chotic symptoms like those in this patient are present in 3%–11% of cases and include isolated delusional states, auditory hallucinations and paranoia.[2]

After establishing the diagnosis of Huntington's disease with genetic analysis, the MRI images for this patient were reevaluated. They were again read as showing only ventricular enlargement evocative of normal pressure hydrocephalus, without evidence of the surrounding caudate atrophy associated with Huntington's disease. Could both diagnoses be present? A review of the literature revealed two case reports of patients diagnosed with Huntington's disease by motor symptoms and family history, who then developed ventricular enlargement accompanied by cognitive symptoms that suggested to the authors an additional diagnosis of normal pressure hydrocephalus.[3] Lumbar punctures revealed normal opening CSF pressure and shunts placed in these patients improved their cognitive symptoms but did not affect their chorea. This finding prompted the authors to speculate a process by which the cortical changes in Huntington's disease contributed to the development of normal pressure hydrocephalus. Nonetheless, despite the suggestive imaging, the genetic diagnosis of Huntington's disease in this patient indicates a convincing single diagnosis whose symptoms were, unlike the earlier case reports, unchanged after a lumbar puncture. It is unlikely that he possesses both diagnoses.

A retrospective look at this patient's case suggests that this patient's behavior and cognitive decline are consistent with the diagnosis of Huntington's disease. It is crucial to consider Huntington's disease as a diagnosis in elderly patients exhibiting cognitive decline with prominent psychiatric symptoms even in the presence of imaging studies that suggest normal pressure hydrocephalus. These elderly patients may be the first in their family to exhibit symptoms of Huntington's disease.

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Two Novel Comments on the Treatment of Huntington's Disease

To the Editor: Huntington's disease is a hereditary brain disorder that affects people of all races, all over the world. Until quite recently, little was known about Huntington's disease. Yet in the last 20 years, much has been learned about the causes and effects of Huntington's disease and about therapies and techniques for managing the symptoms.

Huntington's disease is a degenerative disease whose symptoms are caused by the loss of cells in the basal ganglia. This damage to cells affects cognitive ability (thinking, judgment, and memory), movement, and emotional control. Symptoms appear gradually, usually in midlife, between the ages of 30 and 50 years old. However, the disease can strike young children (juvenile form) and the elderly.[1]

Despite the vast research on the anatomy and physiology of the basal ganglia, there is little agreement on the exact role the basal ganglia play in the behavior and motor control. While numerous models of basal ganglia have been proposed, few are computational in nature; thus, very few simulations of basal ganglia exist for testing the hypotheses and comparing the results to neurological and psychophysical data. The lack of computational models may, however, be partly due to the complexity of basal ganglia anatomy, multiplicity of physiological responses, and subtleties of behavioral responses.[2]

We believe that the mathematical models of brain physiological performance have a great role in elucidating medical knowledge and have expanded our attitude about the illness states. The increase in effectiveness of mathematical models is manifested by two factors: the complexity of brain structure itself and greater complexity of pathological conditions. Certain neurological disorders such as Huntington's disease have a particular importance and have gained the attention of many researchers.[3]

We have implemented a computational model for gait disorder to show how *GABA* level variation and glutamate toxicity can cause the disease.[1,4] Then, in a new unpublished research, we tried to introduce a perfect model, based on recordings taken from Huntington's disease patients.[5] This study shows the effect of changing neurotransmitters on movement symptoms of Huntington's disease. We have considered most available physiologi-

cal information about neuron basic features and basal ganglia.

Basal ganglia are formed of some main parts, each of which is formed from many neurons that work together to do their job (i.e., control of movement). Similarly, our model has blocks that are formed in the same arrangement. In addition, the relation between blocks is modeled by proper gains, and inside each neuronal block we have put a related mathematical model. Each block has a threshold, firing rate, and a first order transfer function which simulates the neuron membrane.

Our model was able to simulate the physiological behavior of basal ganglia in Huntington's disease. In order to find out an effective new treatment, we ran our model with different amounts of gains (i.e., neurotransmitter amounts). Two important finding was obtained:

1. Decreasing the level of glutamate to half its normal level reduces movement disorders.

2. Increasing *GABA*, together with increasing threshold, also decreases movement disorders.

We propose that these two comments may be a good introduction to help ameliorate Huntington's disease, though experimental research is needed to validate our hypotheses.

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Conversion Hemianesthesia: Possible Mechanism

To the Editor: A recent fMRI study of three patients who had a left-sided sensory conversion disorder found that unilateral vibrotactile stimulation of the affected limb did not produce activation of the contralateral primary somatosensory region.[1] Although these authors concluded that their findings implicated selective alterations in primary sensorimotor cortex, they did not posit an explanation for their findings.

Support for the observation that patients can fail to detect stimuli in the absence of defective afferent pathways comes from the observation of patients with attentional neglect. In the absence of injury the failure of the primary sensory cortex to activate could be induced by a reduced ability of the afferent signals to reach the cortex induced by closure of a physiological gate. Sensory information reaches the cortex after relay through specific thalamic nuclei and somatosensory information is transmitted via the ventralis posterolateralis. The thalamus is surrounded by the nucleus reticularis, which sends inhibitory projections to the thalamic relay nuclei, including the ventralis posterolateralis. Activation of the nucleus reticularis inhibits thalamic relay to the cortex.[2] The nucleus reticularis serves as a gate for sensory information. When a stimulus is significant or novel, corticofugal projections inhibit the nucleus reticularis and allow the thalamus to relay sensory input to the cortex. When a stimulus is not significant or novel, corticofugal projections to the nucleus reticularis can selectively excite the nucleus reticularis and inhibit the relay of sensory information.

Unimodal association areas converge upon polymodal or supramodal association cortex such as the prefrontal cortex and the posterior temporoparietal region. These posterior convergence areas may subserve cross-modal associations and sensory synthesis as well as store declarative memories or representations. Thus, these posterior temporoparietal areas are important in detecting novel and significant stimuli. These temporoparietal areas might project either directly or indirectly to the nucleus reticularis, and when there is a significant or novel stimulus the temporoparietal cortex might inhibit the inhibitory nucleus reticularis and allow more sensory information to the cortex. In contrast, the supramodal frontal cortex might be important in exciting the inhibitory nucleus reticularis and thus gate out information. Support for this postulate comes from the study of patients' visual habituation using the Troxlar paradigm.[3] In this paradigm the subject looks at a central fixation point and notes when a lateral dot fades from vision. When these dots are presented contralateral to parietal lesions there was enhanced habituation (rapid fading), but when presented contralateral to frontal lesions there was delayed habituation.