Hypophonia in Parkinson's disease: Neural correlates of voice treatment revealed by PET

Liotti et al. followed up on earlier work on Lee Silverman voice treatment efficacy in Parkinson's disease by studying five Parkinson's disease patients with PET before and after Lee Silverman voice treatment. After treatment, neural activity as reflected by regional cerebral blood flow was reduced in motor-premotor cortex and increased in basal ganglia, prefrontal cortex, and anterior insula.

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Instruct patients with Parkinson's disease: "Speak loud and low!"

de Swart et al. compared the effectiveness of a newly developed pitch-limiting voice treatment with the Lee Silverman voice treatment ("think loud, think shout"). The new method of instruction "speak loud and low" (illustrated with online video) appeared to yield an immediate improvement of loudness and voice quality, and to prevent frequently-encountered adverse therapy effects on vocal pitch and laryngeal muscle tension seen with Lee Silverman voice treatment.

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Speech therapy: Does it work?

Commentary by David Rosenfeld, MD

"Speech" is the motor output of language. Human beings readily acquire speech and language during critical periods of development, but have difficulty learning them as adults. Similarly, it is difficult for heretofore normal patients afflicted with brain disease to overcome their dysarthria. However, there are some effective techniques for these patients.

In this issue, Liotti et al. document in Parkinson's patients taught to "think loud, think shout," a shift in PET activity from premotor cortex to

basal ganglia and anterior insula, speculating a change from effortful to more automatic speech production. de Swart et al., also in this issue, advocate an improved technique, teaching patients to "speak loud, speak low," effectively overcoming the strained high pitch that can result from the previous method. These authors remind us that there are neural correlates for speech-motor production, and compromised patients can learn techniques to improve their speaking.

Speech production is complex,

involving acoustics, mathematics, and physics.¹ If we can develop new speech therapies and ascertain what parts of the brain are involved in these therapeutic process, we can improve therapeutic efficacy for those afflicted with speech compromise.

Reference

1. Raphael L, Harris K, Borden G, Bickley L. *Speech Science Primer: Physiology, Acoustics, and Perception of Speech*, 4th ed. Baltimore: Lippincott Williams & Wilkins, 2002.

Health care utilization for migraine in the UK and the US

Lipton et al. examined migraine diagnosis and treatment in the US and the UK. Though consultation and diagnosis rates are higher in the UK, most patients with migraines never receive a medical diagnosis and are treated with over-the-counter drugs, excluding prescription drugs, despite high levels of disability.

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Early and late seizures after stroke

Lamy et al. (with the PFO and Atrial Septal Aneurysm Study Group) followed 581 patients age 18–55 years with unexplained ischemic stroke for a mean interval of over 3 years. Early (<7d) seizures occurred in 2.4% and were more likely to occur in patients with greater stroke disability and cortical involvement. Late seizures occurred in 5%; risk factors were early seizures and cortical signs and larger infarct size.

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“Based on risk alone, treatment may be indicated after the first seizure no matter when it occurs. . .”

The accompanying editorial by Labovitz and Hauser notes that since stroke is the leading cause of late-onset epilepsy the issue of whether and when to treat stroke patients with antiepileptic drugs is of importance. The Lamy et al. study found such a low incidence of seizures that treatment should be reserved for patients who have had at least one seizure. However, Labovitz and Hauser note that the study population reported by Lamy et al. limits the conclusions that can be drawn. Only young patients were studied and patients were excluded if long term survival or ability to follow up were in question.

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Slowing Parkinson’s disease progression with dopamine agonists

Is the progression of Parkinson’s disease slowed by pramipexole or ropinirole administration? Since slowing has been suggested by recent clinical trials that have assessed Parkinson’s disease progression with striatal dopaminergic SPECT or PET imaging. J. Eric Ahlskog argues that these results are confounded by chronic study-drug influences on regulated proteins that bind, transport, or metabolize the radioligands. It may be premature to conclude that these drugs have a neuroprotective effect in Parkinson’s disease.

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Albin and Frey review the basic science rationale and clinical data for initial dopamine agonist treatment of Parkinson’s disease. They point out that basic science data may not be relevant and concur that clinical data is equivocal. Further clinical studies are needed.

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Fred Wooten’s editorial crisply summarizes the matter:

1. Is there a symptomatic advantage for agonists over levodopa for initial therapy in Parkinson’s disease? . . . “I do not see a clear symptomatic advantage for agonists based on current data.”

2. Are dopamine agonist drugs neuroprotective? Because the β -CIT SPECT and 18F-fluorodopa PET studies are problematic with present paradigms, and because clinical end points are at odds with imaging data, . . . “I [am] not persuaded that the current evidence establishes that dopamine agonists are neuroprotective in patients with Parkinson’s disease.”

He emphasizes the need for “development of a validated clinical scale to address Parkinson’s disease progression that would take in account signs and symptoms, disability/functional capacity, quality of life, and treatment-related complications and side effects.”

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Routine evaluation of children with global developmental delay

Between 1% and 3% of children have global developmental delay. The Shevell et al. AAN Practice Parameter reviews recommendations for their evaluation. Routine metabolic screening has a yield of only ~1%. Because of a higher yield (3.5% to 10%), routine cytogenetic studies and molecular testing for the Fragile X mutation are recommended. Assessment for Rett syndrome should be considered in females with global developmental delay. Additional genetic studies (e.g., subtelomeric chromosomal rearrangements) should be considered in appropriate circumstances. EEG is recommended only in global developmental delay with features suggestive of epilepsy/epileptic syndrome. Routine neuroimaging is recommended: MRI preferred to CT. The high incidence of visual and auditory impairment mandates appropriate visual and audiometric assessment.

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How soon can Rasmussen's encephalitis be identified?

Granata et al. studied 12 patients who proved to have Rasmussen's encephalitis. Characteristic clinical, EEG, and MRI findings would have established tentative diagnosis by 6 months from first symptoms.

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Bayes theorem applied to electrodiagnostic testing in carpal tunnel syndrome

T2-weighted MRI 15 days and 5 years after the first seizure.

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Is menarche epileptogenic?

In a retrospective study, Klein et al. showed that epilepsy in women starts more commonly during perimenarche than during other postnatal childhood periods. Hormonal changes during perimenarche may promote the development of epilepsy.

Three nerve conduction tests combined.

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Are asymptomatic microbleeds a risk factor for aspirin-associated intracerebral hemorrhages?

A gradient-recall echo MRI shows a symptomatic left putaminal hemorrhage (arrow) and multiple asymptomatic microbleeds in the contralateral hemisphere (arrowheads).

Wong et al. examined gradient recall-echo MRI for detection of microbleeds in patients with aspirin-associated intracerebral hemorrhages. Microbleeds were both more frequent and more extensive in aspirin users with intracerebral hemorrhages than seen in aspirin-users without intracerebral hemorrhages.

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