from apathy to shoplifting, compulsive gambling, sexual indiscretions, remarkable lack of common sense, new ritualistic behaviors, and alterations in dietary preferences, usually leading to increased taste for sweets or rigid attachment to specific food items. In many patients with AD, neurofibrillary degeneration eventually spreads to prefrontal cortex and gives rise to components of the frontal lobe syndrome, but almost always on a background of severe memory impairment. Rarely, the bvFTD syndrome can arise in isolation in the context of an atypical form of AD pathology.

Lesions in the caudate nucleus or in the dorsomedial nucleus of the thalamus (subcortical components of the prefrontal network) also can produce a frontal lobe syndrome. This is one reason why the changes in mental state associated with degenerative basal ganglia diseases such as Parkinson's disease and Huntington's disease display components of the frontal lobe syndrome. Bilateral multifocal lesions of the cerebral hemispheres, none of which are individually large enough to cause specific cognitive deficits such as aphasia and neglect, can collectively interfere with the connectivity and therefore integrating (executive) function of the prefrontal cortex. A frontal lobe syndrome is therefore the single most common behavioral profile associated with a variety of bilateral multifocal brain diseases, including metabolic encephalopathy, multiple sclerosis, and vitamin B_{12} deficiency, among others. Many patients with the clinical diagnosis of a frontal lobe syndrome tend to have lesions that do not involve prefrontal cortex but involve either the subcortical components of the prefrontal network or its connections with other parts of the brain. To avoid making a diagnosis of "frontal lobe syndrome" in a patient with no evidence of frontal cortex disease, it is advisable to use the diagnostic term *frontal network syndrome*, with the understanding that the responsible lesions can lie anywhere within this distributed network. A patient with frontal lobe disease raises potential dilemmas in differential diagnosis: the abulia and blandness may be misinterpreted as depression, and the disinhibition as idiopathic mania or acting out. Appropriate intervention may be delayed while a treatable tumor keeps expanding.

CARING FOR PATIENTS WITH DEFICITS OF HIGHER CEREBRAL FUNCTION

Brain damage may cause a dissociation between feeling states and their expression so that a patient who may superficially appear jocular could still be suffering from an underlying depression that needs to be treated. If neuroleptics become absolutely necessary for the control of agitation, atypical neuroleptics are preferable because of their lower extrapyramidal side effects. Treatment with neuroleptics in elderly patients with dementia requires weighing the potential benefits against the potentially serious side effects.

Spontaneous improvement of cognitive deficits due to acute neurologic lesions is common. It is most rapid in the first few weeks but may continue for up to 2 years, especially in young individuals with single brain lesions. Some of the initial deficits appear to arise from remote dysfunction (diaschisis) in parts of the brain that are interconnected with the site of initial injury. Improvement in these patients may reflect, at least in part, a normalization of the remote dysfunction. Other mechanisms may involve functional reorganization in surviving neurons adjacent to the injury or the compensatory use of homologous structures, e.g., the right superior temporal gyrus with recovery from Wernicke's aphasia. Cognitive rehabilitation procedures have been used in the treatment of higher cortical deficits. There are few controlled studies, but some show a benefit of rehabilitation in the recovery from hemispatial neglect and aphasia. Determining driving competence is challenging, especially in the early stages of dementing diseases. The diagnosis of a neurodegenerative disease is not by itself sufficient for asking the patient to stop driving. An on-the-road driving test and reports from family members may help time decisions related to this very important activity.

Some of the deficits described in this chapter are so complex that they may bewilder not only the patient and family but also the physician. It is imperative to carry out a systematic clinical evaluation to characterize the nature of the deficits and explain them in lay terms to the patient and family. An enlightened approach to patients with damage to the cerebral cortex requires an understanding of the principles that link neural networks to higher cerebral functions in health and disease.