The Neurological Examination in Aging, Dementia and Cerebrovascular Disease

Part 1: Introduction, Head and Neck, and Cranial Nerves

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Abstract

This four-part series of articles provides an overview of the neurological examination of the elderly patient, particularly as it applies to patients with cognitive impairment, dementia or cerebrovascular disease. The focus is on the method and interpretation of the bedside physical examination; the mental state and cognitive examinations are not covered in this review. Part 1 begins with an approach to the neurological examination in normal aging and in disease, and reviews components of the general physical, head and neck, neurovascular and cranial nerve examinations relevant to aging and dementia. Part 2 covers the motor examination with an emphasis on upper motor neuron signs and movement disorders. Part 3 reviews the assessment of coordination, balance and gait. Part 4 discusses the muscle stretch reflexes, pathological and primitive reflexes, sensory examination and concluding remarks. Throughout this series, special emphasis is placed on the evaluation and interpretation of neurological signs in light of findings considered normal in the elderly.

Introduction

Maximizing diagnostic accuracy in dementia is important given the increasing availability of treatment options for Alzheimer disease (AD), stroke, vascular dementia (VaD), other neurodegenerative disorders such as Parkinson's disease (PD) and depression.[1-3] Despite increasingly sophisticated diagnostic neuroimaging technologies, the clinical examination remains essential to the proper evaluation of a patient with neurological disease.[4] Together with a thorough medical and neurological history, the diagnostic examination aims to identify the presence or absence of neurological disease, refine the differential diagnosis of dementia type and etiology, and screen for medical comorbidities and functional limitations (Table 1). Physical examination is especially important in the geriatric population where an accurate history may be difficult to obtain, atypical presentations of disease are common, and multiple coexisting diseases, disabilities and polypharmacy are the rule.[5] Indeed, the standard neurological examination often needs to be expanded in the elderly to include functional assessments and other components of a "comprehensive geriatric assessment", where appropriate.[6,7] As the saying goes, "you won't find what you don't look for." Many excellent references on the technique of neurological examination and principles of neurological diagnosis are available for the interested reader.[8-13]

Neurological examination should be guided by hypotheses generated from the patient's history. Just as a radiologist's interpretation of imaging studies is improved by prior knowledge of the clinical history, the neurological examination is most reliable when it aims to confirm findings that are suspected from the history.[14,15] Neurological assessment is based on principles of anatomical local- ization that aim to identify lateralized or focal neurological findings suggestive of local pathology, multifocal disease or bilateral cerebral dysfunction. Determination of the site of nervous system dysfunction (e.g., cortical, subcortical, peripheral nervous system) and which systems are involved (e.g., pyramidal, extrapyramidal, cerebellar, sensory, autonomic) can guide diagnosis toward specific neurodegenerative, vascular or multi-system disorders.

Stroke is suggested by focal neurological signs.[16] Thus, finding evidence of stroke on examination can help support a clinical diagnosis of VaD or mixed AD-VaD.[17,18] Non-neurologists are able to make a correct clinical diagnosis of stroke in the majority of cases.[19] In a study of 1,701 patients assessed in a tertiary referral dementia clinic setting, the clinical examination features that best distinguished patients with mixed AD-VaD from those with probable AD were focal neurological findings (15% vs. 3%) and gait disorder (24% vs. 12%); the prevalence of extrapyramidal signs did not differ significantly between these groups (10% vs. 6%).[20]

Repeated assessments over time are useful for charting the course of illness relative to the expected natural history of the disorder.[21] In the absence of specific diagnostic tests for many of the neurodegenerative disorders, follow-up assessments serve to confirm or refute the provisional clinical diagnosis as new signs or symptoms emerge. Re-examination can also suggest whether there are short-term (diurnal or day-to-day) fluctuations in the clinical condition, as seen for example in delirium, dementia with Lewy Bodies (DLB) and PD. The examination of patients with a chronic progressive condition should also aim to identify additional, unrelated pathology

that may develop elsewhere in the nervous system or in other organ systems, and otherwise be overlooked or attributed to progression of the primary neurological disorder.[22] For example, signs and symptoms of subdural hematoma, cerebral tumour, peripheral neuropathy or myelopathy may go unnoticed in a patient who carries a primary diagnosis of dementia.

Normal Aging and the Neurological Examination

Knowledge of the normal limits of the physical examination in healthy elderly individuals is essential for proper interpretation of the neurological examination (Table 2, page 38). Critchley's classic 1931 paper drew attention to neurological signs that accompany aging,[23] and since then several excellent reviews of this topic have been published.[5,24-28] Many physiological age-related changes occur in the nervous system.[28-32] However, the frequency of these normal "age-related" neurological signs, detectable on routine clinical examination, depends on the definitions of normality and disease.[33-35] Thus, prevalence figures vary widely among studies due to differences in study populations, examination technique, items tested, scoring of abnormal responses and the degree to which medical and neurological diseases are excluded. Many examination findings that show increasing prevalence with aging may reflect common age-associated diseases (e.g., stroke, diabetic neuropathy) rather than normal age-related change.[36]

One study involving 537 community-dwelling individuals 75 years or older suggested that the main neurological signs that can be most confidently attributed to aging per se, rather than disease, are impaired vibration sense, limitation of upward gaze and bradykinesia.[36] Similarly, Odenheimer *et al.* conducted a community survey of 467 individuals aged 65 or over and compared the prevalence of neurological signs in the entire sample to a subgroup of 272 subjects free of medical or neurological disease.[37] They concluded that, although neurological abnormalities are increasingly common with age, most are due to underlying disease and only one-third to one-half can be attributed to aging alone.[37] Of the items tested in this study, primitive reflexes and gait abnormalities were the main age-related neurological findings; muscle tone and strength in the arms, heel taps, muscle stretch reflexes, proprioception and Romberg's sign did not show significant age-related changes in the healthy elderly subjects.[37] Jenkyn *et al.* examined 2,029 healthy elderly subjects aged 50–93 and found age-related increases in the frequency of primitive reflexes, limitation in vertical gaze and abnormal smooth pursuit eye movements, especially in subjects over age 70.[38] Kaye *et al.* characterized the physical findings of the "optimally healthy oldest old", a highly-select group of community-dwelling volunteers aged 85 years or older (mean age 89 years) who were cognitively intact, in excellent health, took no major medications, and were carefully screened to exclude medical or neurological disease.[39] They found that the very old have a high prevalence of neurological signs that cannot be considered pathologic, and their results provide an indication of the "best" neurological function to be expected in the ninth and tenth decades.[39] The find-

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Table 1

Goals of the Physical Examination in Persons with Dementia

Assist in diagnosis and differential diagnosis

Distinguish abnormal physical signs from normal aging of the nervous system

Screen for secondary and reversible causes of dementia[106-109]

Identify medication toxicity

Identify conditions that require prompt intervention

Screen for cardiovascular and cerebrovascular risk factors amenable to preventive treatment

Direct laboratory and imaging investigations

Screen for common medical comorbidities, impaired vision and hearing,[109] dehydration and malnutrition

Detect comorbid neurologic conditions unrelated to the neurological presentation

Help stage the severity of illness, monitor disease progression, and assess prognosis based on initial and longitudinal observation

Assess functional capacity, independence in activities of daily living and safety

Assess gait and mobility, identify individuals at risk for falls or in need of walking aids

Recognize patients who may be unsafe to operate a motor vehicle[75]

Guide referrals to appropriate consultants, nurses, paramedical specialties, community agencies, and to assist in long-term care planning

Establish a therapeutic physician-patient relationship[8]

Provide opportunities for education, counseling and liaison with family members and caregivers

ings that best distinguished the aging changes of this healthy elderly group from healthy younger controls (aged 65–74) were deficits in balance (impaired tandem gait and one-leg standing balance with eyes closed), impaired olfaction and impaired ocular pursuit.[39]

General Examination

Dementia may be the presenting feature or an associated manifestation of a wide variety of systemic and metabolic diseases affecting the nervous system. The examiner must remain alert to signs that suggest a secondary or reversible cause of the dementia. In addition, comorbidity is common and knowledge of the most frequent comorbid diseases in the elderly will help guide the clinician.[40] The average number of associated diseases in patients with AD is three and in VaD it is five.[40]

The examination should begin at first contact with the patient. On general inspection, observe the patient's appearance, body habitus, grooming, hygiene, dress and mannerisms. Note the level of alertness and attentiveness, affect and behaviour, insight and concern, and determine the presence of psychotic features. Look for stigmata of acute or chronic systemic disease such as loss of weight and muscle bulk, pallor, jaundice, organomegaly, signs of thyroid disease or other endocrinopathy, lymphadenopathy, clubbing, as well as signs of chronic alcohol, drug or tobacco abuse. Patients with dementia are at risk for dehydration, weight loss, malnutrition and pressure sores.[41-44] Pay particular attention to the hands, skin, face and eyes as markers of systemic disease.[45-47]

Suspicions of certain neurological disorders can be aroused immediately by visual recognition (spot diagnosis or "augenblickdiagnose").[48] For example, a masked face and reduced eye blink suggest parkinsonism. A wide-eyed, astonished or startled-looking facial expression with lid retraction, furrowed brow, deepened nasolabial folds and facial dystonia suggest Progressive Supranuclear Palsy (PSP).[49] Take note of how the patient enters the room, including stance, gait, posture and mobility when sitting, standing, turning and going through doorways. Note any involuntary movements (dyskinesias) such as tremor, myoclonus, tics, dystonia or chorea.[50] The posture and tone of the neck can reveal characteristic abnormalities, such as nuchal extension (retrocollis) in some patients with PSP, and forward flexion (antecollis) in multiple system atrophy. Inspection of the size and shape of the head, and palpation of the scalp and skull may reveal signs of traumatic injury or remote neurosurgical procedures (burr hole, ventricular shunt) not apparent on history taking.

Neurovascular Examination

In patients with suspected cerebrovascular disease, a focused cardiovascular history should be taken. The history should elicit vascular risk factors such as hypertension, diabetes, smoking, hyperlipidemia, history of stroke or transient ischemic attacks affecting the brain or retina, and presence of coronary artery disease, valvular heart disease, atrial fibrillation, or other cardiac or peripheral vascular disease. The examiner should document heart rate and rhythm, blood pressure lying and standing, and the cardiac examination. It is essential to detect hypertension, including isolated systolic hypertension, as well as atrial fibrillation, as these are the most important treatable stroke risk factors and will require treatment in most cases.

Auscultation for vascular bruits over the head, neck (carotid, vertebral and subclavian arteries) and orbits is an essential part of the neurovascular examination.[8,12] Detection of a carotid bifurcation bruit has special significance: if the patient has had recent symptomatic carotid territory ischemia and ipsilateral high-grade (>70%) internal carotid stenosis, carotid endarterectomy is indicated for the prevention of recurrent stroke in suitable patients.[51] However, if carotid stenosis is moderate

Table 2
Common Neurological Findings in "Normal" Elderly
Decreased olfaction and taste
Decreased visual acuity (presbyopia)
Decreased pupil size and reactivity
Limitation of conjugate vertical gaze (particularly upward gaze) and convergence
Smooth ocular pursuit becomes saccadic
Hearing loss (presbycusis)
Decreased muscle bulk, without visible fasciculations
Mild increase in muscle tone
Decreased muscle strength (but not generally apparent on examination)
Decreased speed and coordination of movement (reaction time, speed of performing a task, coordination and dexterity)
Gait changes (slightly flexed posture, reduced stride length and gait velocity)
Postural instability (presbystasis)
Decreased muscle stretch reflexes (loss of ankle jerks)
Increased prevalence and number of primitive reflexes
Decreased vibration sensation in distal lower extremities

or asymptomatic, the relative benefits of surgery are much lower and more controversial.[51] Ideally, a symptomatic carotid bruit should be promptly evaluated. Depending on the acuity and the specifics of the situation, this may be done non-invasively using carotid Doppler, magnetic resonance or CT angiography to determine the degree of stenosis, or it may mandate timely intra-arterial angiography with a view to surgical intervention or angioplasty.

Inspection and palpation of the facial pulses can also provide clues to the integrity of the carotid circulation. For example, a distended, pulsatile superficial temporal artery may imply ipsilateral internal carotid artery occlusion with increased collateral flow through external carotid artery branches. Conversely, facial pulses are diminished with common carotid or external carotid occlusion. It is important to examine the scalp arteries if giant cell arteritis is suspected, especially in elderly patients with unexplained headache, visual disturbance, stroke or cognitive impairment. A pulseless, distended, hard and tender superficial temporal artery would strongly suggest temporal arteritis.

The peripheral vascular exam may indicate generalized atherosclerotic disease, revealed by auscultation for femoral and abdominal bruits, examination for abdominal aortic aneurysm, palpation of pedal pulses, and assessment of skin temperature and trophic changes in the distal extremities. Bilateral palpation of the radial pulses can detect a pulse delay due to subclavian stenosis. In this case, blood pressure measurements in each arm may differ, reflecting a subclavian steal syndrome (vertebrobasilar ischemia due to diversion of blood from the vertebral artery to a stenotic subclavian artery when the arm is active).

In selected patients, it is prudent to check for autonomic dysfunction by inquiring about orthostatic presyncope/syncope, incontinence, urinary retention, impotence and alterations in sweating. Blood pressure and pulse should be measured in the lying, sitting and standing positions. Orthostatic hypotension is defined as a drop in systolic blood pressure of at least 20 mmHg, or diastolic blood pressure of 10 mmHg, within three minutes of standing.[52] Failure of the pulse to increase in response to postural hypotension suggests a neurogenic cause rather than volume depletion. Neurogenic autonomic dysfunction can be a prominent feature in multiple system atrophy and advanced PD.[52,53]

Examination of Cranial Nerves
Olfaction

On routine examination, testing of olfaction is often omitted but may be useful in patients with cognitive impairment. Diminished smell occurs with increasing age, and more than 50% of those aged 65 to 80 have major olfactory impairment.[54] Olfactory sensation is reduced early in AD and has been reported to help in differentiating AD from major depression.[55-60] It has been suggested that olfactory dysfunction might be a marker of early AD and may help predict the development of AD in patients presenting with mild cognitive impairment.[61,62] Patients with multi-infarct dementia have less olfactory impairment compared to patients with AD. Anosmia is one of the earliest signs in PD, but olfaction is preserved in parkinsonism-plus syndromes; hence, anosmia may help distinguish PD from PSP.[55,63-69] Anosmia occurs with several other neurodegenerative diseases, prior head trauma, chronic sinus disease, long history of smoking and, occasionally, is an early sign of frontal lobe or skull base tumour.

Neuro-ophthalmologic Examination

It is important to screen for visual problems in the elderly, as visual impairment may not be reported by the patient, may contribute to functional difficulties and may represent a potentially correctable problem.[70,71] Using a visual acuity testing card in a geriatric clinic, 36% of 202 patients with impaired vision were identified, many of whom had treatable conditions.[72] The most common age-related change is presbyopia—a decreased ability to focus on near objects due to impaired accommodation of the lens. In seniors aged 75–85 in the Framingham study, 46% had cataracts, 28% had macular degeneration, 7% had glaucoma and 7% had diabetic retinopathy.[73] Cataracts sufficient to impair vision rise in frequency from 1% in the fifth decade to 100% in the ninth decade.[74] On examination, the best-corrected visual acuity should be recorded in each eye, using the Snellen chart for distance vision or a near vision pocket card. Acuity worse than 20/40 is usually associated with some degree of functional visual impairment in daily activities (e.g., reading newspaper), 20/50 acuity is a minimum recommended standard for driving in some jurisdictions,[75] and acuity worse than 20/200 represents legal blindness. Physicians should have a low threshold for referral to an ophthalmologist. Even in very old age, 20/20 acuity should be the goal and is often attainable with correction, depending on the nature of the visual loss.

Bedside assessment of visual fields is performed by confrontation testing (finger counting or hand motion) in the four quadrants of each eye separately.[76-78] For detection of more subtle field loss, comparison of the clarity of the examiner's two hands or of two red objects held in front of each eye in the upper and lower quadrants is recommended. This allows a subjective visual field loss not apparent with finger counting or movement to be appreciated. A homonymous visual field defect (hemianopia or quadrantanopia), which may not be reported by the patient, is an important finding that may indicate a contralateral posterior hemispheric lesion involving the optic radiations or occipital region, often due to stroke. For example, a contralateral temporal lobe lesion can cause a field defect in the upper quadrant, whereas disruption of the optic radiations in the parietal lobe causes a field defect in the lower quadrant.

Fundoscopy can reveal clues to visual loss (cataracts, macular degeneration, glaucoma and optic atrophy), as well as optic disc edema, due to raised intracranial pressure (papilledema). As

the eye is a marker for cerebrovascular disease, fundoscopic examination can also detect central retinal artery or branch occlusions, retinal emboli, signs of diabetic or hypertensive retinopathy, ischemic optic neuropathy and signs of chronic ocular ischemia.[79,80]

Pupil size, symmetry and reactivity should be documented. With aging, there is a 20–40% decrease in pupil size.[81] According to one study, pupil size and reactivity are further reduced in AD.[82] Up to a quarter of the population have mild physiologic anisocoria (1mm or less). Pupillary abnormalities such as Horner's syndrome (unilateral small pupil and ptosis) can suggest prior brainstem stroke or carotid artery disease with interruption of sympathetic fibres. Argyll-Robertson pupils, small pupils that react to accommodation for near objects but not to light, are classically associated with neurosyphilis (at one time, the most common cause of dementia). A relative afferent pupillary defect (Marcus Gunn phenomenon) is an objective sign of an afferent visual defect usually indicative of optic nerve disease (the pupil of the affected eye paradoxically dilates instead of constricting to light on the swinging flashlight test).

Abnormalities of eye movements can be helpful in the diagnosis of certain neurodegenerative disorders and stroke syndromes. For example, a supranuclear vertical gaze paresis is a hallmark feature of PSP and is part of the clinical diagnostic criteria for this disorder.[49,83,84] Early on, there is a characteristic slowing of vertical saccades despite full motility (downward saccades usually affected first); with advancing disease this can progress to complete ophthalmoplegia. Supranuclear gaze palsy may also be seen in corticobasal degeneration, multiple system atrophy and PD.[85] Limitation of vertical gaze and convergence deficits are common age-related changes.[23,38,39,81,86-88] Chamberlain measured vertical gaze in 367 subjects aged five–94 years and found a progressive linear decline in upward gaze, but not downward gaze, with each decade of advancing age.[88] Children aged five–14 years have 40 degrees of ocular elevation; this decreases to 30 degrees by ages 45–54 years, 22 degrees by ages 65–74 years, and less than 17 degrees over age 75 years (with many elderly unable to elevate 10 degrees).[88] Jenkyn *et al.* considered an excursion of 5mm or less for upgaze (35 degrees) or 7mm or less for downgaze (50 degrees) to be abnormal.[38] Using this definition, age-related vertical gaze restriction was found in less than 6% of normal individuals under age 70, in 15% aged 70–74, and in nearly 30% in those 75 or older in one study,[38] and in 65% of the healthy 85 or older population in another study.[39] Benassi *et al.* reported an age-related restriction of conjugate upward gaze in 16–21% of elderly subjects,[86] and Waite *et al.* reported this finding in 20% of their elderly cohort.[36] Saccadic pursuit ("cogwheeling" of pursuit eye movements) increases with aging and also correlates with cognitive impairment.[38,89,90] Patients with dementia may demonstrate a "visual grasp reflex", an inability to suppress reflexive saccades toward a visual target when instructed to look in the opposite direction, and this has been attributed to frontal lobe degeneration.[91] Square-wave jerks, brief involuntary horizontal saccadic oscillations that move the eyes away from fixation, are seen in the majority of patients with PSP, but can also be detected occasionally in multiple system atrophy, AD and normal individuals.[92] Nystagmus may be physiological or pathological and a wide variety of patterns are described.[93] Upbeating or downbeating nystagmus usually signifies central (i.e., brainstem or cerebellar) rather than peripheral vestibular pathology. Blepharospasm, lid levator inhibition, or apraxia of eye opening have been described as features in PSP, corticobasal degeneration and PD.

Visual perceptual disturbances can be features of dementia with occipitoparietal cortical involvement[94] (e.g., posterior cortical atrophy, often due to atypical AD) or bilateral posterior circulation territory ischemic stroke.[95] Balint's syndrome, for example, consists of problems looking toward or reaching toward objects (oculomotor apraxia and optic ataxia), and difficulty grasping the meaning of a complex scene with ability to see only the details, i.e., missing the forest for the trees (asimultanagnosia).[95] Other, related abnormalities that can be detected on examination include the inability to identify objects (visual object agnosia) or recognize faces (prosopagnosia).[95] Visual illusions or hallucinations may be a symptom of the dementia itself,[96] drug toxicity, or a manifestation of sensory deprivation due to poor vision (Charles Bonnet syndrome).[95,97]

Lower Cranial Nerves

Facial sensation and motor function can be assessed by examining the trigeminal and facial nerves. Inspect the face for symmetry, drooping of the corner of the mouth, flattening of the nasolabial fold, widening of the palpebral fissure and eyelid closure. Test the ability to close the eyelids against resistance, and the ability to raise the eyebrows to symmetrically wrinkle the forehead. Have the patient blow out the cheeks, whistle, purse the lips and show the teeth. A unilateral upper motor neuron facial palsy suggests stroke or other lesion in the contralateral cerebral hemisphere. Typically, the weakness affects the contralateral lower face (the upper third of the face is spared because of bilateral supranuclear innervation) for voluntary facial movements, but there is preservation of involuntary, emotionally-mediated movement, which has separate supranuclear control. In peripheral lesions (lower motor neuron lesions) of the facial nerve (e.g., Bell's palsy), the entire face on one side is weak, and both voluntary and involuntary movements are affected. Be wary of a history of "Bell's palsy" in the elderly as this condition can be mimicked by facial palsy due to mild stroke.[98]

Testing hearing ability adds to the overall functional assessment and, if impaired, may be an indication for referral.[99,100] Thirty percent of people aged 65 and over have significant hear-

ing impairment.[101] Presbycusis, bilateral symmetric high-frequency hearing loss, results from age-related loss of cochlear hair cells. Hearing aids can improve quality of life.[99,100] The examiner can screen for high-frequency hearing loss using the "whisper test",[102] in which words are whispered in one ear while hearing is masked in the other ear by rubbing on a card covering that ear. A whispered word can normally be identified at three feet. Unilateral hearing loss is abnormal. Tuning fork tests can distinguish between sensorineural hearing loss (air conduction is greater than bone conduction; the Weber test is midline) from conductive hearing loss (bone conduction is greater than air conduction; Weber test lateralizes to the affected ear). Otoscopic examination can exclude impacted cerumen or ruptured tympanic membrane as a cause for conductive hearing loss.

Abnormalities of tongue protrusion can be caused by hemispheric or brainstem stroke, but mild asymmetries are frequently normal. Inability to initiate tongue protrusion can be a sign of buccofacial ideomotor apraxia, associated with left hemisphere stroke. Inability to sustain tongue protrusion is a characteristic manifestation of the motor impersistence seen in Huntington's disease, and may also be seen with right hemisphere lesions.[103] Inspection of the tongue may reveal atrophy and fasciculations in motor neuron disease.

Dysarthric speech may be a feature in specific neurological disorders. Some examples include the slow, spastic, strained speech of bilateral upper motor neuron lesions (e.g., motor neuron disease or bilateral strokes), the hypophonic monotonous output of PD, the harsh strained voice of PSP, and the staccato scanning dysarthria with altered volume regulation in cerebellar disease.

Pseudobulbar palsy results from bilateral upper motor neuron lesions affecting the bulbar musculature, producing dysarthria, dysphagia, hyperactive gag reflex, hyperactive jaw jerk (elicited by tapping the chin with the mouth slightly open), and brisk facial muscle stretch reflexes (elicited by the examiner tapping his or her finger lightly at the corner of the mouth).[104] Such patients often have an accompanying "pseudobulbar affect" with "emotional incontinence" such as involuntary pathological crying or laughing, a very disturbing symptom that may be responsive to treatment with selective serotonin-reuptake inhibitors.[105] ◆

Part 2, focusing on motor examination of normal aging and in disease, will appear in next month's issue of *Geriatrics & Aging*.

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