Brain imaging in Alzheimer’s disease (AD) has greatly benefited from technological advances. Shifting from computed tomography (CT) to magnetic resonance imaging (MRI) allowed progress from linear to volumetric measurements of brain atrophy (Frisoni et al., 2003). Pioneer functional imaging work with low-resolution positron emission tomography (PET) machines and a method using a priori selected regions-of-interest (ROI) almost anecdotally reported posterior cingulate cortex involvement in AD, among several other associative cortices (Cutler et al., 1985). However, voxel-based analyses of the entire brain metabolism much more convincingly demonstrated a wide metabolic impairment in the posteromedial cortex, comprising posterior cingulate and medial parietal (precuneus) cortices (Minoshima et al., 1994). Voxel-based analyses allowed the comparison of cerebral images between populations and the search for clinico-metabolic comparisons without any a priori anatomical hypothesis. Emerging techniques offer the possibility to measure decreased posterior cingulate biological brain components in AD with proton MR spectroscopy (Hattori et al., 2002; Mielke et al., 2001), but we lack correlations with neuropathology to correctly interpret the data. Diffusion tensor imaging (DTI) provides a technique for the assessment of the integrity or disorganization of white matter tracts, in the temporal subcortical white matter, the cingulum bundle, and the corpus callosum, for example (Takahashi et al., 2002). The technique is still