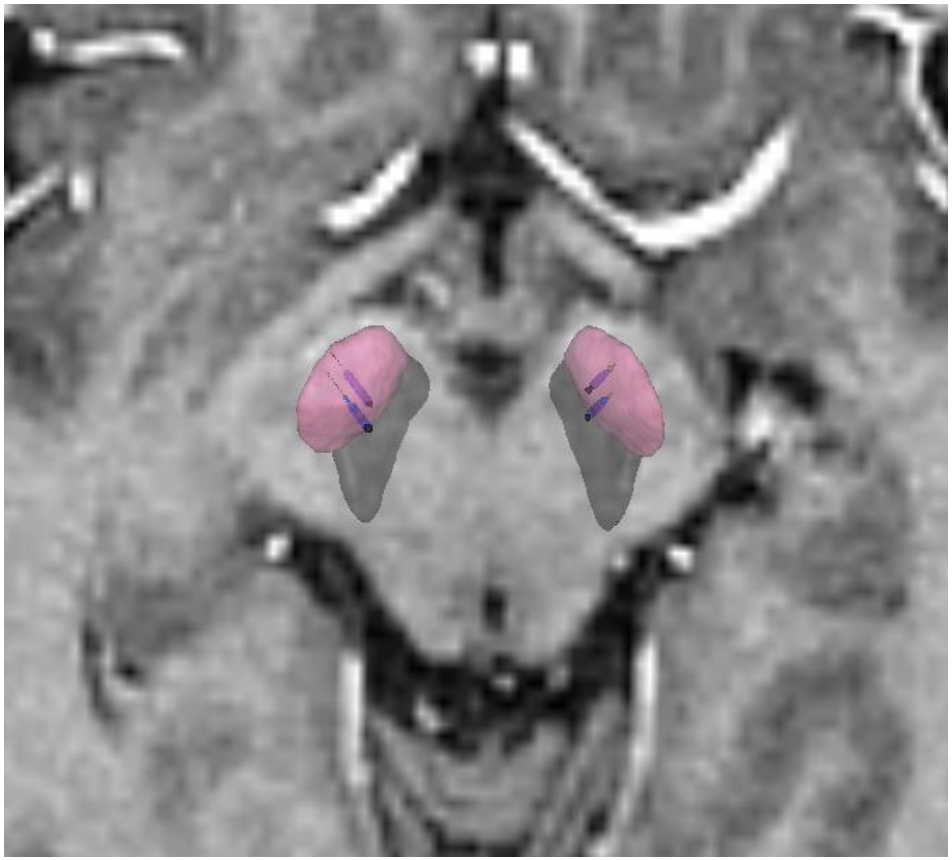


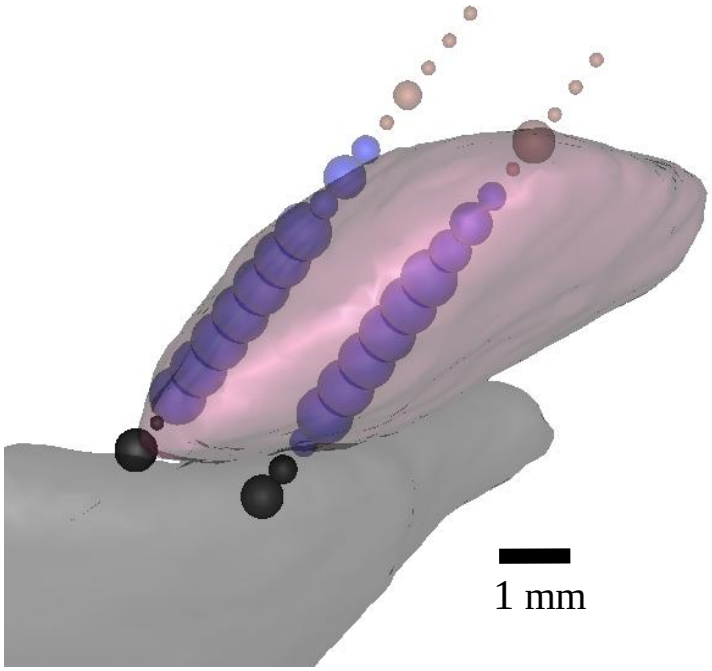
<u>12 patients</u> :

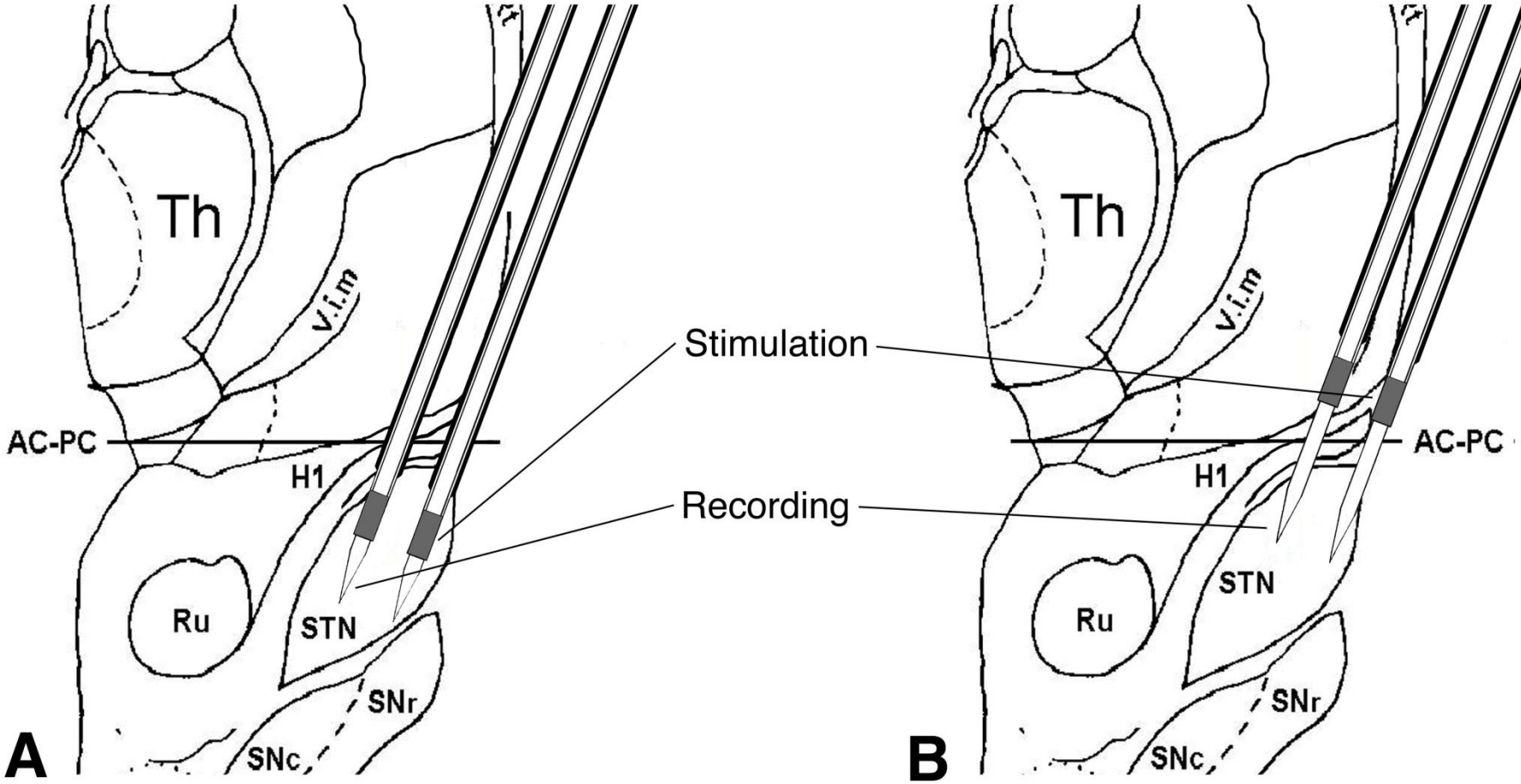
80 % of good match between electrophysiology and atlas

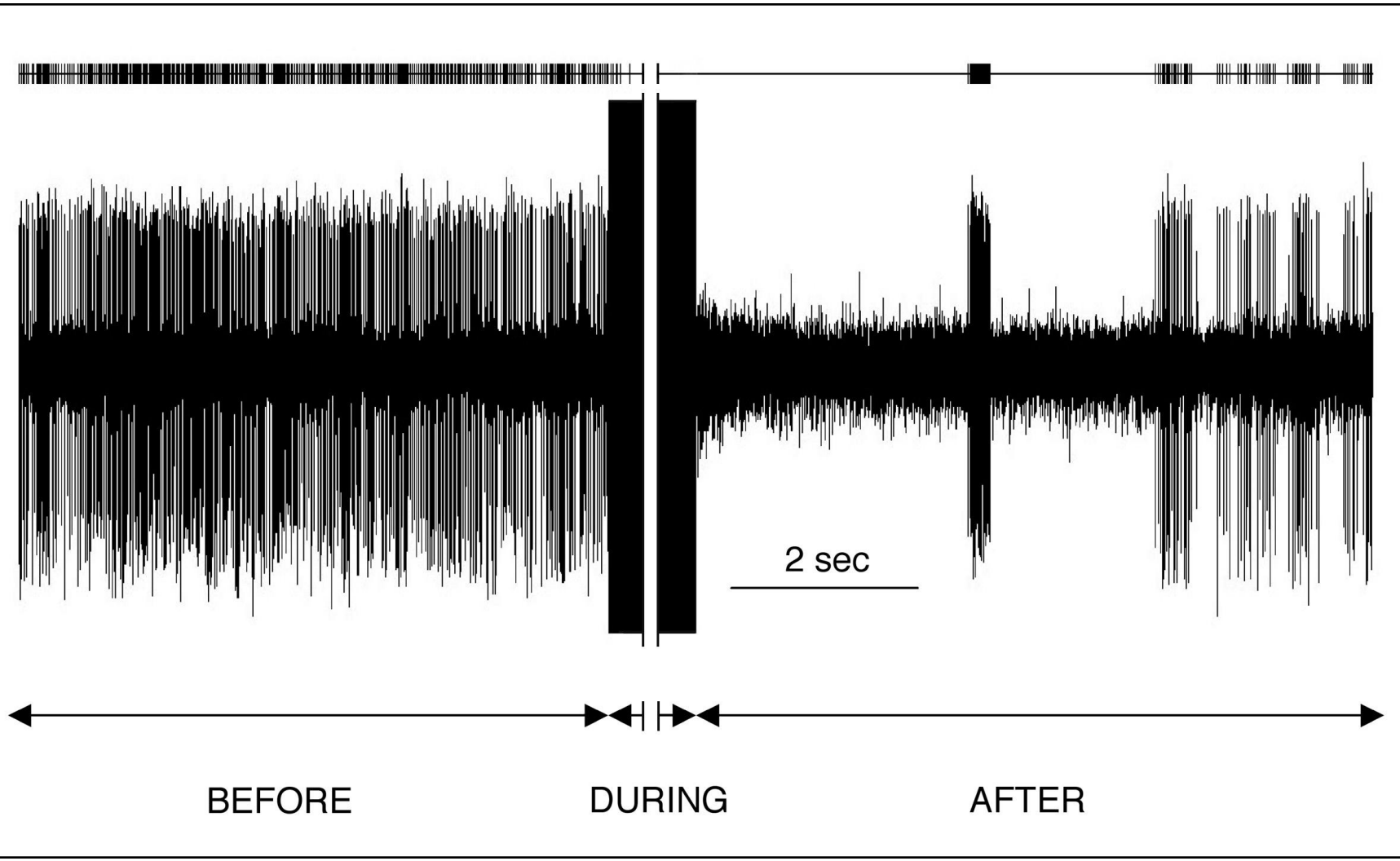
Mean errors = 1 mm

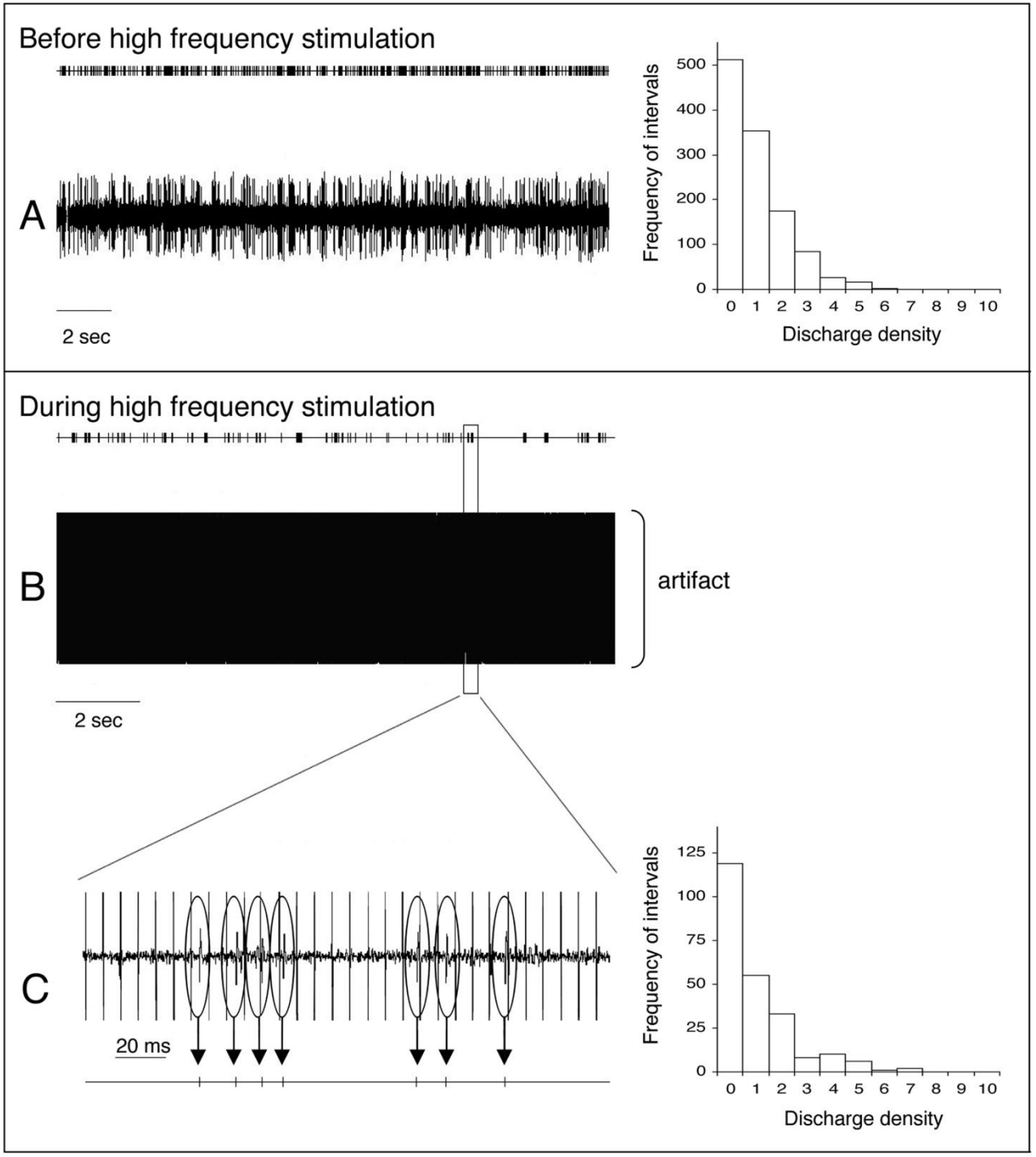
3D Atlas fusion with per op MER electrophysiology






Th
V.i.m
AC-PC
H1
Ru
STN
SNr
SNc
A
Stimulation
Recording
Th
V.i.m
AC-PC
H1
STN
Ru
SNr
SNc
B


2 sec
BEFORE
DURING
AFTER


Before high frequency stimulation
A
2 sec
Frequency of intervals
500
400
300
200
100
0
0 1 2 3 4 5 6 7 8 9 10
Discharge density
During high frequency stimulation
B
artifact
2 sec
C
20 ms
Frequency of intervals
125
100
75
50
25
0
0 1 2 3 4 5 6 7 8 9 10
Discharge density

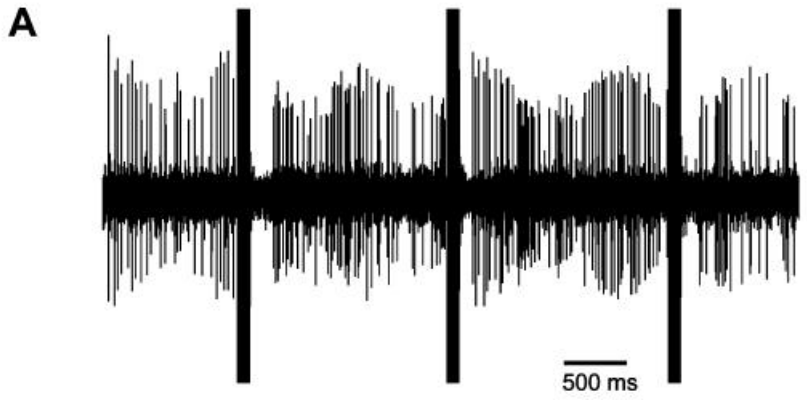
Mean Firing Rate of Subthalamic Nucleus (STN) Neurons Before, During, and After Different Conditions of Stimulation*

Frequency of Stimulation	Before Stimulation	During Stimulation	After Stimulation
140 Hz			
Intra-STN (n = 21)	38.1 ± 14.5	6.7 ± 7.1†	36.0 ± 15.9
Sham procedure (n = 8)	40.4 ± 11.8	35.6 ± 18.0	41.2 ± 14.4
80 Hz (n = 7)	40.9 ± 13.4	5.9 ± 5.5†	40.3 ± 9.9
40 Hz (n = 8)	41.9 ± 12.1	25.6 ± 8.4†	41.6 ± 13.1
14 Hz (n = 10)	29.2 ± 14.9	26.9 ± 14.6	30.1 ± 14.7

*Values are mean ± SD and expressed in Hertz.
†$P<.002$ compared with before stimulation.

STN HFS removes rigidity, akinesia and tremor

STN Inhibition by STN HFS trains



Filali M, Hutchison WD, Palter VN, Lozano AM, Dostrovsky JO

Stimulation-induced inhibition of neuronal firing in human subthalamic nucleus.

Exp Brain Res. 2004 Jun;156(3):274-81.

Effect of HFS of GPi on Thalamus activity

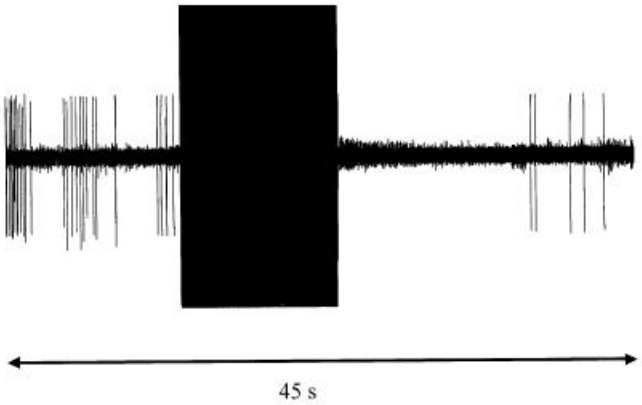


Fig. 2. Effect of ipsilateral GPI electrical stimulation on the spontaneous activity of a type A VOA neuron. Extracellular recording of a neuronal activity located 3 mm anterodorsal from the target in the VOA. Before GPI stimulation, mean firing rate was 17 Hz, DBS (130 Hz) of the GPI for 10 s reversibly inhibited this neuronal activity. Twenty seconds after the end of GPI stimulation, VOA neuronal activity recovered.

Pralong E, Debatisse D, Maeder M, Vingerhoets F, Ghika J, Villemure JG.

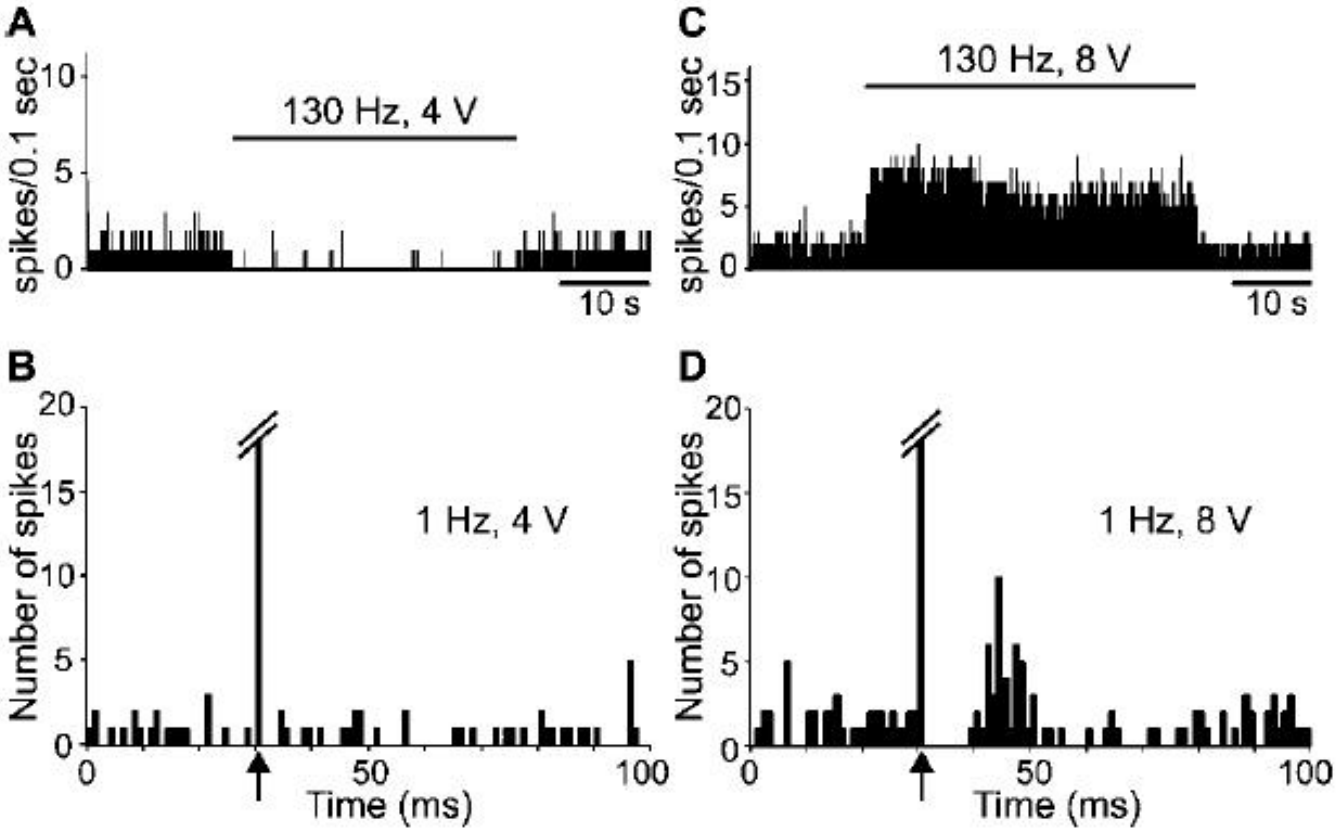
Effect of deep brain stimulation of GPI on neuronal activity of the thalamic nucleus ventralis oralis in a dystonic patient.
Neurophysiol Clin. 2003 Sep;33(4):169-73.

STN HF stimulation

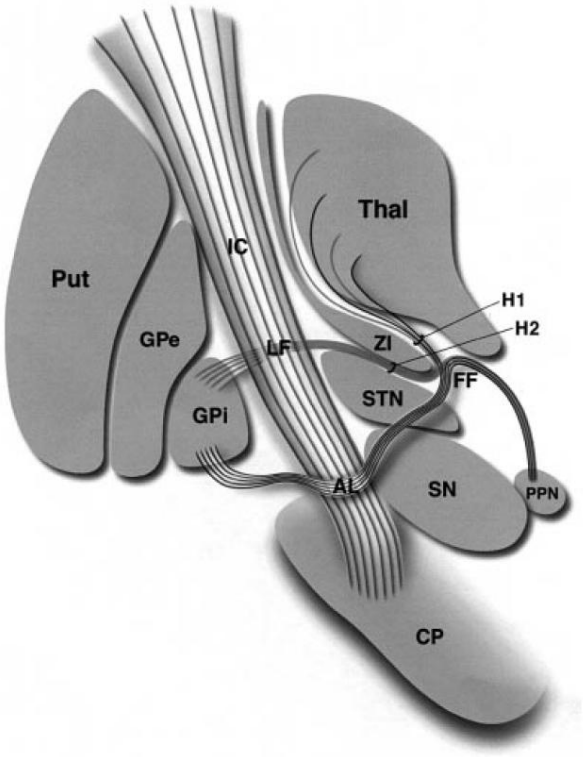
Inhibitory and excitatory effects on the same SNr cell

Maurice et al. • Effects of STN High-Frequency Stimulation on SNR Neurons

9932 • J. Neurosci., October 29, 2003 • 23(30):9929–9936



Basal Ganglia : a complex circuitry connecting many nuclei



Hamani C, Saint-Cyr JA, Fraser J, Kaplitt M, Lozano AM.

The subthalamic nucleus in the context of movement disorders.
Brain. 2004 Jan;127(Pt 1):4-20. Epub 2003 Nov 7. Review.

GPe, GPi, STN, Thalamus projections

*→ GABA

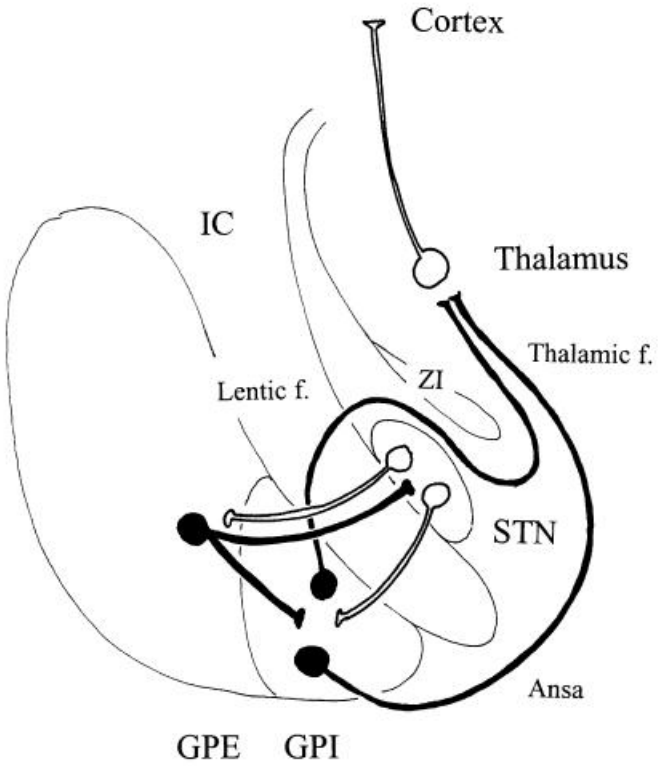


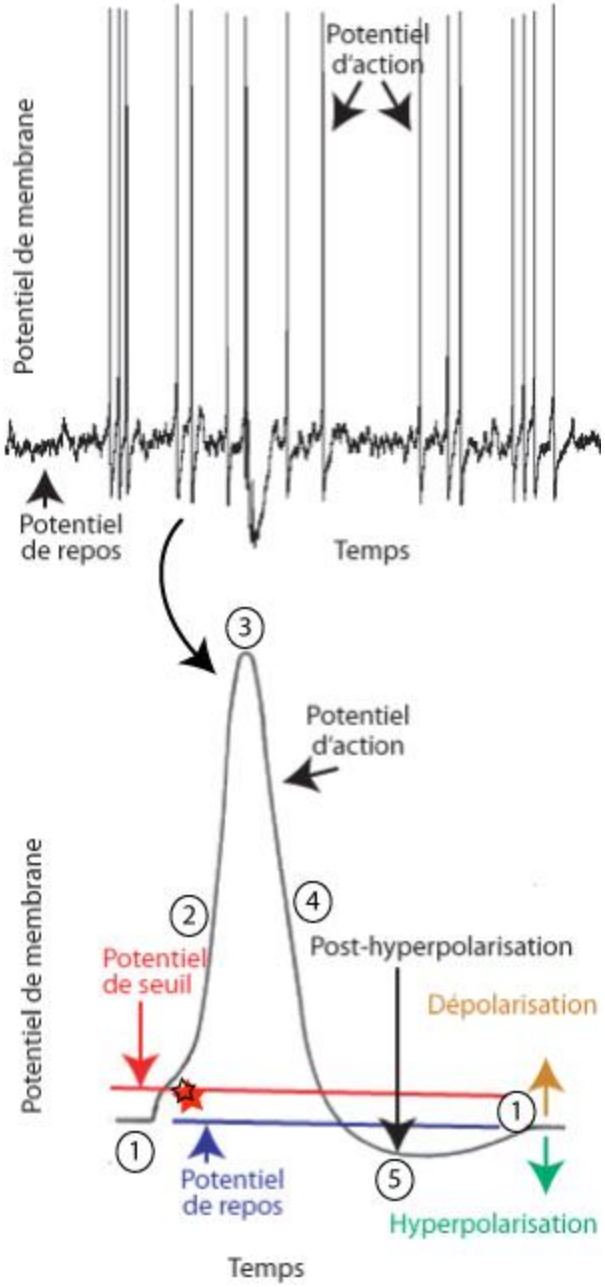
Fig. 11 Diagram of some of the pathways in the vicinity of the STN relevant to the effects of STN stimulation. Facilitatory neurons are white; inhibitory neurons are black. Ansa = lenticularis; f = fasciculus; other abbreviations are defined below the Summary.

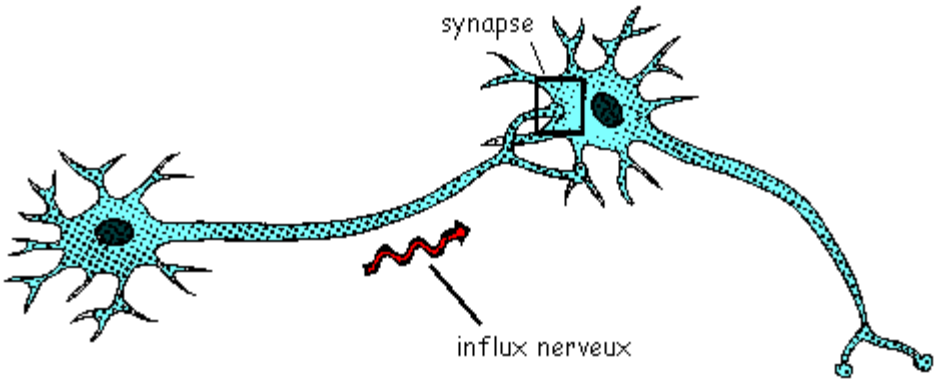
O→ GLU

Ashby P, Kim YJ, Kumar R, Lang AE, Lozano AM.

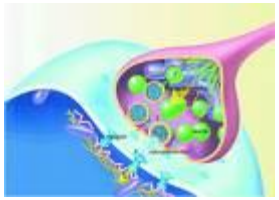
Neurophysiological effects of stimulation through electrodes in the human subthalamic nucleus. Brain. 1999 Oct;122 (Pt 10):1919-31.

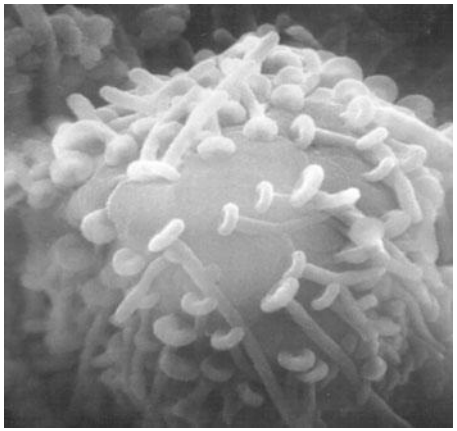
Orthodromic →



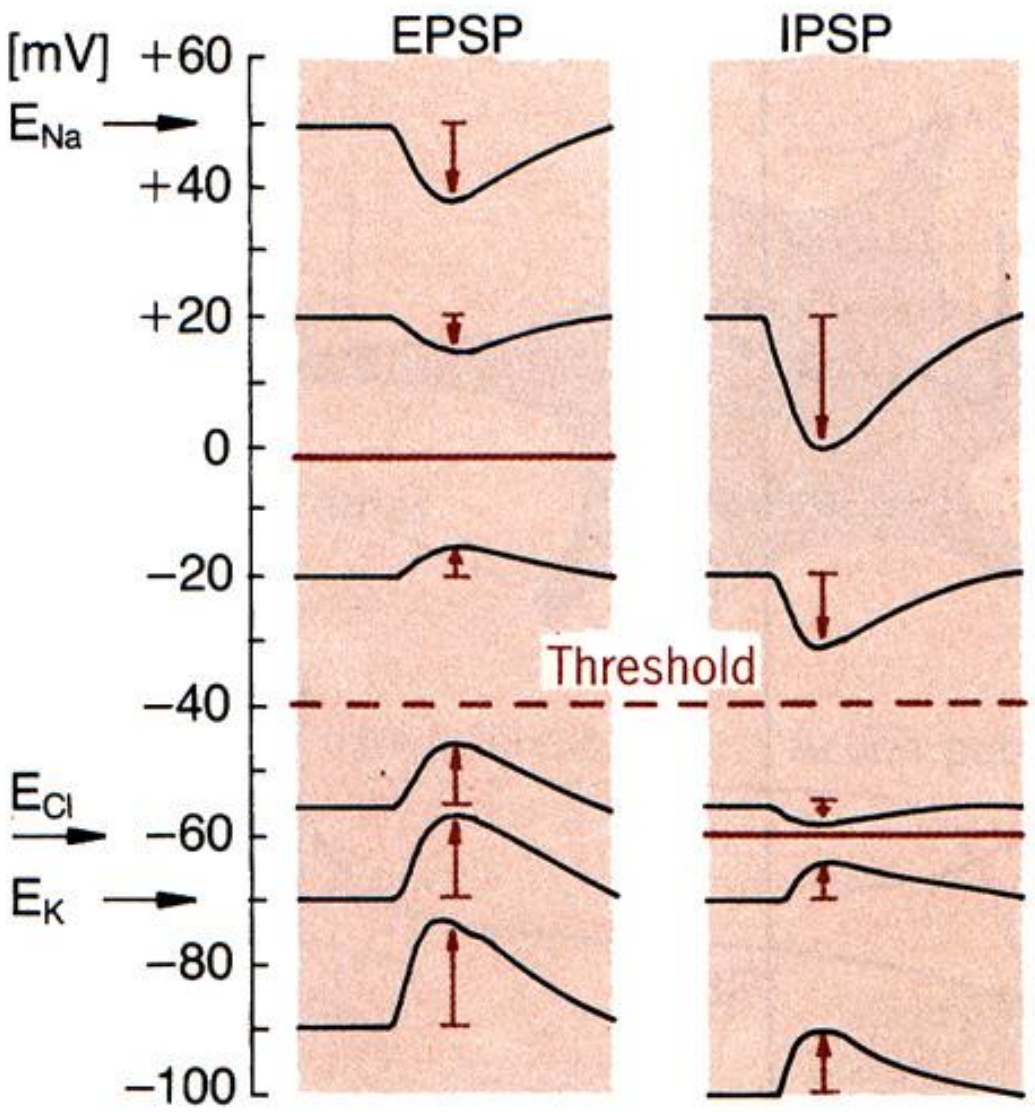


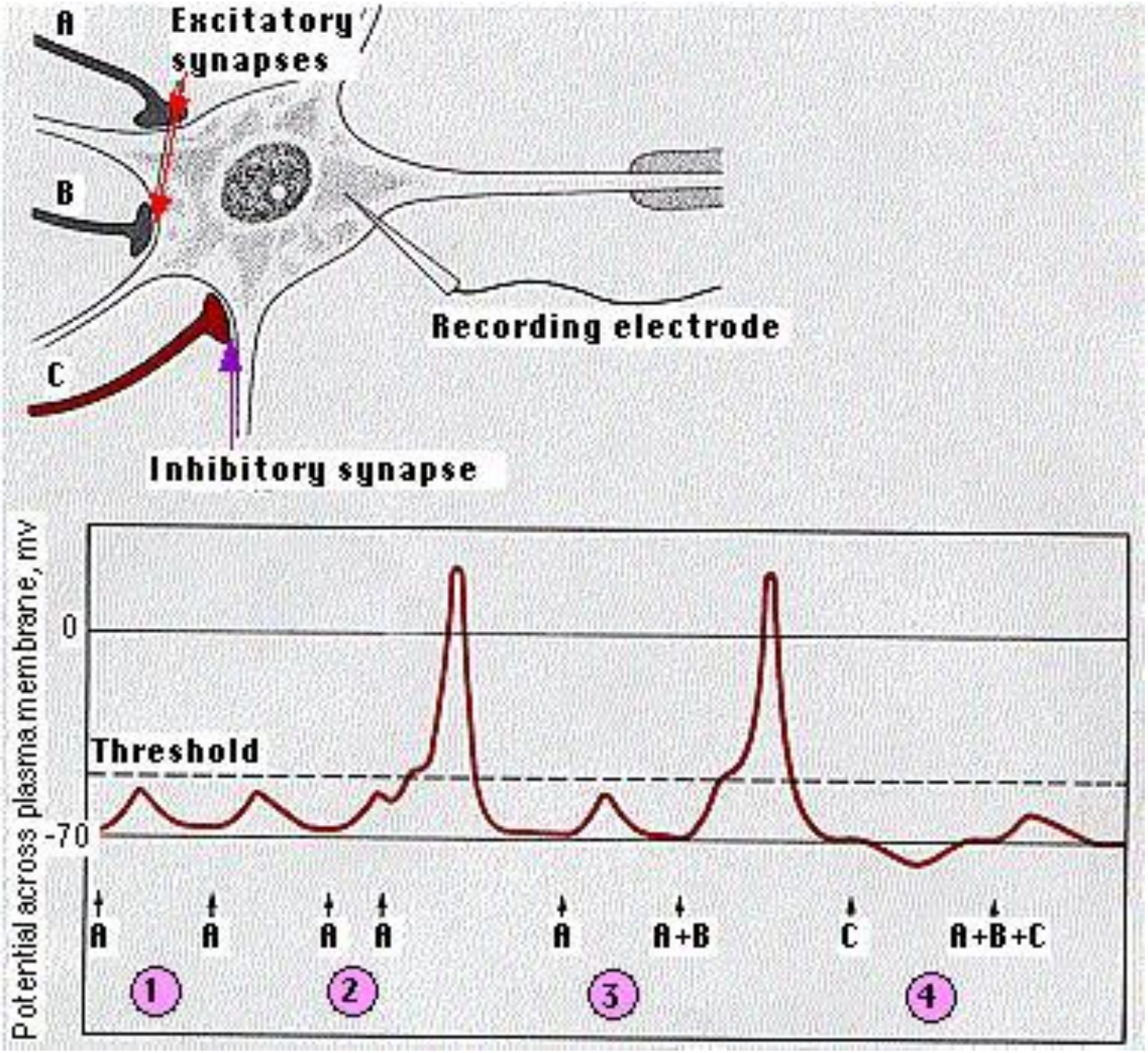
← Antidromic





GLU **GABA**

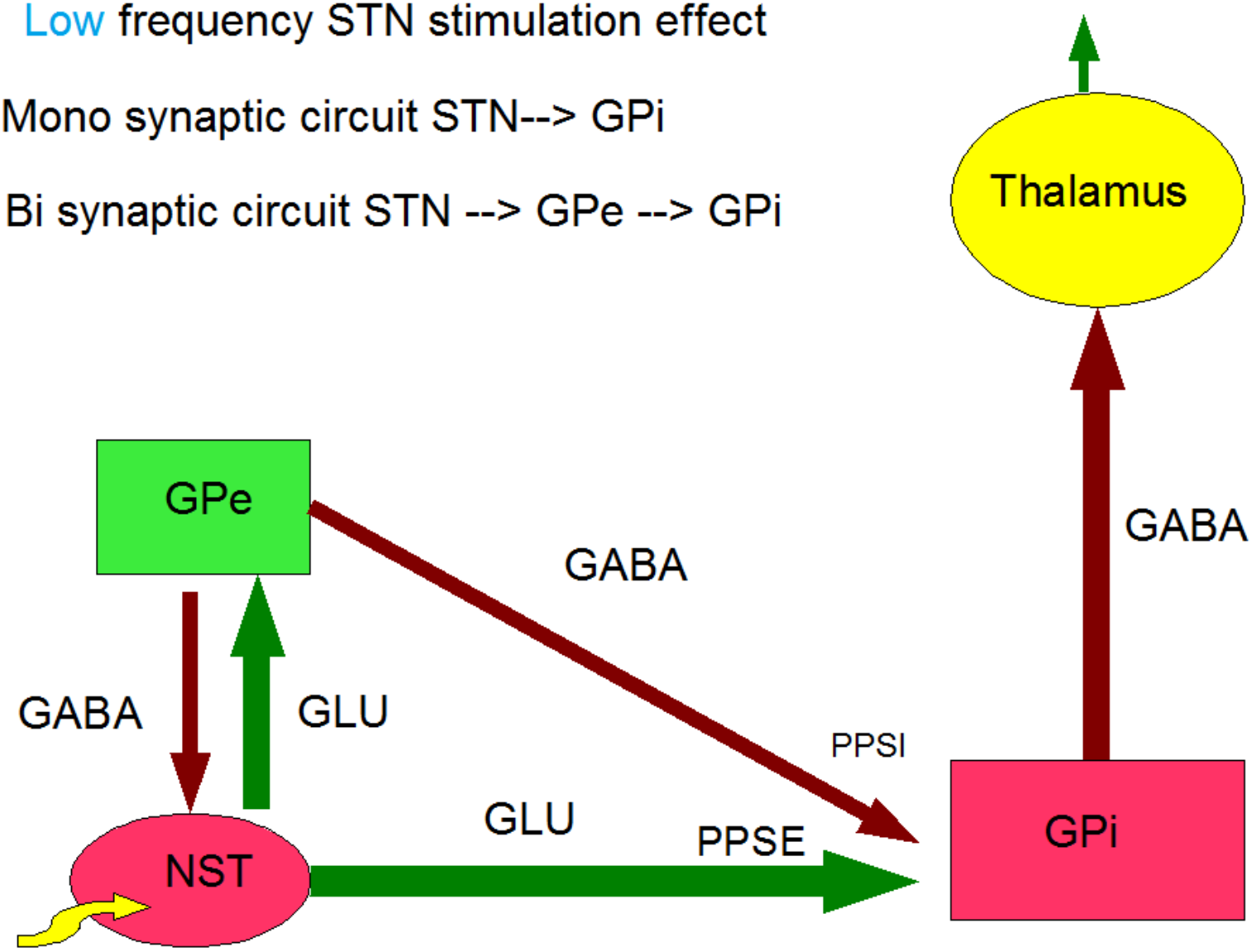



A
Excitatory synapses
B
C
Recording electrode
Inhibitory synapse
Potential across plasma membrane, mv
0
Threshold
-70
A
A
A
A
A
A+B
C
A+B+C
1
2
3
4

Low frequency STN stimulation effect

Mono synaptic circuit STN--> GPi

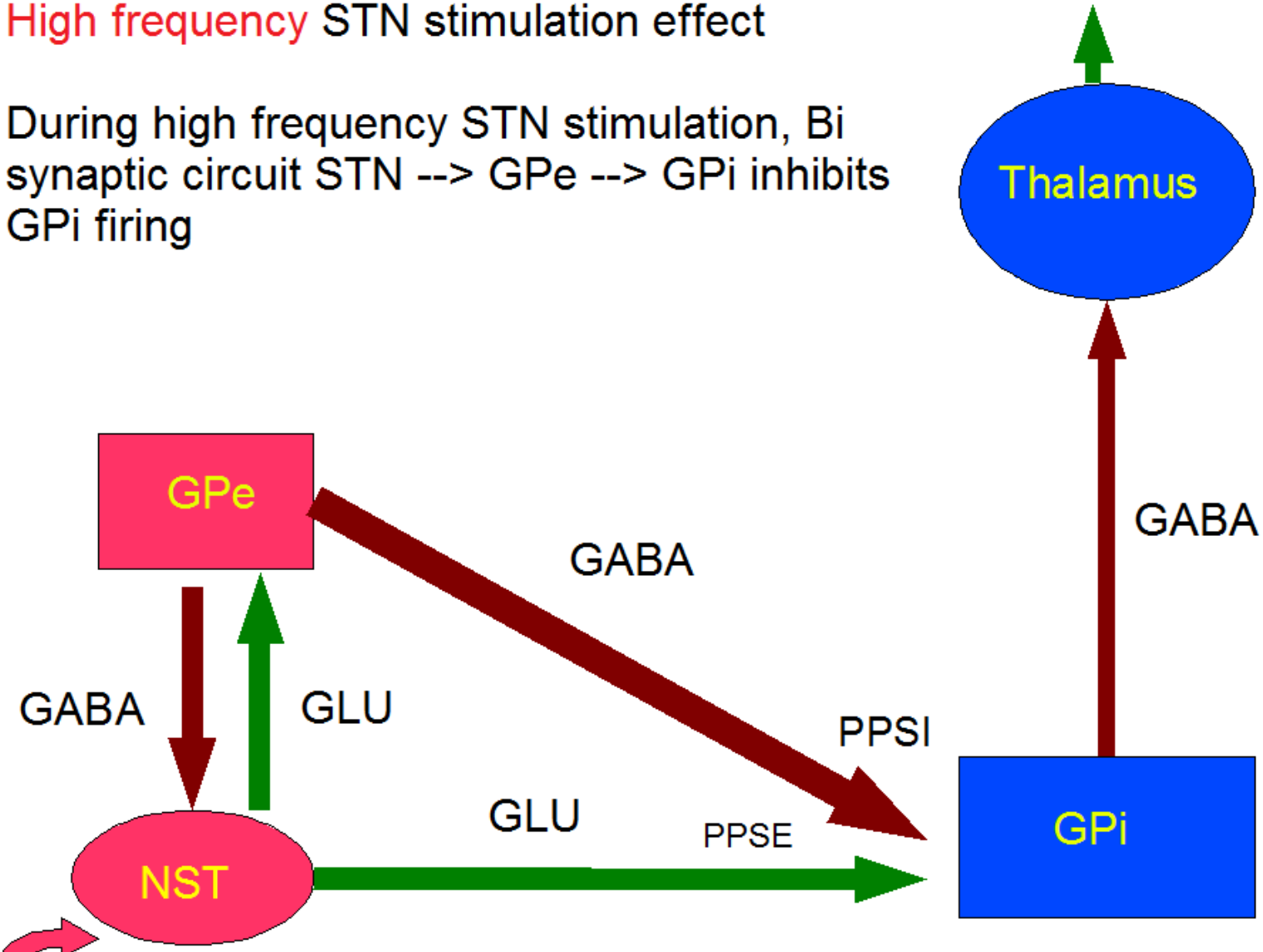
Bi synaptic circuit STN --> GPe --> GPi



Adapted from Hitoshi KITA et al. The Journal of Neuroscience, September 21, 2005 • 25(38):8611– 8619

High frequency STN stimulation effect

During high frequency STN stimulation, Bi synaptic circuit STN --> GPe --> GPi inhibits GPi firing



Adapted from Hitoshi KITA et al. The Journal of Neuroscience, September 21, 2005 • 25(38):8611– 8619

Conclusion

- Fundamental research on basal ganglia contributes to improve our knowledge on the physiopathology of a number of neuro-psychiatric diseases.
- Models of basal ganglia help understanding normal and abnormal organization of neuronal networks.
- However, neurophysiological and clinical exploration of basal ganglia during neurosurgical procedures show much more complexe features as will be illustrated by **following vidéos**.

Faculté de médecine
Pierre et Marie CURIE

Fédération de Neurophysiologie Clinique

Laboratoire de Physiologie

http://www.physio.chups.jussieu.fr

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Groupe Hospitalier Pitié-Salpêtrière