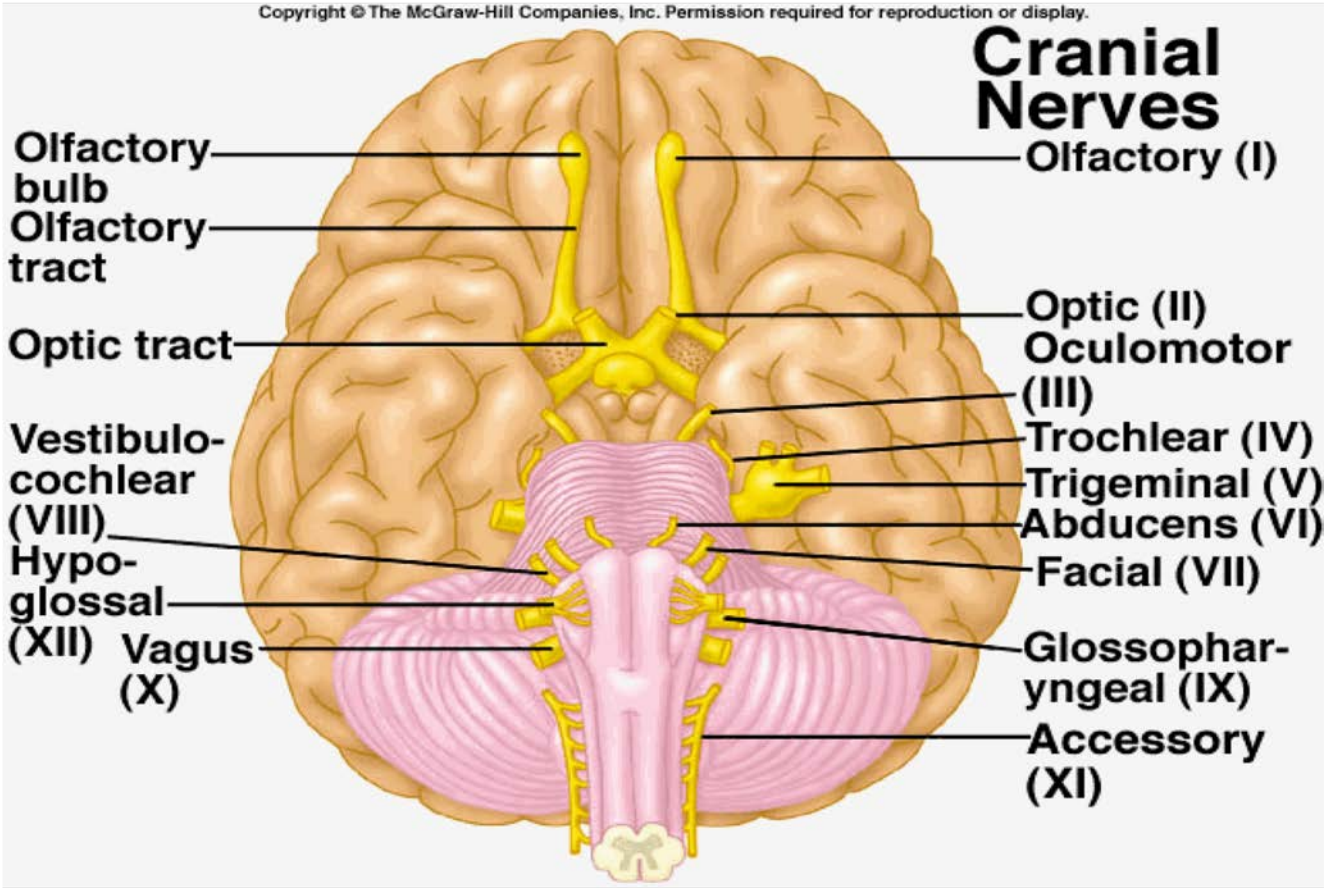
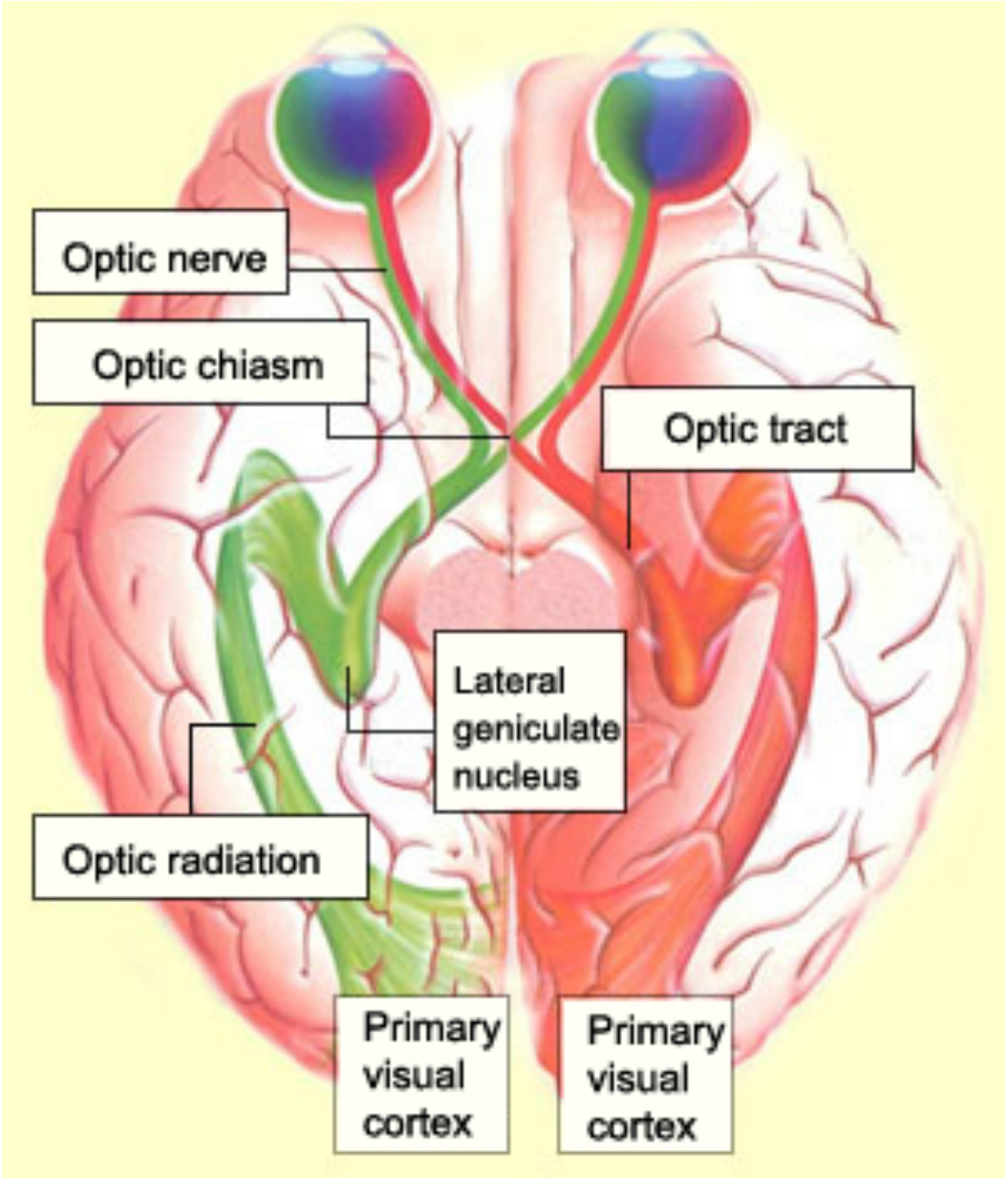
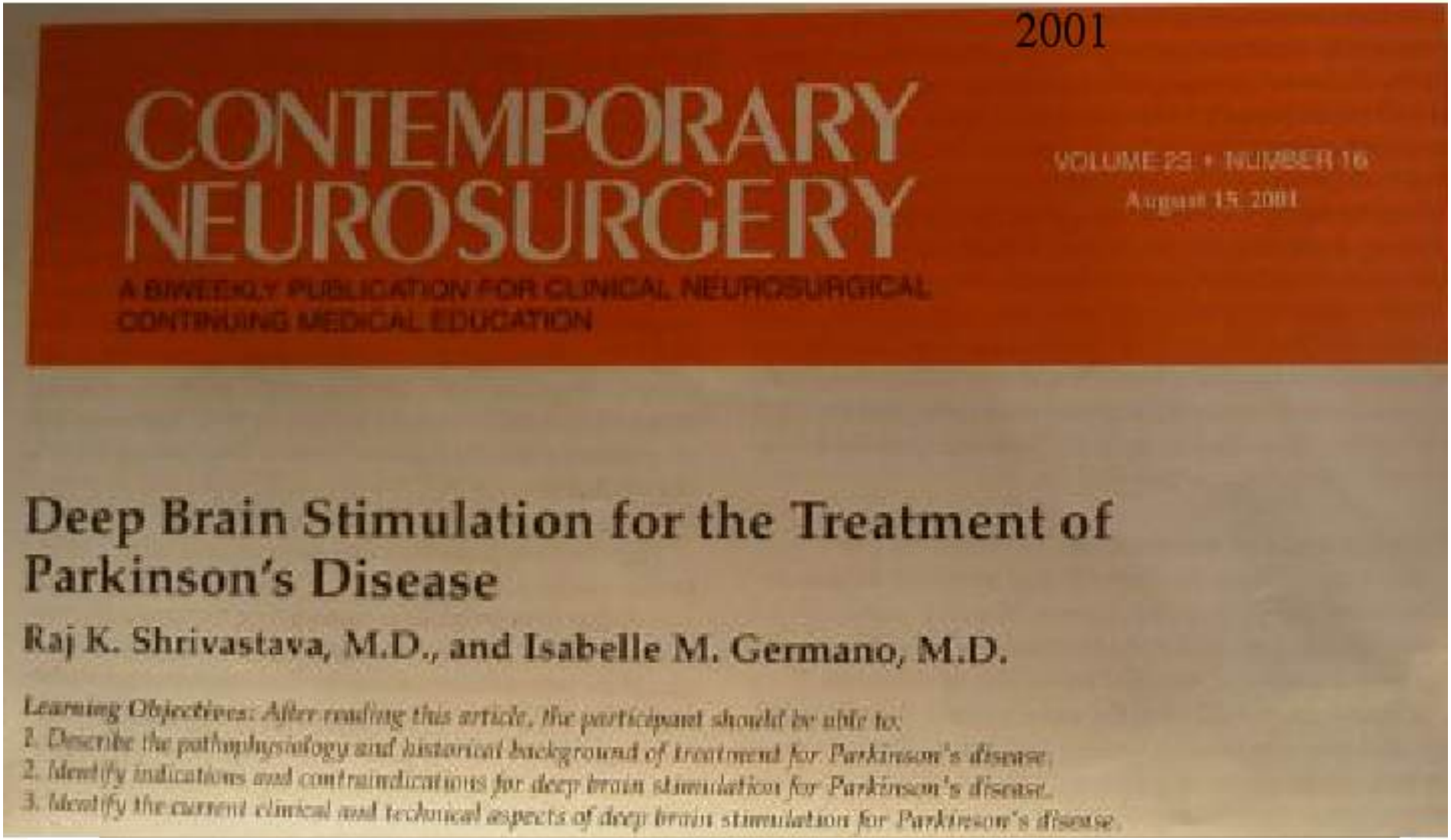
II Optic
III Oculomotor
IV Trochlear
V Trigeminal
VI Abducens
VII Facial
X Vagus




Optic nerve
Optic chiasm
Optic tract
Lateral geniculate nucleus
Optic radiation
Primary visual cortex
Primary visual cortex



Learning objectives: Identify current clinical and technical aspects of DBS for PD

CME publication, 2001

(Publication de Formation Médicale Continue")

2001

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Deep Brain Stimulation for the Treatment of Parkinson's Disease

Raj K. Shrivastava, M.D., and Isabelle M. Germano, M.D.

Learning Objectives: After reading this article, the participant should be able to:
1. *Describe the pathophysiology and historical background of treatment for Parkinson's disease.*
2. *Identify indications and contraindications for deep brain stimulation for Parkinson's disease.*
3. *Identify the current clinical and technical aspects of deep brain stimulation for Parkinson's disease.*

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Parkinson's disease (PD) is one of the most common neurological disorders, with an estimated 1 million individuals affected every year. The average age of onset is 60 years. The classical clinical features of PD are tremor at rest, rigidity, bradykinesia, and postural and gait instability. Tremor at rest is one of the first presenting symptoms in up to 70% of newly diagnosed cases. However, as the disease progresses, the other symptoms usually become the most devastating. Parkinson-like symptoms (i.e., parkinsonism) are present in diseases other than idiopathic PD (Table 1). The differential diagnosis of PD includes a range of parkinsonism-plus syndromes. These include Shy-Drager syndrome, multiple system atrophy, and progressive supranuclear palsy, as well as secondary parkinsonism related to other factors such as vascular disease, drug and toxin exposure, repeated trauma, hydrocephalus, tumors, and infections.

Medical therapy with levodopa is the standard treatment for PD. Long-term therapy (5 years or more) with levodopa, however, results in significant side effects such as dyskinesias and motor fluctuations in most patients. In addition, levodopa-induced dyskinesias can develop earlier in patients who present with early-onset disease.

Pathophysiology

Clinical and laboratory studies support the theory that PD is caused by an increase in basal ganglia output. The pathology underlying PD is based on the loss of dopaminergic input to the striatum, combined and associated with degeneration of the nigrostriatal pathway. This effect leads to decreased activity of the striatal inhibitory neurons via D2 receptors and increased activity of striatal neurons via D1 receptors. The direct-indirect pathway model developed in the late 1980s described two parallel channels of neuronal information converging to influence the output nuclei of the basal ganglia. According to this model, alterations in levels of excitation or inhibition produce changes in the absolute level of neuronal activity in the target structures. The striatal inhibitory neurons project directly to the internal segment of the globus pallidus internus (GPi), and the striatal neurons project indirectly through the globus pallidus externus (GPe) and the subthalamic nucleus (STN).

The GPe and GPi are anatomically adjacent, separated by the internal capsule, and they consist of large, caudally projecting GABA-ergic neurons. These neurons have a high baseline firing rate, probably due to tonic stimulation from the STN, suggesting that they exert tonic inhibition on their target structures. The overall effect for both pathways is an increase in activity of GPi, which suppresses cortical activity via inhibitory projections to the thalamus.

The two pathways are believed to have different roles in the generation of movements, with the indirect pathway exerting, via the GPi, tonic inhibition of target structures in order to block

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The authors have disclosed that they have no significant relationships with or financial interests in any commercial organizations pertaining to this educational activity.

Category: Functional

Key Words: Parkinson's disease, Deep brain stimulation, Image-guided computer-assisted neurosurgery, Subthalamic nucleus, Globus pallidus, Ventrointermediate nucleus of the thalamus

CME publication, 2001

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FOX NEWS, February 2011

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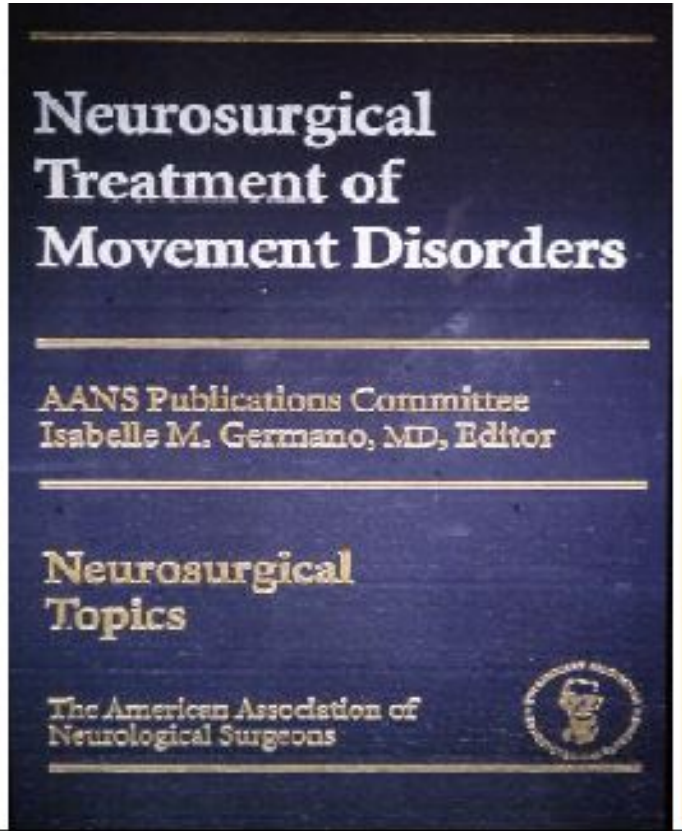

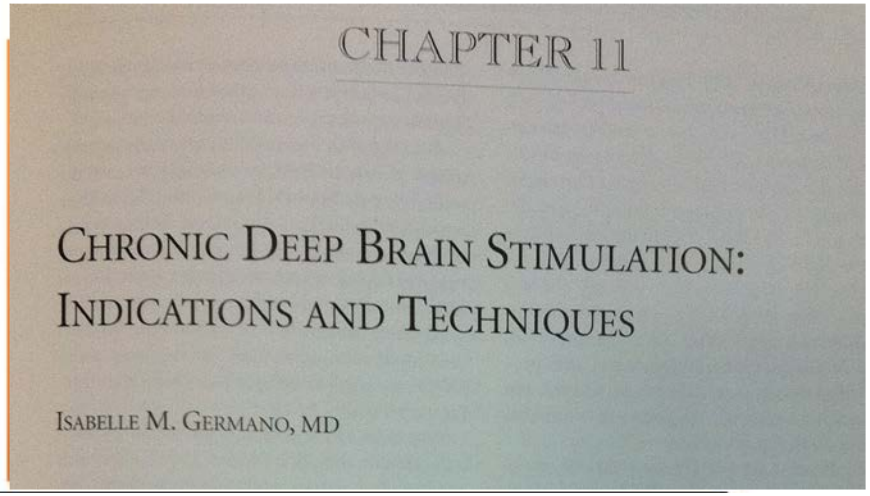

The strategy for implantation of a permanent electrode in the GPi includes finding its lateral boundary, the internal capsule, and its inferior boundary, the optic tract. T

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