Pathologic Gait

Joseph E. Burris, MD
Professor of Clinical PM&R
University of Missouri

Overview: etiology of pathologic gait deviations

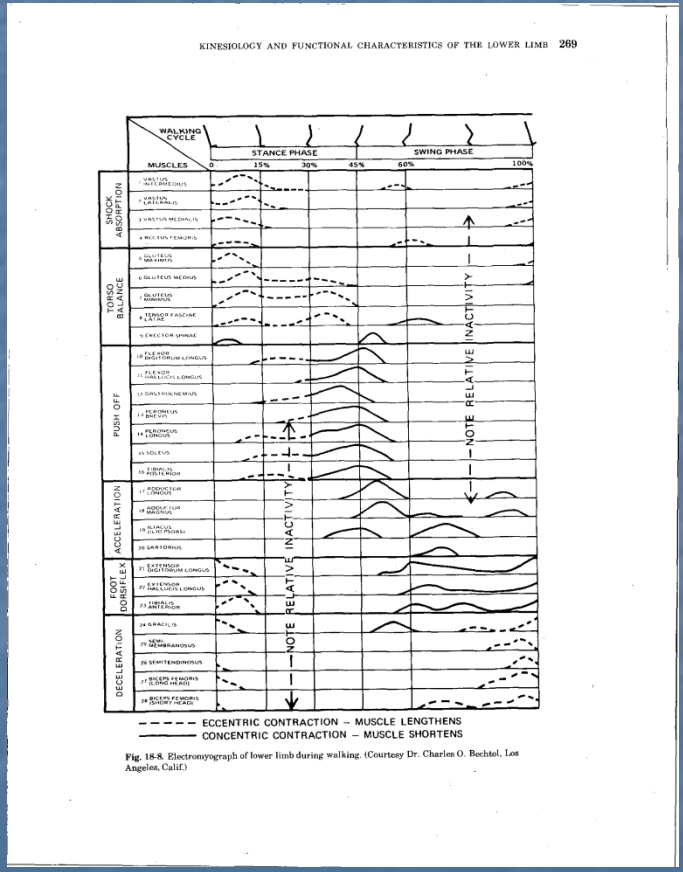
- Compensation of gait:
  - Muscle weakness
  - Pain
  - Soft tissue injury
  - Bony injury
  - Neurologic dysfunction

History and Physical Exam

- History
  - Congenital
  - Acquired
  - Degenerative
- Examination
  - Musculoskeletal
    - ROM
    - Joint
    - Soft tissue
      - Muscle
      - Connective tissue
    - Bone
- Neurologic
  - General
  - Focal
  - Paralysis
    - Spastic
    - Flaccid
  - Sensation
    - Pain
    - Light touch
    - Proprioception
  - Balance
    - Central
    - Vestibular
    - Visual

Motor Control

- Recruitment
  - Timing
  - Quantity
- "Derecruitment"
  - Timing
  - Quantity



Other Factors

- Cardiac
- Pulmonary
- Fatigue

Gait Analysis

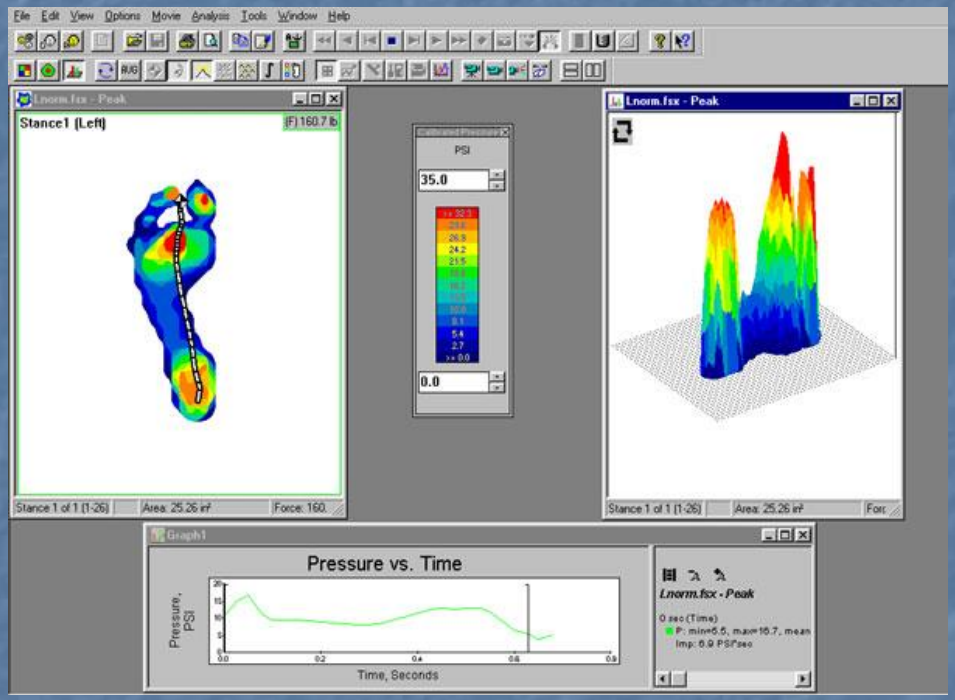
- Kinematics
  - Temporal and spatial joint/limb movement
- Qualitative
  - Observational gait analysis
- Quantitative

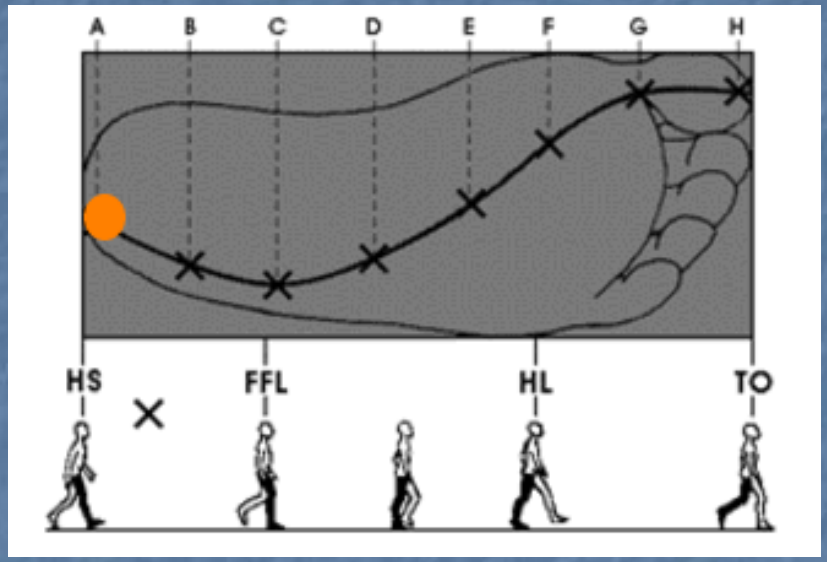
- https://www.youtube.com/watch?v=-WnLC-yJBwo&feature=player_embedded

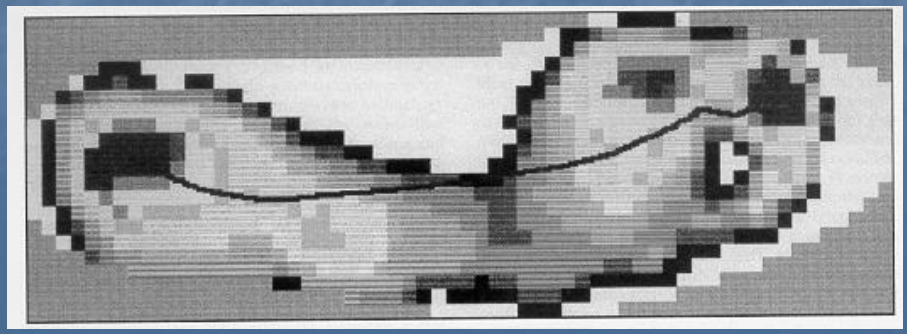
Gait Analysis

- Kinetics
  - Forces/torques that produce joint/limb movement

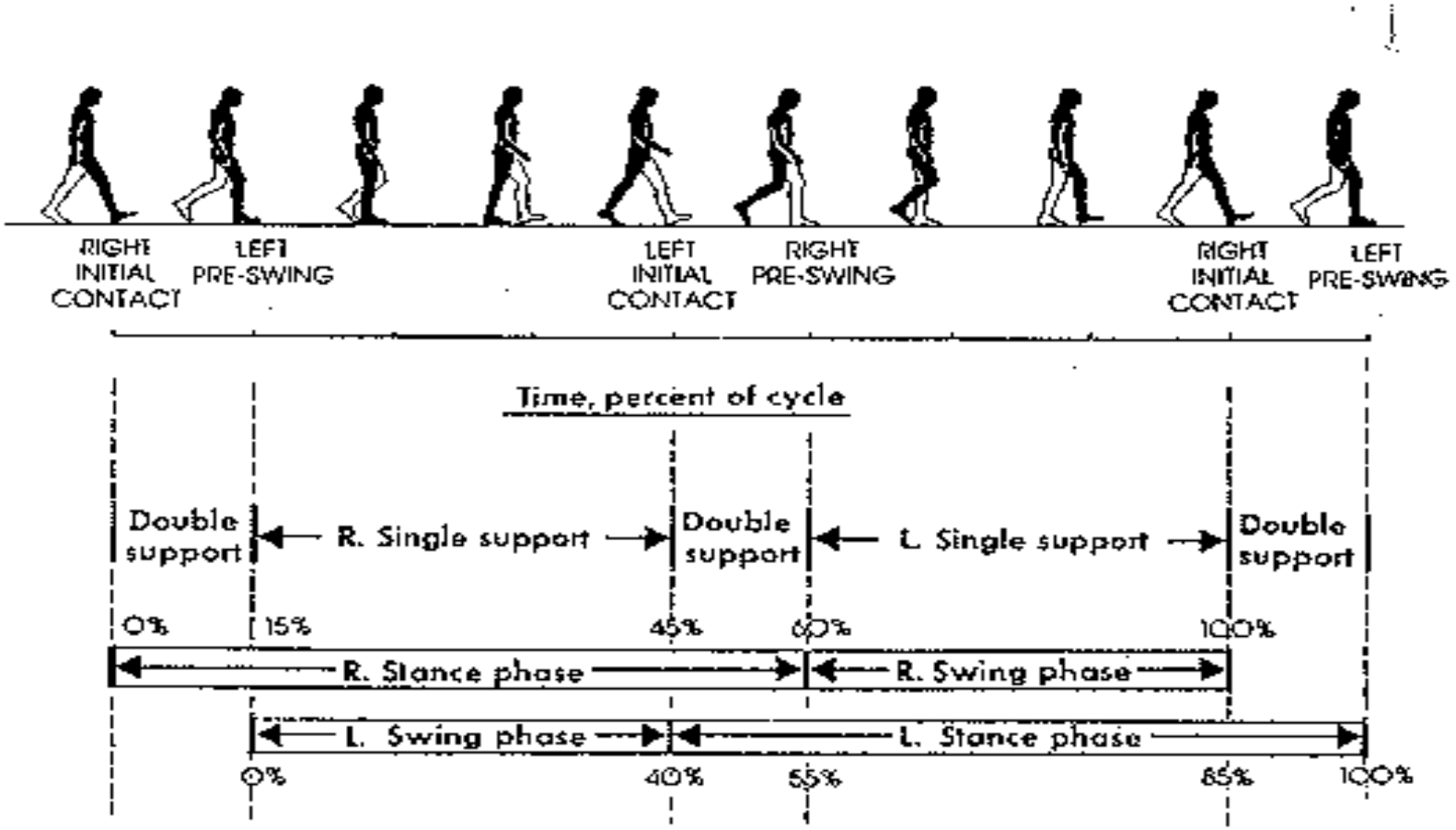
Summary of Pressures during Stance Phase



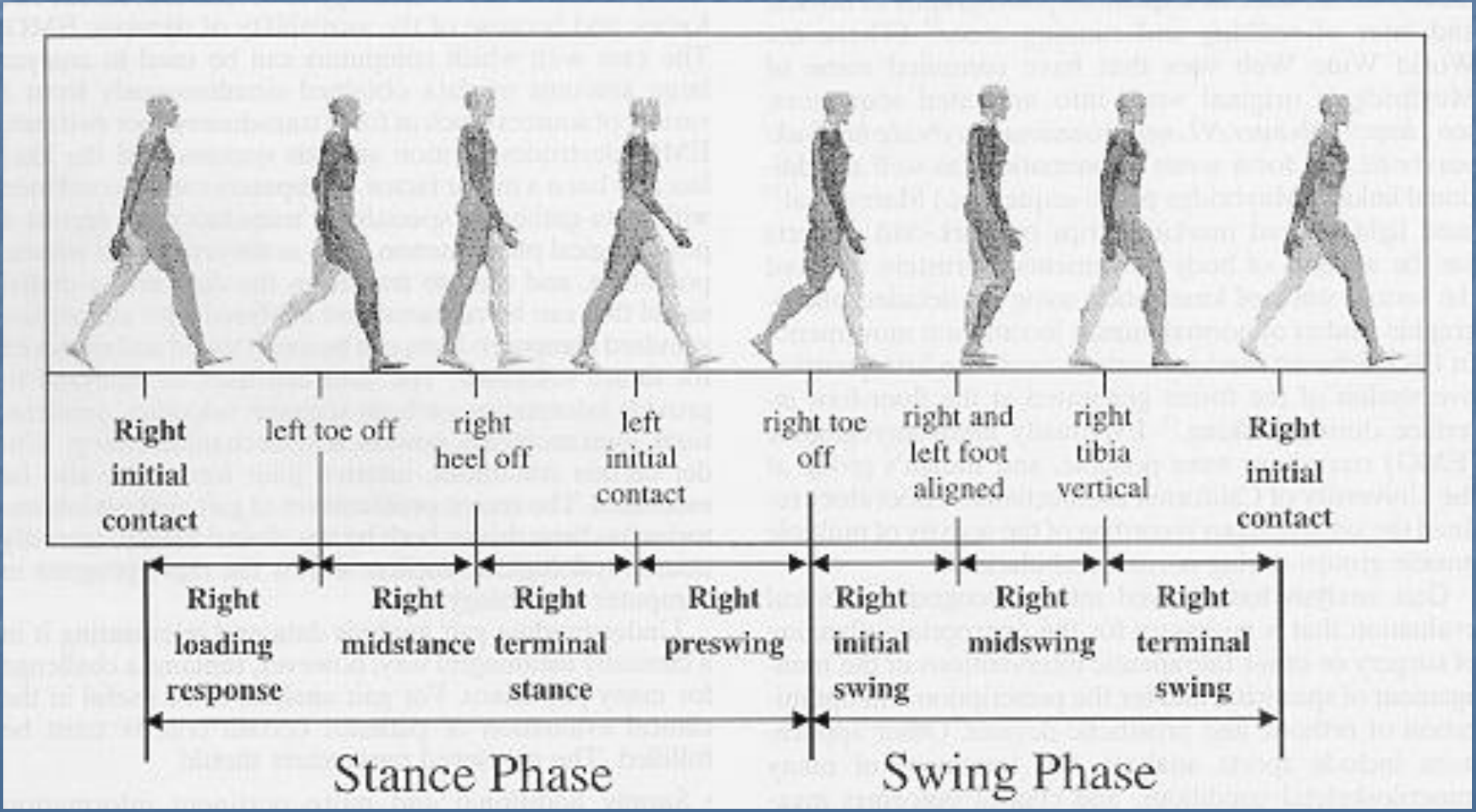




Normal Gait


RIGHT INITIAL CONTACT
LEFT PRE-SWING
LEFT INITIAL CONTACT
RIGHT PRE-SWING
RIGHT INITIAL CONTACT
LEFT PRE-SWING
Time, percent of cycle
Double support
R. Single support
Double support
L. Single support
Double support
0%
15%
45%
60%
100%
R. Stance phase
R. Swing phase
L. Swing phase
L. Stance phase
0%
40%
55%
85%
100%

Normal Gait


Right initial contact
left toe off
right heel off
left initial contact
right toe off
right and left foot aligned
right tibia vertical
Right initial contact
Right loading response
Right midstance
Right terminal stance
Right preswing
Right initial swing
Right midswing
Right terminal swing
Stance Phase
Swing Phase

Hip ROM during normal gait cycle

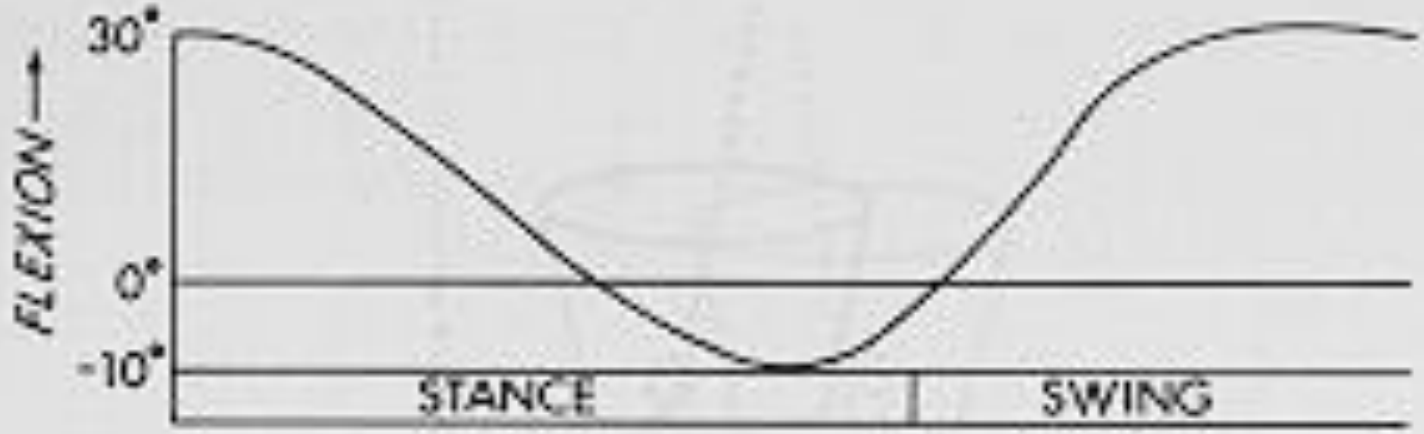


FIG 13–8.
Hip motion involves only 1 arc of flexion and 1 arc of extension. (Adapted from Perry J: *Clin Orthop* 1974; 102:18.)

Knee ROM during normal gait cycle

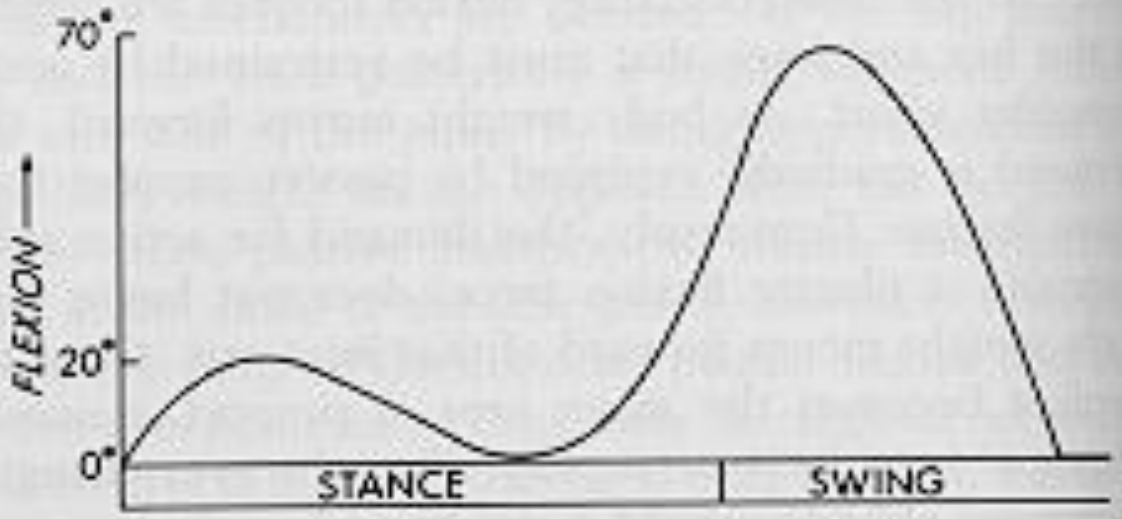


FIG 13–7.
The knee attains 35 degrees of flexion by the end of stance. Peak flexion is reached in the first third of swing while the limb is in a trailing position. (Adapted from Perry J: *Clin Orthop* 1974; 102:18.)

Ankle ROM during normal gait cycle

FIG 13–6.
Ankle motion during the gait cycle ranges from 10 degrees of dorsiflexion to 20 degrees of plantar flexion. (Adapted from Perry J: *Clin Orthop* 1974; 102:18.)

Ankle dorsiflexor weakness/paralysis

- Loading phase "foot slap"
- Footdrop (toe drag) in swing phase
- Excessive swing phase hip/knee flexion

- Ex. Fibular (peroneal) nerve palsy affecting anterior tibialis function

Ankle plantarflexor weakness/paralysis

- Uncontrolled ankle rotation during loading response to midstance
- Uncontrolled heel and toe off in terminal stance and preswing
- Loss of "propulsion" with an appearance of dropoff in latter stance phase

- Ex. Tibial nerve palsy affects gastrosoleus function

Quadriceps weakness/paralysis

- Affects all phases of gait
- Knee extension at initial contact
- 15-20 degrees knee flexion at midstance--loss of control of knee flexion in loading phase
- Loss of knee extension at terminal stance
- Loss of knee extension at terminal swing

- Ex. Femoral neuropathy

Hamstrings weakness/paralysis

- Uncontrolled knee extension and hip flexion—terminal swing
- Uncontrolled swing phase limb deceleration—loss of eccentric hamstrings contraction
- Harsh initial contact
- Difficulty placing the swing limb for initial contact

- Ex. Sciatic neuropathy

Hip extensor weakness/paralysis

- Gluteus maximus—loss of eccentric hip extension control in loading response
- Sudden posterior thrust of trunk after initial contact

- Ex. Inferior gluteal nerve palsy

Hip flexor weakness/paralysis

- Iliopsoas—loss of hip flexion in early swing phase
- Ex. Femoral neuropathy, lumbosacral plexopathy

Hip abductor weakness/paralysis

- Gluteus medius—"dropping" of the pelvis on the affected side in loading and midstance, resulting in trendelenburg gait
- Ex. Superior gluteal neuropathy, myopathy

Ataxia

- Impaired balance
- Lack of motor coordination
- Widened base of support
- Variable step length
- Associated movements are exaggerated (lurch, stagger)
- Watches feet

- Ex. Brainstem CVA, olivopontocerebellar atrophy, Friedreich's ataxia

Parkinsons/Parkinsonism

- Poor posture
- Short step length
- Shuffling
- Lack of associated movements (reciprocating)
- Festination

- Ex. Parkinsons disease

Hemiplegia

- Synergy
- Upper limb flexor
- Lower limb extensor
- Ex. CVA, TBI, MS, CP
- Spasticity
- Velocity-dependent increase in resistance to muscle stretch after upper motor neuron injury
- Spastic dystonia

Hemiplegia
Upper extremity flexion synergy

- Scapular retraction and depression
- Shoulder internal rotation
- Shoulder adduction
- Forearm pronation
- Elbow flexion
- Wrist flexion
- Finger flexion

Hemiplegia
Lower extremity extension synergy

- Pelvic elevation
- Hip extension, adduction, internal rotation
- Knee extension
- Ankle plantarflexion
- Foot inversion
- Toe plantarflexion
- Hallux extension

Hemiplegia

- Stance phase:
  - "slap"/equinovarus
  - Knee flexion/extension
  - Trendelenburg/extension
  - Hip flexion/extension
  - Toe drop off/clenching
- Swing phase:
  - Adductor swing
  - Circumduction
  - Toe drag
  - Sound limb vaulting

Equinus

- Ankle plantarflexion inversion spasticity
- Stance phase
  - Initial contact midfoot/forefoot
  - Weight bearing shifted laterally
- Swing phase
  - Toe drag

- Ex. CVA, TBI, MS, CP

Scissor

- Hip adductor spasticity
- Narrowed base of support
- Knee crosses midline--stance and swing

- Ex. Cerebral palsy

Antalgia

- Deviation dependent on pain location and severity
- Affected side—decreased stance phase
- Non-affected side—decreased step length

Antalgia

- Joint/bone:
  - Arthritis
  - Fracture
- Soft tissue injury
  - Bursitis
  - Tendonitis
  - Sprain/strain
  - Overuse
  - DOMS