deficits on cognitive testing involve executive control or language (speech or naming) function, but some patients lack either finding despite profound social-emotional deficits. PDD or DLB patients have more severe deficits in visuospatial function but do better on episodic memory tasks than patients with AD. Patients with vascular dementia often demonstrate a mixture of executive control and visuospatial deficits, with prominent psychomotor slowing. In delirium, the most prominent deficits involve attention, working memory, and executive function, making the assessment of other cognitive domains challenging and often uninformative.

A functional assessment should also be performed to help the physician determine the day-to-day impact of the disorder on the patient's memory, community affairs, hobbies, judgment, dressing, and eating. Knowledge of the patient's functional abilities will help the clinician and the family to organize a therapeutic approach.

Neuropsychiatric assessment is important for diagnosis, prognosis, and treatment. In the early stages of AD, mild depressive features, social withdrawal, and irritability or anxiety are the most prominent psychiatric changes, but patients often maintain core social graces into the middle or late stages, when delusions, agitation, and sleep disturbance may emerge. In FTD, dramatic personality change with apathy, overeating, compulsions, disinhibition, euphoria, and loss of empathy are early and common. DLB is associated with visual hallucinations, delusions related to person or place identity, RBD, and excessive daytime sleepiness. Dramatic fluctuations occur not only in cognition but also in arousal. Vascular dementia can present with psychiatric symptoms such as depression, anxiety, delusions, disinhibition, or apathy.

LABORATORY TESTS

The choice of laboratory tests in the evaluation of dementia is complex and should be tailored to the individual patient. The physician must take measures to avoid missing a reversible or treatable cause, yet no single treatable etiology is common; thus, a screen must use multiple tests, each of which has a low yield. Cost/benefit ratios are difficult to assess, and many laboratory screening algorithms for dementia discourage multiple tests. Nevertheless, even a test with only a 1–2% positive rate is worth undertaking if the alternative is missing a treatable cause of dementia. Table 35-3 lists most screening tests for dementia. The American Academy of Neurology recommends the routine measurement of a complete blood count, electrolytes, renal and thyroid function, a vitamin B_{12} level, and a neuroimaging study (computed tomography [CT] or MRI).

Neuroimaging studies, especially MRI, help to rule out primary and metastatic neoplasms, locate areas of infarction or inflammation, detect subdural hematomas, and suggest NPH or diffuse white matter disease. They also help to establish a regional pattern of atrophy. Support for the diagnosis of AD includes hippocampal atrophy in addition to posterior-predominant cortical atrophy (Fig. 35-1). Focal frontal, insular, and/or anterior temporal atrophy suggests FTD (Chap. 448). DLB often features less prominent atrophy, with greater involvement of amygdala than hippocampus. In CJD, magnetic resonance (MR) diffusion-weighted imaging reveals restricted diffusion within the cortical ribbon and basal ganglia in most patients. Extensive white matter abnormalities correlate with a vascular etiology (Fig. 35-2). Communicating hydrocephalus with vertex effacement (crowding of dorsal convexity gyri/sulci), gaping Sylvian fissures despite minimal cortical atrophy, and additional features shown in Fig. 35-3 suggest NPH. Single-photon emission computed tomography (SPECT) and PET scanning show temporal-parietal hypoperfusion or hypometabolism in AD and frontotemporal deficits in FTD, but these changes often reflect atrophy and can therefore be detected with MRI alone in many patients. Recently, amyloid imaging has shown promise for the diagnosis of AD, and Pittsburgh Compound-B (PiB) (not available outside of research settings) and ^{18}F-AV-45 (florbetapir; approved by the U.S. Food and Drug Administration in 2013) are reliable radioligands for detecting brain amyloid associated with amyloid

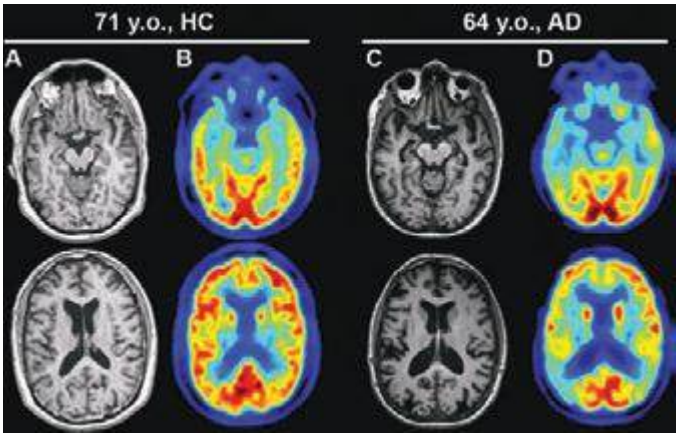

FIGURE 35-1 Alzheimer's disease (AD). Axial T1-weighted magnetic resonance images of a healthy 71-year-old (***A***) and a 64-year-old with AD (***C***). Note the reduction in medial temporal lobe volume in the patient with AD. Fluorodeoxyglucose positron emission tomography scans of the same individuals (***B*** and ***D***) demonstrate reduced glucose metabolism in the posterior temporoparietal regions bilaterally in AD, a typical finding in this condition. HC, healthy control. *(Images courtesy of Gil Rabinovici, University of California, San Francisco and William Jagust, University of California, Berkeley.)*

angiopathy or neuritic plaques of AD (Fig. 35-4). Because these abnormalities can be seen in cognitively normal older persons, however (~25% of individuals at age 65), amyloid imaging may also detect preclinical or incidental AD in patients lacking an AD-like dementia syndrome. Currently, the main clinical value of amyloid imaging is to exclude AD as the likely cause of dementia in patients who have negative scans. Once disease-modifying therapies become available, use of these biomarkers may help to identify treatment

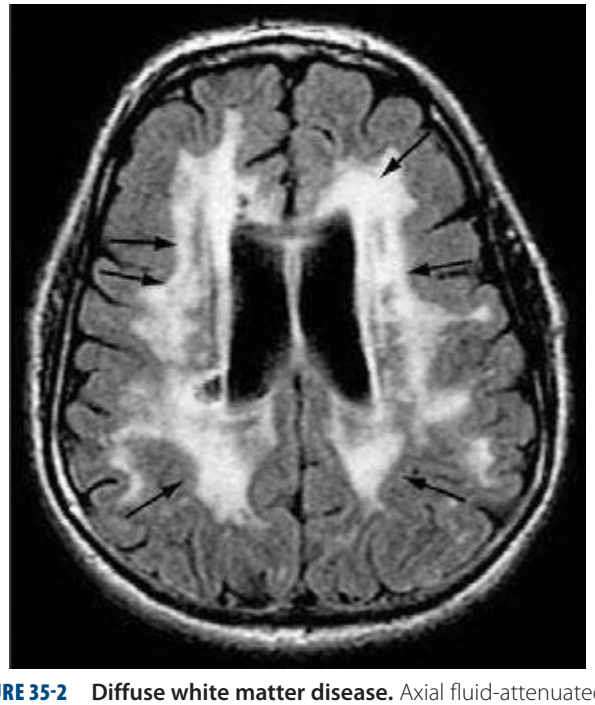

FIGURE 35-2 Diffuse white matter disease. Axial fluid-attenuated inversion recovery (FLAIR) magnetic resonance image through the lateral ventricles reveals multiple areas of hyperintensity (*arrows*) involving the periventricular white matter as well as the corona radiata and striatum. Although seen in some individuals with normal cognition, this appearance is more pronounced in patients with dementia of a vascular etiology.