

Impaired Control

Pathophysiology – Stroke – Gait Implications

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Normal Motor Control

Normal walking requires:

- **Precision**
- **Coordination**
- **Speed**
- **Versatility**

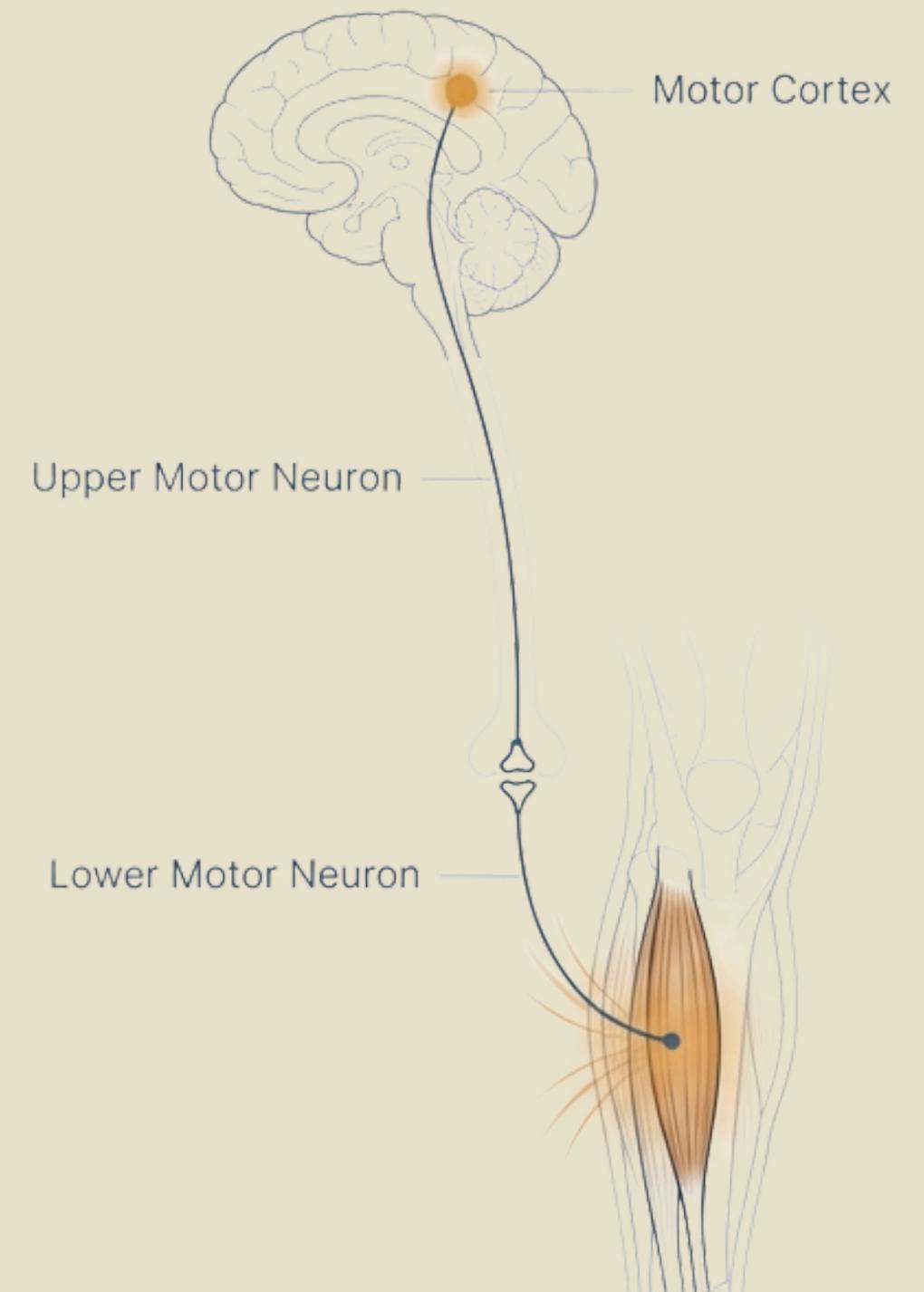
These features depend on selective muscle activation

Motor control is coordinated by:

- **Motor cortex**
- **Sensory feedback from:**
 - Peripheral nervous system
 - Cerebellum
 - Basal ganglia
 - Brainstem

Upper & Lower Motor Neurons

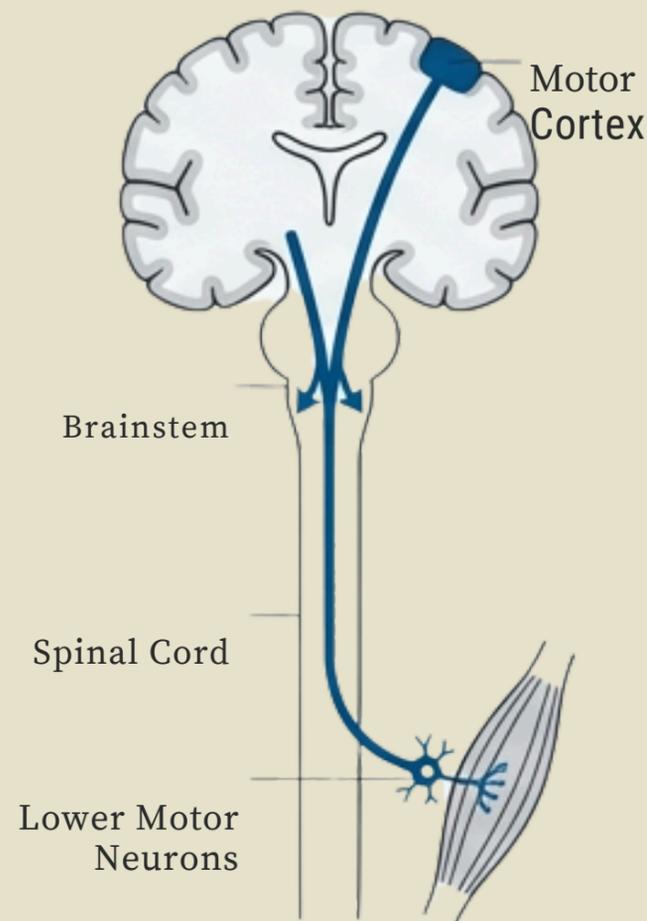
Purposeful movement is generated in the motor areas of the cerebral cortex and transmitted through long upper motor neuron axons to the spinal cord. These upper motor neurons synapse directly with lower motor neurons in the anterior horn, which activate motor units in skeletal muscle. This direct connection allows fast and precise execution of voluntary movement.



The Two Systems of Motor Control

Lateral Corticospinal Tract (Pyramidal Tract)

The Lateral Corticospinal Tract



- Main pathway for selective motor control
- Acts as a “motor expressway”

Features:

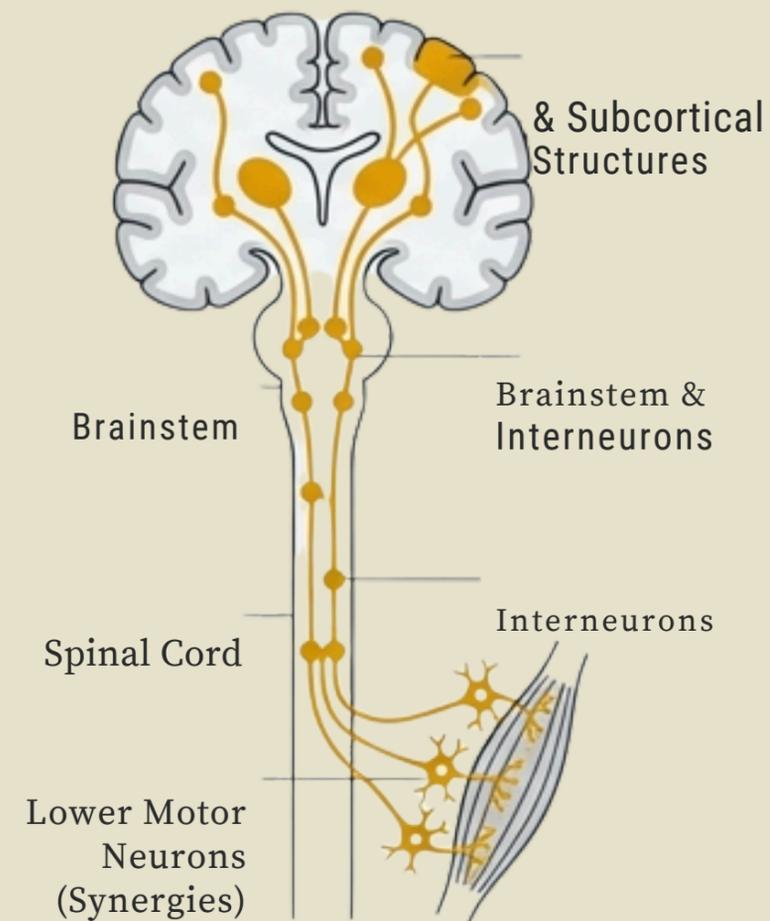
- Direct UMN to LMN synapses
- Near one-to-one muscle representation

Responsible for:

- **Precise**
- **Fast**
- **Isolated movements (especially distal muscles)**

Extra-Pyramidal Motor Systems

The Medial Corticospinal Tract



- More primitive motor system

Origin:

- Deeper motor cortex
- Midbrain
- Basal ganglia
- Brainstem

Characteristics:

- Less selective
- Activates muscles in synergy

Main functions:

- Postural control
- Multisegmental reflexes
- Flexor & extensor limb synergies

Postural control and multisegmental reflexes are their main function. The neurons involved with limb motion combine the hip, knee, and ankle muscles into flexor or extensor synergies. Cortical input provides voluntary activation of the synergies but cannot select a single joint.

Causes of Impaired Selective Motor Control

Damage to:

- Motor cortex
- Cervical or thoracic spinal cord

Clinical conditions:

- Stroke (CVA)
- Traumatic brain injury
- Tetraplegia / paraplegia
- Multiple sclerosis
- Cerebral palsy
- Hydrocephalus
- Infections
- Tumors

Stroke

*Hemiplegia, defined as unilateral paralysis of the **arm and leg**, is the **most common** motor impairment following a **cerebrovascular accident (CVA)**. A **stroke** occurs when **blood flow to a specific area of the brain is abruptly interrupted**, either by arterial occlusion (**thrombus**) or by vessel rupture (**hemorrhage**).*

Causes and Vascular Involvement of Stroke

Thrombotic occlusion Most common cause 76%:

- Internal carotid artery
- Middle cerebral artery

Vertebral artery occlusion:

- Causes brainstem strokes
- Represents ~15% of cases
- **Hemorrhagic stroke**
- Less common
- Accounts for ~9% of cases
- **Clinical implication**
- Brain has variable vascular supply
- Lesion location and size vary
- Early prediction of disability is difficult



Early Motor Control & Neural Mechanisms

During early recovery:

Hyper-reflexia persists

Spasticity is mild

Contractures are generally minimal

Knowledge of motor control:

Largely derived from animal studies

These may be misleading:

Humans lack primitive neural pathways

Animals can walk independently at birth

Human motor control is more complex and less primitive

Pyramidal Tracts, Primitive Control & Plasticity

Travis & Woolsey (1955):

Bilateral section of pyramidal tracts in monkeys

Initial result:

Severe loss of mobility (stroke-like rigidity)

True cause:

Contractures, not loss of neural control

With repeated ROM exercises:

Independent mobility returned

Pyramidal Tracts, Primitive Control & Plasticity

Primary deficit after pyramidal tract damage:

Loss of precise distal motor control

Basic movements preserved via primitive control centers

in humans after severe stroke:

Primitive flexor and extensor synergies may appear

Modern research shows neuroplasticity:

Brain recruits adjacent neurons

Possible formation of new neural connections

Recovery remains highly variable, complicating rehab planning

Classification of Stroke Disability & Gait Patterns

Stroke disability varies due to:

Extensive and variable cerebral blood supply

Mulroy's cluster analysis (gait-based classification)

Key parameters:

Walking velocity

Mid-swing ankle dorsiflexion

Mid-stance knee flexion

Classification of Stroke Disability & Gait Patterns

Four functional walking groups:

FAST: 44% of normal velocity

MODERATE: 21% of normal velocity

SLOW-EXTENDED: 11%

Uses gluteus maximus

Mild knee hyperextension ($\sim 6^\circ$)

SLOW-FLEXED: 10%

Adequate quadriceps strength

Knee flexion $\sim 23^\circ$

Clinical value:

Improves gait analysis

Guides rehabilitation strategies

1. Gait Assessment

Classification: Considered a "fast walker" post-stroke.

Performance: Walking speed and stride length (63%) are below normal, while cadence (87%) is close to normal.

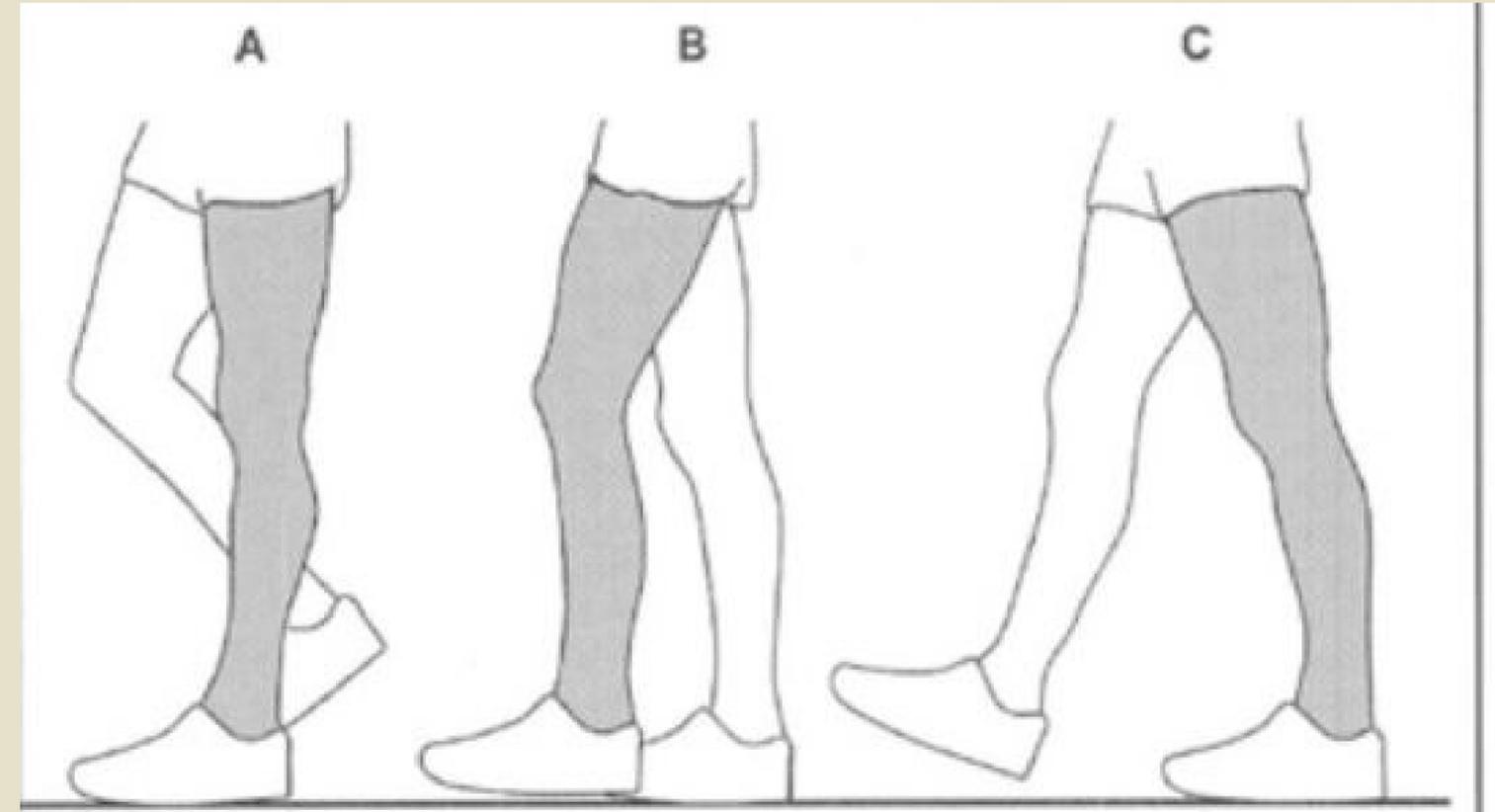
Normal Phases: Mid-stand and mid-swing events were completely normal.

2. Mechanical Problem (Major Malfunction)

Heel Lift Failure: The primary problem is the failure of the heel to lift off the ground at the end of the stand phase.

Poor Timing: The heel only lifts after the other foot has touched the ground, reducing the fluidity of the movement.

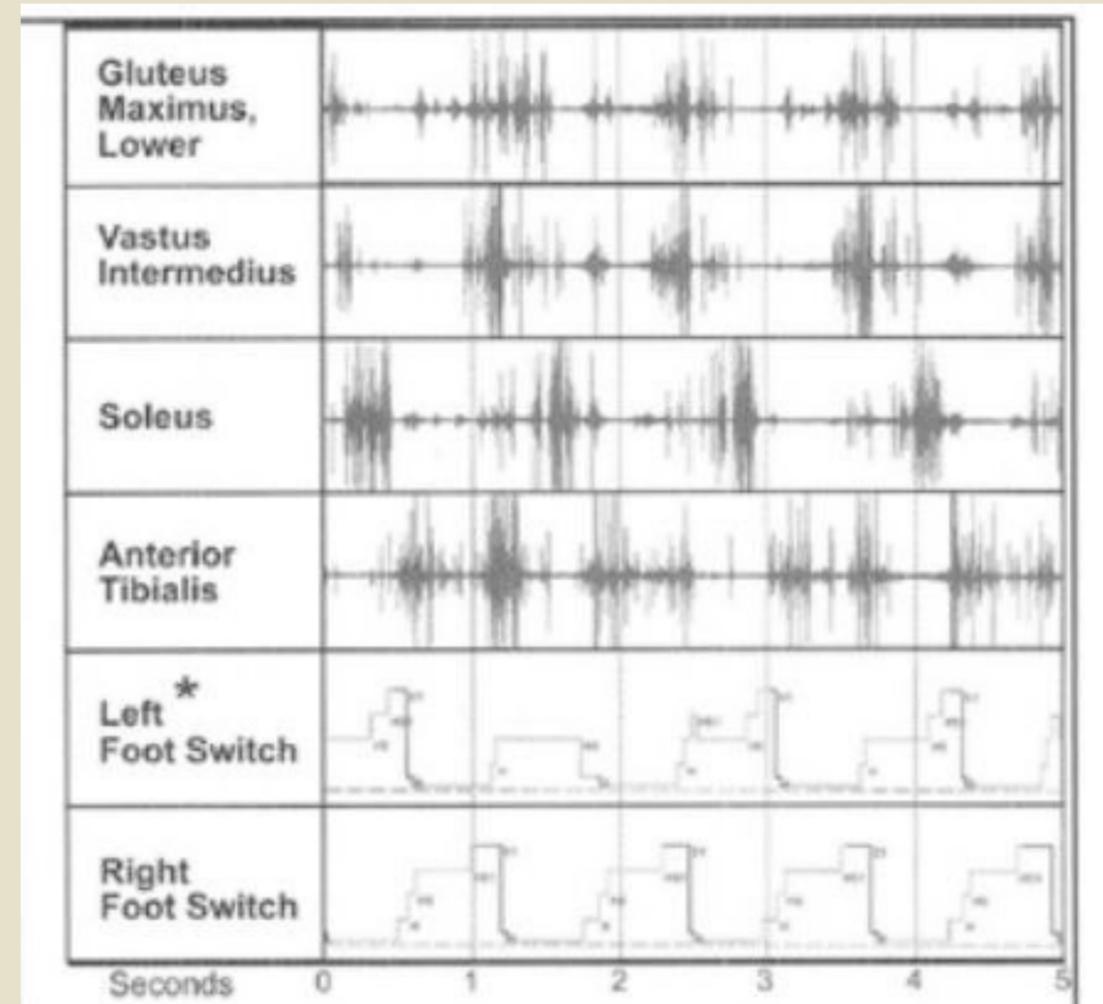
Knee Hyperextension: A tendency towards knee hyperextension was observed to compensate for the lack of propulsive force.



3. Muscle Analysis (EMG & MMT)

Impaired Muscle: Weakness in the plantar flexors, specifically the soleus muscle, was assessed as "moderate". Locking failure: The weak soleus muscle was unable to lock the ankle, preventing smooth weight transfer to the forefoot.

Activity of other muscles: Slightly prolonged activity was recorded in the gluteus maximus and hamstring (VI) muscles in an attempt to stabilize the ankle.



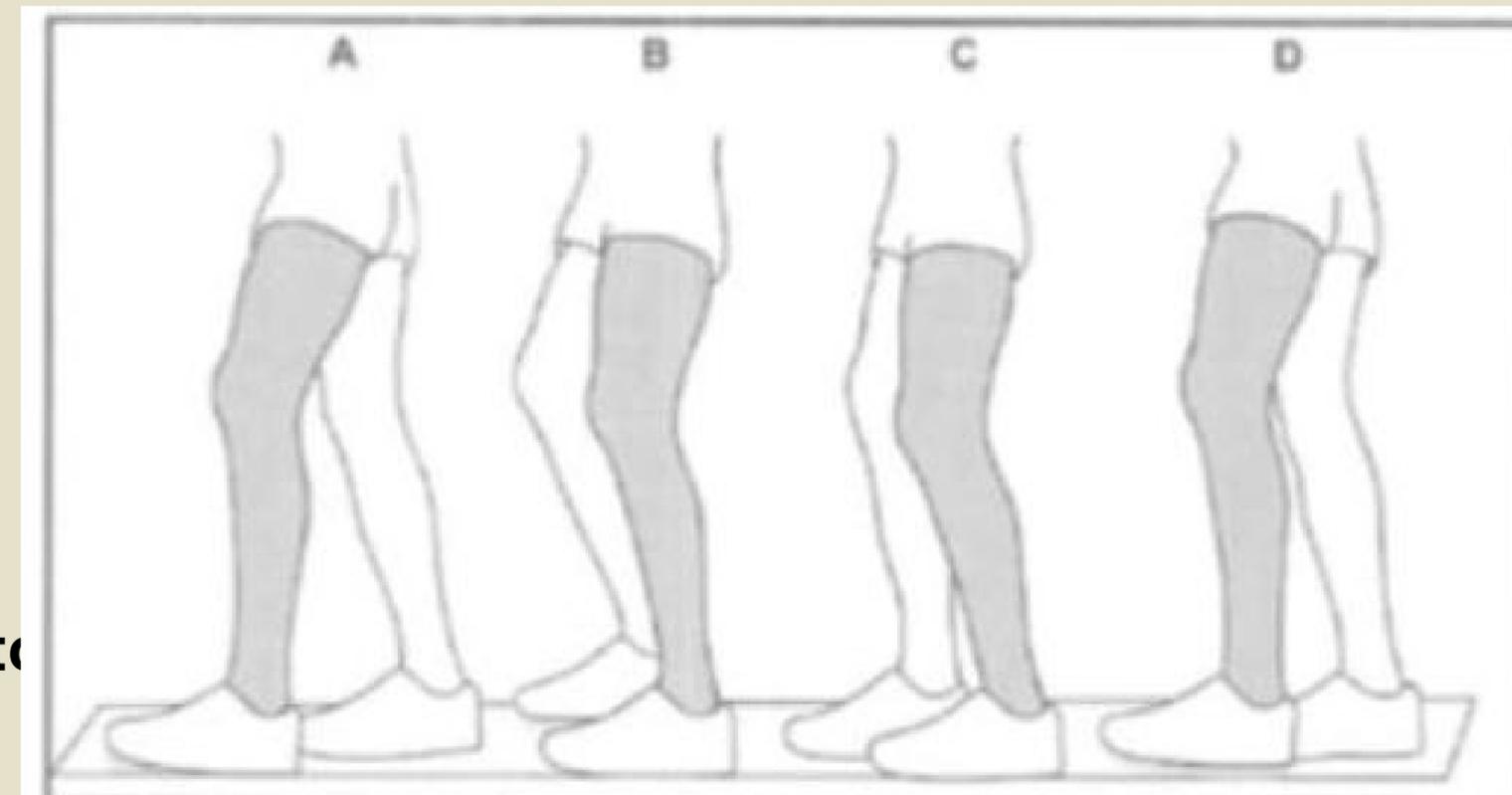
Stages of Walking (Figure 15-18 A–D)

A – Initial Contact (IC): Heel low, knee flexed.

B – Intermediate Standing: Increased knee and hip flexion, and excessive dorsiflexion of the ankle.

C – Push-Off Stage: Weak forward advance, decreased ankle and knee flexion, and no heel lift.

D – Intermediate Swing: Ankle reaches neutral position to provide space for the foot.



Electromyography (Figure 15-18E)

Solis: Weak activity, insufficient to propel the leg forward.

VI (medial quadriceps): Continuous activity stabilizes the flexed knee during single-leg standing.

TA (tibialis anterior): Continuous activity achieves neutral ankle position during swing and assists in trunk forward advancement.

Note: Muscle activity begins before heel contact with the ground.

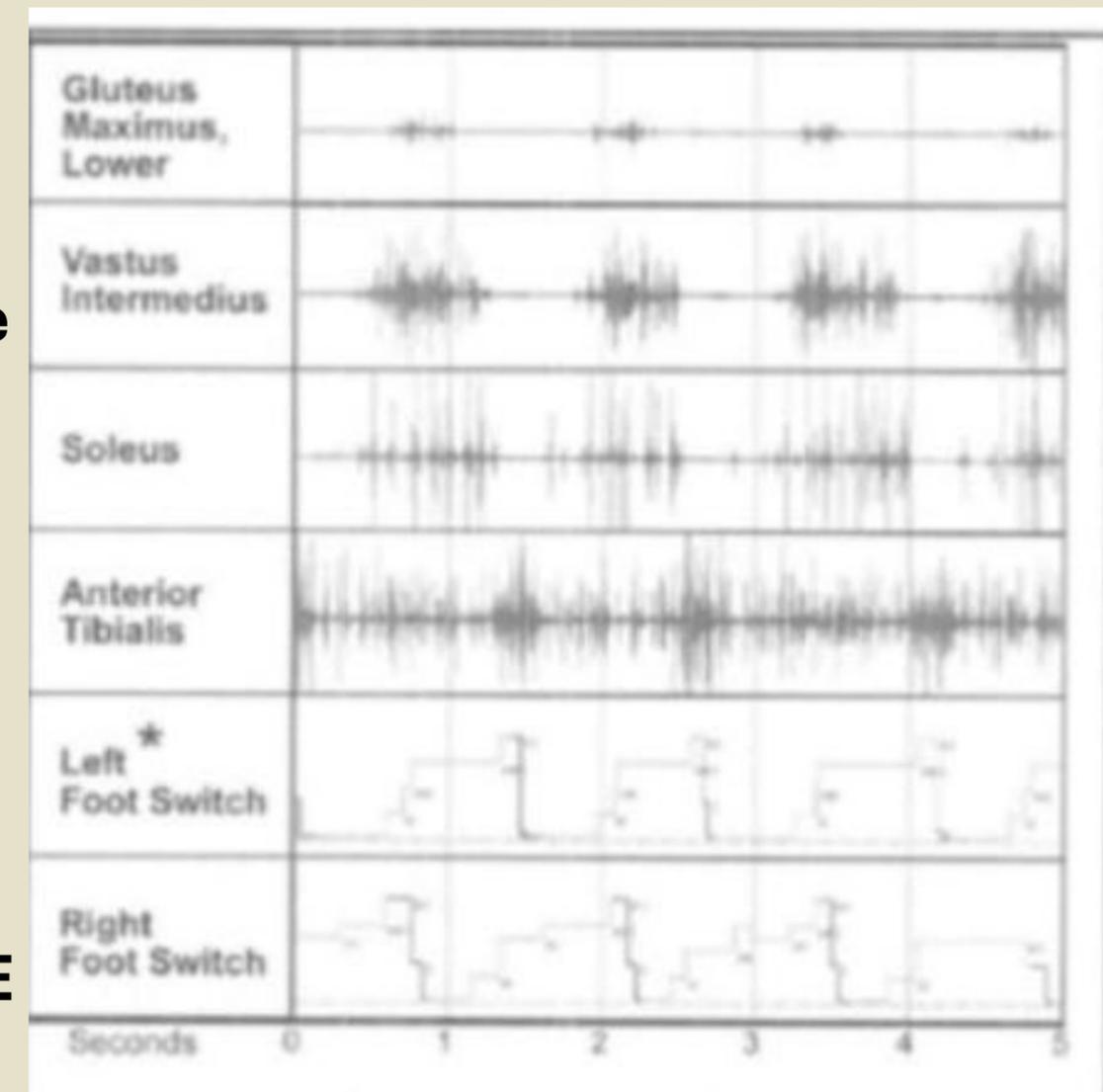
Patient K is described as having a moderate gait (Class II: MODERATE Walker)

Age: 44 years.

Walking speed: 32% of normal.

Stride length: 38% of normal.

Cadence: 83% of normal.



Kinesthetic characteristics:

Heel is low on initial contact.

Excessive knee flexion persists during standing.

Foot proximity reduces stride length.

The propulsion phase is limited, and knee flexion is weak.

The ankle reaches a neutral position during swing.

Manual muscle testing (MMT)

Plantar flexors: Weak, solitary flexor very weak (26%).

Gluteus maximus: Moderate assessment, low activity does not provide sufficient hip stability.

VI: Moderate to above average assessment (27%), stabilizes flexed knee.

TA: Good assessment, achieves sufficient ankle flexion and helps in thigh advancement.

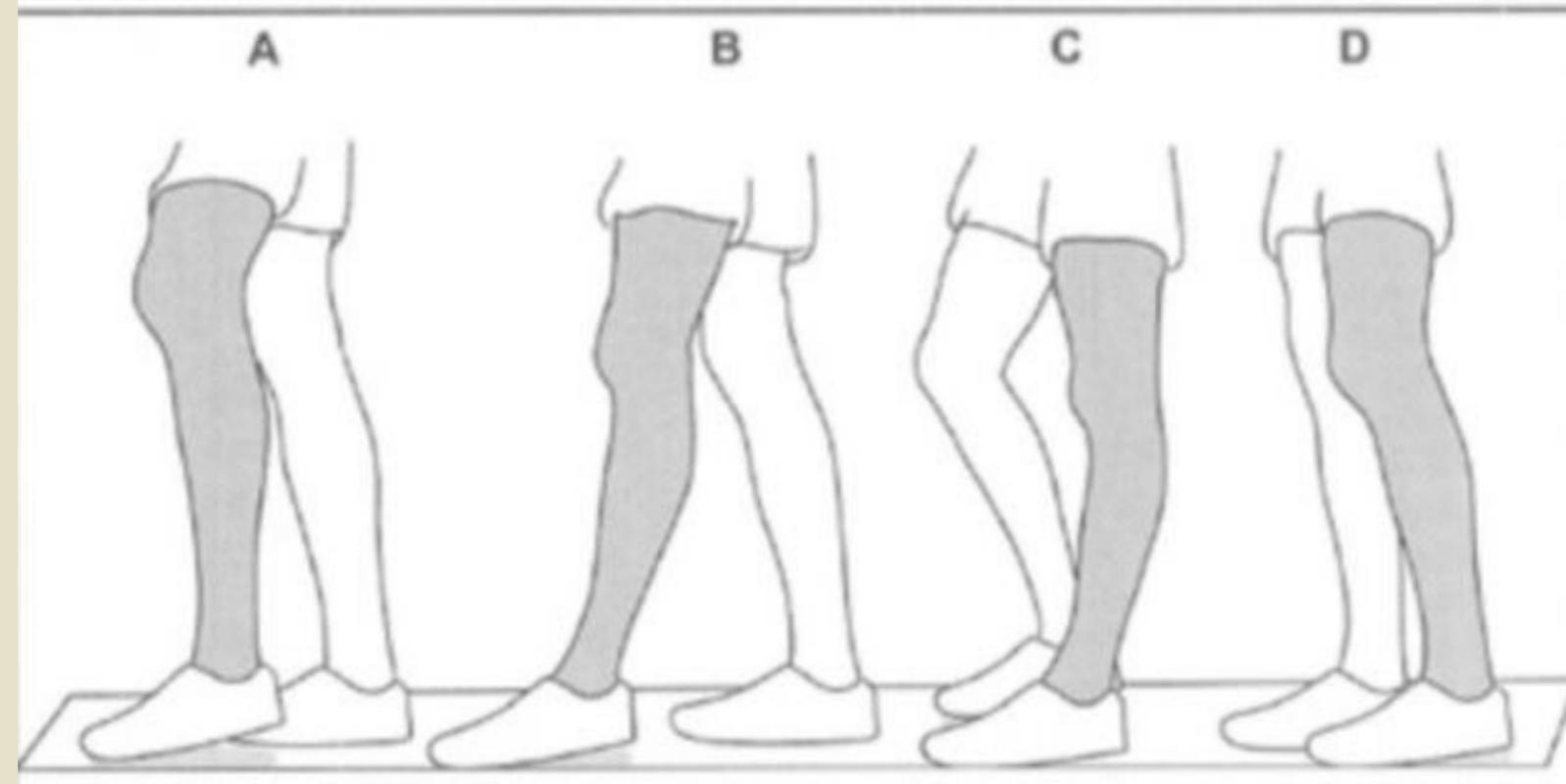
Gait Phases (Figure 15-19 A–D)

A – Moderate Swing: Excessive ankle flexion (plantar flexion) due to weakness in the pretibials.

B – Initial Contact (IC): Excessive ankle flexion continues, resulting in the forefoot first touching the ground.

C – Moderate Standing: Gluteal contraction leads to knee hyperextension and protects the weakened quadriceps.

D – Push-Off Phase: Excessive ankle flexion continues, resulting in a shortened stride.



 **Gait Analysis of Patient L (Classification: SLOW-EXTENDED Walker)**

Age: 45 years.

Walking Speed: 17% of normal.

Kinesthetic Characteristics

Excessive ankle flexion during moderate swing.

Forefoot touching the ground at initial contact.

Knee hyperextension with flat feet during standing.

Retroversion of the shins while standing limits forward advance and shortens the stride.

Manual Muscle Testing (MMT) and EMG Results

Gluteus Maximus: "Fair minus" rating, normal dense overlap pattern.

Vastus Intermedius and Soleus: Weak EMG signals, "Fair minus" and "Poor minus" ratings, respectively.

Anterior Tibialis: No EMG signal, 0/5 rating (complete absence of activity).

Additional Notes:

Gluteus maximus activity begins early in the intermediate swing, followed by a brief second burst of activity during standing.

The gluteus maximus contributes to thigh extension during single-leg standing and compensates for quadriceps weakness.

The sciatic nerve (V) and soleus show no functional activity.

The ground contact pattern varies between the varus (H-5), valgus (H-1), and neutral (H-5-1) positions.



Post-Stroke Gait Patterns: Slow-Extended and Slow-Flexed Walkers



introduction& Walking Speed

Post-Stroke Gait Patterns

- Two hemiplegic gait patterns:
- Slow-Extended Walker
- Slow-Flexed Walker
- Walking velocity severely reduced:
- Slow-Extended: 17% of normal
- Slow-Flexed: 9% of normal
- Both patterns show:
- Shortened step length
- Reduced cadence
- Inefficient gait

Slow-Extended Walker

Key Kinematic Features

- Persistent ankle plantarflexion (equinus)
- Forefoot-first initial contact
- Knee hyperextension during mid-stance
- Posterior tibial tilt limits contralateral step length

Compensation strategy: mechanical stabilization of the limb

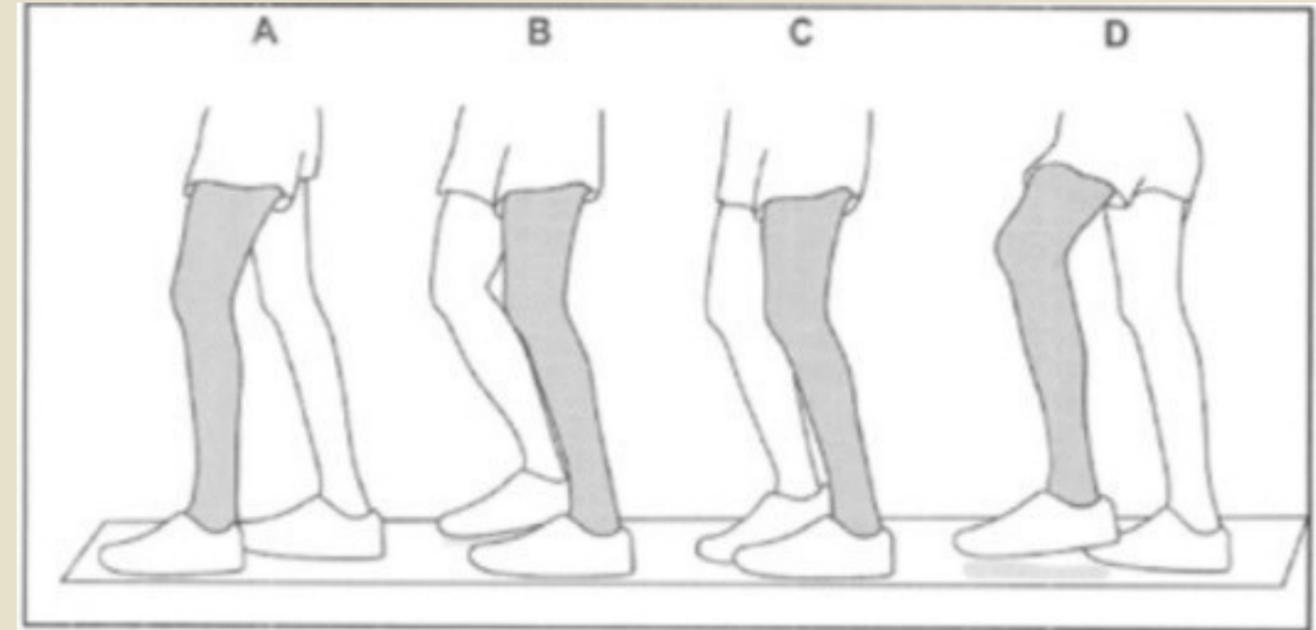
Slow-Flexed Walker

Key Features

- Flexed knee posture from initial contact
- Excessive ankle dorsiflexion
- Forward tibial collapse during stance
- Very short step length and slow cadence

EMG Pattern

- Weak extensor muscles
- Prolonged soleus activity
- Extensor synergy (poor selective control)



Clinical Interpretation & Conclusion

Clinical Implications

- Both patterns result in:
- Slow, inefficient gait
- Poor stability
- Main difference:
- Slow-Extended → knee hyperextension for stability
- Slow-Flexed → limb collapse due to weak extensors
- EMG helps guide:
- Rehabilitation planning
- Targeted muscle retraining

Hip Deviations: The Core of Impaired Mobility



Excessive Flexion

- Caused by spasticity or contracture of the iliopsoas, sartorius, or rectus femoris.
- Limits the "trailing limb" posture in terminal stance, shortening step length.
- Increases anterior pelvic tilt, often leading to compensatory lumbar lordosis.



Femoral Anteversion & Lever Arm Syndrome

- Children with CP often retain persistent femoral anteversion due to failed derotation.
- Results in Lever Arm Syndrome, where muscles operate biomechanically inefficiently.
- Often causes "intoeing," sometimes misdiagnosed as tight adductor muscles.
- May lead to compensatory external tibial torsion and valgus foot/ankle positioning.





Comprehensive Classification for Locomotor Function



GMFCS: Gross Motor Function Classification Scale

The gold standard for describing locomotor function, ranging from Level 1 (independent walking/running) to Level 5 (requiring extensive assistance for mobility).



FMS: Functional Mobility Scale

Complements GMFCS by classifying walking ability over specific distances: 5, 50, and 500 metres, providing practical insights into functional ambulation.

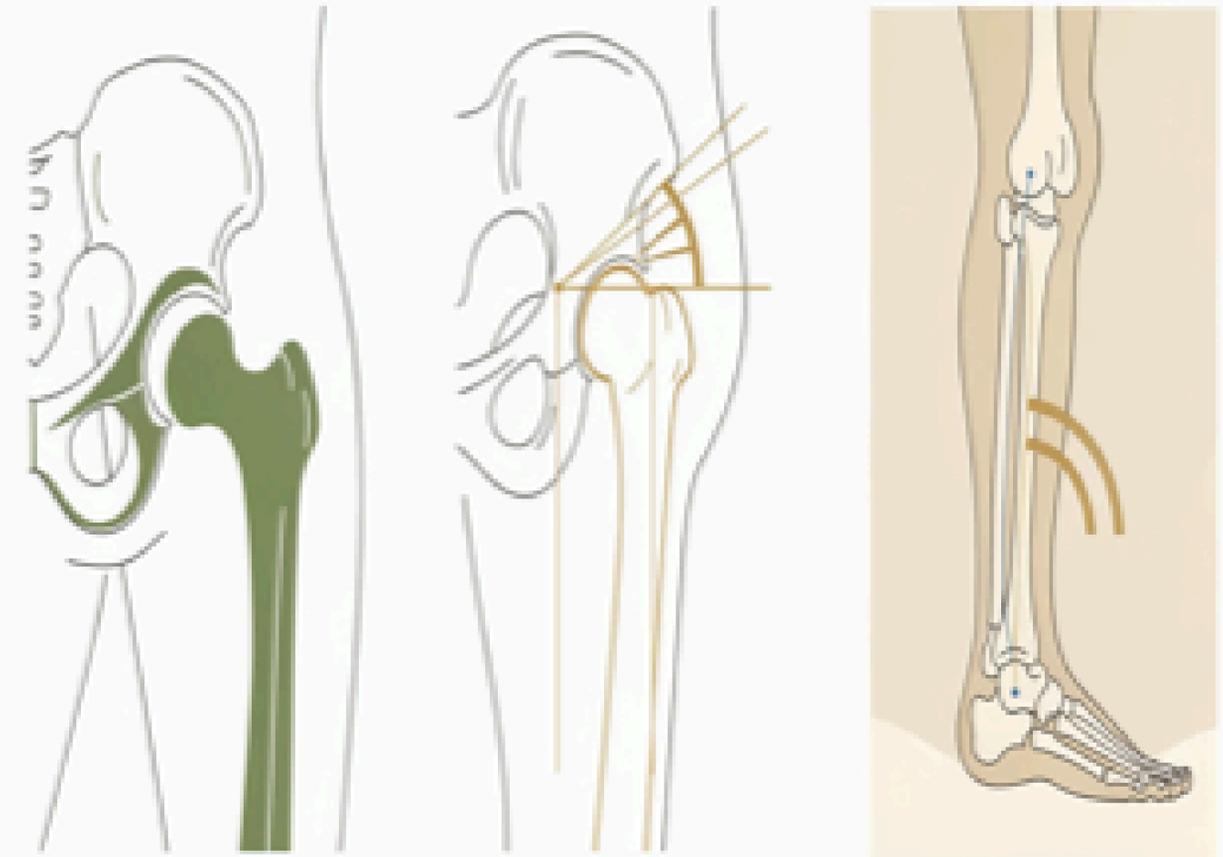


Gillette Gait Index

Quantifies gait "normalcy" using 3D analysis to measure deviations from standard patterns, offering objective data for clinical assessment.

Unravelling Paediatric Gait Deviations in Cerebral Palsy

Understanding the intricate gait patterns in children with cerebral palsy (CP) is paramount for effective rehabilitation. This presentation delves into the classifications, common hip, knee, and foot/ankle deviations, and the crucial role of quantified motion analysis.



Femoral Anteversion: A Detailed Look

Persistent femoral anteversions significantly impact gait, manifesting in several ways:



Misleading Presentation

The appearance of crossed hips due to intoeing can lead to incorrect diagnoses of adductor tightness.



Biomechanical Inefficiency

Muscles function sub-optimally, leading to increased energy expenditure and reduced power during ambulation.



Compensatory Adaptations

Over time, the body adapts through external tibial torsion and valgus positioning, further complicating gait mechanics.



Knee Deviations: Impact on Stance and Swing



Jump Gait

Characterised by increased anterior pelvic tilt, hip flexion, knee flexion, and ankle plantarflexion (equinus), creating a "bouncing" appearance.

These patterns highlight the complex interplay of muscle spasticity and weakness affecting knee joint stability.



Crouch Gait

A challenging pattern involving excessive knee flexion during stance and inadequate flexion during swing, often coupled with ankle calcaneus (hyperdorsiflexion).

These patterns highlight the complex interplay of muscle spasticity and weakness affecting knee joint stability.

Foot and Ankle Deviations: Foundation of Gait Instability

Equinus

The most common abnormality in CP, caused by gastrocnemius-soleus spasticity/contracture, leading to forefoot or toe walking.

Calcaneus

Excessive ankle dorsiflexion during stance, frequently an iatrogenic problem due to over-lengthening of the Achilles tendon post-surgery.

Varus and Valgus

Coronal plane deviations that destabilise the foot, often occurring alongside equinus or calcaneus, further compromising balance.



Equinus: The Prevalent Abnormality

Equinus gait significantly impacts a child's ability to achieve a flat-foot stance, leading to:

- Reduced ground contact area, affecting balance and stability.
- Increased pressure on the forefoot, potentially causing skin breakdown and pain.
- Compensatory strategies in the knee and hip, further propagating gait deviations.
- Difficulties with shoe wear and participation in daily activities.

Early identification and intervention are crucial to mitigate its long-term effects.





Iatrogenic Calcaneus: A Surgical Consequence

"Excessive ankle dorsiflexion during stance is frequently an iatrogenic problem, resulting from the over-lengthening of the Achilles tendon during surgery."

While surgical intervention aims to correct gait abnormalities, precise execution is vital. Over-lengthening of the Achilles tendon can lead to a new set of challenges:

- Loss of plantarflexion power, crucial for push-off during gait.
- Increased energy cost of walking due to inefficient biomechanics.
- Potential for secondary deformities in the foot and ankle.

The Need for Quantified Motion Analysis



Limitations of Visual Inspection

While useful for initial assessment, visual observation alone cannot capture the subtle, complex nuances of paediatric neurological control in gait.



Precision of 3D Analysis

Quantified Motion Analysis provides objective, measurable data on joint angles, forces, and temporal parameters, revealing hidden patterns.



Informed Decision-Making

This data is indispensable for guiding surgical planning, optimising therapeutic interventions, and monitoring treatment efficacy.

It moves beyond mere observation to provide an evidence-based foundation for personalised care.

Conclusion: Advancing Paediatric Gait Rehabilitation

The complexities of paediatric gait deviations in cerebral palsy necessitate a multi-faceted approach, integrating robust classification systems with detailed biomechanical understanding.

Holistic Assessment

From GMFCS to understanding specific hip, knee, and ankle pathologies, a thorough evaluation is key.

Evidence-Based Intervention

Leveraging Quantified Motion Analysis ensures that interventions are precisely tailored to each child's unique needs.

Optimising Outcomes

Ultimately, this comprehensive understanding empowers clinicians to enhance mobility and improve the quality of life for children with CP.

