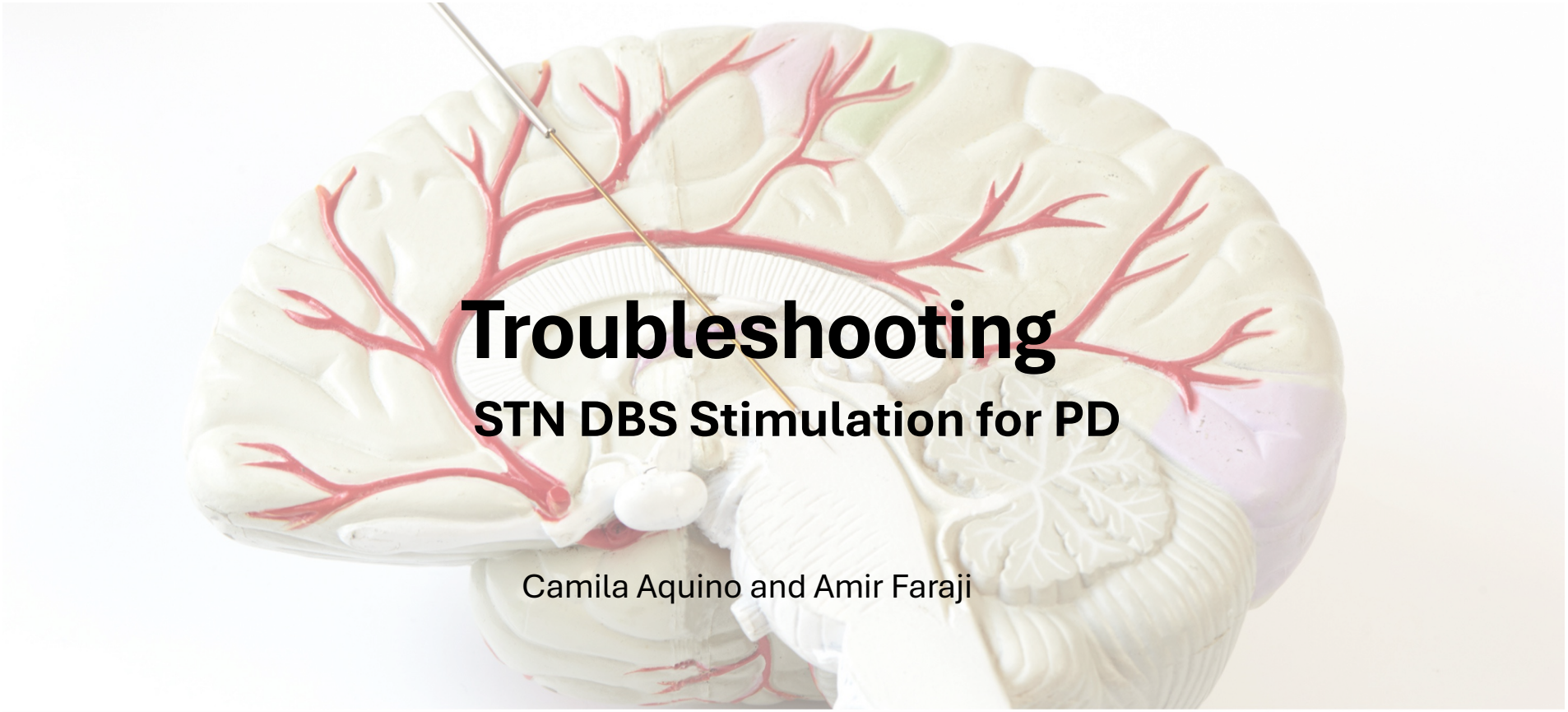


Troubleshooting

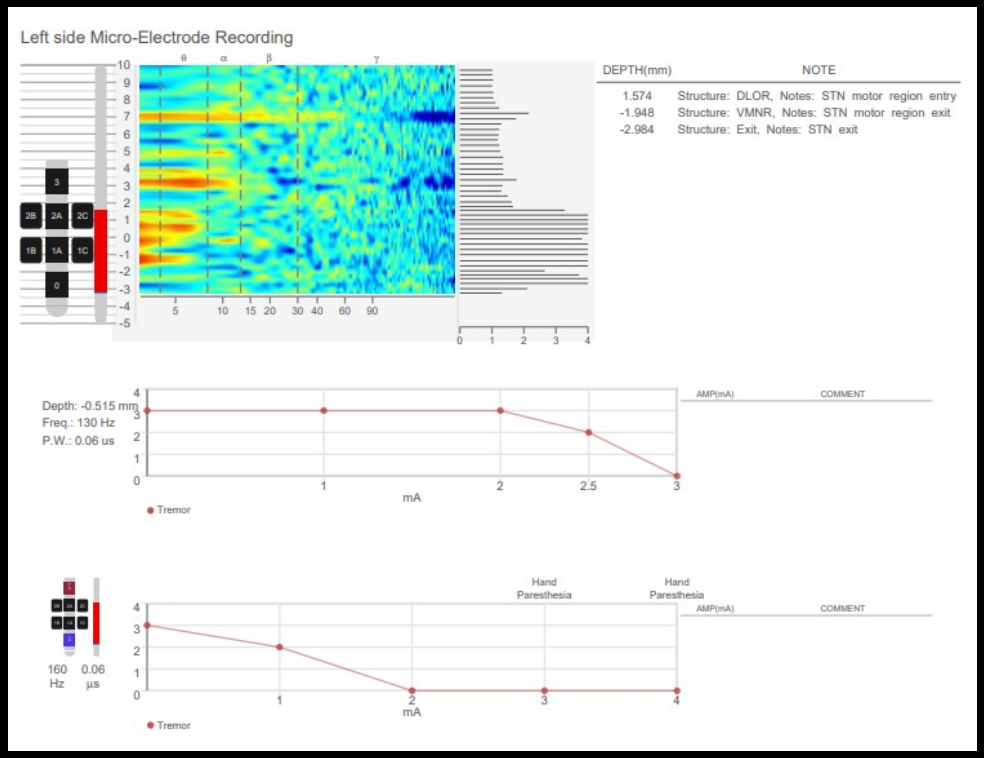
STN DBS Stimulation for PD

Camila Aquino and Amir Faraji

5th Neuromodulation School for Movement Disorders
New York, New York, USA | August 13-14, 2026

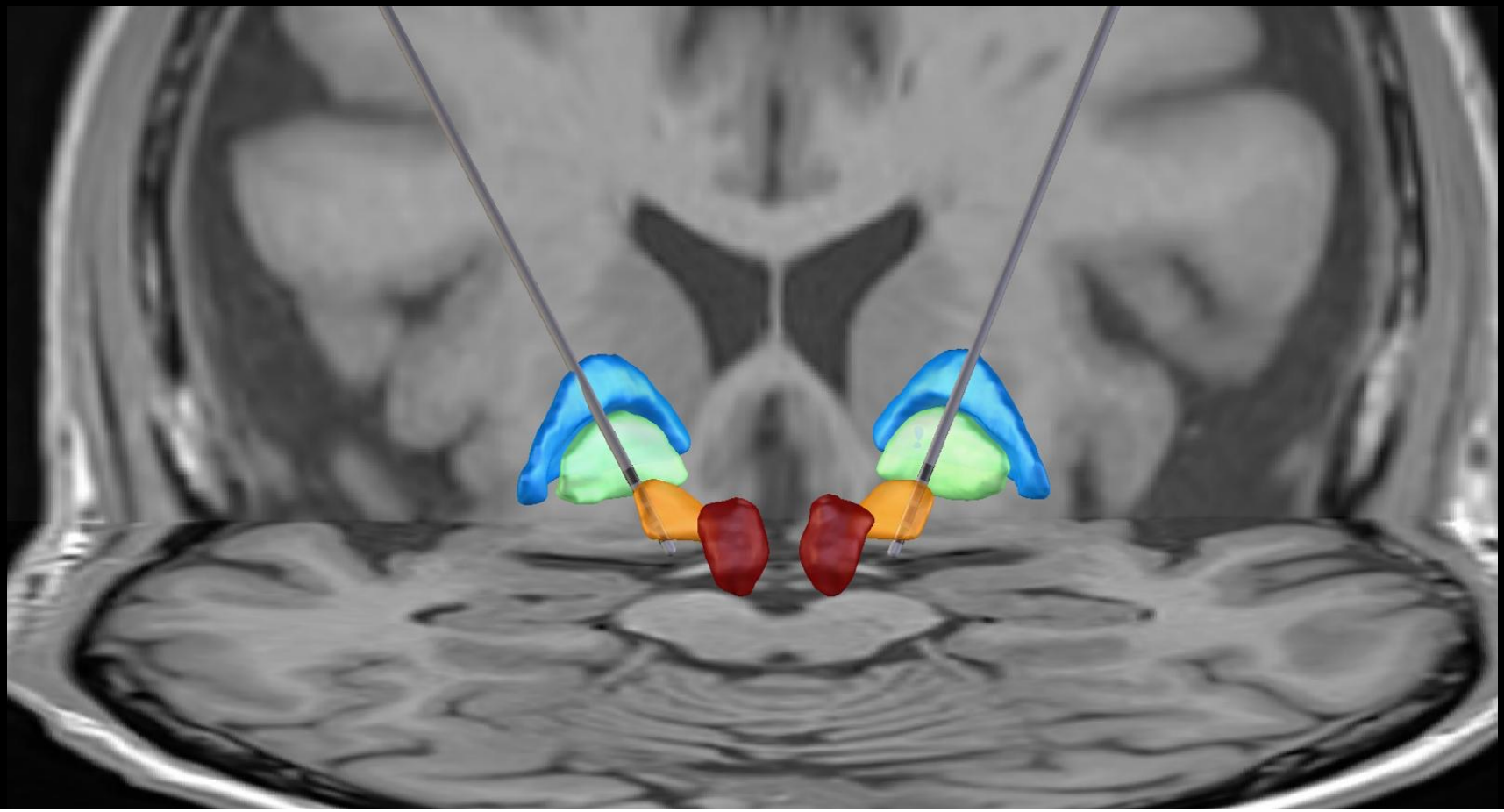
International Parkinson and Movement Disorder Society
Pan American Section

Customized Reports Aid DBS Programming

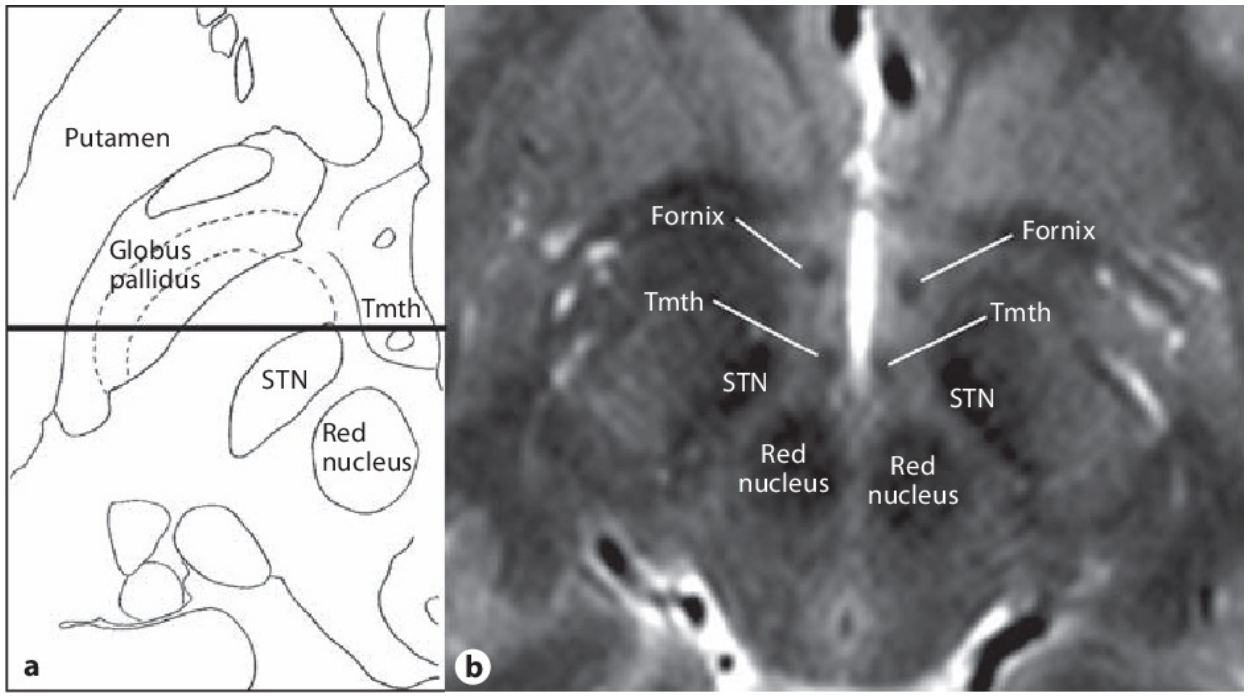


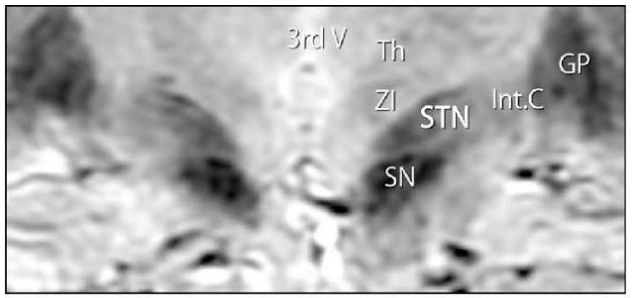
HOUSTON Methodist NEUROLOGICAL INSTITUTE

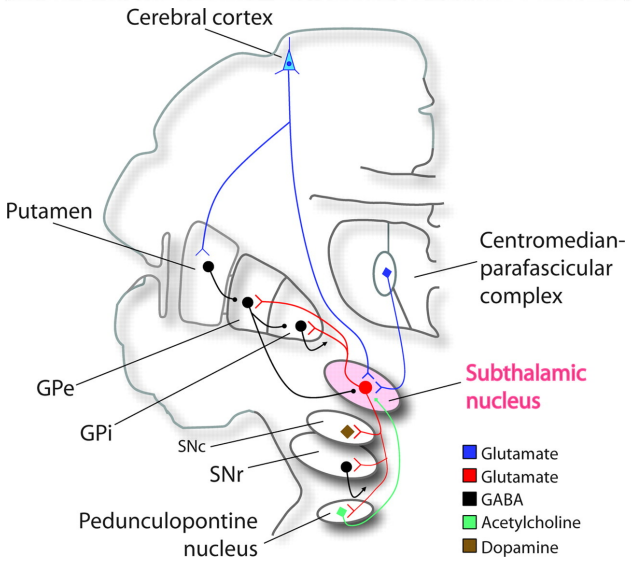
Advanced Imaging to Verify Electrode Placement



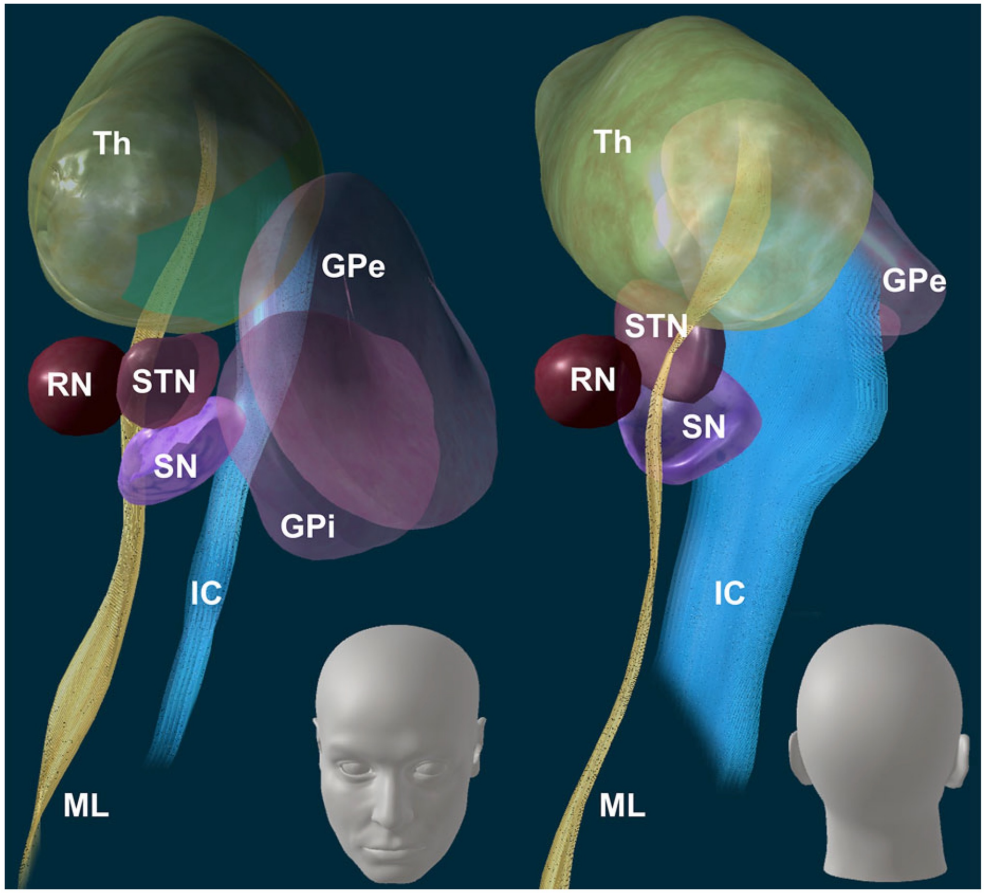
Anatomy Around the Subthalamic Nucleus







Relationship of DBS Responses to STN Anatomy



- **Lateral (or Anterior) to STN -** Internal Capsule (**IC**): **contralateral muscular contraction (facial pulling, eye deviation, etc.)**
- **Medial and Posterior to STN –** Cranial III Nerve: Diploipa, **Ispilateral** Eye Deviation
- **Posterior to STN –**Medial Lemniscus (**ML**): **Paresthesias/Tingling**
- **Medial to STN –** Red Nucleus (**RN**): **Dizziness, Nausea, Sweating**

Relationship of DBS Responses to STN Anatomy

CLINICAL OBSERVATION	STIMULATED ANATOMY	RELATIVE TO OPTIMAL LOCATION IN THE STN
PD symptom relief (tremor, bradykinesia, rigidity)	Dorsolateral STN (Motor Aspect)	Optimal location
Diplopia, Ipsilateral eye deviation, dizziness, nausea, sweating, depression	Cranial Nerve III, Red Nucleus, Limbic Aspect	Medial
Facial pulling, **limb contraction contralaterally**, **contralateral gaze deviation**	Corticospinal tract of the **Internal Capsule**, frontal eye field fibers of the internal capsule	Lateral or Anterior
Paresthesia (tingling, electrical sensations, numbness)	**Medial Lemniscus**	Posterior
Possible improvement in Dyskinesia	Zona Incerta	Superior

Montgomery EB Jr. Intraoperative Neurophysiological Monitoring for Deep Brain Stimulation: Principles, Practice, and Cases. Oxford University Press; 2014
Montgomery EB Jr. Deep Brain Stimulation Programming: Mechanisms, Principles, and Practice. New York, NY: Oxford University Press; 2017
Tarsey D et al., Deep Brain Stimulation in Neurological and Psychiatric Disorders. Totowa, NJ: Humana Press; 2008
Krack P et al., Postoperative management of subthalamic nucleus stimulation. Movement Disord. 2002;17:S3,S188-S197.
Greenberg BD et al., Deep brain stimulation of the ventral internal capsule/ventral striatum for obsessive-compulsive disorder: worldwide experience. Mol Psychiatr. 2010;15(1):64-79
Krause M et al., Deep brain stimulation for the treatment of Parkinson's disease: subthalamic nucleus versus globus pallidus internus. Journal of Neurology, Neurosurgery & Psychiatry. 2001;70(4):464-470
Blomstedt P et al., Deep brain stimulation of the subthalamic nucleus versus the zona incerta in the treatment of essential tremor. Acta neurochirurgica. 2011;153(12):2329-2335.

A General Framework for Troubleshooting

No formal guideline exists, but a consistent, stepwise approach reduces trial-and-error

1 Localize

Is it on-stim / off-stim?
On-med / off-med?
Timing separates stimulation effects from medication and disease-progression effects.

2 Check hardware

New, delayed, or asymmetric symptoms warrant an impedance check ± imaging

3 Map systematically

Full monopolar / directional review:
Define the therapeutic window and side-effect threshold at every contact.

A Reshape

B Redirect

C Reduce

Why Side Effects Happen Where They Do

Current spread beyond the sensorimotor STN into neighboring structures produces a predictable topography of side effects

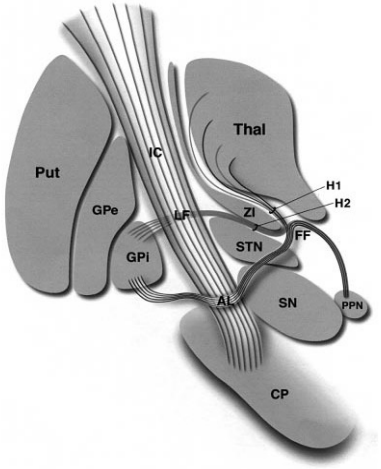
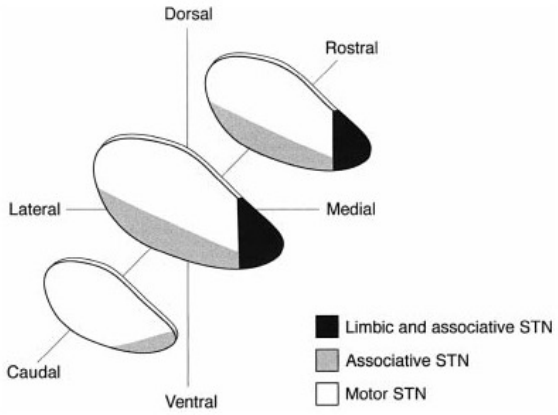
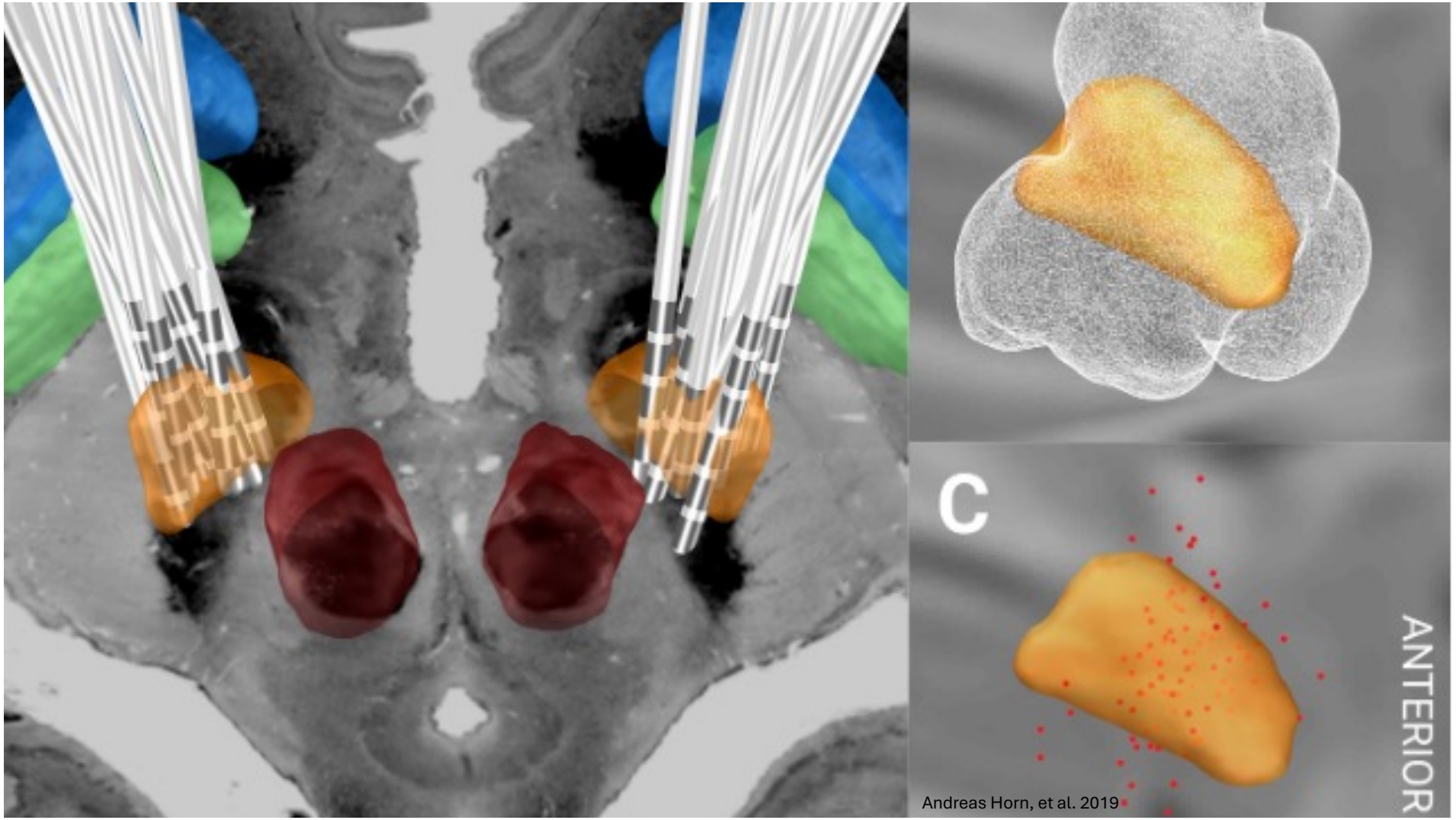


Fig. 1 Representation of the major anatomical structures and fibre tracts associated with the subthalamic nucleus. AL = ansa lenticularis; CP = cerebral peduncle; FF = Fields of Forel; GPe = globus pallidus externus; GPi = globus pallidus internus; H1 = H1 Field of Forel (thalamic fasciculus); IC = internal capsule; LF = lenticular fasciculus (H2); PPN = pedunculopontine nucleus; Put = putamen; SN = substantia nigra; STN = subthalamic nucleus; Thal = thalamus; ZI = zona incerta.



Hamani C, et al. *Brain*. 2004;127(Pt 1):4-20.



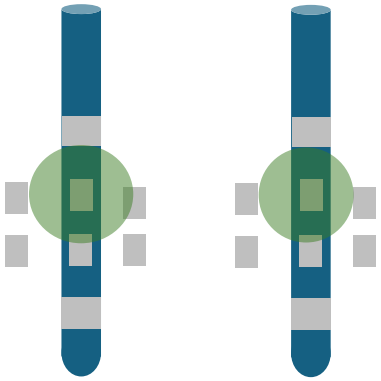
Andreas Horn, et al. 2019

Stimulation-Induced Dysarthria

62-year-old male. Bilateral STN DBS for PD.

Dysarthria noted when stimulation ON.
No improvement with or without medication.

Entry stimulation parameters:
Left STN: C+ 2- ; 3.2 mA; 80 µs; 130 Hz
Right STN: C+ 10- ; 2.9 mA; 80 µs; 130 Hz



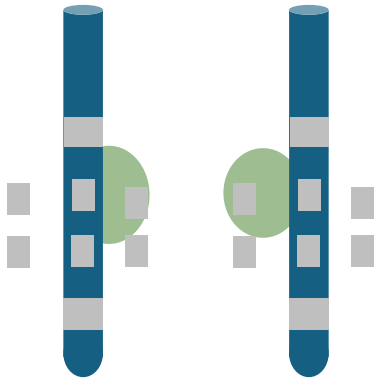
Approach:

1. Test each hemisphere's contribution independently - unilateral vs. bilateral effect. Start by turning off or reducing the stimulation on dominant hemisphere.
2. Lower PW (by 10 µs at a time, each side individually) and reassess if benefit still present and if speech improves.
3. Repeat monopolar review of full ring and segments. Identify the contact/segment with largest therapeutic window.
4. Steer current away from the internal capsule – medially or using the contact with largest therapeutic window.
5. If persistent, consider bipolar settings; longitudinal steering with MICC, interleaved.
6. Try low frequency stimulation.
7. Referral for SLP.

Stimulation-Induced Dysarthria

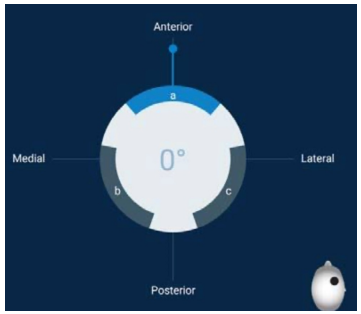
Final stimulation parameters:

Left STN: C+ 2ab- ; 3.2 mA; 60 µs; 130 Hz
Right STN: C+ 10c- ; 2.9 mA; 70 µs; 130 Hz



Approach:

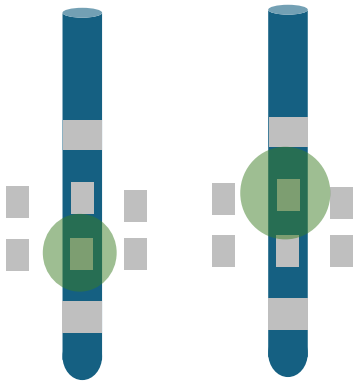
1. Lower PW (Repeat monopolar review of full ring and segments.
   Identify the contact/segment with largest therapeutic window.
2. Directional stimulation – medially



Tonic contractions / muscle pulling

49 year-old female. Bilateral STN DBS for PD. During monopolar review, patient experiences low threshold for stimulation-induced muscle contractions on the right body.

Entry stimulation parameters:
C+ 1- ; 1.5 mA; 60 µs; 130 Hz
C+ 10- ; 2.3 mA; 60 µs; 130 Hz



Approach:

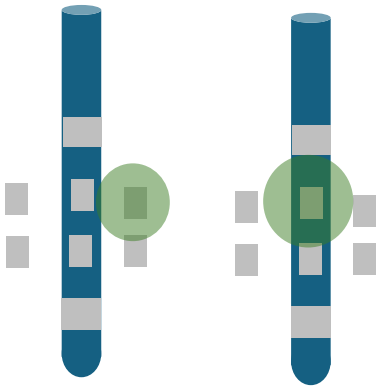
1. Consider imaging reconstruction to confirm verify lead location.
2. Repeat monopolar testing on the affected side with lower PW (40 µs).
3. Review contacts and segments. Identify the contact / segment with largest therapeutic window.
4. Try adjacent contact or contact with largest therapeutic window
5. Steer current medially
6. Use lowest effective stimulation amplitude (compensate with medication)
7. Consider bipolar settings, longitudinal steering with MICC, interleaved.
8. Neurosurgery consultation for lead revision.

Tonic contractions / muscle pulling

Final stimulation parameters:

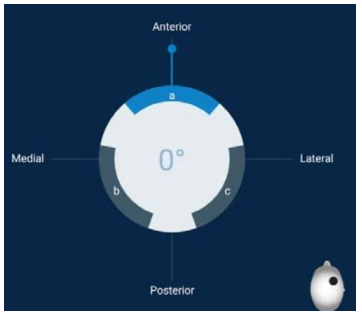
C+ 2b- ; 2.5 mA; 40 µs; 125 Hz

C+ 10- ; 2.3 mA; 60 µs; 125 Hz



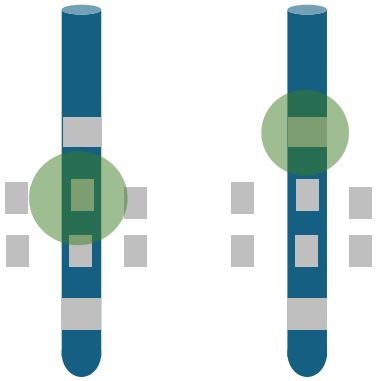
Approach:

1. Lower PW (40 µs).
2. Try adjacent contact or contact with largest therapeutic window
3. Steer current medially
4. Use lowest effective stimulation amplitude (compensate with medication)



Persistent Paresthesia

55-year-old female.
Bilateral STN DBS for tremor predominant PD.
During monopolar review experiences persistent tingling on the left body side with contact 8, 9, 10 before meaningful benefit. Contact 11 is only partially effective for tremor.



<u>Entry stimulation parameters:</u>
C+ 2- ; 2.8 mA; 60 µs; 130 Hz
C+ 11- ; 2.3 mA; 60 µs; 130 Hz

Approach:

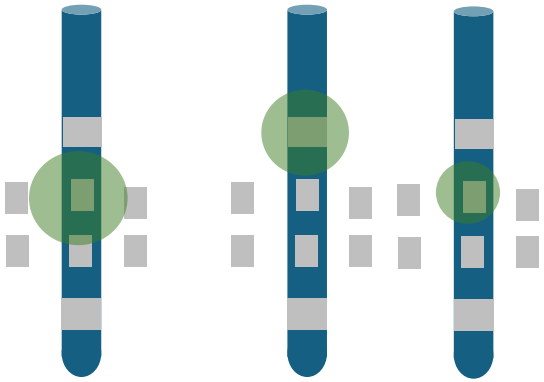
1. Consider imaging reconstruction to confirm verify lead location.
2. Repeat monopolar testing on the affected side with lower PW.
3. Review contacts and segments. Identify the contact / segment with largest therapeutic window.
4. Steer current more anteriorly / laterally
5. Consider bipolar settings, longitudinal steering with MICC, interleaved.
6. Use lowest possible stimulation amplitude (may required higher medication doses)
7. Neurosurgery consultation for lead revision

Persistent Paresthesia

.

Final stimulation parameters:

C+ 2- ; 2.8 mA; 60 µs; 125 Hz
C+ 11- ; 2.1 mA; 60 µs; 125 Hz
C+ 10a- ; 1.0 mA; 60 µs; 125 Hz



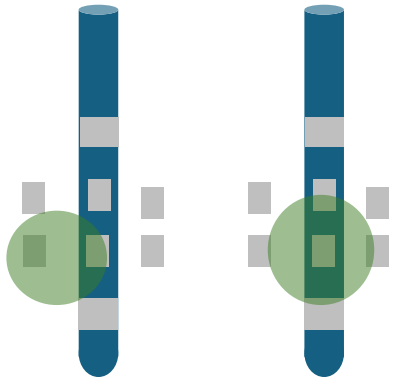
Approach:

1. Try adjacent contact or contact with largest therapeutic window
2. Steer current more anteriorly / laterally
3. Use lowest possible stimulation amplitude in each contact
4. Longitudinal steering

Hypomania

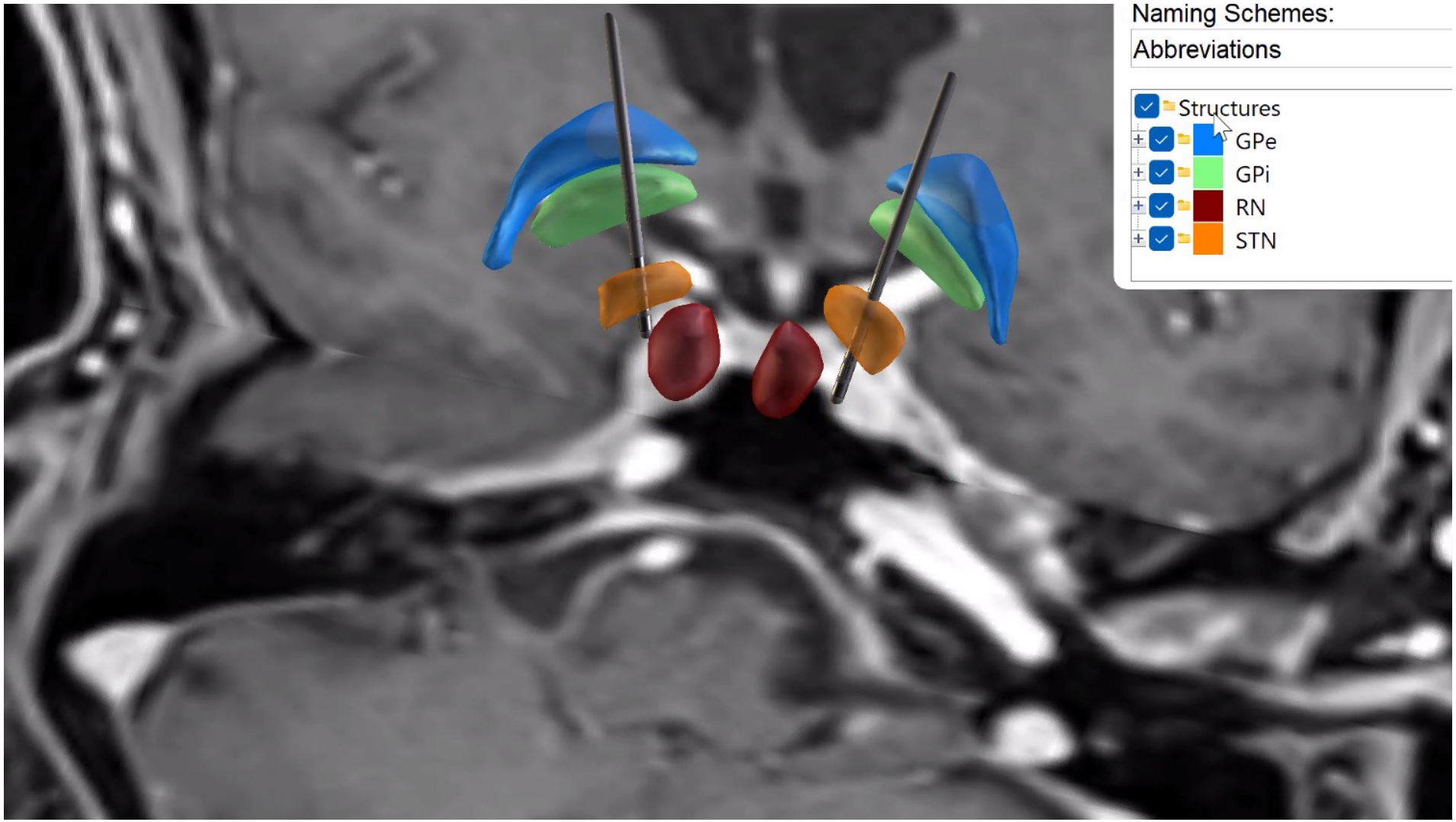
56-year-old male.
Bilateral STN DBS for tremor predominant PD.
Since initial programming, patient excessively shopping on Amazon, irritable, and arguing with neighbors over their kids throwing things in his backyard.

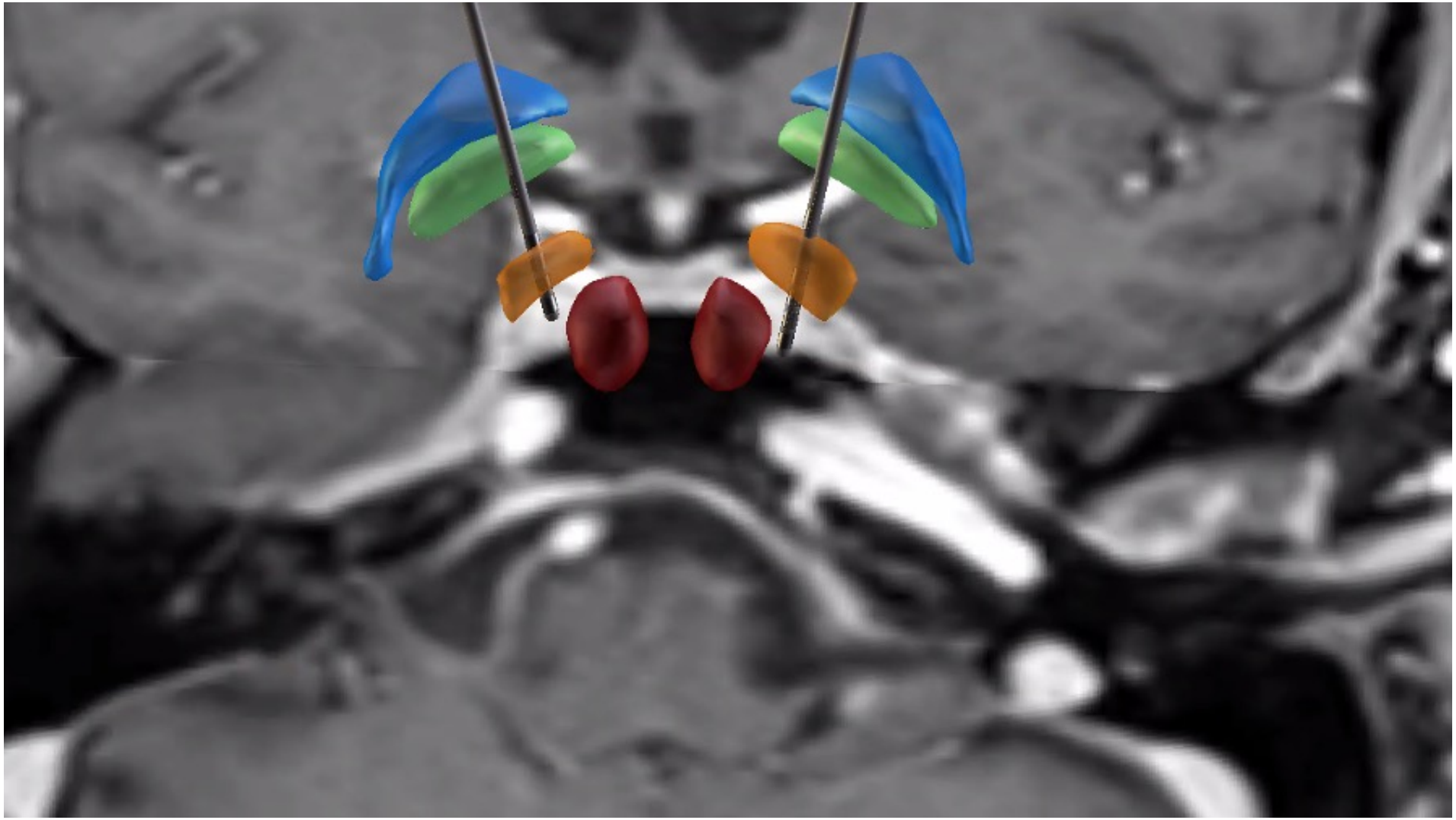
Entry stimulation parameters:
C+ 1ac- ; 2.4 mA; 60 µs; 130 Hz
C+ 9- ; 2.6 mA; 60 µs; 130 Hz



Approach:

1. Test each contact/amplitude systematically and document mood and behavior in real time (often contact-specific)
2. Move stimulation to the next dorsal contact, and further if persists.
3. Reduce stimulation amplitude
4. Steer current laterally, away from limbic / ventromedial STN
5. Reduce dopaminergic agents that could be contributing to behavior
6. Reconstruct imaging to assess lead location
7. Involve psychiatry for management of risky behaviors
8. Neurosurgery consultation for lead revision

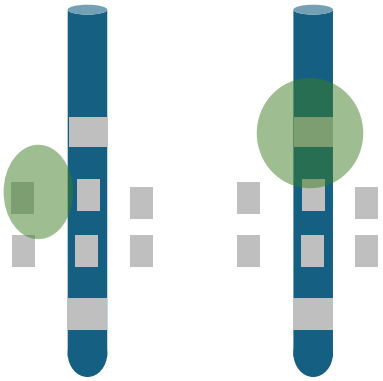

Naming Schemes:
Abbreviations
Structures
GPe
GPi
RN
STN



Hypomania

Stimulation parameters tried before lead revision:

C+ 2c- ; 2.5 mA; 60 µs; 125 Hz
C+ 11- ; 3.0 mA; 60 µs; 125 Hz



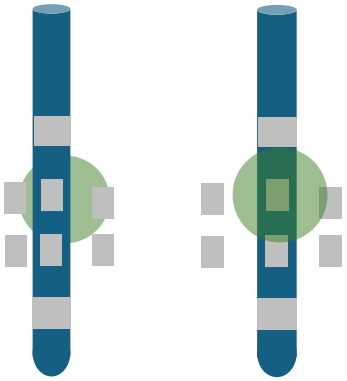
Approach:

1. Moved stimulation to the next dorsal contact and further with improvement of mood but **loss of motor control.**
2. Steered current away from medial STN without benefit
3. Reduced dopaminergic agents – no behavioral improvement and worse tremor.
4. Reconstructed imaging to assess lead location
5. Involved psychiatry for management of risky behaviors and frustration
6. **Neurosurgery revised both leads and patient is successfully programmed.**

Gait dysfunction (FOG and falls)

70-year-old patient. PD for 14 years, PIGD phenotype.
Prior to surgery, FOG mostly during off med.
Since STN DBS, unpredictable FOG and falls.

Entry stimulation parameters:
C+ 2bc-; 2.1 mA; 60 µs; 130 Hz
C+ 10- ; 2.6 mA; 60 µs; 130 Hz



Approach:

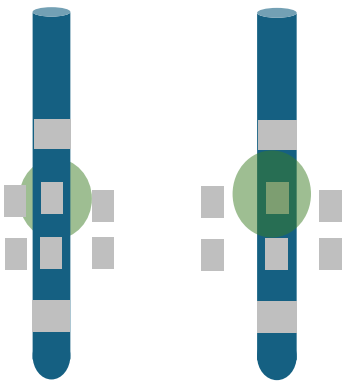
1. Define if gait problems are worse during ON med, OFF med, Stim ON, Stim OFF.
2. Test gait acutely **with increased stimulation** and reduced stimulation. Observe each leg during walking, turning, doorways. Test each hemisphere's contribution independently.
3. If stimulation induced, reduce amplitude on the side less affected by PD.
4. Consider using ventral contacts closer to SNr – longitudinal steering or interleaved.
5. Explore reduced PW.
6. Test adaptive settings (aDBS)
7. Try low frequency stimulation (<100 Hz) with compensatory amplitude increase
8. Medication increase – L-Dopa; amantadine. Exploratory options.
9. Referral for physical therapy and occupational therapy.

Gait dysfunction (FOG and falls)

Final stimulation parameters:

C+ 2bc; 2.6 mA; 60 µs; 90 Hz

C+ 10- ; 2.9 mA; 60 µs; 90 Hz



Approach:

1. Low frequency stimulation (<100 Hz) with compensatory amplitude increase to keep total energy delivered.
2. Amantadine started BID.
3. Referral for physical therapy and occupational therapy.

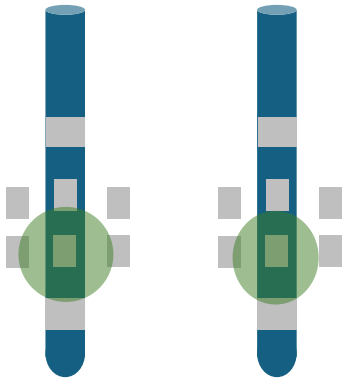
Stimulation-Induced Dyskinesia

63-year-old female. Bilateral STN DBS for management of PD with motor fluctuations and dyskinesias on moderate levodopa doses. Challenging programming due to dyskinesias.

Entry stimulation parameters:

C+ 1-; 1.9 mA; 60 µs; 130 Hz

C+ 9- ; 1.6 mA; 60 µs; 130 Hz



Approach:

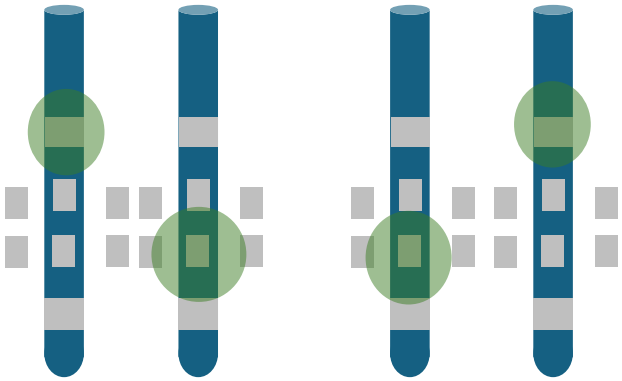
1. Distinguish between peak-dose, biphasic, or stimulation induced dyskinesia. Cycle stimulation on/off across the dose cycle.
2. Stimulate with low amplitude and slow increments
3. Lower dopaminergic medication if dyskinesias parallel motor benefit.
4. Move stimulation to the next adjacent dorsal contact.
5. Lower stimulation frequency by decrements of 10 Hz.
6. If suboptimal, consider activating a the more dorsal contacts (Zi) with one of the following options: double monopolar, interleaving, longitudinal steering.
7. Consider adaptive settings (aDBS)
8. If biphasic dyskinesia, consider adding a dopamine agonist.

Stimulation-Induced Dyskinesia

Final stimulation parameters:

C+ 1-; 1.9 mA; 60 µs; 110 Hz
C+ 3-; 1.0 mA; 60 µs; 110 Hz

C+ 9- ; 1.6 mA; 60 µs; 110 Hz
C+ 11- ; 0.9 mA; 60 µs; 110 Hz



Approach:

1. Lowered stimulation frequency by decrements of 10 Hz.
2. Activated the more dorsal contacts (Zi) with interleaving

Take home messages

Localize first: on-stim vs. off-stim, on-med vs. off-med. Don't reprogram before you know what you're treating.

Map before you move: A full monopolar/directional review defines the real therapeutic window.

Steer, don't just shrink – directional contacts, pulse width, and frequency reshape the field; amplitude reduction is a last resort.

Programming and pharmacotherapy are titrated together, not stimulation alone.

New, delayed, or asymmetric symptoms deserve a hardware/imaging work-up before reprogramming.

Build a multidisciplinary safety net – PT, OT, SLP and psychiatry are part of the algorithm.