The Neurological Examination in Aging, Dementia and Cerebrovascular Disease

Part 2: Motor Examination

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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 (featured in the September issue of Geriatrics & Aging) began with an approach to the neurological examination in normal aging and in disease, and reviewed components of the general physical, head and neck, neurovascular and cranial nerve examinations relevant to aging and dementia. Part 2, featured here, 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, and 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

With normal aging, motor function declines and leads to reduced strength and speed of movements, although these changes tend to be mild and are symmetrical.[1,2] There is a reduction in the number and size of muscle fibers (especially type II, fast twitch) as well as a reduction in the number of peripheral nerve fibers and conduction velocity.[3-6] For example, the sural nerve of an 80-year-old contains half the number of large myelinated axons compared to a 20-year-old. On clinical examination, careful comparisons between right and left sides for asymmetry of power, tone and reflexes is critical for distinguishing normal aging from true neurological abnormalities—*asymmetry is almost always suggestive of pathology*.

Motor examination comprises assessment of muscle bulk, tone, power, inspection for fasciculations, assessment for features of a hypokinetic or hyperkinetic movement disorder, evaluation of limb and trunk coordination, gait, balance and functional mobility. In the evaluation of a patient with dementia, motor examination will focus primarily on identifying motor dysfunction due to central nervous system pathology (pyramidal, extrapyramidal and cerebellar systems). In particular, one should search for features of an upper motor neuron syndrome that may point to the presence of a multi-infarct state. Upper motor neuron[7] signs are not normally a feature of Alzheimer disease (AD) until the terminal stages. Lower motor neuron[7] findings in a peripheral nerve distribution may indicate a mononeuropathy (e.g., carpal tunnel syndrome, ulnar or peroneal pressure palsy, vasculitic neuropathy). Polyneuropathy may be an important clue to some secondary causes of dementia (e.g., hypothyroidism, B12 deficiency), unusual neurodegenerative conditions (e.g., metachromatic leukodystrophy, Machado-Joseph disease) or concomitant disease (e.g., diabetes). Muscle atrophy and fasciculations suggestive of motor neuron disease can be seen in a subgroup of patients with Fronto-Temporal Dementia and are associated with a worse prognosis.[8]

Muscle tone, the resistance of a muscle to passive movement, is rated as normal, increased or decreased. Increased muscle tone may take one of three forms: spasticity, rigidity or paratonia. Spasticity, a velocity-dependent "clasp-knife" response that affects primarily flexors in the arm and extensors in the leg, is indicative of upper motor neuron pathology (e.g., prior stroke).[9] It is often best appreciated by brisk supination of the relaxed forearm or by rapid extension of the elbow or knee. Rigidity, a sustained "lead pipe" resistance throughout the range of motion affecting flexor and extensor muscles equally, is a feature of parkinsonism. There may be a cogwheel component and the hypertonia is frequently increased by reinforcement maneuvers (e.g., opening and closing the fist of the opposite arm). Paratonia (gegenhalten), a progressively increasing and irregular resistance to passive movement in any direction, is a pathological finding occurring in dementia patients, possibly reflecting bilateral frontal lobe dysfunction. The hypertonia is proportional to the amount of force applied, and often becomes more pronounced by instructing the patient to relax. These patients actively resist changes in limb position and appear unable to relax the limbs (e.g., the patient's arms remain elevated after being released by the examiner's hand, even after instructions to relax).[10,11] Gegenhalten usually occurs in the lower limbs and is often classified as

Table 1

Movement Disorder Phenomenology[26,30]

Hypokinetic Disorders		Example
Parkinsonism	Characterized by akinesia (lack of movement), often accompanied by rigidity	Parkinson's disease, parkinsonism-plus disorders, drug-induced parkinsonism
Hyperkinetic Disorders (Dyskinesias)		
Tremor[40]	Rhythmic oscillation of a body part	
	Resting tremor—tremor present when body part is fully at rest	Classical tremor of Parkinson's disease (when severe, parkinsonian tremor can also have a postural and kinetic component)
	Action tremor Postural tremor—tremor present when trying to hold a body part still against gravity	Essential tremor, enhanced physiological tremor
	Kinetic tremor—tremor present during voluntary movement	Intention tremor of cerebellar disease
Chorea	Irregular, random, flowing movements	Levodopa-induced choreiform dyskinesias, neuroleptic-induced tardive dyskinesia, Huntington's disease
Dystonia	Sustained muscle contractions causing twisting, repetitive movements or abnormal postures—may be focal (e.g., torticollis), segmental, multifocal or generalized	
Athetosis	Slow, writhing, twisting, dystonic movements of distal limbs	
Ballismus	Rapid, large-amplitude, flailing movements	Hemiballism due to basal ganglia stroke
Myoclonus	Brief, shock-like muscle jerks—may be spontaneous or stimulus-sensitive;	Hiccough (a physiological diaphragmatic myoclonus)
	Negative myoclonus—sudden loss of postural tone	Asterixis
Tics	Repetitive, stereotyped movements or vocalizations (simple or complex) associated with an inner urge to make the movement; may be temporarily suppressed by the patient	Tourette syndrome

a cortical disinhibition sign; in the upper limbs an association with limb apraxia has been reported.[11] In a community survey, the prevalence of marked paratonic rigidity was 32% in demented patients and 1% in normal elderly subjects.[12]

Muscle strength testing aims to identify patterns of weakness by looking for asymmetries between the right and left sides, upper and lower extremities, and distal and proximal distributions.[13] The six-point Medical Research Council (MRC) grading scale is commonly employed by neurologists since it allows comparisons among different observers and over time: 5 = normal power; 4 = weak movement against resistance (grades 4+, 4 and 4- refer to movement against strong, moderate and slight resistance); 3 = can move against gravity but

Table 2
Unified Parkinson's Disease Rating Scale (UPDRS): Motor Assessment

Speech
Facial expression
Resting tremor
Action tremor
Rigidity
Hand movements (rapid large-amplitude tapping of the index finger with the thumb; opening and closing of the hand)
Rapid alternating (pronation-supination) movements of the hands
Rapid tapping of the heel on the ground
Trying to arise from a chair with arms across the chest (i.e., without pushing off the arms of the chair)
Posture
Gait
Postural stability on the backwards pull test
Body bradykinesia/hypokinesia

not against resistance; 2 = can move but not against gravity; 1 = muscle contraction but no joint movement; 0 = no contraction.[14] Descriptive terms are also used, such as mild but definite weakness, moderate paresis, severe paresis or complete paralysis.[13] Upper motor neuron (pyramidal, corticospinal) weakness typically affects the extensor muscles of the upper limb more than the flexors, the lower limb flexor muscles more than the extensors, and distal more than proximal muscle groups, particularly finger dexterity. This pattern contrasts with the weakness of peripheral neuropathy, which typically follows a myotomal or radicular distribution and, if diffuse, often affects distal more than proximal muscles, or the weakness from a myopathic process, which is most often proximal and symmetric. Hemiparesis due to stroke is recognised by its unilateral distribution and accompanying features of the upper motor neuron syndrome: spasticity, hyperreflexia, clonus and extensor plantar response (Babinski sign).

Mild or recovered hemiparesis may only be evident with special testing to elicit subtle signs of corticospinal tract dysfunction. Several such tests have been described:

- downward drift or pronator drift of the outstretched arm with eyes open or closed;
- downward drift of the elevated leg when lying supine or prone;
- the digiti quinti sign[15] (abduction of the little finger on the side of a mild hemiparesis when arms are outstretched and palms down);
- Souques's sign[16] (abduction of all the fingers on the hemiparetic side);
- the Hachinski upgoing thumb sign[17] (extension of the thumb of the affected hand when the arms are outstretched and palms facing each other);
- asymmetry on the forearm rolling test[18] (when rotating both forearms around each other in a circular motion);
- asymmetry on rapid finger tapping.[19]

Weaver recommends a 30-second maneuver that incorporates many of the above tests: the patient hold both arms outstretched, palms up and fingers together for 15 seconds, then fingers apart for 15 seconds. The examiner watches for abduction of the little finger, spreading of the fingers and pronation or drift of the arm (pronator drift, when present, always occurred within 30 seconds).[20] The forearm rolling test is reported to have greater sensitivity (87%) than upper extremity drift (79%) or the Babinski sign (45%) for detecting contralateral hemispheric dysfunction in patients with a proven cerebral lesion.[18] Rapid finger tapping on a hard surface is an excellent way to see (and hear) mild motor impairment.[19] As the corticospinal tract is especially important for controlling fractionation of finger movements, slowing of finger tapping speed and impaired finger dexterity are common residual signs of a motor stroke.[21]

Movement Disorder Symptomatology

A variety of movement disorders may appear as part of certain neurodegenerative dementias or as co-existing disorders. Their recognition may have implications for diagnosis, prognosis and treatment (Table 1).

Parkinsonism is common in the elderly.[22,23] In a population-based study of 467 community dwelling seniors, the prevalence of parkinsonism was 15% in those aged 65–74, 30% in those 75–84, and 52% in those aged 85 and older.[23] Parkinsonism may develop in patients with primary degenerative dementia (AD, vascular dementia or Lewy body disease), may be drug-induced (e.g., neuroleptic exposure)[24,25] or may represent idiopathic Parkinson's disease (PD) or an atypical parkinsonian disorder (e.g., progressive supranuclear palsy, corticobasal degeneration, multiple system atrophy).[26-28] The prevalence of true idiopathic PD is approximately 3% in the over-65 population, and increases with age.[29]

The classic parkinsonian features are characterized by the acronym "TRAP": tremor, rigidity, akinesia/bradykinesia and postural disturbances.[26,28,30] Clinical diagnosis of PD usually requires two of bradykinesia, rigidity or tremor, and

diagnosis is supported by asymmetry of symptoms at onset, unilateral resting tremor, levodopa responsiveness and absence of atypical features (see below).[27,31,32] Neuroleptic-induced parkinsonism and some other akinetic-rigid syndromes are often bilaterally symmetric at onset, although many exceptions exist. Postural instability is common in the elderly and is usually a feature of late, not early, PD. Bradykinesia refers to slowness in initiation and execution of movements, usually with progressive reduction in amplitude of movements (hypokinesia), early fatigability and a fading out of repeated movements (diminuendo). Rigidity in PD is manifest predominantly in the limbs, in contrast to progressive supranuclear palsy (PSP) where axial rigidity usually predominates.

To screen for extrapyramidal signs, we recommend performing a brief examination using the motor section of the Unified Parkinson's Disease Rating Scale (UPDRS).[33,34] A video instructional tape of this examination is available[35] and rating forms can be accessed online (www.wemove.com). This rating scale takes only a few minutes to administer and includes assessments that quantify the motor aspects of Parkinson's disease (Table 2).

The UPDRS also includes an activities of daily function questionnaire that can be a useful guide for eliciting features of parkinsonism, such as difficulties with speech, salivation, swallowing, handwriting, cutting food, using utensils, dressing, hygiene, turning in bed, gait, falling, freezing while walking and tremor. It also includes a rating for dyskinesias.

Normal elderly individuals can occasionally be mistaken for having PD as some aging changes resemble parkinsonism, particularly bradykinesia/hypokinesia and changes in posture and gait.[36] Prettyman confirmed that extrapyramidal signs are a common age-related phenomenon in cognitively intact, community-dwelling elderly; over 50% of seniors aged 80 or over had at least one extrapyramidal sign.[37] Similarly, in another survey nearly 50% of healthy elderly individuals in the community and nearly all geriatric day hospital patients had at least one extrapyramidal sign on examination.[38] The mild extrapyramidal signs of normal aging are usually symmetrical and do not respond to levodopa,[39] whereas in PD, symptoms begin unilaterally in most patients and respond to levodopa.[31,32] In the elderly, posture becomes flexed at the neck and trunk but knees and elbows are straight; in PD, there is flexion of the neck, trunk, hips, knees and elbows.[29] Resting tremor—a tremor that is maximal at rest and attenuates with action—is not usually seen as part of normal aging *per se*, and its presence suggests PD or other parkinsonian disorder.[29,40] A classical 3-5Hz pill-rolling resting tremor is relatively specific for PD and is reported to be the most reliable sign for the diagnosis of PD in the elderly, although it may also be seen in atypical parkinsonian disorders.[29]

In contrast to the parkinsonian resting tremor, some elderly individuals have essential tremor[40] (formerly called benign essential tremor), a disorder frequently confused with PD or incorrectly considered as a "senile tremor". Essential tremor is not a feature of normal aging and may become a source of functional disability for the patient. It is characterized as a 4–12Hz postural or kinetic tremor, seen best in the outstretched arms bilaterally and often worsening with goal-directed action.[41,42] This tremor can be brought out by having the patient reach for a target, pour a glass of water or try to bring a drink to the mouth without spilling.

A handwriting sample is an important component of the examination that will characteristically reveal the bradykinesia and micrographia in parkinsonism, and will highlight the action tremor in essential tremor. The examiner can keep a record of the patient's handwritten sentences, signatures and drawings of multiple loops across the page to compare changes over time and in response to treatment. The Archimedes spiral, in which the patient attempts to draw a spiral with each hand without crossing the lines, is a particularly useful technique for recording action tremor.

In AD, extrapyramidal features of bradykinesia and rigidity (often paratonic) commonly develop in patients with moderately severe and severe dementia, and have been associated with greater cognitive and functional impairment than in AD patients without parkinsonism.[43-55] Tremor is uncommon in AD. The frequency of parkinsonian signs increases with progression of AD.[44,46,47,55] In one study, over a three-year follow-up the prevalence of bradykinesia increased from 39% to 72% and rigidity increased from 11% to 61%.[44] In another longitudinal AD study, parkinsonism developed in 23% after 66 months compared to only 5% in the control group—the emergence of parkinsonism in AD was nearly five times that expected in the general population.[46] Higher frequencies of extrapyramidal signs are reported in series that include patients taking neuroleptic medication.[44,56] The development of extrapyramidal features in AD has been associated with faster progression of disability and predictive of poorer survival.[22,44,46,57-59] Some authors suggest that these patients represent a subtype of AD with a more rapid course.[54,59-61] These patients are also at greater risk of developing extrapyramidal side effects if exposed to neuroleptic medications.

When parkinsonism is an early or prominent feature in a patient with dementia, one should consider the diagnosis of dementia with Lewy bodies (DLB).[62,63] It is possible that some AD patients with extrapyramidal features in earlier studies may in fact have had DLB, prior to the recognition of DLB as a separate entity or as a pathology concomitant with AD. In autopsy series, parkinsonism was present in 75% of patients with DLB (vs. 20% of AD patients) and represents one of the core diagnostic features of this disorder, along with cognitive fluctuations and visual hallucinations, according to recent consensus criteria.[62,64] The parkinsonism in DLB can resemble PD but usually has more severe rigidity, less resting tremor and less asymmetry at onset.[65] In a

patient with an akinetic-rigid syndrome, the presence of any one of four features—absence of tremor, myoclonus, no response to levodopa or no perceived need to treat with levodopa—was 10 times more likely in DLB than in PD.[66]

Other movement disorder symptomatology may suggest specific dementia syndromes, such as chorea in Huntington's disease, dystonia in corticobasal degeneration, asterixis in metabolic encephalopathy and myoclonus in Creutzfeld-Jacob disease. Myoclonus (sudden brief shock-like muscle jerks) occurs in AD and DLB and can generate diagnostic confusion between these conditions and Creutzfeld-Jacob disease.[67,68] The presence of myoclonus in AD is less common (5–10% of patients) than extrapyramidal signs in the early stages, but increases with progression of disease and has been associated with a younger age of onset and more severe dementia.[54,55,69-71] Stimulus-sensitive myoclonus in the hands or face is also seen in multiple system atrophy.[72]

The motor features of corticobasal degeneration are characterized by a progressive asymmetric-rigid syndrome, limb dystonia, focal myoclonus, ideomotor apraxia and alien limb phenomenon.[73-77] The presenting complaint is often clumsiness, impaired dexterity and rigidity in one arm. Dementia can also be the initial presentation of this disorder.[78]

Tardive dyskinesia occurs in about a quarter of patients chronically treated with neuroleptics for psychotic disorders, though this may become less frequent with the arrival of the newer atypical neuroleptics.[26,79-81] These are recognizable as repetitive stereotypic orofacial and tongue movements such as lip smacking, puckering, chewing, tongue protrusion and pushing the tongue against the inside of the cheek.[26]

Huntington's disease is an autosomal dominant disorder characterized by a triad of generalized choreiform movements, psychiatric/behavioural disturbance and dementia.[26,28] In the early stages of the movement disorder, patients may appear excessively animated, restless, fidgety and clumsy, with characteristic eye movement abnormalities and a wide-based, "disco-dancing" gait.

A variety of movement disorders may occasionally follow stroke affecting the basal ganglia/thalamic/subthalamic regions, including hemichorea, hemiballismus, hemidystonia, tremor and hemiparkinsonsism.[82] ◆

Part 3, focusing on assessment of coordination, balance and gait, will appear in next month's issue of *Geriatrics & Aging*.

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