

# APRAXIA OF GAIT

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## Apraxia of Gait: Definition and Core Characteristics

Apraxia of Gait (AOG) refers to the severe impairment in or complete loss of the ability to execute the complex, coordinated motor patterns required for independent ambulation. This is a highly specific neurological condition where the difficulty in walking cannot be attributed to primary motor deficits such as gross muscle weakness, commonly known as paralysis or paresis, nor is it explained by primary sensory impairment, pain, or cerebellar ataxia. Fundamentally, AOG is a disorder of motor planning and execution; the individual possesses the necessary strength and sensation but lacks the ability to organize the sequential, fluid motor movements required to ambulate effectively. It is crucial to distinguish this central planning failure from peripheral or mechanical causes of walking difficulty, placing AOG firmly within the category of higher-order cortical dysfunctions.

The core challenge faced by a person with AOG lies in the inability to formulate the motor program necessary for walking. While the individual may understand the command to walk and desire to comply, the brain struggles to translate this intention into a successful sequence of steps. The patient is unable to organize motor movements in order to walk in a fluid manner, resulting in a characteristic presentation that is often described as shuffling, clumsy, or hesitant. This impairment is often magnified during complex tasks, such as turning, navigating tight spaces, or dual-tasking while walking, highlighting the breakdown in the automatic, procedural memory systems that typically govern locomotion.

A key diagnostic feature of AOG is the discrepancy between the patient's ability to perform stepping motions while lying down (supine) versus standing up (orthostatic). When asked to mimic the stepping motion while supine, patients can often execute the movement with normal range and coordination, demonstrating that the primary motor pathways controlling leg movement are intact. However, upon transitioning to a standing position, the apraxia becomes immediately evident, suggesting that the integration of postural control, balance maintenance, and locomotor planning, which are primarily managed by the frontal and subcortical structures, is compromised. This dissociation underscores the nature of AOG as a planning disorder rather than a strength deficit.

## Neuroanatomical Basis and Etiology

The neural substrate for Apraxia of Gait involves damage to specific areas of the brain responsible for the initiation, modulation, and complex sequencing of motor movements. The most commonly implicated areas include the frontal lobes, particularly the supplementary motor area (SMA) and the prefrontal cortices, the basal ganglia, and their extensive white matter connections. The frontal lobes are essential for motor planning and the execution of voluntary, goal-directed movements. Damage to these areas disrupts the cortical input necessary to trigger and sustain the rhythmic patterns of walking, leading to the characteristic start hesitation and shuffling associated with the

condition.

A significant proportion of AOG cases are associated with conditions that affect the subcortical white matter and the deep gray matter structures. Among the most frequent causes is **Normal Pressure Hydrocephalus (NPH)**, a condition characterized by enlarged ventricles and a triad of symptoms including gait disturbance, urinary incontinence, and dementia. In NPH, the mechanical pressure from the expanded ventricles compresses the periventricular white matter tracts, which contain crucial fibers originating from the frontal motor regions. This compression interferes with the communication between the cortex and the brainstem locomotor centers, resulting in a highly distinctive gait pattern often termed "magnetic gait" due to the appearance that the feet are glued to the floor.

Other etiologies contributing to AOG include widespread ischemic or vascular disease, typically affecting the small vessels that supply the deep white matter, leading to Binswanger's disease or multi-infarct dementia. Degenerative disorders, such as certain forms of atypical parkinsonism like Progressive Supranuclear Palsy (PSP) or Corticobasal Degeneration (CBD), can also manifest with severe apraxic features, although these presentations are often complicated by other neurological signs. Furthermore, large or strategically located tumors, subdural hematomas, or chronic demyelinating diseases that interrupt the crucial cortico-subcortical loops can also precipitate the development of AOG, emphasizing that the disorder arises from the disruption of integrated circuits rather than localized peripheral damage.

## Clinical Presentation and Symptomology

The clinical presentation of Apraxia of Gait is distinct and often evolves over time. Initially, patients may report subtle difficulties with balance or a reduced speed of walking. As the condition progresses, the most prominent symptom becomes the severe impairment in gait initiation, known as **start hesitation**. The patient may stand motionless, appearing frozen at the beginning of the walk, struggling to lift the foot and propel the body forward. When movement is finally initiated, the steps are typically small, shuffling, and hesitant, lacking the normal heel-to-toe sequence and amplitude found in healthy ambulation.

Another hallmark feature is the difficulty with turning. While walking in a straight line is challenging, attempting a pivot or a 180-degree turn often results in a series of extremely small, choppy steps, or even a complete standstill, increasing the risk of falls significantly. The base of support is typically widened, reflecting the patient's attempt to compensate for poor balance and instability. In severe cases, the gait becomes highly unstable, requiring continuous support, and the characteristic "magnetic gait" is observed: the feet seem to drag or stick to the floor, often described as if they are weighted down or under the influence of a strong opposing force. This magnetic quality is highly suggestive of pathology involving the frontal-subcortical circuits, often

seen in NPH.

Crucially, the symptoms of AOG are often inconsistent and highly dependent on environmental context and internal demands. Patients may exhibit a phenomenon known as **kinesia paradoxa**, where they can momentarily walk better or even normally under specific external stimuli, such as stepping over an obstacle, walking toward a visible target, or following rhythmic auditory stimulation (RAS), like marching to music. This temporary improvement suggests that while the internal, automatic motor program is damaged, the ability to utilize external cues to bypass the damaged circuit remains partially intact, distinguishing AOG from pure motor weakness where performance remains consistently impaired regardless of external input.

## Differential Diagnosis

Accurately diagnosing Apraxia of Gait requires careful clinical examination to differentiate it from other common causes of gait disturbance, particularly those related to Parkinson's disease, cerebellar disorders, and sensory neuropathies. The distinction is vital because the treatment and prognosis vary dramatically based on the underlying etiology. Unlike Parkinsonian gait, which is characterized by bradykinesia, rigidity, and a forward-flexed posture, AOG often lacks the severe resting tremor and the typical response to dopaminergic medication seen in Parkinson's disease, although overlap certainly exists, particularly in atypical parkinsonism. While both conditions involve shuffling and start hesitation, the magnetic quality and the profound difficulty in lifting the feet in AOG are often more pronounced.

AOG must also be separated from **cerebellar ataxia**, which results from damage to the cerebellum and its pathways. Cerebellar ataxia presents with broad-based stance, decomposition of movement, and significant intention tremor, leading to an irregular, staggering gait characterized by poor coordination and severe disequilibrium. In contrast, AOG is primarily a problem of motor planning and initiation, not of coordination or balance maintenance per se, although secondary balance issues arise due to the ineffective stepping pattern. Furthermore, the ability of the AOG patient to perform stepping movements when supine is usually preserved, a feature not typically seen in severe cerebellar dysfunction.

Finally, AOG must be differentiated from gait disturbances caused by peripheral neuropathy or spasticity. Peripheral neuropathy causes sensory loss, leading to a high-stepping, slapping gait (steppage gait) as the patient attempts to compensate for lack of proprioceptive feedback. Spasticity, due to upper motor neuron lesions, results in stiffness, circumduction, and scissoring gait patterns. In AOG, motor strength and deep tendon reflexes are generally normal or near-normal, ruling out primary spasticity, and the underlying impairment is clearly cognitive or planning-based rather than related to primary muscle tone or sensory input failure.

## Assessment and Diagnostic Procedures

The assessment of Apraxia of Gait relies heavily on detailed clinical observation and standardized neurological testing, followed by neuroimaging. The clinical evaluation includes observing the patient during various tasks: walking straight, turning rapidly, walking heel-to-toe, and performing tandem gait. Specific scales, such as the Tinetti Performance Oriented Mobility Assessment (POMA) or the Berg Balance Scale, can quantify functional deficits, but specialized gait scales focusing on initiation, step length, and fluidity are often more informative for AOG. The differential assessment, particularly the supine versus standing stepping test, is paramount for confirming the apraxic nature of the impairment.

Neuroimaging, typically Magnetic Resonance Imaging (MRI) or Computed Tomography (CT) scans, is essential to identify the underlying pathology. MRI is crucial for visualizing the characteristic structural changes associated with AOG, such as periventricular white matter hyperintensities indicative of small vessel disease, or ventricular enlargement suggestive of NPH. In cases where NPH is suspected, the diagnosis is often supported by the presence of enlarged ventricles without significant cortical atrophy, and specific flow void signs on MRI.

For patients presenting with the classic triad suggestive of NPH, invasive diagnostic procedures may be employed. The **Cerebrospinal Fluid (CSF) tap test** involves removing a large volume of CSF (typically 30-50 mL) and immediately reassessing the patient's gait performance. A significant, albeit temporary, improvement in gait metrics following the tap is highly predictive of a positive response to CSF shunting, confirming NPH as the etiology of the AOG. Similarly, continuous external lumbar drainage over several days may be used to provide a more prolonged test of CSF dynamics and potential response to surgical intervention.

## Classification and Subtypes

While Apraxia of Gait is often treated as a unified syndrome, its classification is usually determined by the underlying etiology, which dictates both the specific clinical presentation and the subsequent therapeutic strategy. The most clinically relevant distinction separates AOG associated with Normal Pressure Hydrocephalus from AOG resulting from widespread degenerative or vascular disease.

The **NPH-related AOG** subtype is characterized by the classic magnetic gait, profound start hesitation, and often involves accompanying cognitive decline (subcortical dementia) and urinary urgency/incontinence. This subtype is considered potentially reversible, as the mechanical compression leading to the apraxia can be alleviated by ventricular shunting. The specific presentation of NPH gait tends to be symmetrical and often appears more severe when the patient is standing, contrasting with the often preserved motor function while lying down.

The **Vascular/Degenerative AOG** subtype arises from chronic cerebral ischemia, small vessel disease, or neurodegenerative processes affecting the frontal-subcortical loops. This form is often less responsive to interventions and tends to progress more steadily. Depending on the extent and location of the lesions, the gait disturbance may be accompanied by more prominent executive dysfunction, frontal release signs, or signs of pseudobulbar palsy. Furthermore, some authors classify AOG as part of a broader spectrum of frontal gait disorders, emphasizing the common neuroanatomical origin in the frontal lobes and their descending tracts, regardless of whether the etiology is vascular, compressive, or degenerative.

## Treatment and Rehabilitation Strategies

The management of Apraxia of Gait is multifaceted and highly dependent on identifying the treatable components of the underlying disease. When AOG is confirmed to be secondary to Normal Pressure Hydrocephalus, the primary treatment is neurosurgical intervention involving the placement of a ventricular shunt, typically a ventriculoperitoneal shunt. This procedure aims to drain excess CSF, thereby reducing the pressure on the periventricular white matter tracts. For appropriate candidates, shunting can lead to dramatic and sustained improvement in gait function, often significantly reversing the apraxia and improving quality of life.

For AOG stemming from non-reversible vascular or degenerative causes, treatment focuses primarily on optimizing physical function and compensating for motor planning deficits. **Physical therapy** is a cornerstone of rehabilitation. Therapists utilize specific techniques aimed at bypassing the automatic motor planning deficit by employing external cues. These techniques include rhythmic auditory stimulation (RAS), where patients walk to the beat of music or a metronome; visual cueing, such as stepping over lines taped on the floor or focusing on targets; and verbal instructions, which help the patient consciously process each step, compensating for the failed subconscious motor program.

Pharmacological management is generally supportive and less curative than in Parkinson's disease, but trials of certain medications may be warranted, especially if there is an overlapping vascular or subcortical component. Dopaminergic agents, traditionally used for Parkinsonism, are sometimes trialed, particularly if there is a suspicion of vascular parkinsonism. Cholinesterase inhibitors, used in dementia management, may also be employed to potentially improve cognitive function, which can indirectly aid gait performance by improving attention and execution of complex motor commands necessary for safe walking. Assistive devices, such as walkers or canes, are often necessary to prevent falls, but their effectiveness can be limited due to the severe planning deficits characteristic of AOG.

## Prognosis and Long-Term Outlook

The long-term prognosis for individuals with Apraxia of Gait is highly variable and directly linked to the underlying cause and the timeliness of intervention. The most favorable prognosis is reserved for patients whose AOG is clearly attributable to Normal Pressure Hydrocephalus and who demonstrate a positive response to the CSF tap test. For these individuals, shunt surgery offers a high probability of significant functional recovery, often allowing them to regain independent ambulation and substantially slow the progression of their gait impairment and associated cognitive decline. However, even in successful shunting cases, continued physical therapy is essential to maximize and maintain functional gains.

In cases where AOG is secondary to widespread chronic vascular disease, neurodegenerative conditions, or advanced cortical atrophy, the prognosis is generally guarded. The progressive nature of these underlying pathologies means that the gait impairment is likely to worsen over time, despite symptomatic management. While rehabilitation and compensatory strategies can temporarily improve mobility and safety, the relentless nature of the disease often leads to increasing dependence on assistive devices and, eventually, wheelchair reliance. Therefore, early diagnosis and aggressive rehabilitation are critical to preserve functional independence for as long as possible.

Ultimately, the effective management of AOG requires continuous monitoring and a multidisciplinary approach involving neurologists, neurosurgeons, physical therapists, and occupational therapists. The goal is not merely to treat the physical symptoms but to address the broader implications of the motor planning deficit, ensuring patient safety, maximizing residual mobility, and maintaining the highest possible quality of life despite the challenges imposed by this complex neurological disorder. Research continues into non-invasive brain stimulation techniques and novel pharmacological agents that might target the damaged frontal-subcortical circuits responsible for the impairment in motor sequence execution.