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GAIT AND ITS ASSESSMENT IN PSYCHIATRY

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In this series, Drs. Sanders and Gillig explain how aspects of the neurological examination can aid in differential diagnosis of some common (and some uncommon) disorders seen in psychiatric practice.

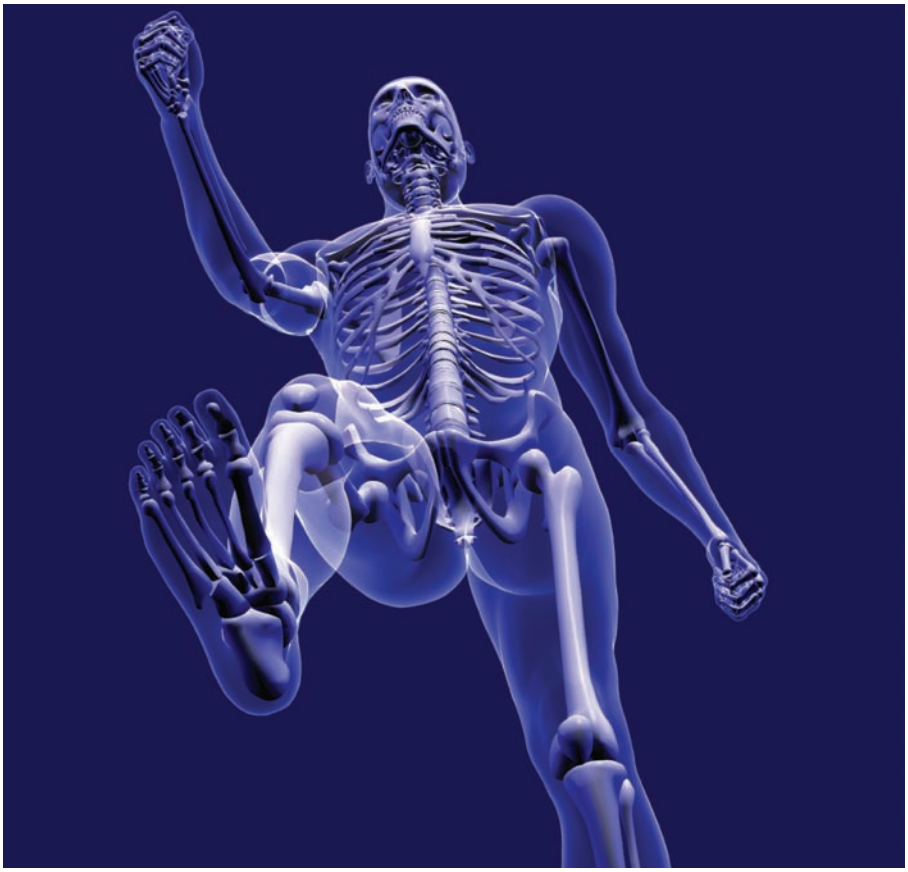
ABSTRACT

Gait reflects all levels of nervous system function. In psychiatry, gait disturbances reflecting cortical and subcortical dysfunction are often seen. Observing spontaneous gait, sometimes augmented by a few brief tests, can be highly informative. The authors briefly review the neuroanatomy of gait, review gait abnormalities seen in psychiatric and neurologic disorders, and describe the assessment of gait.

INTRODUCTION

As much as it amuses humans to see dogs, bears, elephants, and so forth walking on their hind legs, for all practical purposes only humans do it. Bipedal walking is a complex activity that developed along-side higher cortical structures and capabilities.[1] Among individual humans, we shall see that gait develops and then declines in parallel with higher cortical structure and function.

Because gait reflects the integrity of higher brain systems, it has much to tell psychiatrists. In fact, the unaided observation of gait can tell the informed observer a great deal in



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a few seconds. A fairly complete assessment of balance and gait can be complete within two minutes, yielding a tremendous amount of diagnostic and prognostic information. This may be the most important examination in psychiatry outside of the mental status.

GENERAL INFORMATION

Balance is the ability to stand, and gait is rhythmic stepping movements for travel (locomotion). Balance and gait problems tend to be found in the same individuals, so they tend to be discussed together.

Parkinsonian gait has several features, including short steps (*petit pas*, in which the heel lands less than one foot-length ahead of the toes of the other foot), reduced arm swing, stooped posture, anteropulsion/ retropulsion (center of gravity is ahead of or behind the feet, causing forward or backward acceleration), and festination (hasty but short steps attempting to compensate for displaced center of gravity). Postural instability is also a central feature of Parkinsonism, evidenced when the patient attempts to stand up without the use of his or her arms (he or she tends to fall back into the seat) or when the physician pushes on the chest or back of the standing patient (the patient will have more difficulty than most maintaining position).

Turning problems are common with any gait disorder; turning is generally more difficult than walking otherwise. People without balance or gait problems usually can do an "about-face" in one or two steps. Those with nonspecific problems may need three or four steps. People needing five or more steps are likely to have cerebral or basal ganglia dysfunction. If a patient has less trouble turning than walking forward, a psychogenic disturbance is likely.[2]

One sometimes hears of the wide-based gait. Technically, base width is the distance between the medial malleoli during walking (a wide-based stance is a different matter). Base width is normally negligible, but is wider in balance and gait disorders. With frank ataxia, base width is about 12 inches (equal to the width of the average floor tile). A mildly widened base is roughly half a tile. If base width approaches two feet, the likelihood of psychogenic gait disorder rises, unless the patient has morbid obesity or some structural explanation.[3]

Ataxic gait consists of arrhythmic steps (irregular), unsteadiness, wide base, and highly impaired tandem gait.[4] It can be found with injury to the cerebellum, pons, and sometimes thalamus; with loss of position sense; and can be simulated by cortical damage.

Hemiparesis can be seen in many aspects of gait, but most clearly in the affected leg. This leg cannot be shortened to avoid hitting the floor, so is swung around or circumducted.

ANATOMY

The entire nervous system is involved in balance and gait.[5,6] Balance and gait require intact brain, spinal cord, and sensory systems. Walking messages are initiated by the motor and premotor cortex and modified by the subcortical nuclei, brainstem, and cerebellum. These all activate the spine's central pattern generator, which coordinates arm and leg movements into rhythmic gait. Proprioceptive, visual, and vestibular inputs reach the spinal central pattern generator and affect its output.

Frontal lobe systems involved in balance and gait include primary motor cortex, supplementary motor area, and prefrontal cortex.[7] The frontal lobe projects to subcortical structures through periventricular white matter tracts in which the leg fibers are the most medial, hence the legs are most vulnerable to injury starting periventricularly.[8] The extent of white matter disease when examined by magnetic resonance imaging (MRI) predicts the likelihood of balance and gait problems.[9,10]

Particular features of disturbed gait correspond to particular malfunctioning structures (Table 1). Some of these correspondences are more obvious than others.

SPECIFIC CONDITIONS

Psychiatrists see more than their share of inscrutable gaits, particularly in the elderly. One major reason for this is that psychiatrists treat patients with forebrain disorders, and gait disturbances based on forebrain damage are highly variable. Add to that the shifting effects of "hysteria," fear of falling, restlessness, mania, depression, and drug effects, and psychiatrists can be excused for sometimes being at a loss to explain a patient's peculiar gait.

Most disturbed gait secondary to forebrain dysfunction, having evolved through a splitter's paradise of terms reflecting its various features—among them magnetic gait, apraxic gait, frontal ataxia, higher level gait disorder, frontal lobe gait disorder, lower body Parkinsonism, and Parkinsonian ataxia, to name a few[1]—is now lumped under terms such as *cortical balance* and *gait disorder*. Cortical balance and gait disorders are caused by frontal and occasionally parietal lesions.[1] Findings include a highly individualized admixture of Parkinsonism, ataxia, spasticity, "magnetic" aspects (feet stuck to ground), and apraxia (inability to perform the complex act in spite of intact component abilities). Because white matter tracts communicating with the frontal and parietal lobes are vulnerable to injury, they are the most common site of injury responsible for this group of gait disorders.[11]

Dementia is closely related to the above. Cognitive deficits are related to balance and gait disorders.[12–15] Slow gait predicts cognitive decline prospectively.[16–18] Even without considering dementia, balance and gait disorders are common in the elderly and are estimated to be 14 percent in individuals over 65 years, and 50 percent in individuals over 85 years.[19]

Alcoholism has an enormous presence in psychiatry and

TABLE 1. Disturbances of gait, corresponding anatomy, and etiology

AFFECTED STRUCTURE	CLINICAL FEATURE	COMMON ETIOLOGIES
Cortical (mostly frontal) or subcortical	Cautious, Parkinsonian (short steps, short arm swing), magnetic (feet barely leave the floor), apractic (lacking the skill of walking), ataxic (impaired control), spastic (hypertonic, jerking)	Periventricular white matter disease, normal pressure hydrocephalus, frontal vascular lesions, frontal tumors, parietal lesions
Basal ganglia	Parkinsonian (most specific are turning problems, hesitation/freezing, festination)	Parkinson disease, drug-induced, vascular
Thalamus (less common cause of gait disturbance)	Astasia (subjective imbalance, positive Romberg sign), ataxia (contralateral ataxia and sensory loss)	Vascular injuries in posterolateral thalamus
Cerebellum	Ataxia (midline and legs with vermal damage, appendicular and arms with hemispheric damage)	Toxic (EtOH, benzodiazepines, anticonvulsants), vascular, infectious/ inflammatory, demyelinating, metabolic, tumor, trauma, paraneoplastic
Brainstem	Variations on ataxia	Midbrain, pontine, and medulla infarcts
Spinal cord	Spastic (stiff appearance, "kicking" steps, bilateral leg circumduction), scissoring (with more severe spasticity)	Osteoarthritis, trauma, B12 deficiency, inflammation, demyelination, tumor, abscess, several others
Proprioceptive nerves	Sensory ataxia, sensory gait (impaired with eyes closed), positive Romberg sign	Diabetic neuropathy, Guillain Barre, other demyelinations
Vestibular input	Cautious, slightly wide base, tandem impaired by wide base, positive Romberg sign	Unilateral: vestibular neuritis, Meniere disease; bilateral: gentamicin toxicity
Visual	Visual disequilibrium: subjectively off balance, but balance and gait normal	Post-cataract or lens surgery, new visual corrective lenses
Muscle, neuromuscular junction	Waddling, steppage gait/foot drop, hyperextended knee	Proximal myopathies, myasthenia
Orthopedic	Antalgic gait	degenerative joint disease, acute orthopedic injury

neurology. It affects gait at every level of the nervous system.[20] Major relevant alcohol-related deficits include cognitive deficits, weakness due to myopathy, asterixis (sudden loss of muscle tone), cerebellar ataxia, chorea, and loss of position sense (sensory ataxia). The Wernicke-Korsakoff syndrome of thiamine deficiency includes confusion and ataxia, both of which impact gait (the third is extraocular movement problems). Alcoholic neuropathy is a distal, predominantly sensory or sensorimotor polyneuropathy. The dysesthesia of alcoholic neuropathy sometimes discourages walking. Alcoholic cerebellar degeneration affects mostly the vermis. Consistent with this, one finds a wide-based gait, poor tandem gait, and perhaps leg ataxia, but usually no arm ataxia.

Schizophrenia is consistently associated with mild Parkinsonism and ataxia, regardless of medications. Often the gait is slower, stride length shorter,[21,22] and tandem gait mildly impaired.[23,24]

Depressed patients occasionally have noticeable Parkinsonism that resolves with recovery from the depression. As a group, depression (especially among patients of at least middle age and those with melancholic depression) have slow gait with small steps.[25] Gait normalizes as the mood disorder improves.[26]

Psychogenic or "hysterical" gait disorders have been described in the literature for the last 150 years, when astasia (inability to stand) and abasia (inability to walk) was noted in patients with intact leg function.[27] "Astasia-abasia" eventually became a euphemism for hysterical gait disturbance, sometimes

characterized by acrobatic near-falls that appear to require more strength and balance than normal standing and walking. Gait may be very slow, and buckling of the knees is common.[28] Although gait is often slow, turns are often normal.[29] Useful clues suggesting psychogenic balance and gait disorders are abrupt onset, selective disability, relation to minor trauma, and improbable longitudinal courses.[30]

MEDICATION-INDUCED PROBLEMS

As we all know, medications contribute to balance and gait problems, particularly in vulnerable populations. Medications are a factor in at least 30 percent of elderly with balance or gait problems.[31] Polypharmacy (more than four medications) is a risk factor for falls, and psychiatric medications are major offenders[32]—and not just the "usual suspects" (tricyclics, benzodiazepines, barbiturates, and antipsychotics). For example, selective serotonin reuptake inhibitors (SSRIs) are associated with falls[33] and can cause more postural instability than tricyclics. Viewed mechanistically, we can imagine that medications affecting virtually any nervous function can interfere with balance and gait, and thereby contribute to falling. Most medication-related falling can be attributed to cognitive impairment, ataxia, Parkinsonism, and hypotension. Cognitive impairment is of course most often seen with antihistaminic and anticholinergic drugs. Ataxia is typically caused by sedative-tranquilizers and anticonvulsants. Parkinsonism, in which postural instability or tripping are responsible, is usually caused by antipsychotics, less often by other dopamine-blocking agents such as metoclopramide and prochlorperazine, still less often by SSRIs, valproate, and calcium blockers. Hypotension can be caused by any number of agents, including several antipsychotics, antidepressants, and of course antihypertensive drugs. Clozapine and valproate, by causing asterixis (negative myoclonus), can cause falling at conventional doses/levels (when the legs lose tone). Gabapentin may also cause asterixis-related falls.[34] Vestibular functioning can be suppressed to bad effect by meclizine and benzodiazepines and can be ablated by aminoglycosides. Drug-induced excessive walking will be discussed at another point.

Most psychiatric readers likely ask themselves whether any examination at all might better be delegated to an extender or consultant. However, even the most constrained specialists get to watch most of their patients walk a few steps.

HOW TO EXAMINE STABILITY AND GAIT

Most psychiatric readers likely ask themselves whether any examination at all might better be delegated to an extender or consultant. However, even the most constrained specialists get to watch most of their patients walk a few steps.

First, observe the patient while he or she walks for a few steps. Specialists might study the gait at some length, taking time to focus methodically on each of several body parts. More important, however, is to observe the gait overall, keeping open to general impressions. The image then may be mentally replayed to pick up the features responsible for these impressions, or the patient might be asked to walk more.

In psychiatric practice, cortical- and basal ganglia-related gait abnormalities are the most commonly encountered. The most common abnormal gait findings are Parkinsonian (due to drug side effects, idiopathic Parkinsonian disorders, and occasional Parkinsonian effects of depression), the group of cortical gait abnormalities (particularly in the elderly), and ataxic gait disturbances (in chronic alcoholic patients and due to drug side effects). Asymmetry should be investigated further with pyramidal system tests; it is often found to be isolated and without any apparent pathological basis, sometimes referable to asymmetric Parkinsonism or dystonia.

Casual gait can be characterized in terms of speed (rapid in mania, slow in basal ganglia and several other conditions, and depression), stride length (short in cortical and basal ganglia disorders), arm swing (reduced in cortical and basal ganglia disorders), stride height (increased in steppage gait associated with foot drop, decreased with cortical "magnetic" gait), rhythmicity (arrhythmic in ataxia).

As mentioned previously, these samples of "casual gait" can be supplemented with brief tests, which require no special facilities or equipment. Ask the patient to stand from a sitting position in a seat of average height with arms crossed across the chest. Inability to do so indicates axial or leg weakness or impaired balance. Ask the patient to walk to a certain point (about 10 feet away), turn around and return. Count the steps required to make the turn: more than two is abnormal, more than four suggests a basal ganglia disorder). While walking, note any significant widening of the base (6 inches or half a floor tile between medial malleoli is significant). Since the inability to continue walking while talking predicts risk for falling,[35] it can be useful to attempt to elicit speech while the patient is walking. Note the natural posture (erect or stooped as in Parkinson disease or depression). Of course, when assessing gait, one should also take cognition, anxiety, and depression into consideration.

Other neurological tests can be useful in the assessment of abnormal

balance and gait. Successful execution of tandem gait requires one to be able to ambulate with a narrow base and to have accurate leg control, so it can be useful in clarifying these two gait parameters. A positive Romberg test reveals abnormal proprioception and/or vestibular function. Trunk movements sometimes bear assessment. Occasionally a patient strikingly lacks control of trunk movement, as evidenced by using the arms to roll over or sit up rather than using the trunk. Limb apraxia (ask the patient to pretend to kick a ball or use a hammer) can be key to assessing the higher order apraxia of gait.

CONCLUSION

Gait abnormalities, particularly those affecting psychiatric patients, can be quite difficult to assign to simple causes. Like psychiatric symptoms, gait abnormalities often resist categorization, change over time, and in other ways defy clear understanding. Like most of psychiatry, studying an abnormal gait requires that we persistently strive to clarify the case while tolerating its ambiguities. No matter how little we personally engage in the rest of the neurological exam, simply watching when the patient walks can be most informative.

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