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Review Article

Gait: The First Indicator of Many Neurological Diseases and Disorders

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Abstract

The intricate interplay of the primary elements of the neurological, musculoskeletal, and cardiorespiratory systems underpins human gait. In elderly individuals, a preferred brisk walk is a strong indicator of overall health and quality of life (QoL). However, the physiology of walking is complicated. Thus, diagnosis of gait problems requires a thorough understanding of normal gait physiology.

Gait abnormalities are typically a debilitating side effect of aging. Irrespective of whether neurological or non-neurological in origin, they are a significant source of disability, morbidity, and mortality in the elderly population. Gait abnormalities result in the loss of personal freedom, falls, and injuries, significantly decreasing an individual's QoL. This review discusses the origins and concepts of instrumented gait analysis.

A method for examining gait and essential aspects of prevalent gait abnormalities and their underlying causes are also covered. In addition, a clinically focused strategy for treating neurological gait concerns in elderly individuals, and the future of gait analysis is presented.

Keywords: *Abnormal Walking; Antalgic; Cerebral Cortex; Cognitive Impairment; Frequent Falling; Nerve Degeneration*

Abbreviations

3-D: Three-Dimensional; AD: Alzheimer's Disease; bvFTD: Behavioral Variation FTD; EMG: Electromyography; FTD: Frontotemporal Dementia; HD: Huntington's Disease; PD: Parkinson's Disease; QoL: Quality of LIfe; RF: Radiofrequency; SCA: Spinocerebellar Ataxia

Introduction

Walking, a typical everyday action, is highly complex [1]. Since recorded history, physicians and philosophers have been contemplating how humans move. Aristotle (384–322 BCE) was the first to describe how people walk [2]. In his first written reference regarding the analysis of walking, Aristotle explained that the line traced by a reed, soaked in ink, connected to a man's head, walking over the ground, alongside a wall, would not be straight, but zigzag—because the head becomes lower when he bends and higher when he stands straight and elevates himself [2]. Aristotle's *On the Gait of Animals*, written in 350 BC, was the first work to describe gait analysis [3].

Galileo Galilei (1564–1642) contributed to gait analysis, examining the behavior of falling bodies and laying the groundwork for mechanical movement analysis. Giovanni Alfonso Borelli, one of Galileo's students, performed the first experiment in gait analysis in 1680. He extensively researched the mechanics of animal movement[2–4].

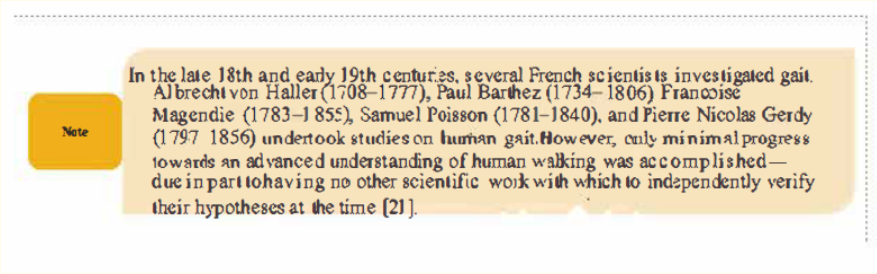

Figure 1: *Adapted from physio.co.uk [21}. GAIT Note: rv 190422.1912.*

Wilhelm Eduard Weber (1804–1891) was the next key contributor, as part of a team, that studied these issues. He was best known for his research on the eponymous SI unit of magnetic flux [2].

Étienne-Jules Marey (1830–1904), from Paris, published the next significant study on human mobility. Marey was a contemporary gait analyzer to who created the first sphygmograph in 1860 and the first cardiogram in 1863. His earliest gait research was performed with a student, Gaston Carlet (1849–1892).

Carlet (1845-1892) designed a shoe with three pressure sensors in the sole that measured the forces applied by the leg on the surface. He was the first to record the synchronous bump of the ground response. Carlet's thesis in 1872 finished with a simple and true depiction of the average human gait cycle [2].

Muybridge and Marey invented animated photography in the 1880s that recorded the many motion stages utilizing a photographic plate. Muybridge recorded movement with several cameras, and displayed it through presentations and demonstrations [3].

Braune and Fischer devised a technique of three-dimensional (3-D) insight into human mobility in the 1890s, marking a turning point in the history of experimental gait analysis. Pictures at a frequency of 26 frames/s were created, using light-emitting markers and trigonometric measurement. Braune and Fischer used this method to examine the rotational displacements of the lower extremity joints [5].

During the 1940s and 1950s, a significant advancement was observed in research on human gait. Verne Thompson Inman and his team's pioneering work highlighted the importance of including technology, orthopedics, and anatomy in gait analysis [6]. The use of this method enabled investigations on limb deformations and rotations, kinematics and accelerations, external factors impacting the limbs, energy expenditure while walking, and myoelectric muscle stimulation during motion (with dynamic electromyography).

Shortly thereafter, force plates for gait kinetics assessment, accelerometers for limb motion measurement, single-channel electromyography, and eventually, multichannel electromyography were launched. In the 1970s, goniometry was first used in clinical practice. A three-plane, exoskeletal, electrogoniometric approach was developed to measure the sagittal, coronal, and transverse spins of the hip and knee joints [7–14].

In 1972, the Vanguard motion analyzer, consisting of 50 frames/s cameras to capture the subject's movements, was released. This approach benefits from not requiring the participant to wear any equipment and allowing several assessments to be taken in the same period. Electromyographic activity may be overlaid on a motion picture film to enable contemporaneous monitoring, allowing both legs to be recorded simultaneously. To this technology, six-channel electromyography (EMG) with telemetry and a high-speed cine (movie) camera that filmed at the same pace as the mobile patient, delivering more reliable results.

The 2-D system was improved further with the application of video surveillance to monitor the activities of markers attached to the study participant's body part, and a video-recording device recorded measurements. Also, a computer-assisted estimation of the values of the angular position was applied in generating a concise overview of the locomotion system throughout walking [15]. A list of significant contributors to gait analysis is provided in Table 1 [2].

Date	Name	Contribution
384–322	Aristotle	Theories on the movement of humans and animals
1608–1679	Giovanni Borelli	Muscle and tendon biomechanics
1708–1777	Albrecht von Haller	Physiology of walking
1734–1806	Paul Barthez	
1783–1855	François Magendie	
1781–1840	Samuel Poisson	
1797–1856	Pierre Gerdy	
1804–1891	Wilhelm Eduard Weber	Anatomy of mechanics
1806–1871	Eduard Friedrich Weber	
1806–1875	Guillaume Duchenne	Founder of electrophysiology. Reported Duchenne gait pattern
1844–1924	Friedrich Trendelenburg	Orthopedic surgeon. Reported Trendelenburg gait pattern
1830–1904	Étienne-Jules Marey	Physiologist. Force and pressure measurement. Chronophotography
1849–1892	Gaston Carlet	First essentially correct description of the gait cycle
1850–1918	Georges Demenÿ	Pioneer of the photographic method
1830–1904	Eadweard Muybridge	Photography of movement
1831–1892	Wilhelm Braune	First 3-D gait analysis
1861–1917	Otto Fischer	
1896–1966	Nikolai Bernstein	Development of motor control theories
1878–1935	Jules Amar	Pneumatic three-component force-plate
1893–1971	Wallace Fenn	Mechanical one-component force-plate
1905–1980	Verne Inman	Founded biomechanics laboratory at the University of California
1906–1993	Howard Eberhart	
1925–1984	Pat Murray	Instrumental studies of regular walking in men and women
1918–unknown	Jacquelin Perry	Pioneer of clinical electromyography and observational gait analysis
1923–2006	David Sutherland	Digitalization of data from cine film. Development of walking in children
1926–2000	Jürg Baumann	Integration of cine photography with electromyography

Table 1: *Gait analysis research history.*

In the mid-1970s, 3-D motion analysis techniques were introduced. Further advances in motion analysis were made possible by introducing microchip digital technology. Current techniques provide a digital visual representation of body movement on a computer screen in less than a minute. Furthermore, the emergence of small, lightweight markers—such as active small infrared light-emitting diodes, passive infrared reflecting spheres, and EMG telemetry—enabled rapid collection of massive gait data [16].

Discussion

Gait is the walking pattern of an individual. Walking requires muscular coordination and balance to push the body forward in a pattern known as the stride. Gait and sophisticated cognitive functioning are inextricably linked, and walking is no longer seen as a simple mechanical action apart from cognition. Walking involves the combination of concentration, preparation, recollection with motor, visual, and cognitive functions. Lower cognitive functioning is associated with decreased gait velocity, slower pace, lower frequency, and reduction in gait diversity [17].

Gait may be categorized into normal and abnormal gaits [18]. Normal gait is the walking style that humans use when not disturbed [19]. It requires careful regulation of limb motions, orientation, and muscle definition—a highly complicated process involving the entire neurological system. Rhythmic activity is generated by specialized neuron groups in the spinal cord and brainstem, which send signals to motor neurons, activating limb muscles.

The cerebral cortex combines information—from the optical, auditory, and proprioceptive systems; hypothalamus, basal ganglia, cerebellum; and afferent neurons—that transport sensory signals from muscle and sensory organs. These systems work in harmony, allowing humans to allow people to walk in a straight, unimpeded line, and alter their stride to avoid obstacles while maintaining balance. Abnormalities in any part or aspect of the neurological system can cause gait disorders [20].

In unusual or pathological gait, the bodily systems regulating walking do not function normally. Nerve abnormalities, impaired motor control, adverse biological factors, and inflammation contribute to pathological gait [18]. Abnormal gait is more common in elderly individuals, affecting one in three people older than 60 years. Gait problems are associated with this population's worsening life quality of life (QoL), and frequent nursing home placement. Aberrant gait may also signal the development of dementia in people with moderate cognitive impairment [20]. Numerous factors may be responsible for an irregular gait (Figure 2) [21,22].

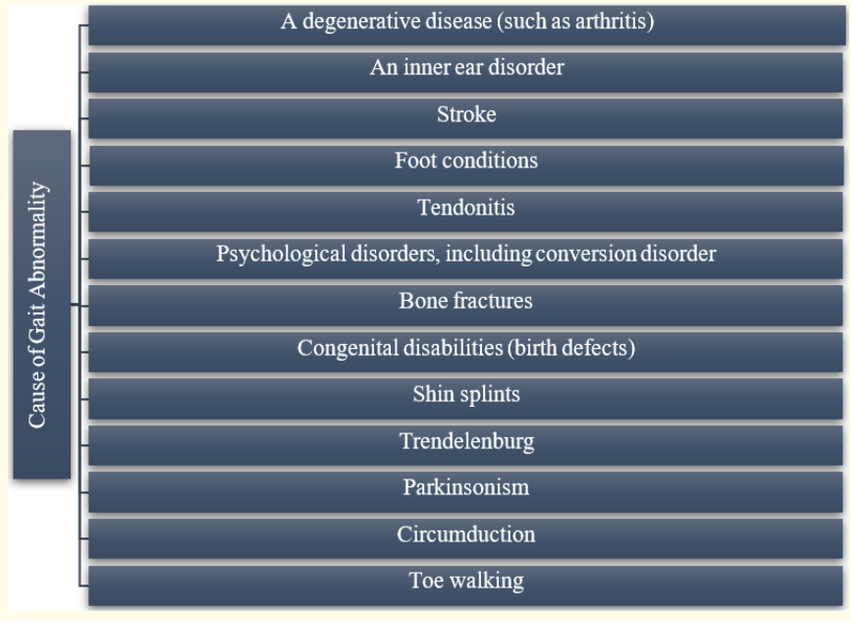

Figure 2: *Examples causing gait abnormality.*

The risk of gait and balance abnormalities increases with age, affecting 10% of those aged 60–69 years and >60% of those aged 80 years. The incidence increases to 82% in those > 85 years [23]. Variances distinguish different forms of gait in the upper and lower extremities, total speed, forces, kinetic and potential energy sequences, and variations in surface interactions [24]. A phenomenological categorization of common gait abnormalities is provided in Figure 3 [25–28].

Gait disorder	Characteristics/Identification	Associated signs & symptoms
Hemispastic gait	Unilateral extension and circumduction of 1 leg	Weakness on the affected side; hyperreflexia; extensor plantar response; flexed arm
Paraspastic gait	Stiffness, extension, adduction, and scissoring of both legs	Bilateral leg weakness, hyperreflexia, spasticity, and extensor plantar responses
Ataxic gait	Wide-based gait; incoordination; staggering; decomposition of movements	Dysmetria; dysdiadochokinesia; tremor; postural instability
Sensory ataxic gait	Unsteadiness of walking when visual input is withdrawn	Positive Romberg sign; decreased position sense
Steppage gait	Weakness of foot dorsiflexors; footdrop; excessive flexion of hips and knees when walking; short strides; unilateral or bilateral	Atrophy of distal leg muscles; decreased ankle reflex; possible sensory loss
Cautious gait	Wide-based, careful, slow steps; reaching for support; as in walking on ice; better at home than in open spaces	Associated often with anxiety, fear of open spaces, and fear of falling
Freezing gait	Blockage, e. g. on turning	Can fall easily
Apraxic gait	Difficulty initiating a step; freezing; feet almost stuck to floor; turn hesitation; shuffling gait	Hypokinesia; muscular rigidity; grasp reflexes; possible resting tremor, dementia, or urinary incontinence
Propulsive or retropulsive gait	Body's center of gravity appears to be either in front of or behind the patient, who is struggling to keep the feet up to center of gravity; festination	Hypokinesia; muscular rigidity; postural instability
Astasia	Primary impairment of stance/balance	Postural instability
Dystonic gait	Sustained abnormal posture of the foot or leg; distorted gait; hyperflexion of hips	Action-related gait disturbance; atypical presentations
Choreatic gait	Irregular, dancelike gait; slow and wide-based; spontaneous knee flexion and leg raising	Athetotic and choreic movements of the upper extremities
Steppage gait	Weakness of foot dorsiflexors; footdrop; excessive flexion of hips and knees when walking; short strides; unilateral or bilateral	Atrophy of distal leg muscles; decreased ankle reflex; possible sensory loss
Waddling gait	Wide-based gait; swaying; toe walk; lumbar lordosis; symmetric	Proximal muscle weakness of lower extremities
Antalgic gait	Limping; avoidance of bearing full weight on the affected leg; limitation of range of movement	Pain in lower extremity aggravated by leg, hip, and thigh movement as well as weight bearing
Vertiginous gait	Unsteady gait; falling to one side; postural imbalance	Vertigo; nausea; nystagmus
Psychogenic gait disorder	Astasia-abasia; bizarre and nonphysiologic gait; lurching; rare fall or injury	Give-way weakness; Hoover sign; other signs of conversion
Parkinsonian gait	Short-stepped; shuffling; hips, knees, and spine flexed; festination; en bloc turns	Bradykinesia; muscular rigidity; postural instability; reduced arm swing; rest tremor

Figure 3: *Gait disorders.*

Identification signs and markers of gait disorders

The identification signs [29] and markers [30–34] of gait disorder are listed in Figure 4.

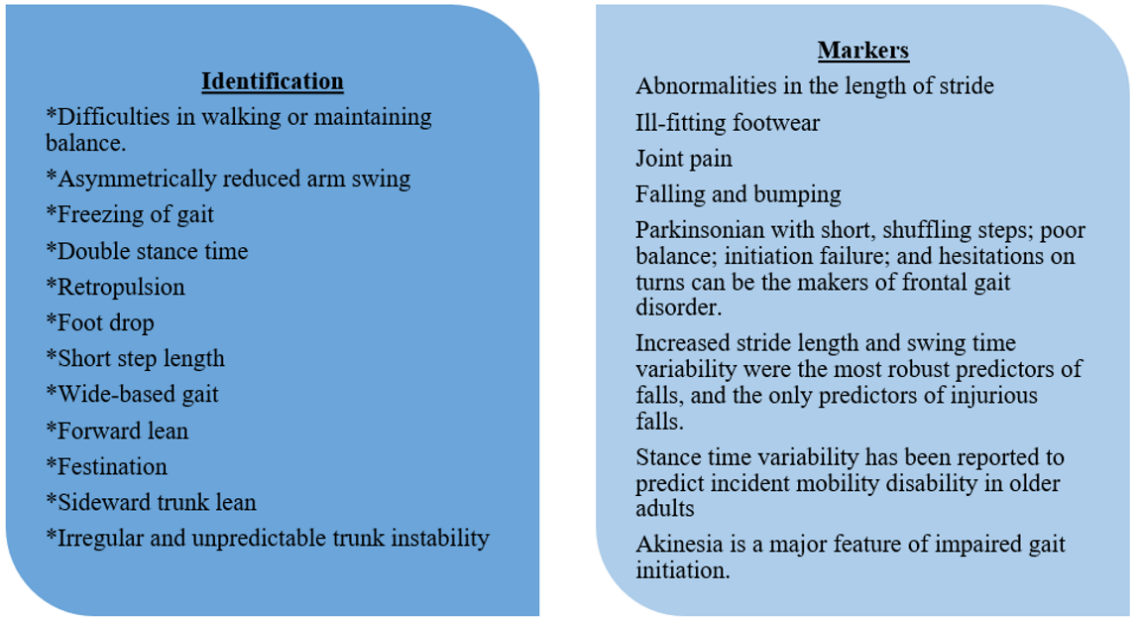

Figure 4: *Identification signs and markers of gait disorder.*

Causes of gait disorders

Biomechanical [1,35] and neurological [36,37] causes of gait disorder are presented in Figure 5.

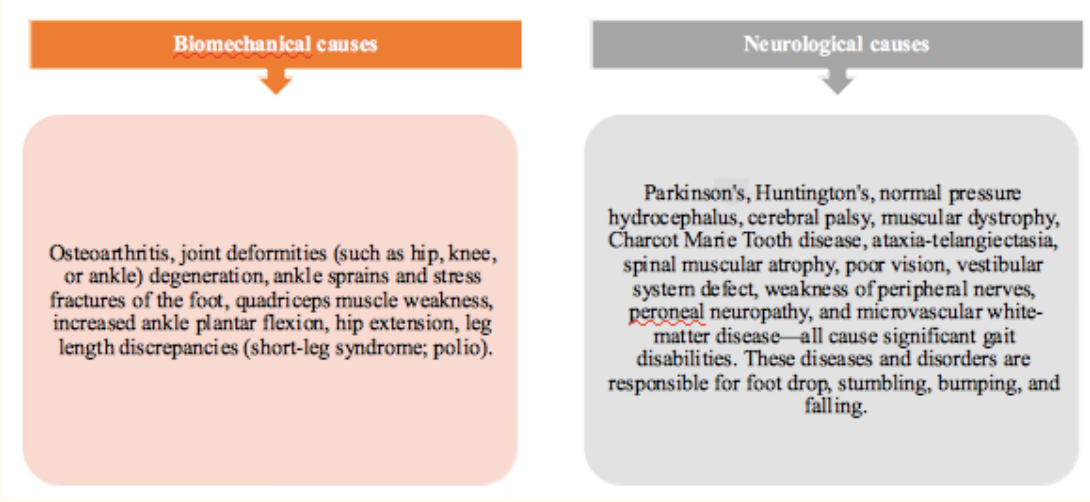

Figure 5: *Biomechanical and neurological causes of gait disorder.*

Distinguishing features of gait in neurodegenerative conditions

Gait was once thought to result from central pattern generators created by spinal neural circuits. This proposition led to the notion that basic neural circuits responsible for producing and controlling locomotion are primarily found in the spinal cord. Therefore, any lesion to the relevant region of the cerebral cortex is likely to impact the walking pattern significantly [38].

Neurodegenerative disease is an abnormal change in the neural system, causing tremors in the limbs, jaw, or face and stiffness and slowness of movement due to liver function disruption. Typically, the condition develops over time and causes mobility impairments and walking difficulties [39]. Several NDDs unique to gait abnormalities are listed in Figure 6 [40].

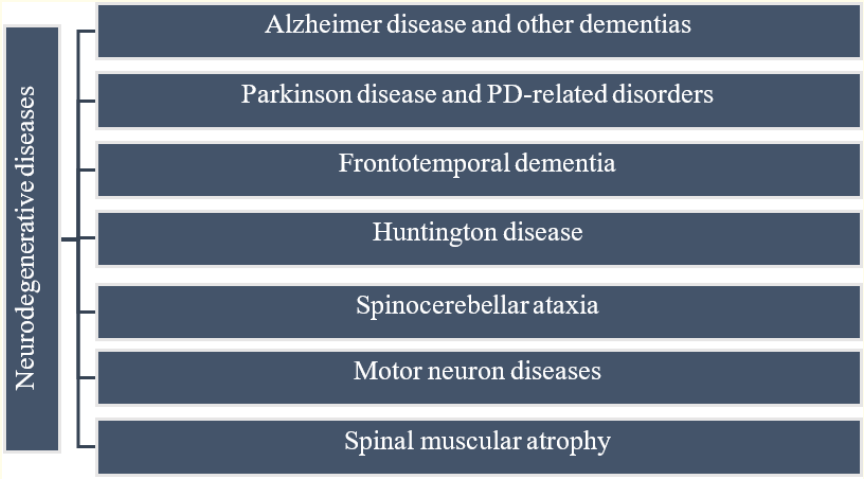

Figure 6: *Neurodegenerative diseases specific to gait disorders.*

Alzheimer's disease (AD)

Gait abnormalities characterize Alzheimer's disease (AD) and other dementias. Although the cognitive decline is the most prevalent clinical feature of dementia, motor abnormalities such as bradykinesia, extrapyramidal stiffness, and gait difficulties have also been reported, often in the later phases. Major epidemiological studies conducted in the past two decades have revealed that gait problems, especially slowing gait, may be evident in the early phases of the disease and even predict patients who may progress to dementia. The cohabitation of specific cognitive impairments and gait irregularities is noted in older persons with mild cognitive impairment, a pre-dementia stage.

Regarding potential factors involved in gait-cognitive interrelations in dementia syndromes, significant contributors to concurrent cognitive and gait impairment include structural and functional brain disruption, such as atrophy of specific subcortical areas, white matter disease, and amyloid-deposition burden, and accentuated neurotransmitter depletion [41].

Frontotemporal dementia (FTD)

Frontotemporal dementia (FTD), a type of cortical dementia, develops in younger adults and is more frequently associated with neuropsychiatric symptoms [42]. It is the second most frequent type of young-onset dementia after AD [43]. The pathologically diverse FTD is

characterized by frontal and temporal lobe degeneration. Behavioral variation FTD (bvFTD), semantic dementia, and progressive nonfluent aphasia are the three types of FTD.

The most prevalent is bvFTD, characterized by behavioral problems (dysregulation, apathy, aspontaneity, lack of flexibility, impatience, loss of sensitivity, perseverations, stereotypes, obsessive behavior, hyperorality, and sexual dysfunction). FTD is also characterized by gait disturbance. It can be caused by gait apraxia, extrapyramidal syndrome, or muscular weakness in a neuronal motor condition [44].

Parkinson's disease (PD)

Parkinson's disease (PD) is primarily a neurodegenerative brain condition that destroys the parts of the brain that govern motion. Shuffling gait, decreased balance, and frozen gait are the most common motor dysfunctions observed in patients with PD. Postural control and gait impairment may emerge in the early stages of PD. They are distinguished by slowing of gait, arm motion reduction, walking speed, balance problems, and loss of disassociated arm and trunk movements during walking [45]. Patients with PD have a high risk of falls because of gait disruption and freezing. According to prospective studies, 70% of people with PD have at least 1 fall/year, and 39% have frequent falls [46].

Huntington's disease (HD)

Huntington's disease (HD) is an autosomal dominant hereditary neurological condition characterized by typical gait alterations such as reduced walking pace, difficulty initiating steps, and varied walking style. As the disease progresses, mobility deteriorates, the risk of falls increases, and functional ability decline, expanding the need for care. Hypokinesia and increased gait variability are frequent gait abnormalities in HD.

Disruption in stride length control, increased step-to-step variance, and interrupted gait begins before clinical indications of HD manifest and intensifies as the severity of the symptoms increases [47]. Because of abnormal gait and frequent falls, patients with HD have a lower life quality [48].

Spinocerebellar ataxia (SCA)

Spinocerebellar ataxia (SCA) is a rare condition affecting about 3/100,000 individuals [49]. SCAs are a category of clinically and genetically diverse neurodegenerative illnesses characterized by loss of coordination and motor control due to malfunction of the cerebellum and its afferent sensory linkages [50]. Cerebellar atrophy is one of the most conspicuous signs of SCA. Abnormal gait is the most common symptom at SCA onset. Ataxia scales, such as semiquantitative scales, are frequently used to assess clinical gaits in patients with SCA [49].

Gait instability in SCA is due to abnormalities in the cerebral regions that regulate stability during slow walking and a more significant effect of defective intralimb synchronization during rapid walking. As such, in clinical settings, it is essential to examine the ideal walking speed and moderate and rapid walking speeds to identify features of gait instability [51].

Friedreich ataxia

Multiple system atrophy and autosomal recessive ataxias, such as Friedreich ataxia, were found in most cerebellar ataxia gait investigations [49].

Diagnosis of gait disorder

Figure 7 provides a diagnostic approach to categorizing gait abnormalities [52].

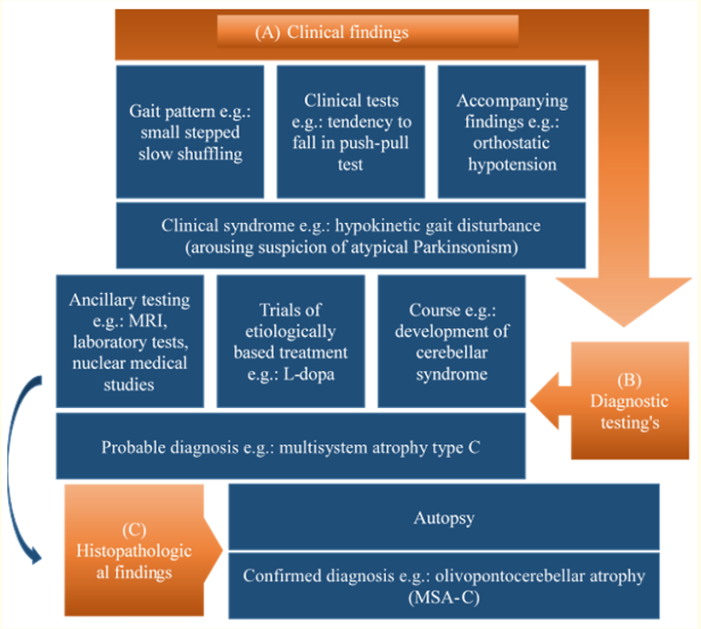

Figure 7: *Diagnostic method for categorization of gait disturbances. Key: A: Initially, classification is based on the clinical description of gait abnormalities. B: A probable diagnosis is formed based on the findings of ancillary tests and the patient's subsequent course. C: Only an autopsy can provide a definitive diagnosis.*

Additional clinical tests for gait, balance, and posture abnormalities, the most significant of which are mentioned in Figure 8, can help discern the differential diagnoses [53].

Test	Function	Implications
Quiet standing with eyes closed; Romberg test	Helps differentiate	Increased sway, or inability to stand independently, can be observed in sensory ataxia (sensory neuropathy) Involuntary movements suggest drug-induced dyskinesias or chorea Excessive sway corrected by distraction is observed in patients with anxiety or functional gait disorders
Response to external perturbation (pull test or push-and-release test)	Detection of symptoms	Patients with postural instability respond with more than two steps or take no steps at all
Walking with eyes closed	Helps differentiate	Deviation to one side is seen in unilateral vestibular ataxia and unilateral cerebellar ataxia
Turning of the head during gait	Helps differentiate	Worsening of gait is seen in vestibular ataxia
Walking backwards compared with walking forwards	Helps differentiate	Discrepant features are seen in dystonia (suggesting task specificity) and functional gait disorders (suggesting inconsistency)
Regular walking compared with running	Helps differentiate	Better running than regular walking can be seen in dystonia, PD and functional gait disorders
Tandem walking	Helps differentiate	Tandem walking without side steps is preserved in PD but impaired in atypical parkinsonism, ataxia and other conditions characterized by mediolateral instability
Rapid 360° turns on the spot	Detection of symptoms	Frequently evokes freezing of gait in PD and atypical parkinsonism
Walking rapidly with short steps	Detection of symptoms	Frequently evokes freezing of gait in PD and atypical parkinsonsim
Forwards dropping of the head (disproportionate antecollis)	Helps differentiate	• MSA, ALS, myasthenia gravis, polymyositis, focal posterior cervical myositis • Drug-induced (dopamine receptor antagonists, amantadine)
Drifting backwards	Detection of symptoms	• PD, atypical parkinsonism (PSP) • Higher-level gait disorders • Trunk dystonia (opisthotonus), often drug-induced (dopamine receptor antagonists)
Excessive trunk flexion that persists when lying down	Detection of symptoms	Vertebral column deformities
Excessive trunk flexion that disappears when lying down	Detection of symptoms	• Trunk or hip weakness (myasthenia gravis, motor neuron disease, myopathies) • Camptocormia (PD, atypical parkinsonism)

Figure 8: *Additional gait and balance tests to obtain details on functional gait and balance characteristics.*

Medical specialties most likely to see patients with gait disorders

Antalgic gait disturbances (e.g. arthritis) and paretic gait disturbances (e.g. radiculopathy following disk herniation) represent a significant proportion of gait abnormalities observed by physicians [52]. The most prevalent neurological reasons are sensory ataxia due to peripheral neuropathy, PD, and cortical gait problems related to subcortical vascular encephalopathy or diseases associated with dementia [1].

Treatment of gait disorders

Figure 9 details different treatment options for gait disorders [54].

	Pathologic Gait	Treatment
Musculoskeletal etiologies	Pain: "Antalgic gait"	Treat underlying cause, analgesia, assistive device (AD) in contralateral hand
	Leg length discrepancy (LLD)	For <2cm: no treatment. For >2cm: shoe lift or consider surgery. Lift <2cm inside shoe, >2cm outside shoe. Surgical options: shortening surgery (epiphysiodesis or femoral shortening), lengthening surgery (femur/tibia), correction muscle or joint contracture.
Neuromuscular etiologies	Lower Motor Neuron Lesion (LMNL) (or other causes of muscular weakness) 1) Hip abductor weakness (Trendelenburg gait)	AD in contralateral hand, gluteus medius strengthening.
	2) Hip extensor weakness (Posterior lurch gait)	Strengthen gluteus maximus. Keep compensatory mechanism, do not fix lumbar hyperlordosis
	3) Knee extensor weakness: Knee buckling (uncompensated), genu recurvatum (compensated)	Ankle Foot Orthosis (AFO):11(p342) Solid AFO set at a few degrees of plantar flexion or a hinged AFO with a neutral dorsiflexion stop. Quadriceps strengthening.
	4) Ankle dorsiflexion weakness (Steppage gait and foot slap)	Posterior leaf spring AFO or hinged AFO with dorsiflexion assist or plantar flexion stop for mediolateral instability. Role of electrical stimulation (ES) to prevent atrophy of anterior tibialis and/or functional ES (FES) to activate ankle dorsiflexor.
	5) Ankle Plantar Flexor Weakness (Calcaneal gait)	Hinged AFO with dorsiflexion stop or solid AFO set at a few degrees plantar flexion to pass GRF anterior to knee to prevent buckling.
	6) Myopathic gait (Waddling gait)	Submaximal aerobic and low-resistant strength training, prevent fixed contractures by stretching and night-time braces. Lengthening surgery may be considered to prolong ambulation in patients with good muscle strength limited by contractures.
	7) Neurogenic Claudication: Pain and neurological deficits with ambulation	Lumbar brace and physical therapy to correct lordotic posture. May need surgical decompression of spine if symptoms not better with conservative measures
	Upper Motor Neuron Lesion (UMNL) (or other causes involving the central nervous system) 1) True Equinus Gait	Hinged AFO with dorsiflexion assist and/or plantar flexion stop; chemoneurolysis of gastroc-soleus muscle; surgical treatment is Tendo-Achilles Lengthening (TAL).
	2) Jump Gait	Hinged or solid AFO, according to the integrity of the plantar-flexion, knee-extension couple. Single event multi-level chemoneurolysis or single event multi-level surgery of hamstring, iliopsoas, gastroc-soleus +/- rectus femoris. Selective dorsal rhizotomy. It is important to distinguish jump knee with true equinus from apparent equinus.
	3) Apparent Equinus	Single-event multi-level chemoneurolysis or surgical lengthening of iliopsoas/hamstrings +/- gastrocsoleus.
	4) Crouch Gait	Single-event multi-level chemoneurolysis in younger and less involved children. Lengthening of hamstrings and iliopsoas, adequate correction of bony problems (medial femoral torsion, lateral tibial torsion), foot stabilization in older children with contractures. Solid ground reaction AFO (GRAFO) or articulated GRAFO with dorsiflexion stop only with fully knee extension (after chemoneurolysis or surgery), otherwise solid AFO.

Figure 9: *Treatment options for gait disorders.*

Summary

Statistical gait measures, such as stride-to-stride variability, are sensitive predictors of neurological deficits [55]. They can also predict future mobility impairment [56] and recurrent dementia [57,58]. Steadily increasing gait variability is observed in patients with neurological diseases that impact cognition, indicating the association of brain circuits engaged in gait regulation with cognitive abilities [59].

For example, gait variation increases from an early phase of AD dementia when mesial-temporal regions are highly affected, among other regions [60–64]. High gait variability is observed in patients with Lewy body dementia, in which the subcortical areas and the basal ganglia are primarily damaged, and bvFTD, in which the frontal and subcortical regions are primarily affected than other regions [65,66]. Gait variation increases in patients with PD as cognitive impairment manifests [67,68].

Future of gait analysis

Gait assessment has considerably advanced in the past century. Significant efforts have been made to build the equipment necessary for human movement analysis since the pioneering work of Braune and Fisher. Hand digitizing has been supplanted by automated motion tracking systems. Hand palpitation has been supplanted by computer-controlled EMG equipment. Gait analysis technology and expertise have evolved to the point where quick analysis is possible [69]. Two new vital technologies in the current gait analysis are detailed in Figure 10 [70].

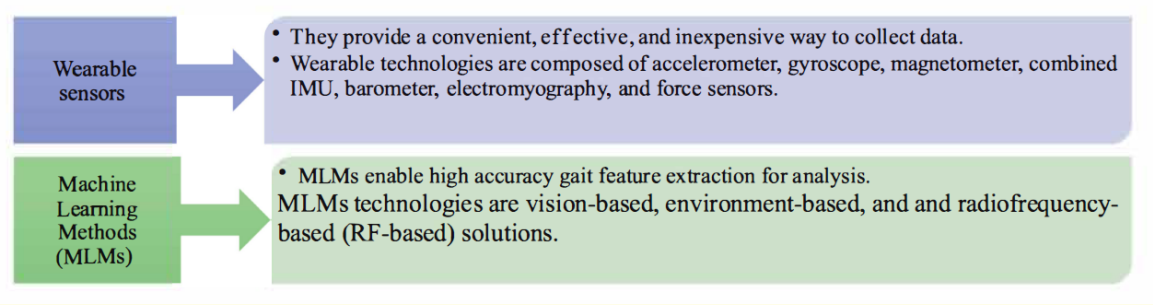

Figure 10: *Contemporary technologies critical for gait analysis.*

Conclusion

This article aimed to review studies regarding the associations between neuropathology and gait abnormalities. Moreover, gait can be used to potentially identify—early on—some of the most severe neurological conditions, such as AD, PD, and HD. Patients with these neurological conditions often have extremely unusual gaits, which motivates researchers and clinicians to target a patient's gait for possible identification. Importantly, gait abnormalities are not an unavoidable result of aging, rather they can indicate underlying conditions that require particular diagnostic procedures.

Wearable technology and machine learning approaches are currently being applied in gait analyses. Continuing and novel research will strengthen the fundamental knowledge of gait disorders, opening new possibilities for better managing this prevalent and debilitating condition.

Conflict of Interest Statement

The authors declare that this paper was written without any commercial or financial relationship that could be construed as a potential conflict of interest.

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