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APPENDIX IV - USING CLINICAL BRAIN PET FOR EVALUATION OF DEMENTIA

Indication: Changes in memory, language function, personality, or behavior—noted by the patient, friends and family, and/or physician – for which the etiology is not evident, or the symptoms have not been reversed, within a reasonable period of time after initial presentation.
Acquisition Protocol: ^{18}FDG (10 mCi for 2D acquisitions, 3.5-10 mCi for 3D acquisitions) administered i.v., 40 min uptake period with eyes open in dimly lit quiet room; followed by transmission and emission scans, reconstructed with attenuation correction.
Interpretation: A pattern of focal cortical inhomogeneities on PET, all accounted for by areas of infarction on MRI, implies dementia secondary to cerebrovascular disease, which also often affects subcortical structures. A pattern of focal cortical inhomogeneities on PET unmatched by MRI findings is consistent with a neurodegenerative disorder (e.g., Alzheimer's disease, Pick's disease, other frontotemporal dementia, dementia with Lewy bodies, dementia of Parkinson's disease, Huntington's disease, Creutzfeldt-Jacob disease, or progressive subcortical gliosis – see Table on page 1, for differential diagnostic considerations.)

Etiology of Dementia	Regional Hypometabolism Identified by FDG-PET
Alzheimer's Disease	Parietal, temporal and posterior cingulate cortices affected early; relative sparing of primary sensorimotor and primary visual cortex; sparing of striatum, thalamus, and cerebellum.
Vascular Dementia	Hypometabolic foci affecting cortical, subcortical, and cerebellar areas.
Frontotemporal Dementia	Frontal cortex, anterior temporal and mesiotemporal areas affected earlier and/or with greater initial severity than parietal and lateral posterior temporal cortex; relative sparing of primary sensorimotor, posterior cingulate, and visual cortex.
Huntington's Disease	Caudate and lentiform nuclei affected early, with gradual development of diffuse cortical hypometabolism.
Parkinson's Dementia	Similar to Alzheimer's Disease, but less sparing of visual cortex. In early, untreated Parkinson's disease, basal ganglia may appear hypermetabolic.
Dementia with Lewy Bodies	Similar to Alzheimer's Disease, but less sparing of occipital cortex.