peripheral gangliocytes in a few cases. We concur with Dr. Goldstein that the distinction between multiple-system atrophy and Parkinson's disease with dysautonomia has been improved by monoaminergic radiolabeling studies.[3]

Dr. Schatz identifies an editing error by Dr. Schlossmacher, who misused the word "pure" in characterizing autonomic failure in multiple-system atrophy (the Shy–Drager syndrome). As a disease of the peripheral nervous system, pure autonomic failure lacks involvement of the central nervous system and, thus, signs of parkinsonism or cerebellar dysfunction. Adding to the complexities in nomenclature and pathophysiology, pure autonomic failure shares with Parkinson's disease the predilection for postganglionic neurons and the characteristic, intraneuronal accumulation of α-synuclein.[4]

Dr. Martin asks whether our patient exemplified the known inaccuracy of predicting death within six months of hospice enrollment. During the last six months of her life, she resided in a nursing home that provided full palliative care and did not need a hospice referral. In general, we favor a hospice approach for patients with advanced neurodegenerative diseases.[5]

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Somatic Mutations of *EGFR* in Colorectal Cancers and Glioblastomas

TO THE EDITOR: Recent reports suggest that mutations in the epidermal growth factor receptor (*EGFR*) gene predict sensitivity to EGFR kinase inhibitors. In particular, patients with lung cancers containing mutations in the EGFR kinase domain have had responses to gefitinib.[1,2] Are similar *EGFR* mutations present in a significant fraction of other tumor types for which gefitinib might be suitable therapy? To answer this question, we screened DNA from 293 colorectal tumors and 59 glioblastomas for alterations in the EGFR kinase domain (exons 17 to 24). These tumors were chosen for analysis because they have been linked to EGFR signaling: EGFR-targeted antibodies (cetuximab) have been approved for use in patients with colorectal cancer, and structural alterations of the *EGFR* gene (amplifications and rearrangements) have been described in glioblastomas.[3] However, our analysis showed that only one of the colorectal cancers and none of the glioblastomas harbored a mutation. The single mutation was a G-to-S substitution at amino acid 719, which is identical to an activating mutation previously reported in lung tumors.[2] Our results show *EGFR* mutations occur at a very low frequency in colorectal cancers and glioblastomas and suggest that gefitinib is unlikely to be effective in patients with these tumors. Furthermore, these data suggest that mutations in the EGFR kinase domain are unlikely to be responsible for the reported success of cetuximab against many colorectal cancers.[4,5]

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