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Troubleshooting Algorithms and Individualized Strategies for the Subthalamic Nucleus in Parkinson's Disease

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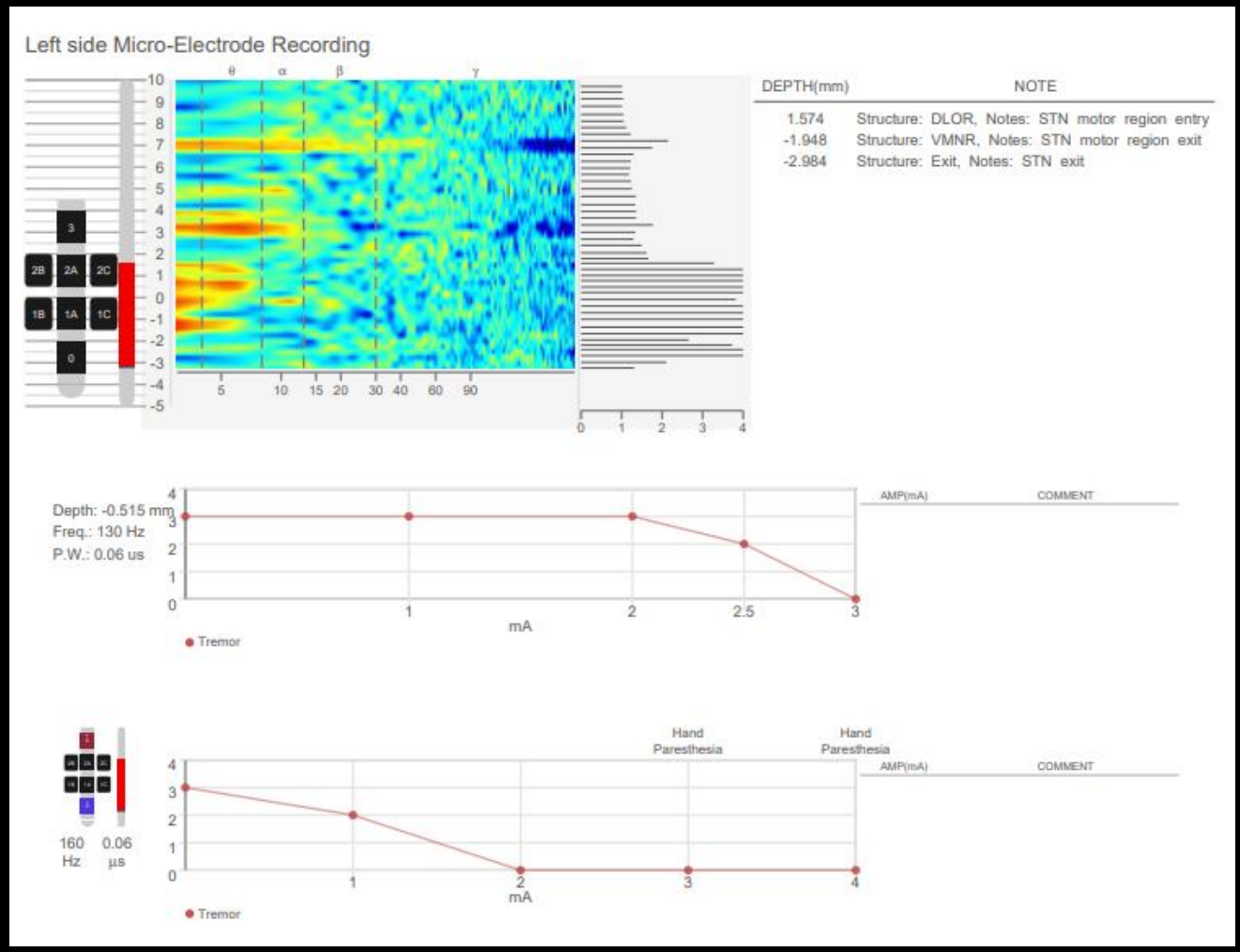
Customized Reports Aid DBS Programming



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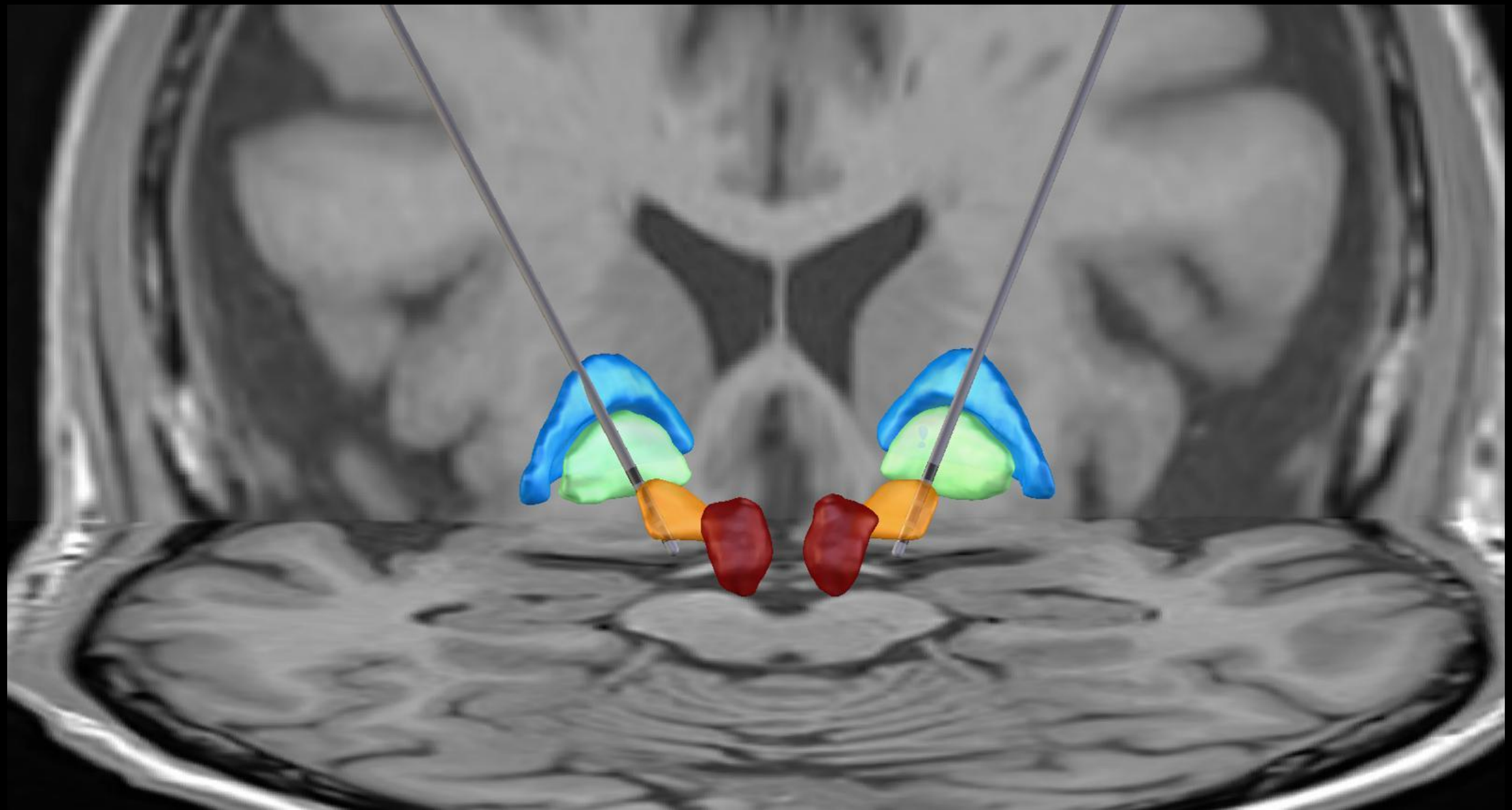
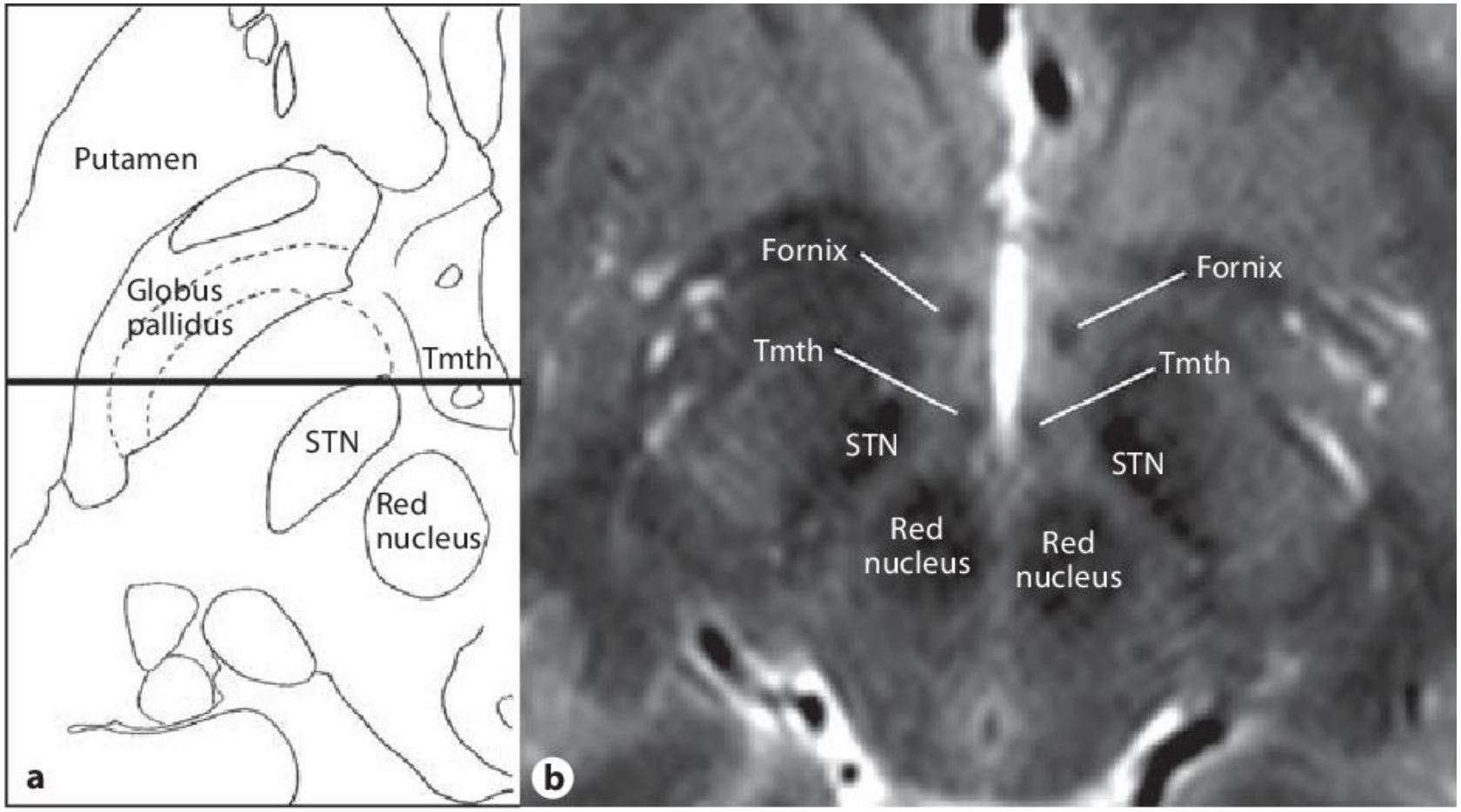
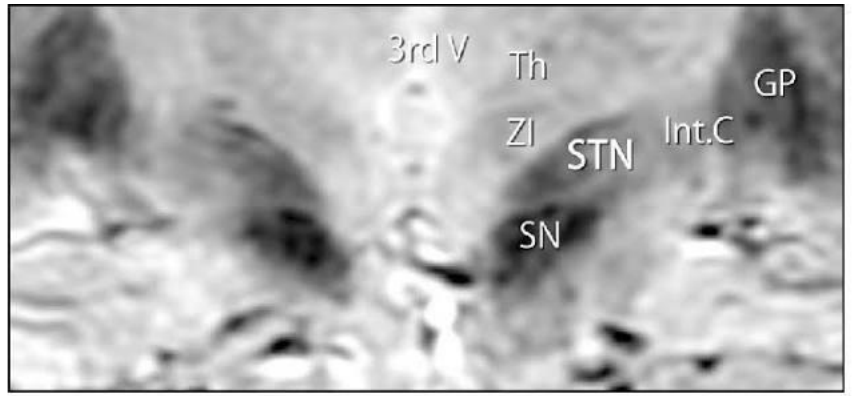
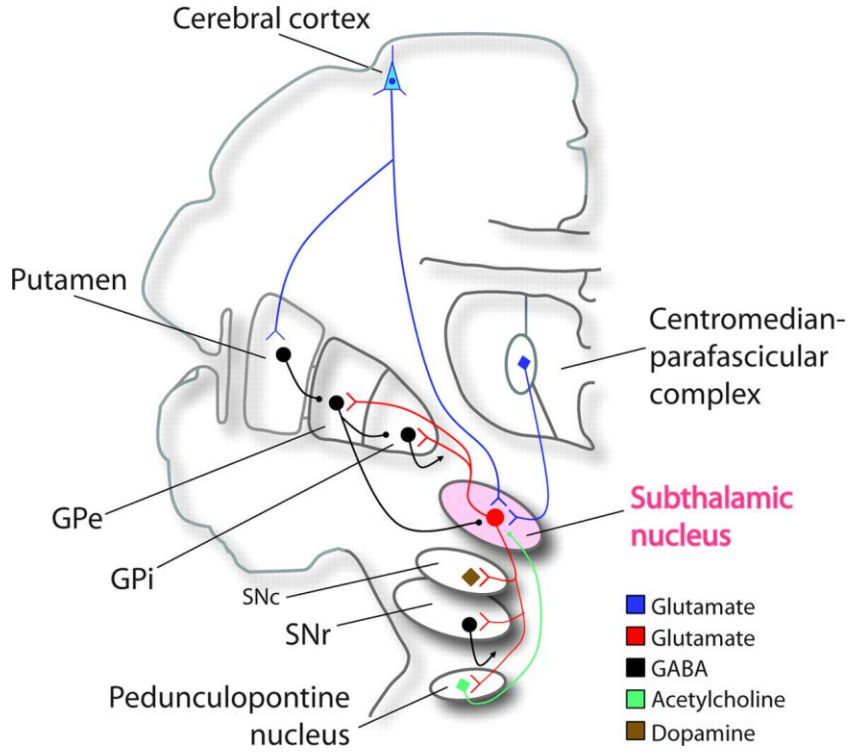
Advanced Imaging to Verify Electrode Placement



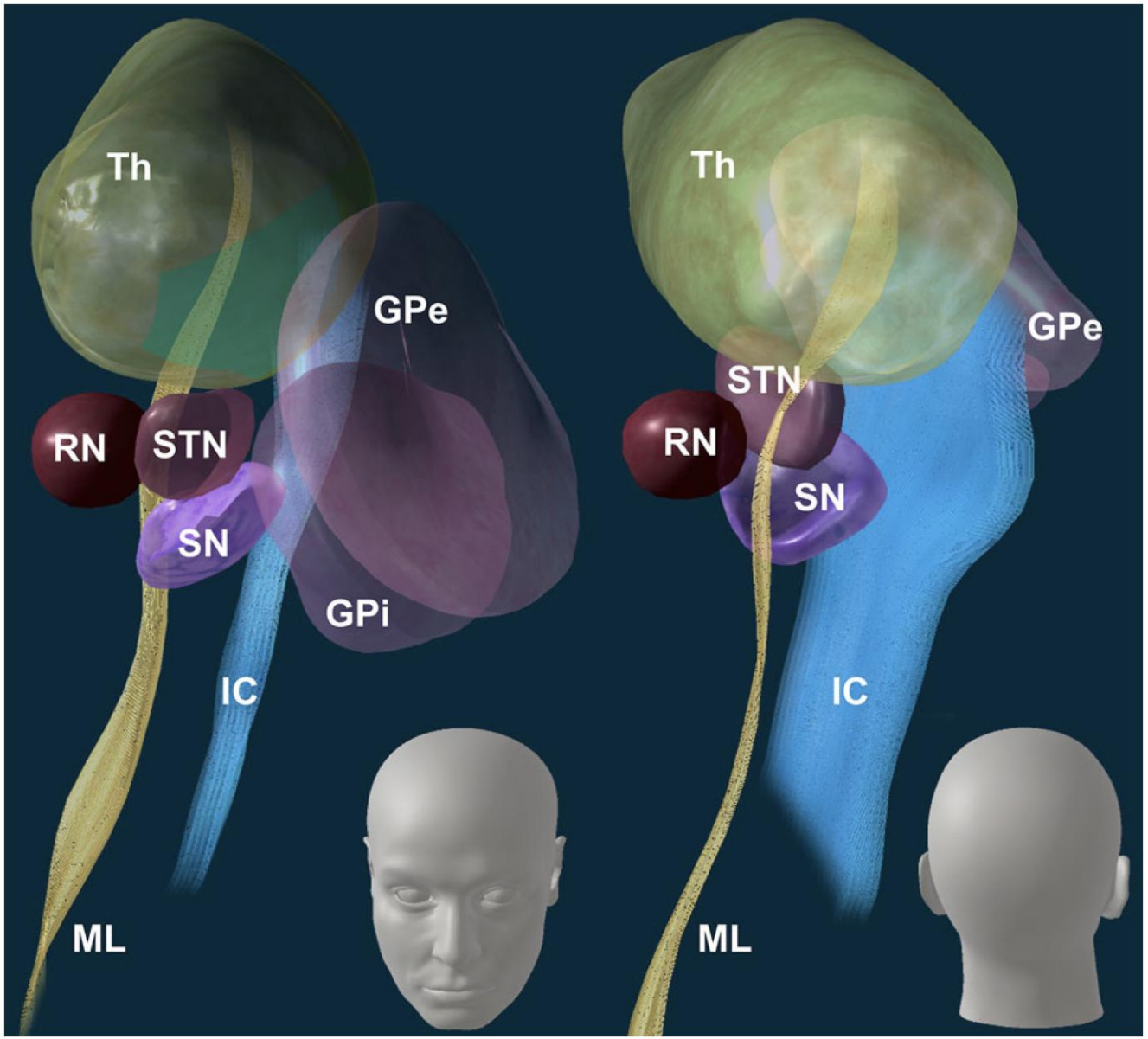
Anatomy Around the Subthalamic Nucleus







Relationship of DBS Responses to STN Anatomy



- **Lateral (or Anterior) to STN -** Internal Capsule (**IC**): **<u>contralateral</u> muscular contraction (facial pulling, eye deviation, etc.)**
- **Medial and Posterior to STN –** Cranial III Nerve: Diploipa, **<u>Ispilateral</u>** 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

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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
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STN Functional Anatomy

Dorsolateral STN: sensorimotor target.
Anterior/medial spread: cognitive, psychiatric effects.
Lateral spread: internal capsule.
Ventral spread: substantia nigra/zona incerta.

Monopolar Review

Test every contact systematically.
Record efficacy threshold, adverse-effect threshold, therapeutic window.
Directional leads: evaluate each segment individually.

Parameter Optimization

Amplitude: largest effect on VTA.
Pulse width: shorten (30–40 μs) to widen therapeutic window.
Frequency: 130–185 Hz for appendicular symptoms; 60–100 Hz for selected gait freezing patients.

Persistent Tremor

Increase amplitude if therapeutic window allows.
Move dorsally or use directional steering.
Rule out medication-resistant tremor and lead location issues.

Bradykinesia and Rigidity

Optimize sensorimotor contact.
Increase amplitude before pulse width.
Assess medication state to avoid underestimating benefit.

Stimulation-Induced Dyskinesia

Decrease amplitude.
Slow titration over weeks.
Move dorsally, shorten pulse width, or use bipolar/current steering.
Adjust levodopa after stabilization.

Capsular Spread

Facial pulling, dysarthria, limb contraction.
Reduce amplitude, shorten pulse width, steer medially/posteriorly if possible.

Oculomotor & Sensory Side Effects

Eye deviation suggests oculomotor fiber activation.
Paresthesias often indicate medial lemniscus spread.
Modify contact orientation or reduce VTA.

Axial Symptoms

Differentiate disease progression from stimulation effect.
Consider 60–100 Hz trial for freezing of gait in selected patients.
Evaluate bilateral symmetry and medication interaction.

Directional Programming

Use image-guided orientation.
Steer current away from internal capsule and medial lemniscus.
Maintain equivalent total current when comparing segments.

Hardware Troubleshooting

High impedance: open circuit/connector issue.
Low impedance: possible short circuit.
Verify battery status, extensions, and imaging when indicated.

When Programming Fails

Review postoperative imaging.
Fuse MRI/CT to assess lead position.
Consider revision if chronic inadequate benefit with narrow therapeutic window.

Evidence Summary

Programming remains individualized.
Short pulse widths and directional stimulation frequently widen the therapeutic window.
Programming should integrate anatomy, MER, imaging, and clinical examination.