Quick Reference

FOR GERIATRIC PSYCHIATRY

The tables in this section are drawn with permission from Spar JE, La Rue A: Concise Guide to Geriatric Psychiatry, 3rd ed. Washington, DC, American Psychiatric Publishing, 2003.

Table 1. Areas to Consider in a Sleep Assessment Interview

Nature of the complaint: what the problem is and when it occurs (e.g., sleep onset, sleep maintenance, early-morning wake up, daytime fatigue, nightmares)

Current sleep-wake schedule

History of sleep complaint (transient disturbance vs. long-standing complaint)

Symptoms of sleep disorders that may not be initially volunteered (e.g., restless legs, periodic limb movements, narcolepsy, gastroesophageal reflux, parasomnias, disruption of sleep-wake schedule)

Symptoms of sleep-disordered breathing (disturbed breathing at night, complaints of snoring, headache on waking, partner sleeps in another room)

Daytime states, routines, activities (sleepiness, fatigue, functioning, mood, activities, satisfaction with daily routines)

Naps, frequency, time of day, length

Sleep hygiene (daytime activity, exercise, sleep environment, activity in bed, diet, use of stimulants/depressants)

History of professional treatment of the sleep complaint and a review of what the client has tried to remedy the sleep problem

Medical/physical problems

Use of prescription and nonprescription drugs

Psychiatric history and mental status review (symptoms of depression, anxiety, thought disorder, other psychological maladjustment)

Stressful circumstances (currently and when sleep problems began)

Information regarding antecedents, consequences, secondary gains, precipitating factors, perpetuating factors

Source: Adapted from Trevorrow T: Assessing Sleep Functioning in Older Adults. In Handbook of Assessment in Clinical Gerontology. Edited by Lichtenberg P. New York, Wiley, 1999, pp 331-350. Copyright @ 1999 John Wiley & Sons, Inc. Reprinted by permission of John Wiley & Sons, Inc.



System	Anatomical Changes With Age	Functional Changes With Age	
Cardiovascular			
Heart	Decreased size, flexibility of collagen matrix; lipofuscin and fat deposition in myocardium; fatty infiltration and calcification of aortic and mitral valves	Impaired left ventricular diastolic filling, reduced β -adrenergic (i.e., chronotropic and inotropic) response to catecholamines, lead ing to decreased peak exercise cardiac index and ejection fraction	
Arteries	Redistribution and molecular rearrangement (cross-linking) of elastin and collagen in arterial walls; calcification	Increased systolic blood pressure	
Respiratory			
Lungs	Enlarged alveolar ducts and alveoli; loss of elasticity Reduced ventilatory capa especially during exercises		
Musculoskeletal	Increased chest wall and joint rigidity; increased kyphosis; degeneration and calcification of cartilage	Same as above	
Gastrointestinal	Some loss of smooth muscle cells of intestine; atrophy of gastric mucosa; increase in gastric pH; some loss of hepatocytes; reduction in hepatic blood flow	Reduced eliminatory efficiency: constipation; reduced metabolism of drugs	
Genitourinary	Loss of renal mass, loss of glomeruli, thickening of basement membrane of glomeruli and tubules, development of tubular diverticula, intimal thickening of arteries, development of afferent-efferent shunts in juxtamedullary glomeruli and obliteration of arterioles in cortical glomeruli; reduced bladder elasticity, especially in women; prostate enlargement in men	Reduced glomerular filtration rate and renal plasma flow; loss of bladder emptying capacity	
Endocrinologic	Atrophy and fibrosis; loss of vascularity; changes may be very minimal	General decline in secretory rate, but resting hormone blood levels may remain constant as clear- ance also declines	
Nervous	Loss of brain weight and volume in most studies; loss of neurons, depending on brain area studied; loss of dendritic arbor with reduced interneuronal connectivity; interneuronal accumulation of lipofuscin and loss of organelles; neurofibrillary degeneration of neurons; accumulation of senile plaques, especially in hippocampus, amygdala, and frontal cortex	ns, depending on brain area studied; loss of dendritic arbor blood flow; reduced metabolism of glucose and oxygen; intellectual changes generation of neurons; accumulation of senile plaques,	
Musculoskeletal	Reduced muscle and bone mass; demineralization of bone; Loss of muscular strengt increased fat in muscles and calcium in cartilage; degeneration of cartilage; loss of elasticity in joints		
Immunologic	Involution of thymus, reduction of the proportion of naïve T cells, increased proportion of activated/memory T cells, decreased expression of IL-2 receptors, decreased cellular proliferative response to T-cell receptor stimulation		
Special senses	Yellowing of lens in eye	Loss of auditory and visual acuity especially night vision	

0	Physical Findings	Behavioral Function	Imaging/Laboratory Findings
Gradual onset and progression; ± family history	Typically none until mid/late stages	Language deficits early (word finding, anomia, fluent apha- sia); clues not helpful with retrieval; visu- ospatial deficits early	Cortical atrophy, ventric- ular enlargement on CT, MRI; temporal/parietal hypometabolism on PET hypoperfusion on SPECT
Abrupt onset, step- wise decline; history of hyper- tension, athero- sclerosis	Neurologic signs and symptoms (e.g., gait abnormalities, falls, incontinence)	Patchy impairment; depression, relative preservation of per- sonality common	Stroke; lacunae in basal ganglia, white matter; periventricular lesions very common, required for diagnosis if focal neurologic signs absent
HIV-positive blood test; gradual onset of cognitive changes	Neurologic signs and symptoms may be present (e.g., ataxia, tremor, frontal release signs)	Forgetfulness, apathy, slowness, poor con- centration common	Elevated CSF protein; mild lymphocytosis may be present; neuroimag- ing nonspecific; HIV usu ally present in CSF
Head injury	Depends on location of injury; dysarthria, hemiparesis common	Memory impairment usually present; impulse dyscontrol, irritability, personality change may be seen; nonprogressive unless head trauma repeated (e.g., in dementia pugilistica)	Depends on location, extent of injury
Dementia in later stages of neuro- logic syndrome	Extrapyramidal signs (e.g., tremor, gait dis- turbance, rigidity, bradykinesia)	Cognitive slowing, poor recall, frontal signs (e.g., perseveration, decreased word list generation, impaired behavioral sequencing); clues helpful with memory retrieval	Subcortical atrophy on CT (e.g., increased inter caudate distance, ventricular enlargement) common; global cerebra metabolism also may be diminished on PET
	wise decline; history of hyper- tension, athero- sclerosis HIV-positive blood test; gradual onset of cognitive changes Head injury Dementia in later stages of neuro-	wise decline; history of hyper- tension, athero- sclerosis HIV-positive blood test; gradual onset of cognitive changes Head injury Dementia in later stages of neuro- logic syndrome Symptoms (e.g., gait abnormalities, falls, incontinence) Neurologic signs and symptoms may be present (e.g., ataxia, tremor, frontal release signs) Extrapyramidal signs (e.g., tremor, gait dis- turbance, rigidity,	Abrupt onset, stepwise decline; history of hypertension, atherosclerosis HIV-positive blood test; gradual onset of cognitive changes Head injury Depends on location of injury; dysarthria, hemiparesis common Dementia in later stages of neurologic syndrome Extrapyramidal signs (e.g., in dementia pugilistica) Extrapyramidal signs (e.g., perseveration, impaired behavioral sequencing); clues helpful with memory



Туре	History	Physical Findings	Cognitive and Behavioral Function	lmaging/Laboratory Findings
Huntington's disease	Autosomal dominant pattern of inheritance; onset generally in 30s–40s; offspring of affected parent 50% likely to be affected	"Fidgeting" progressing to choreoathetosis	Personality change, loss of judgment, irri- tability early, memory impairment later; psy- chosis common	CT or MRI may show striatal atrophy; PET may show striatal hypome- tabolism
Pick's disease	Onset in 50s–60s	Frontal release signs (e.g., snout, grasp reflex) common	Personality change, emotional blunting, deterioration of social skills, language deficits early; memory impairment, dyspraxia later	CT or MRI may show frontal and temporal atrophy; PET may show frontal hypometabolism
Creutzfeldt-Jakob disease	Onset in 40s-60s; 5%-15% have family history; rapid progression (i.e., 1-year course) typical; can be transmitted by corneal trans- plant or contact with infected brain tissue or CSF	Myoclonus early, seizures later; ataxia, visual symptoms, gait disturbance variably present	Nonspecific symptoms (e.g., fatigue, diminished sleep and appetite early; global cognitive deficits late)	CT and MRI may be nor- mal; EEG may show sharp, triphasic synchro- nous discharges at 0.5–2 Hz

Table 4. Screening Laboratory Tests for Evaluation of Depression in the Elderly				
Test	Potential Diagnosis			
Complete blood count with differential white cell count	Folate deficiency anemia, viral infection			
Serum thyroid-stimulating hormone, thyroxine, serum cortisol (A.M. and P.M.)	Hypothyroidism and hyperthyroidism; hypoadrenocorticalism and hyperadrenocorticalism			
Sequential multiple analysis of 18 chemical constituents of blood (SMA-18)	Hypercalcemia, hypokalemia, hyperglycemia			
Urinalysis, blood urea nitrogen	Uremia			
Computed tomography or magnetic resonance imaging of head (as indicated by results of above tests, physical examination)	Brain tumor, stroke			