Clinical Gait Analysis

Biomechanics & Etiology of Common Walking Disorders

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Teaching Points

• Phases of the Gait Cycle
• Primary Muscle Actions during Gait
• Common Gait Disorders
Motion Analysis at Stanford
Edweard Muybridge & Leland Stanford 1878

The Gait Cycle

Phases
STANCE
SWING

Tasks
Weight Acceptance
Single Limb Support
Limb Advancement

Periods
Initial Contact
Loading Response
Mid Stance
Terminal Stance
Pre Swing
Initial Swing
Mid Swing
Terminal Swing
Muscle Activity During Gait

- Initial Contact
  - Heel First Contact
Toe Walking
Diplegic Cerebral Palsy

Mid-Stance

• Controlled Tibial Advancement
3 Foot & Ankle Rockers

Terminal Stance
- Locked Ankle
- Heel Rise
- Trailing Limb

Calf Muscle Weakness
No Fixed Ankle or Heel Rise
Spastic Cerebral Palsy

Swing Phase

Peak knee flexion in initial swing
Ankle dorsiflexion to achieve foot clearance
Gait Analysis

- Video
- Kinematics and Kinetics
- Dynamic EMG
- Postural Balance
- Energy Expenditure

Musculoskeletal Computer Models of Gait

Computer models are generated from gait kinematics (joint motion) and kinetic (torque forces) and reveal the biomechanical features that influence gait.

The changing muscle lengths during gait are calculated using the computer model. Muscles that are too short and limit gait can be identified and selected for treatment.
Diplegic Cerebral Palsy

Diplegic Cerebral Palsy
Kinematics & Kinetics

- Kinematics: 3-D Joint Motion
  8 Digital Motion Capture Cameras Record
  Position of Light Reflective Markers

- Kinetics: Forces Passing Through the Joints
  Force Plate Embedded in the Floor Records
  Ground Reaction Force Vectors

Kinematics

- Nearly normal hip motion
- Increased knee flexion at IC and stance
- Reduced peak knee flexion in swing
- Increased plantar flexion in terminal stance
- Internally rotated foot progression
Kinetics

• Normal ankle plantarflexor moment peaks in terminal stance

• Increased plantar flexor moment in loading response “double bump” associated with increased plantar flexion at IC

• Decreased moment in terminal stance associated with a reduced forefoot rocker
Dynamic EMG

- Footswitch or Markers
- Electrodes
  - Surface
  - Fine Wire
- Interpretation

Muscle EMG Timing During Gait
Dynamic EMG & Kinematics

Postural Balance

- Force Plate Center of Pressure
- Postural Sway with Eyes Open / Closed
Pathologic Gait
Neuromuscular Conditions

- Equinus
- Equinovarus
- Pseudo equinus (knees bent, ankles at neutral, forefoot contact)
- Jumped (knees bent, ankles true equinus)
- Crouch (knees bent, ankles dorsiflexed)
- Stiff–knee gait
Pathologic Gait
Musculoskeletal Conditions
Polio, Dislocation, Arthritis, Muscular Dystrophy

• Pain
• Muscle weakness
• Structural abnormalities (joint instability, short limb)
• Loss of motion
• Combinations of above

Antalgic Gait
Pain

• Any gait that reduces loading on an affected extremity by decreasing stance phase time or joint forces

• Examples
  – “stone in your shoe”
  – Painful hip, knee, foot, etc
Pathologic Hip Gait
Painful due to Arthritis

- Coxalgic gait
  - Intact hip abductors; structural stability
  - Lateral shift, hip compression, abductor load
  - Contralateral pelvic elevation

Hip Biomechanics
Single-limb Stance Lurch Shifts Center of Mass

Hip Joint is Fulcrum: Hip Joint Reaction Force = pull of abductors + body weight
Antalgic Gait

Painful Side:

- Shorten stance phase time
- Lengthen swing phase time
- Lengthen step length

Pathologic Hip Gait

Weakness

- Trendelenburg Gait
  - Weak hip abductors
  - Contralateral pelvic drop
Pathologic Hip Gait

Trendelenburg Coxalgic Gait

Gluteus Maximus Lurch muscular dystrophy
- Weak gluteus max no pain
- Lean backwards to prevent falling forward

Quadriceps Avoidance polio, SCI, ACL
- Weak quadriceps no pain
- Increased knee extension

Drop Foot polio, stroke, SCI
- Weak dorsiflexors no pain
- Increased ankle plantarflexion
Cane & Able

Cane is used on able side - contralateral side

1. Allows for reciprocal arm swing
2. Widens base of support
3. Reduces demand on affected side - long lever arm
Spastic Cerebral Palsy

- Loss of Selective Motor Control
- Short Muscle-tendon Length & Joint Contracture
- Muscle Weakness
- Muscle Spasticity

- Mixed CP: Ataxia, Dystonia, Chorea, Athetosis

Neuromuscular Mechanisms underlying Motor Deficits in Spastic Cerebral Palsy

- EMG Test of Obligatory Muscle Co-activation in Spastic CP
- Muscle Pathology in Spastic CP
- Neuromuscular Activation & Motor-unit Firing Characteristics in CP
- Neonatal Brain Abnormalities & Gait Deficits in Preterm Children
EMG Test to Differentiate Mild Diplegic Cerebral Palsy & Idiopathic Toe Walking

Obligatory Co-activation of Quadriceps & Gastrocnemius

Rose et al. J Pediatric Orthopaedics (1999)
Policy et al. J Pediatric Orthopaedics (2001)
Obligatory Co-activation Quads & Gastrocnemius contributes to Toe-walking & Loss of Selective Motor Control in Cerebral Palsy
Muscle Pathology in Spastic Cerebral Palsy

Increased proportion of type-1: type-2 muscle fibers
Increased fiber size variation
Type-1 fiber proportion vs. EMG prolongation ($r = .77, p = .03$)
Fiber size variability vs. energy expenditure ($r = .69, p = .05$)

Muscle Fiber Architecture
Muscle Atrophy

Neuromuscular Activation & Motor-Unit Firing in Spastic Cerebral Palsy

Torque, EMG, Max M-wave & Neuromuscular Activation

**Torque, EMG, Max M-wave & Neuromuscular Activation**

<table>
<thead>
<tr>
<th>MVC: Torque</th>
<th>CP control</th>
<th>Plantarflexion (GAS)</th>
<th>CP control</th>
</tr>
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<tbody>
<tr>
<td>10 N·m</td>
<td>2 s</td>
<td>12 mV</td>
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<th>Maximum M-wave:</th>
<th>CP control</th>
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<tbody>
<tr>
<td>2.4</td>
<td>11.3</td>
<td>0.8</td>
<td>3.7</td>
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**Maximum Neuromuscular Activation**

**Maximum Neuromuscular Activation**

- Tibialis Anterior
- Gastrocnemius

**Maximal Neuromuscular Activation (% M-wave)**

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<th>CP control</th>
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Sub-maximal Voluntary Isometric Contractions

Neuromuscular Activation Feedback

Motor-Unit Firing
Submaximal isometric contractions

![Graphs showing motor-unit firing levels](image-url)
Maximum Motor-Unit Firing Rates in CP

Projected Max FR
CP = 16 Hz
Control = 25 Hz

CP = 2.4 Voluntary Max Muscle Activation
Muscle Activation Level (% M-wave)

CP = 1.04 Voluntary Max Muscle Activation
Muscle Activation Level (% M-wave)

Gastrocnemius
Projected Max FR
CP = 13 Hz
Control = 9.7 Voluntary Max Muscle Activation
Muscle Activation Level (% M-wave)

Tibialis Anterior
Projected Max FR
CP = 5.7 Voluntary Max Muscle Activation
Muscle Activation Level (% M-wave)

Short-term Synchronization

0.14 extra synchronous firings per motor unit pair per second

Control subject

no extra synchronous firings

CP subject

VA Palo Alto HCS
Neonatal Microstructural Development of Internal Capsule on DTI correlates to Severity of Gait & Motor Deficits in Preterms

J Rose*, M Mirmiran', EE Butler*, CY Lin*, PD Barnes*, R Kermoian* & DK Stevenson'

Developmental Medicine & Child Neurology (2007)

VLBW preterm infants < 32 wks GA, <1500g; 15% have motor deficits

• Neonatal brain MRI-DTI (37 wks PGA)

• Gait analysis at 4 yrs: Gillette Gait Index (NI)

Motor Cortex

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Sagittal Views of the Brain

1. Genu of corpus callosum
2. Forceps minor
3. Internal capsule, anterior limb
4. Septum pellucidum
5. Caudate nucleus
6. Putamen
7. Globus pallidus
8. Internal capsule, posterior limb
9. Thalamus
10. Splenium of corpus callosum
11. Forceps major

Axial View of the Brain

1. Genu of corpus callosum
2. Forceps minor
3. Internal capsule, anterior limb
4. Septum pellucidum
5. Caudate nucleus
6. Putamen
7. Globus pallidus
8. Internal capsule, posterior limb
9. Thalamus
10. Splenium of corpus callosum
11. Forceps major
DTI Fractional Anisotropy (FA)

- Measures directionality of water molecule movement relative to neuronal fibers in units, 0-1 (e.g., CSF approaches 0, corpus callosum = 0.83)

- Decreased FA in internal capsule
  - Fewer nerve fibers
  - Decrease in thickness of fibers
  - Less myelination
  - Reduced Development

Posterior Limbs of the Internal Capsule (PLIC)

Neonatal DTI Fractional Anisotropy (FA) of PLIC

- Preterm control children (n=17)
- Preterm children with normal FA (n=14)
- Preterm children with low FA (n=10)
Preterm Child with Moderate Gait Abnormalities

Gillette Gait Index (NI)

- 3D Kinematics - single score for severity of gait deficits
- Principal Component Analysis: 16 kinematic measures of pelvis, hip, knee & ankle
- Quantifies amount gait deviates from normal
- A higher value indicates more severe gait deficits
### Gait Graphs for Three Children

**Mild**

- Hip
- Knee
- Ankle
- Foot

- NI = 372, FA = 670
- NI = 1083, FA = 647
- NI = 2000, FA = 623

**Moderate**

**Severe**

**Neonatal Brain MRI-DTI Internal Capsule Posterior Limbs & Gait NI at 4 years of Age**

- Normal FA (n=14)
- Low FA (n=10)

Spearman correlation: 

\[ \rho = -0.89, p < 0.01 \]
Acknowledgments

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Thank You