Post-Stroke Locomotor Rehabilitation: Evidence-Based Frameworks for Clinical Decision Making

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Overview

• What is required for successful locomotor rehabilitation?
  – Systematic outcome measurement
  – Intensity
  – Training to be specific to the task
  – Adherence to principles of neuromuscular plasticity

• What does evidence-based practice tell us about locomotor rehabilitation?

• Where to go from here and what will future generations of therapeutic practice include?
Significance of Problem

- Stroke is one of the most debilitating medical conditions in America, affecting approximately 795,000 each year with a surviving cohort of almost 6.5 million people.
- 73% of those surviving stroke will have some degree of long term disability.
- Even among those who achieve independent ambulation, significant residual deficits persist in balance and gait speed, with 60% of persons post-stroke reporting limitations in mobility related to walking.
Stroke Deficits

- Disruption of motor pathways leads to decreased motor unit activation
- Disuse atrophy in the skeletal
- “Maladaptive plasticity”

All lead to inadequate force generation and abnormal timing
  - Decreased support
  - Poor limb advancement
  - Poor weight advancement

Chronic Conditions

- Continued atrophy and plastic changes
- Contractures
- Spasticity (??)
- Decreased cardiovascular fitness and endurance
Where are we currently with locomotor rehabilitation?
Since the 1928 work of Santiago Ramón y Cajal, famed neuroscientist, the prevailing assumption has been that the central nervous system (CNS) is hard-wired, non-malleable, and incapable of repairing itself.

Clinicians have selected compensation as a rehabilitation strategy for non-remediable deficits of strength, voluntary motor control, sensation, and balance.
1. Promote maximum capacity muscle strength, range, and endurance in muscles that can be voluntarily-activated above and below the lesion.

2. Compensate for non-remediable deficits of paralysis and weakness by using braces and assistive devices.

3. Teach new behavioral strategies or skills for ambulation and mobility.
ICF model of Rehabilitation

Recovery versus Compensation

Movement
Neural

Functional
What & when is “recovery”

Figure 1. Graph showing recovery of motor function after stroke based on Fugl-Meyer motor scores. Patients are stratified into groups based on the initial severity of motor deficit measured with Fugl-Meyer Assessment (see text). Regardless of initial severity of stroke, the most dramatic recovery occurs within the first 30 days. Moderate and most severe stroke patients continue to experience some recovery for 90 days. Graph represents mean Fugl-Meyer scores.
Neural Control of Walking

How we walk: central control of muscle activity during human walking.
Nielsen JB.
Control of movement

• Descending drive from the brain via corticospinal tracts

• Network in spinal cord, capable of generating basic rhythmic patterns of movement
  – Supraspinal influences (more than other animals)
  – Peripheral sensory influences

• Direct corticospinal projects to spinal motoneurons has developed for bipedalism
Role of motor cortex in human walking

- Activation increased for:
  - Obstacles
  - Starting/stopping
  - Complicated environments

- Information from cortex to spinal cord may go through corticospinal (CS) tracts, but may use other tracts as well.

Adapted from Dobkin, 2004
Effect of locomotor training on descending drive
Spinal Rhythm Generator?

- Definitely present in cats
- May be activated in deafferented animals in the brainstem (mesencephalic and subthalamic locomotor regions: MLR and SLR)

**Therefore:** basic patterns can be activated by nonpatterned and nonspecific descending drive from the brainstem. This network can recruit and coordinate muscles on its own.

However, does this same network exists in humans?
Do bipedal human have a central pattern generator in the same way as seen in the cat?

Studies in humans with complete SCI (no descending drive to peripheral muscle) demonstrate walking patterns and muscle activation consistent with normal walking.

None of these individuals has learned to walk off of the treadmill.

Harkema, 1997
Role of sensory feedback

1. Sensory feedback may help to drive the active motoneurons

2. May contribute to corrective reflexes following sudden perturbations

3. May provide essential error signals that inform the brain of differences between the intended movement and the actually executed movement...may update future movements (motor learning).
“There is no reason to suggest that human walking is generated only by the spinal cord and that the corticospinal tract only makes a significant contribution in relation to voluntary modification of the gait pattern.

Nor is there any reason to suggest that the motor cortex alone is responsible for the activation of the muscles during walking without any contribution from the spinal cord neuronal circuitries.

Human bipedal walking...is based on an integration of the activity of spinal neuronal circuitries with sensory feedback signals and descending motor commands.”

Nielson, 2003
Central Control Centers for control of walking. (from Nielsen, 2003) Muscle activation comes directly from the cortex as well as spinal CPGs and is modulated at each location by afferent input.
The most effective locomotor rehab should:

• Train volitional, cognitive approaches;
• Normalize sensory inputs; and
• Take advantage of pattern generation circuitry
Experience-Dependent Plasticity

Principles of experience-dependent neural plasticity: implications for rehabilitation after brain damage.
Kleim JA, Jones TA.
Neuroplasticity

• “Persistent changes within the central nervous system that result from prior experiences and influence future motor behaviors (Wolpaw and Tennissen, 2001)”.

• Brain activation
• Axonal growth
• Sprouting
• Synaptic transmission
• Protein upregulation
• Therapy can capitalize on the central nervous system’s ability to learn and reorganize to restore function that has already been compromised or lost.
• Driven by changes in behavioral sensory, and cognitive experiences.
• Learning happens even in the absence of rehabilitation. Therefore, as therapists, we must drive plasticity in the direction to promote recovery of function.

The following are 10 principles of neuromotor change:
Principle 1: Use it or lose it
- Neural circuits not used will degrade and change

Principle 2: Use it and improve it
- Training provides enhancement of function
Principle 3: Specificity

- The type of rehabilitation dictates the type of plasticity

Hodgson et al. 1994
Principle 4: Repetition matters

- Repetition is required for lasting neural changes (i.e. motor learning)

- Not just acquisition of the skill, but repeated use of the skill over time.

  Mean number of steps per therapy session: 357
  (95% CI = 296–418) (Lang 2009)

  Mean number taken per day in healthy individual: 5117 (Bassett, 2010)
Principle 5: Intensity matters

- Sufficient training intensity is required for lasting neural changes (i.e. motor learning)

**A** Prolonged 5 Hz rTMS (1800 stimuli)

**B** Short 5 Hz rTMS (150 stimuli)

WHAT IS INTENSITY FOR WALKING?
Principle 6: Time matters

- Plastic changes require a process over time and do not alter instantaneously.

- Certain time periods post injury may be most beneficial.

Gene expression → Synapse formation → Motor map reorganization
Principle 7: Salience matters

• The system must weigh the importance of any given situation in order to facilitate motor learning (motivation, attention, emotion, etc relate to degree of plasticity).

Principle 8: Age matters

• Potentiation, synaptogenesis, reorganization all hampered
• Aging marked by neuronal and synaptic atrophy
Principle 9: Transference

• “The ability of plasticity within one set of neural circuits to promote concurrent or subsequent plasticity.”

Principle 10: Interference

• “Refers to the ability of plasticity within a given neural circuitry to impede the induction of new, or expression of existing, plasticity within that same circuitry...impairing learning.” (ex. maladaptive plasticity)
So...

The most effective locomotor rehab should:

- Activate desired neural circuitry
- Be specific to the task
- Be repetitive, intense, for appropriate duration
- Be important to the individual
What does neuroplasticity look like in a clinical intervention?
• **Guideline 1:** Maximize weight bearing on the legs
• **Guideline 2:** Afford the sensory experience consistent with walking
• **Guideline 3:** Optimize kinematics for each motor task
• **Guideline 4:** Maximize recovery and independence; minimize compensation

“Train like you walk”
• Systematic
• Intensive
• Progressive
• Repetitive
• Document outcomes
...plus Plasticity

- Use it or lose it
- Use it and improve it
  - Specificity
- Repetition matters
- Intensity matters
- Time matters
- Salience matters
- Age matters
- Transference
- Interference
So, what is “evidence-based practice”? 

Tilson JK, Settle SM, Sullivan KJ.

“Rehabilitation effectiveness is enhanced when the clinician can:
1. determine the best available interventions;
2. for the appropriate subset of patients and;
3. apply them at the correct dose and timepoint of recovery.”
Step 1: Convert the need for information into a focused and searchable clinical question.

Narrow question for:
• Patient or patient population
• Intervention
• Outcome of interest

“PICO”
- Population
- Intervention
- Comparison (alternate intervention)
- Outcome of interest
Step 2: Find the best possible research evidence to answer your question.

Pubmed
PEDro (Physiotherapy Evidence Database)
National Guidelines Clearinghouse
Canadian Stroke Network.
Cochrane Collaboration
(www.cochrane.org/reviews)
Step 3: Critically appraise the research evidence for validity and applicability

Figure 1. The Evidence Pyramid illustrates a hierarchy of “best” sources of evidence for searchable clinical questions. Categories of research evidence are ordered from top to bottom; higher levels on the pyramid represent the most likely sources of high-quality research evidence. The corresponding level of evidence is included for each category.
Table 1. Evidence levels and grades of recommendation

<table>
<thead>
<tr>
<th>Levels of evidence(^a)</th>
<th>Grades of recommendation(^b)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1a: Systematic review of RCTs</td>
<td><strong>Grade A</strong>: Evidence from one or more RCTs of a statistically significant, clinically important benefit (&gt;15%)</td>
</tr>
<tr>
<td>1b: Individual RCT of high quality</td>
<td><strong>Grade B</strong>: Statistically significant, clinically important benefit (&gt;15%) if the evidence was from observational studies or controlled clinical trials (CCTs)</td>
</tr>
<tr>
<td>2a: Systematic review (with homogeneity) of cohort studies</td>
<td><strong>Grade C+</strong>: Evidence of clinical importance (&gt;15%) but not statistical significance</td>
</tr>
<tr>
<td>2b: Individual cohort study (including low quality RCT, e.g., &lt;80% follow-up)</td>
<td><strong>Grade C</strong>: An appropriate outcome was measured in a study that met the inclusion criteria but no clinically important difference and no statistical significance were shown.</td>
</tr>
<tr>
<td>3a: Systematic review (with homogeneity) of case-control studies</td>
<td><strong>Grade D</strong>: Evidence from one or more RCTs of a statistically significant benefit favoring the control group (&lt;0%: favors controls)</td>
</tr>
<tr>
<td>3b: Individual case-control study</td>
<td></td>
</tr>
<tr>
<td>4: Case-series (and poor quality cohort and case-control studies)</td>
<td></td>
</tr>
<tr>
<td>5: Expert opinion without explicit critical appraisal, or based on physiology, bench research or “first principles”</td>
<td></td>
</tr>
</tbody>
</table>

Note: RCT = randomized controlled trial.
\(^a\)From Oxford Centre for Evidence-based Practice\(^17\)
\(^b\)From Ottawa Panel\(^9\)
Step 4: Integrate the critically appraised research evidence with clinical expertise and the patient's unique biology, circumstances, and values.

“Clinical trials do not provide the answers on how to rehabilitate patients.”

- Inform
- Provide a framework for practice
- Integrate with clinical expertise
- Integrate with patient's unique circumstances, values, and preferences.

Partner with patient, family, and caregivers.
Step 5: Evaluate the effectiveness and efficacy of our efforts in Steps 1-4 and identify ways to improve them in the future.

- Monitor impact
- Use standardized outcome measures
- Build on each experience
- PubMed’s MyNCBI service: set up auto alerts for emerging literature of interest
Examples in stroke literature

Question 1: Aerobic conditioning and walking recovery poststroke.

<table>
<thead>
<tr>
<th>Resource</th>
<th>Quality of summary</th>
<th>Aerobic training interventions</th>
<th>Dose recommendations</th>
<th>Patient gait speed severity/poststroke chronicity</th>
<th>Outcomes addressed</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gordon et al., 2004&lt;sup&gt;23&lt;/sup&gt; (CPG)</td>
<td>Level 5 Recommendations based on expert opinion and studies with physiologic outcome measures</td>
<td>Studies involved large muscle activities (walking, treadmill, stationary cycle, combined arm-leg ergometry, arm ergometry, seated stepper)</td>
<td>40%-70% HRR; 50%-80% MHR; RPE 11-14 (6- to 20-point scale); 3-7 days/wk; 20-60 min/session (or multiple 10 min sessions)</td>
<td>No specific recommendations by severity or chronicity</td>
<td>Goals for program: increased independence with ADLs, increased tolerance for prolonged physical activity, increased walking speed/efficiency, reduced risk of cardiovascular disease</td>
</tr>
<tr>
<td>Brosseau et al., 2006&lt;sup&gt;25&lt;/sup&gt; (CPG)</td>
<td>Level 1b, grade A-C Clear description of methods used to develop CPG</td>
<td>None specifically stated</td>
<td>None stated</td>
<td>No specific recommendations by severity or chronicity</td>
<td>Evidence for improvement in: functional status (walking), gait speed, mobility (stair climbing and walking distance)</td>
</tr>
<tr>
<td>Foley et al., 2007&lt;sup&gt;12&lt;/sup&gt; (CPG)</td>
<td>Level 1a, grade A Clear description of evidence used to develop CPG</td>
<td>Cycling, water-based exercise, treadmill training</td>
<td>Not stated</td>
<td>No specific recommendations by severity or chronicity</td>
<td>Evidence for improvement in: gait performance (velocity and endurance)</td>
</tr>
<tr>
<td>van de Port et al., 2007&lt;sup&gt;27&lt;/sup&gt; (SR)</td>
<td>Level 1a, grade A-C+ Meets criteria for quality SR</td>
<td>Cycling, water-based exercise, gait-oriented training</td>
<td>Ranges: 8-90 min; 4-19 wks</td>
<td>No specific recommendations by severity or chronicity</td>
<td>Evidence for improvement in: mobility, gait speed, walking distance, quality of life</td>
</tr>
<tr>
<td>Mead et al., 2007&lt;sup&gt;28&lt;/sup&gt; (RCT)</td>
<td>Level 1b, grade A PEDro score = 7/10</td>
<td>Tx: Combined endurance and strengthening group exercise; C: Deep breathing, progressive muscular relaxation; Intensity: “Brisk efforts encouraged”</td>
<td>Participants had chronic stroke; moderate severity gait impairment</td>
<td>Evidence for improvement in: walking economy, TUG, role-physical SF-36 (directly after treatment); role-physical SF-36 (7-month follow-up)</td>
<td></td>
</tr>
</tbody>
</table>

Note: C = control group; CPG = clinical practice guideline; HRR = heart rate reserve; MHR = maximal heart rate; RPE = rate of perceived exertion; SR = systematic review; RCT = randomized controlled trial; TUG = Timed Up & Go; Tx = treatment group.
Recommendations:

• High quality (level 1a) and clinically relevant (grade A) research evidence that aerobic conditioning is effective for improving walking speed and endurance poststroke.
• Small body of evidence (conflicting grades A and C) of clinical relevance of impact on disability and QOL
• Unclear of dose
• Recommend breaking down by severity and chronicity.

<table>
<thead>
<tr>
<th>Resource</th>
<th>Quality of summary</th>
<th>Orthotic intervention</th>
<th>Dose recommendations</th>
<th>Patient gait speed severity/poststroke chronicity</th>
<th>Outcomes addressed</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>AFO devices without FES</strong></td>
<td></td>
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<tr>
<td>Foley et al., 2007¹² (CPG)</td>
<td>Level 2b, grade B</td>
<td>AFO</td>
<td>None stated</td>
<td>No specific recommendations by severity or chronicity</td>
<td>Evidence for improvement in: gait parameters, postural stability</td>
</tr>
<tr>
<td></td>
<td>Clear description of methods used to develop CPG</td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Wang et al., 2005¹³ (RCT)</td>
<td>Level 1b, grade A</td>
<td>AFO, aggressive bracing</td>
<td>Intervention duration: &quot;short training time&quot;</td>
<td>Participants had moderate severity; subacute chronicity</td>
<td>Evidence for improvement in: gait speed, balance measures</td>
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<tr>
<td></td>
<td>PEDro score = 6/10</td>
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<tr>
<td>Tyson et al., 2001¹⁴ (RCT)</td>
<td>Level 1b, grade A</td>
<td>Off-the-shelf AFO</td>
<td>Intervention duration: 1 mo</td>
<td>Participants had high severity; subacute chronicity</td>
<td>Evidence for improvement in: functional ambulation category, gait parameters, perception of safety and confidence</td>
</tr>
<tr>
<td></td>
<td>PEDro score = 6/10</td>
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<tr>
<td>deWit et al., 2004¹⁵ (RCT)</td>
<td>Level 2b, grade A</td>
<td>Custom hinged AFO</td>
<td>Intervention duration: more than 6 mo</td>
<td>Participants had moderate severity; chronic chronicity</td>
<td>Evidence for improvement in: walking speed, TUG, stairs, self-confidence</td>
</tr>
<tr>
<td></td>
<td>PEDro score = 5/10</td>
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<tr>
<td><strong>AFO devices with FES (external and implanted electrodes)</strong></td>
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<td></td>
</tr>
<tr>
<td>Foley et al., 2007¹² (CPG)</td>
<td>Level 1b, grade A</td>
<td>AFO with FES (external electrodes)</td>
<td>Not stated</td>
<td>No specific recommendations by severity or chronicity</td>
<td>Evidence for improvement in: gait parameters, physiologic cost index, independence measures</td>
</tr>
<tr>
<td></td>
<td>Clear description of methods used to develop CPG</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Robbins et al., 2006¹⁶ (SR)</td>
<td>Level 1a, grade A</td>
<td>AFO with FES (external electrodes)</td>
<td>Intervention duration: 3 wks to 3 mo</td>
<td>Study participants had moderate to high severity; subacute and chronic stroke</td>
<td>Evidence for improvement in: gait parameters, Fugl-Meyer scores, physiologic cost index, spasticity, motor scale</td>
</tr>
<tr>
<td></td>
<td>Clear description of methods used to develop CPG</td>
<td></td>
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<tr>
<td>Kottink et al., 2007¹⁷ (RCT)</td>
<td>Level 1b, grade A</td>
<td>Implanted electrodes – peroneal nerve stimulation only</td>
<td>Intervention duration: 6 mo</td>
<td>Participants had low severity; chronic chronicity</td>
<td>Evidence for improvement in: walking speed, 6-min walk test, assessment of activity level</td>
</tr>
<tr>
<td></td>
<td>PEDro score = 6/10</td>
<td></td>
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</table>

Note: AFO = ankle-foot orthosis; FES = functional electrical stimulation; CPG = clinical practice guideline; RCT = randomized controlled trial; TUG = Timed Up & Go; SR = systematic review.
Recommendations:

• Level 2, grade B evidence that non-FES AFOs are effective for small changes in walking speed.

• Increased:
  – patient independence
  – Perception of safety
  – Confidence

• Level 1a, grade A evidence for improved gait velocity with FES-enabled AFO devices.

• Implanted FES has one RCT supporting level 1b, grade A evidence for improved walking speed.

Questionable functional independence, QOL, and long-term implications.
### Question 3: Treadmill training with body weight support for walking recovery post-stroke?

<table>
<thead>
<tr>
<th>Resource</th>
<th>Quality of summary</th>
<th>Treadmill training with BWS interventions</th>
<th>Dose recommendations</th>
<th>Patient gait speed severity/poststroke chronicity</th>
<th>Outcomes addressed</th>
</tr>
</thead>
<tbody>
<tr>
<td>Moseley et al., 2005 (SR)</td>
<td>Level 1a, grade C+</td>
<td>Studies reviewed were pooled with variability in patient subsets, because patients from acute and chronic phases and nonambulators and ambulators were included.</td>
<td>Variable across studies for speed (0.01 – 0.89 m/s), BWS (not reported to 40% decreased with training), TM walking time (not reported to 20 min), frequency (3x to 5x/wk), duration (2–10 wks)</td>
<td>No significant difference between TM with BWS and other PT interventions in walking dependency or speed for dependent ambulators prior to treatment initiation; nonsignificant trend for independent walkers in walking speed</td>
<td>Inconclusive evidence for improvement in walking speed (gait velocity) or walking dependency</td>
</tr>
<tr>
<td>Brosseau et al., 2006 (CPG)</td>
<td>Level 1b, grade C+</td>
<td>Studies reviewed from acute and chronic phases and nonambulators and ambulators were included.</td>
<td>None stated but noted positive effects of training with increased TM speed</td>
<td>No specific recommendations by severity or chronicity</td>
<td>Evidence for improvement in walking speed (gait velocity) and walking endurance (distance) for those patients trained at higher treadmill speeds</td>
</tr>
<tr>
<td>Foley et al., 2007 (CPG)</td>
<td>Level 1b, grade C</td>
<td>Studies reviewed from acute and chronic phases and nonambulators and ambulators were included.</td>
<td>None stated</td>
<td>No specific recommendations by severity or chronicity</td>
<td>Inconclusive evidence that TM-BWS is more effective than other gait interventions for increasing walking speed or endurance</td>
</tr>
<tr>
<td>Sullivan et al., 2007 (RCT)</td>
<td>Level 1b, grade A</td>
<td>TM-BWS plus UE ergometry on alternate days compared to resisted cycling plus UE ergometry on alternate days</td>
<td>1-hr sessions, 4 days/wk, for 6 wks; moderately high exercise intensity parameters specified for each group</td>
<td>Stratified by severity: severe 0.25 m/s, moderate 0.71 m/s average walking velocity at baseline; Chronicity: 24 mos average time poststroke</td>
<td>Statistically significant and clinically meaningful increase in walking velocity for TM-BWS group compared to cycle group; statistically significant and clinically meaningful increase in walking distance for both TM-BWS group and cycle group</td>
</tr>
</tbody>
</table>

Note: SR = systematic review; TM = treadmill; PT = physical therapy; CPG = clinical practice guideline; RCT = randomized controlled trial; UE = upper extremity.
Locomotor Experience Applied Post-Stroke
• 5 year
• Phase-III
• Single-blinded
• Multi-site
• Randomized Clinical Trial

• If in addition to usual care, to determine if a specialized locomotor training program that includes BWST as a treatment modality can produce clinically significant changes in walking speed in individuals post-stroke compared to Home Exercise Program.

• To determine effect of timing (early vs. late; severity (moderate vs. severe), and duration (12-, 24-, 36-sessions).
Walking speed trajectory by intervention group and severity at screening, 2-(baseline), 6-, and 12-months post-stroke.*

*Figure 1. Timing of Locomotor Training and Changes in Walking Speed 1 Year after Stroke. Screening (Scr) was performed at a mean (±SD) of 26.0±11.6 days after stroke. Randomization was performed at baseline, 2 months after stroke. Bars indicate 95% confidence intervals. HE denotes home exercise, LT locomotor training, and P12, P24, and P36 post-training assessments at weeks 12, 24, and 36, respectively.

Duncan et al. 2011
Walking Speed at 6 Months

- Six months after stroke, Early-LTP (0.25±0.21 m/s) and HEP (0.23±0.20 m/s) groups had similar gains in walking speed and sustained these gains at 1 year.

- The Late-LTP group (which only received usual care from 2 to 6 months) improved by 0.13±0.14 m/s at 6 months.
Statistically and Clinically Significant Changes in Outcomes from 2 mos to 12 mos post-stroke

<table>
<thead>
<tr>
<th>Item</th>
<th>Early-LTP (n=139)</th>
<th>Late-LTP (n=143)</th>
<th>HEP (n=126)</th>
<th>Overall p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Comfortable Walking Speed (m/sec)</td>
<td>0.23±0.20</td>
<td>0.24±0.23</td>
<td>0.25±0.22</td>
<td>0.67</td>
</tr>
<tr>
<td>6 minute Walking Distance (m)</td>
<td>73.2±69.4</td>
<td>79.0±75.1</td>
<td>85.2±72.9</td>
<td>0.45</td>
</tr>
<tr>
<td>Step Activity Monitor (SAM) – Median of average number steps/day [25th-75th percentile]</td>
<td>858 [-253, 2422]</td>
<td>1022 [-111, 3009]</td>
<td>1471 [435, 3481]</td>
<td>0.10</td>
</tr>
<tr>
<td>Stroke Impact Scale (SIS) Participation</td>
<td>17.1±25.9</td>
<td>13.1±22.0</td>
<td>14.4±20.6</td>
<td>0.38</td>
</tr>
<tr>
<td>SIS ADL/IADL (range = 0-100)</td>
<td>9.6±19.5</td>
<td>9.4±17.2</td>
<td>14.5±19.0</td>
<td>0.07</td>
</tr>
<tr>
<td>SIS Mobility (range = 0-100)</td>
<td>13.7±21.6</td>
<td>12.0±19.1</td>
<td>14.2±20.3</td>
<td>0.685</td>
</tr>
<tr>
<td>Fugl-Meyer LE Score (range = 0-34)</td>
<td>1.7±3.9</td>
<td>1.5±3.7</td>
<td>2.5±4.3</td>
<td>0.13</td>
</tr>
<tr>
<td>Berg Score (range = 0-56)</td>
<td>8.0±7.8</td>
<td>5.9±9.1</td>
<td>8.3±8.78</td>
<td>0.06</td>
</tr>
</tbody>
</table>

No differences in improvements across treatment groups

HIGHLY clinically relevant improvements
### Preplanned Secondary Analysis of 6 Month Outcomes

Late-LTP (usual care) experienced approximately **HALF** the improvement of early intervention groups

<table>
<thead>
<tr>
<th>Item</th>
<th>LTP (n=139)</th>
<th>HEP (n=126)</th>
<th>UC (n=143)</th>
<th>Overall p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Comfortable Walking Speed (m/sec)</td>
<td>0.25±0.21</td>
<td>0.23±0.20</td>
<td>0.13±0.14</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>6 minute Walking Distance (m)</td>
<td>81.8±62.8</td>
<td>75.9±69.3</td>
<td>41.0±47.4</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Step Activity Monitor (SAM) – Median of average number steps/day [25th - 75th percentile]</td>
<td>1017 [-102, 2209]</td>
<td>1357 [84, 3382]</td>
<td>566 [-362, 2043]</td>
<td>0.0367</td>
</tr>
<tr>
<td>Stroke Impact Scale (SIS) Participation (range = 0-100)</td>
<td>11.8±26.7</td>
<td>14.6±22.9</td>
<td>7.7±20.5</td>
<td>0.0384</td>
</tr>
<tr>
<td>SIS ADL/IADL (range = 0-100)</td>
<td>9.8±17.2</td>
<td>13.0±16.9</td>
<td>7.0±17.8</td>
<td>0.0516</td>
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<tr>
<td>SIS Mobility (range = 0-100)</td>
<td>15.3±21.4</td>
<td>14.9±20.0</td>
<td>7.0±15.7</td>
<td>0.0006</td>
</tr>
<tr>
<td>Fugl-Meyer LE Score (range = 0-34)</td>
<td>2.2±3.4</td>
<td>2.4±4.1</td>
<td>1.3±3.3</td>
<td>0.1196</td>
</tr>
<tr>
<td>Berg Score (range = 0-56)</td>
<td>8.8±8.1</td>
<td>7.9±8.5</td>
<td>5.3±7.0</td>
<td>0.0018</td>
</tr>
<tr>
<td>Activities Specific Balance Confidence Score (range=0-100)</td>
<td>13.8±20.8</td>
<td>15.6±19.4</td>
<td>6.2±20.2</td>
<td>0.0013</td>
</tr>
</tbody>
</table>

Falls and Falls Rate

The most common minor adverse event was falls

- 57.6% of individuals experienced 1 fall
- 34% experienced multiple falls
- 6% experienced an injurious fall
- More multiple falls in early-LTP group than late-LTP or HEP (p<0.07)
For patients in the first year post-stroke who can walk 10 feet but are not walking at speeds >0.8 m/s (1.8 mph):

This randomized trial provides strong and high quality evidence that:

- Structured progressive locomotor training (including BWSTT) is not superior to a structured strengthening and balance exercise program for walking recovery.

- Either structured program is more effective than usual care at 6 months post-stroke.

- Both interventions have low risks of adverse events, but structured exercise program in the home results in fewer adverse events compared to locomotor training.
Current Therapeutic Approaches

- Activity-based therapies
  - Robotic interventions
- Task-specific interventions
- Aerobic training
- Muscle Strengthening
- FES (neuroprosthetics)
- Virtual Rehabilitation
Results. Similar outcomes for final walking speed were found for the different prevailing treatment methods. Treatment gains were likewise comparable and generally insufficient for upgrading patients’ functional community walking capacity.

Conclusions. Different treatment methods exist for poststroke gait rehabilitation. Their availability, mode of application, and costs vary, yet outcomes are largely similar. Therefore, choosing an appropriate method may be guided by a pragmatic approach. Simple “low technology” and conventional exercise to date is at least as efficacious as more complex strategies such as treadmill and robotic-based interventions.
Similarities for success:
• Intensity
• Repeatability

Obstacles:
• Adverse personal effects
• Decreased cardiovascular potential
• Condition of peripheral muscle
• Heterogeneity of sample
• Responders versus non-responders
Current Therapeutic Approaches

• Activity-based therapies
  – Robotic interventions
• Task-specific interventions
• Aerobic training
• Muscle Strengthening
• FES (neuroprosthetics)
• Virtual Rehabilitation
Responders versus Non-Responders
What does EBP tell us about current clinical decision making?
“Pre-gait” Sequencing

Bridging → Quadruped → Sitting

Modified plantigrade ↔ Half-kneeling ↔ Kneeling

Standing → WALKING!!
Training symmetrical standing did not lead to symmetrical gait performance...locomotor pattern was only minimally affected by static balance exercises.

Remember Hodgson’s cats: those trained to stand could stand and not step, while those trained to step could not stand.
Standing ≠ Walking

What about BALANCE?

A direct comparison of local dynamic stability during unperturbed standing and walking.
Kang HG, Dingwell JB.

• Local dynamic stability properties were significantly different when comparing standing versus walking.
• COP divergence measures only significantly correlated with standing.
TASK SPECIFICITY MATTERS!

...But is that all that matters?
Elements of Optimized Walking Programs

- Strengthening and power training
- Cardiovascular training
- Dynamic balance training
- Motor control training
Advancing Locomotor Rehabilitation Measurement in Physical Therapy Practice
Guiding Questions

• Recovery versus compensation: how can measurement distinguish the two?

• Physical performance vs. mechanisms of response?

• How does your standardized “measurement” help guide clinical decision making?
Numeric Rating Scale (NRS)

The patient rates pain on a scale from 0 to 10.
Questions regarding traditional “Measurement”

1) Does improvement in a measure imply that the patient “recovered” neuromotor function or simply more efficiently compensated for impaired underlying body functions?

2) Does the measurement assist clinical decision making by differentiating between a variety of treatment options?
Historically, locomotor rehabilitation measurement has incorporated assessments using:

1) rating scales
2) timed movement tasks
3) laboratory based outcomes.

How may technological advances improve clinical measurement capabilities?
Rating Scales

• Often based on summary scores
  – Lose individual item sensitivity
  – Unable to measure subtle changes in control

• Makes no comments on mechanisms

• No information on recovery vs. compensation or guiding of treatment plan
Timed Movement Tasks

• Timed up and Go
• 6 minute walk test (distance)
• 10 meter walk test (speed)

Many, many very positive elements of these tests
  – Simple to measure
  – Inexpensive
  – Reliable and valid
  – Sensitive
  – Specific
Walking Speed is….

• “...almost the perfect measure” *(Wade 1992)*
  – Reliable *(Richards 1996)*
  – Valid *(Steffen 2002)*
  – Sensitive *(van Iersel 2008)*
  – Specific *(Harada 1995)*
  – Correlates with
    • Functional ability *(Perry 1995)*
    • Balance confidence *(Mangione 2007)*
Walking Speed: Predictive

- Future health status (Studenski 2003; Purser 2005)
- Functional decline (Brach 2002)
  - Hospitalization (Montero-Odasso 2005)
  - Discharge location (Salbach 2001; Rabadi 2005)
  - Mortality (Hardy 2007)
- Functional & Physiological changes (Perry 1995)
  - Potential for rehabilitation (Goldie 1996)
  - Aids in prediction of:
    - Falls (Guimaraes 1980)
    - Fear of falling (Maki 1997)

**Walking Speed**

[ meter per second (m/s) ]

- 0: Dependent in ADL's and IADL's
- 0.2: More likely to be Hospitalized
- 0.4: Need Intervention to Reduce Falls Risk
- 0.6: Less likely to have Adverse Event
- 0.8: D/C to SNF
- 1: D/C to Home more likely
- 1.2: Household Walker
- 1.4: Limited Community Ambulator

- Cross Street & Normal WS
Suggested Way to Measure

Acceleration zone (5 meters) → 10 meter Walk -Timed Section → Deceleration zone (5 meters)
HOWEVER...

Do these tests answer either of our two introductory questions regarding recovery and treatment planning?

What is the role of a vital sign?
<table>
<thead>
<tr>
<th>Diagnostic Test</th>
<th>Differential Exams</th>
</tr>
</thead>
<tbody>
<tr>
<td>BP</td>
<td>Cholesterol</td>
</tr>
<tr>
<td></td>
<td>Blood sugar</td>
</tr>
<tr>
<td></td>
<td>Adrenal gland fxn</td>
</tr>
<tr>
<td></td>
<td>Thyroid fxn</td>
</tr>
<tr>
<td></td>
<td>Kidney fxn</td>
</tr>
<tr>
<td>Walking speed</td>
<td>??</td>
</tr>
</tbody>
</table>
Gait Analysis
Laboratory Measures
Angles

GRFs

Moments
Laws of Motion

d’Alembert approach to inverse dynamics

General laws of motion (sum forces, sum moments):

\[ \vec{F}_{\text{contact}} + \vec{F}_{\text{distance}} + \vec{F}_{\text{inertia}} = 0 \]
\[ \vec{M}_{\text{contact}} + \vec{M}_{\text{distance}} + \vec{M}_{\text{inertia}} = 0 \]

General inertial forces and moments:

\[ \vec{F}_{\text{inertia}} = -m \cdot \vec{\alpha} \]
\[ \vec{M}_{\text{COM}}^{\text{inertia}} = \vec{\alpha} \cdot \vec{I} - \vec{\omega} \times \vec{I} \cdot \vec{\omega} \]

**NOTE:** \( \vec{M}_{\text{COM}}^{\text{inertia}} \neq \vec{\alpha} \cdot \vec{I} \)

**in general!!!**

General (3 dimensional) Parallel Axis Theorem:

\[ \vec{M}_{\text{inertia}}^{O} = \vec{p}^{O/\text{COM}} \times \vec{F}_{\text{inertia}} + \vec{M}_{\text{inertia}}^{\text{COM}} \]

Moments calculated at the proximal end of each segment:

\[ \vec{M}_{\text{contact}}^{F} = \left( \vec{p}^{\text{T} \text{COM}} \times (-m_{F} \cdot \vec{\alpha}_{F}) + \vec{R}_{F}^{-1} \cdot \left( -\left( \vec{R}_{F} \cdot \vec{\alpha}_{F} \right) \cdot \vec{I}_{F} - \left( \vec{R}_{F} \cdot \vec{\omega}_{F} \right) \times \vec{I}_{F} \cdot (\vec{R}_{F} \cdot \vec{\omega}_{F}) \right) \right) - \vec{p}^{\text{T} \text{COM}} \times (m_{F} \cdot \vec{g}) - \text{COP} \times \text{GRF} - \text{GRM} \]
\[ \vec{M}_{\text{contact}}^{S} = \left( \vec{p}^{\text{T} \text{COM}} \times (-m_{S} \cdot \vec{\alpha}_{S}) + \vec{R}_{S}^{-1} \cdot \left( -\left( \vec{R}_{S} \cdot \vec{\alpha}_{S} \right) \cdot \vec{I}_{S} - \left( \vec{R}_{S} \cdot \vec{\omega}_{S} \right) \times \vec{I}_{S} \cdot (\vec{R}_{S} \cdot \vec{\omega}_{S}) \right) \right) - \vec{p}^{\text{T} \text{COM}} \times (m_{S} \cdot \vec{g}) + \vec{M}_{\text{contact}}^{F} \]
\[ \vec{M}_{\text{contact}}^{T} = \left( \vec{p}^{\text{T} \text{COM}} \times (-m_{T} \cdot \vec{\alpha}_{T}) + \vec{R}_{T}^{-1} \cdot \left( -\left( \vec{R}_{T} \cdot \vec{\alpha}_{T} \right) \cdot \vec{I}_{T} - \left( \vec{R}_{T} \cdot \vec{\omega}_{T} \right) \times \vec{I}_{T} \cdot (\vec{R}_{T} \cdot \vec{\omega}_{T}) \right) \right) - \vec{p}^{\text{T} \text{COM}} \times (m_{T} \cdot \vec{g}) + \vec{M}_{\text{contact}}^{S} \]

\( F, S, \) and \( T \) reflect the moment at proximal foot, shank, and thigh segments respectively.

Joint reaction forces:

\[ \vec{F}_{\text{contact}}^{F} = -m_{F} \cdot \vec{\alpha}_{F} - m_{F} \cdot \vec{g} - \text{GRF} \]
\[ \vec{F}_{\text{contact}}^{S} = -m_{S} \cdot \vec{\alpha}_{S} - m_{S} \cdot \vec{g} + \vec{F}_{\text{contact}}^{F} \]
\[ \vec{F}_{\text{contact}}^{T} = -m_{T} \cdot \vec{\alpha}_{T} - m_{T} \cdot \vec{g} + \vec{F}_{\text{contact}}^{S} \]
### Gait Analysis: Full Body

**Reference Limb:**
- L
- R

<table>
<thead>
<tr>
<th>Major Deviation</th>
<th>Weight Accept</th>
<th>Single Limb Support</th>
<th>Swing Limb Advancement</th>
</tr>
</thead>
<tbody>
<tr>
<td>Minor Deviation</td>
<td>IC</td>
<td>LR</td>
<td>MSI</td>
</tr>
</tbody>
</table>

**Trunk:**
- Lean: B/F
- Lateral Lean: R/L
- Rotates: B/F

**Pelvis:**
- Hikes
- Tilt: F/A
- Lacks Forward Rotation
- Lacks Backward Rotation
- Excess Forward Rotation
- Excess Backward Rotation
- Iliopelvic Drop
- Contralateral Drop

**Hip:**
- Flexion: Limited
- Excess
- Inadequate Extension
- Past Retract
- Rotation: IRER
- ADD/ABduction: Add/Ab

**Knee:**
- Flexion: Limited
- Excess
- Inadequate Extension
- Wobbles
- Hyperextension
- Extension Thrust
- Varus/Varus: V/V
- Excess Contralateral Flex

**Ankle:**
- Forefoot Contact
- Flat Foot Contact
- Foot Slop
- Excess Plantar Flexion
- Excess Dorsiflexion
- Inversion/Eversion: IvEv
- Heel Off
- No Heel Off
- Drag
- Contralateral Vaulting

**Toes:**
- Up
- Inadequate Extension
- Clawed

**Major Problems**
- Weight Acceptance
- Single Limb Support
- Swing Limb Advancement

**Excessive UE Weight Bearing**

**Name:**

**Patient #:**

**Diagnosis:**
Spatiotemporal Analysis
BREAK
Elements of Optimized Walking Programs

• Motor control training
• Cardiovascular training
• Strengthening and power training
• Dynamic balance training
Examination of Walking Specific Motor Control
Ground Reaction Forces
The percentage of total anterior GRF (propulsive) impulse generated by the paretic leg, referred to as paretic propulsion (Pp), was calculated by dividing the propulsive impulse of the paretic leg by the sum of the paretic and non-paretic propulsive impulses.

\[ Pp = \frac{\text{Paretic Propulsive Impulse}}{\text{Paretic Propulsive Impulse} + \text{Non-Paretic Propulsive Impulse}} \]
Bowden, et al, 2006

Mild Hemiparesis
Propulsion_{Paretic} = 52.8%
Gait Velocity - 1.20 m/s

Moderate Hemiparesis
Propulsion_{Paretic} = 54.2%
Gait Velocity - 0.60 m/s

Severe Hemiparesis
Propulsion_{Paretic} = 8.9%
Gait Velocity - 0.44 m/s
Increased flexor activity in preswing (e.g., TA and RF) was correlated with decreased paretic propulsion.

Increased plantarflexor activity in late swing (e.g., MG and SO) was correlated with increased paretic propulsion.

Turns, et al, 2007
What correlates with propulsion?

- Propulsion_{Paretic} cannot be measured in a conventional clinical setting without access to force plates.

- Step length asymmetry relates to propulsive forces generated by the legs.
Paretic Step Ratio (PSR)

\[
PSR = \frac{\text{Paretic Step Length}}{\text{Stride Length}}
\]
$r = -0.78, p < .001$

Balasubramanian, et al, 2007
Mechanics of Propulsion and PSR

ACTIVE propulsion: generated by appropriately timed ankle power

PASSIVE propulsion: generated by properly positioned foot COM posterior to the pelvic COM (larger horizontal portion of the red arrow).

Therefore, emphasize hip extension!
Clinical Usefulness

Beyond simply promoting symmetry, PSR can be used to develop individual goals to train propulsive force production, equalize bilateral biomechanical involvement by improving hip extension, or promote paretic step initiation.

– Longer paretic steps may be due to compensation by greater non-paretic propulsion
– Weak relation with speed suggests compensatory strategies to attain functional speeds
– PSR may serve as a useful surrogate to assess propulsion during hemiparetic walking
• These measures are capable of distinguishing “recovery” from “compensation”
• They also are capable of distinguishing motor control problems and provide critical information for treatment.

BUT?

Of what value is this measure to a clinician without biomechanical laboratory capabilities?
Raw GRFs

Summed GRFs

GRFs vs. Acceleration
PERSON WITH STROKE

Raw GRFs

- Paretic leg
- Non paretic leg

Summed GRF

Summed GRF

GRFs vs. Acceleration

- Normed GRFs
- Acceleration
COMa as an outcome measure

Pre- and post-training accelerations of individuals participating in a locomotor training intervention. While COM acceleration increases, it does not approach normative values, particularly in the critical phase 3 and 4 period of the gait cycle, implying specific therapies need to target specific mechanistic elements of the walking pattern.

COMa before (blue line) and 2 minutes after (red line) incline training. A brief (5 minute) period of adaptation training yielded a 48% increase in both peak COMa and the time integral of the COMa curve in phase 3 and 4. The x-axis represents percentage of the gait cycle.
Portable applications

- segmental accelerations
- studies into shock absorption
- spatiotemporal gait parameters
- control of stability
- age-related changes in movement patterns
- relating quantifiable movement patterns to falls
COMa to diagnose motor control deficits

Figure 6. COMa sorted by Pp. Sorting by Pp yields three distinctly different acceleration profiles, although the double support phase (shaded area) are similar in shape. The low Pp group (b) generates very little acceleration in the double support phase. However, the high Pp group (c) demonstrates only positive acceleration through the first half of the gait cycle, while remaining negative throughout late double support and swing. (Bowden, Behrman et al. In Press). The x-axes represent percentages of the gait cycle.
Examination of Power Training
NORMAL ANKLE Joint Motion, Torque Demand & Muscle Action

IC

RANGE OF MOTION

Dorsiflexion

Plantar Flexion

TORQUE DEMAND

Dorsiflexion

Plantar Flexion

MUSCLE ACTION

Soleus

Gastrocnemius

Tibialis Anterior

Extensor Digitorum Longus

Extensor Hallucis Longus

GAIT CYCLE %

0 12 31 50 62 75 87 100

Ankle Moment (Nm/kg)
JOINT POWER

\[ P = \text{moment times angular velocity} \]

\[ P(+) = \text{concentric (moments and velocity same direction): shortening contraction} \]

\[ P(-) = \text{eccentric (moments and velocity in opposite direction): lengthening contraction} \]
Resistance training and locomotor recovery after incomplete spinal cord injury: a case series

CM Gregory*¹,², MG Bowden¹, A Jayaraman², P Shah², A Behrman¹,², SA Kautz¹ and K Vandenborne²

¹NF/SG Veterans Health System, Brain Rehabilitation Research Center, Gainesville, FL, USA; ²Department of Physical Therapy, University of Florida, Gainesville, FL, USA
Figure 2  Relative gains in muscle max-CSA in the PF (black bars) and KE (gray bars) muscle groups

Table 2  Pre- and post-RPT spatio-temporal characteristics of gait for maximal and self-selected gait speeds

<table>
<thead>
<tr>
<th></th>
<th>Pre</th>
<th>Post</th>
<th>Pre</th>
<th>Post</th>
</tr>
</thead>
<tbody>
<tr>
<td>Speed (m/s)</td>
<td>0.77 ± 0.04</td>
<td>1.03 ± 0.14</td>
<td>1.08 ± 0.09</td>
<td>1.47 ± 0.18</td>
</tr>
<tr>
<td>Step length (cm)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>More-involved</td>
<td>46.7 ± 6.1</td>
<td>61.8 ± 4.2</td>
<td>52.8 ± 6.7</td>
<td>72.6 ± 6.7</td>
</tr>
<tr>
<td>Less-involved</td>
<td>38.8 ± 6.1</td>
<td>57.8 ± 4.2</td>
<td>46.3 ± 6.7</td>
<td>72.1 ± 6.7</td>
</tr>
<tr>
<td>% Single support</td>
<td>53.3 ± 5.9</td>
<td>53.7 ± 4.1</td>
<td>40.4 ± 3.2</td>
<td>38.4 ± 3.1</td>
</tr>
<tr>
<td>% Double support</td>
<td>28.3 ± 3.6</td>
<td>32.1 ± 2.9</td>
<td>20.5 ± 1.1</td>
<td>23.0 ± 2.5</td>
</tr>
<tr>
<td>Cadence (steps/min)</td>
<td>89.0 ± 1.9</td>
<td>95.5 ± 2.6</td>
<td>120.2 ± 3.4</td>
<td>124.3 ± 6.1</td>
</tr>
</tbody>
</table>

Figure 4: Pre- (blue) and post-training (red) hip, knee and ankle powers plotted over the gait cycle in the more- and less-involved limbs of a person following incomplete-SCI.
Fig. 1. Mean joint power profiles of the affected and less-affected hip, knee and ankle joints of subjects with stroke before treatment (grey line), after treatment (black line) compared with normal elderly subjects (dashed line).
• H1 (hip extension during initial stance), H3 (hip flexion during early swing), and A2 (ankle plantarflexion during late stance and pre-swing) all increased after therapy and the A2 and H3 bursts were significantly correlated with walking speed (Richards et al., 2004).

• Furthermore, the A2 peak burst improvement was responsible for 25% of the gain in walking.

• Examining the interlimb coordination effects demonstrated that pre-training, paretic A2 and H3 accounted for 84% of the variance in walking speed, while post-training the non-paretic H3 replaces the paretic H3 to account for 82% of the post-training variance in patients that improved from 0.40 m/s to 0.58 m/s as a result of the intervention (Richards et al., 2004).
Examination of Cardiovascular Status
• Cardiorespiratory fitness is approximately 50% below age-matched controls
• Decreased fitness combines with increased energetic demands to decrease efficiency
• O$_2$ use normalized to walking speed 50% higher
• Due to reduced peripheral muscle activation, change in fiber type, reduced enzyme activities, and altered hemodynamics

*all responsive to exercise interventions
• Peak oxygen consumption ($\text{VO}_2$) values in patients with stroke are below the level required for many basic activities of daily living (ADL)

• Peak cardiovascular fitness levels are roughly half those of age-matched individuals

• Decreased CV fitness likely limits amount of walking, thereby limiting daily physical activity and contributing to a downward spiral

“Stroke is a cardiac event...”  
_Pam Duncan_
Voluntary Fatigue

Cadence < 40 rpm

Borg = 18

Dyspnea

Angina

HTN

Other

90% THR

Hypotension

ST segment depression

0.5%

32%

22%

15%

7%

0.5%

0.5%

0.5%

1%

Courtesy of Dorian Rose PT, PhD
Figure 1. Mean percent change in 6-min walk distance in T-AEX (solid line) and R-CONTROL (dotted line) groups. There was a significant group-by-time interaction in 6-min walk distance by repeated-measures ANOVA (†P<0.02) with progressive gains across the 6-month intervention period (⁎P<0.05). Values are mean±standard error.
TABLE 2. Effects of 6 Months T-AEX and R-CONTROL on Cardiovascular Fitness, Timed-Walk Performance, and Functional Mobility

<table>
<thead>
<tr>
<th>Variables</th>
<th>Treadmill Group</th>
<th>Control Group</th>
<th>P, Group-by-Time Interaction</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Baseline</td>
<td>Posttreatment</td>
<td>Baseline</td>
</tr>
<tr>
<td>Peak Vo2, L/min</td>
<td>1.22±0.47</td>
<td>1.43±0.57†</td>
<td>1.25±0.36</td>
</tr>
<tr>
<td>Peak Vo2, mL/kg/min</td>
<td>14.9±0.9</td>
<td>17.3±1†</td>
<td>14.7±1</td>
</tr>
<tr>
<td>Economy of gait, Vo2 (mL/kg/min)</td>
<td>10.7±0.6</td>
<td>9.9±0.6*</td>
<td>10±0.5</td>
</tr>
<tr>
<td>6-min walk, ft</td>
<td>761±73</td>
<td>922±79†</td>
<td>848±109</td>
</tr>
<tr>
<td>30-ft walk usual pace, m/s</td>
<td>0.63±0.06</td>
<td>0.74±0.06*</td>
<td>0.67±0.07</td>
</tr>
<tr>
<td>30-ft walk fast pace, m/s</td>
<td>0.82±0.08</td>
<td>0.95±0.09*</td>
<td>0.9±0.10</td>
</tr>
<tr>
<td>WIQ distance subscale, patients</td>
<td>45±7</td>
<td>70±7†</td>
<td>50±7</td>
</tr>
<tr>
<td>Rivermead Mobility Index, patients</td>
<td>11.3±0.4</td>
<td>12±0.3</td>
<td>11.7±0.4</td>
</tr>
</tbody>
</table>

*P<0.01 significance for time main effect after dropping group-by-time interaction.
†P<0.001 for significant improvement within T-AEX group.

Figure 2. Between-group comparison of peak aerobic capacity across 6 months. There was a significant time by group interaction in Vo2 peak (mL/kg/min) by repeated-measures ANOVA (†P<0.005). Vo2 peak was significantly different from baseline at both the 3-month and 6-month time points within the T-AEX group (*P<0.05). Values are mean±standard error.

Figure 3. Between-group comparison of WIQ distance scores across 6 months of training shows a significant and progressive improvement (*P<0.05) in the T-AEX group only. Values are mean±standard error.
Examination of Dynamic Balance Control
???
Figure 1. Clinical Balance Outcomes after a Locomotor Training Intervention. All three clinical measures improved post-training, with the BBT (p=0.003) and ABC (p=0.05) significantly. However only two participants by the BBT and three by the DGI changed falls risk status. No data were collected on actual falls frequency.
Figure 2a and 2b. COM and COP captured on the GAITRite Msqr. 1a depicts a normal pivot approach to the DGI stop and turn task, while 1b demonstrates a multiple step, decreased speed approach. Note the decrease smoothness in the COM tracing (green line) and COP tracing (red line).
Balance Control

Weerdesteyn, 2008

Sherrington et. al. 2008

Proposed Model
Treatment

• Increase task complexity and difficulty (IRT example)
• Athletic training
• Intensity of training
• Must know if people continue to fall
• STILL evolving
Putting it All Together
Walking Performance
Appendix – Components of the mobility intervention

<table>
<thead>
<tr>
<th>Task</th>
<th>Target</th>
<th>Description and progression</th>
</tr>
</thead>
<tbody>
<tr>
<td>Warm-up</td>
<td>ROM and flexibility</td>
<td>Marching on the spot, arm lifts, ankle circles, stretching of the trunk, thigh, and calf muscles.</td>
</tr>
<tr>
<td>Step-ups</td>
<td>Balance</td>
<td>Five minutes of placing each foot alternately on a step, progressing to stepping onto a step (step-ups), to a higher step, and to decreasing UE support; time divided evenly between leading with right versus left foot.</td>
</tr>
<tr>
<td>Balance beam</td>
<td>Balance</td>
<td>Five minutes of walking forwards, sideways, and backwards between two parallel lines, 20 cm apart, progressing to using one line, to using a balance beam, and finally to lateral stepping on the floor, feet crossing over in front or in back, and then alternating.</td>
</tr>
<tr>
<td>Kicking ball</td>
<td>Balance</td>
<td>Five minutes of kicking a ball against a wall, progressing to decreasing UE support, to increasing the distance from the wall, to kicking to a target, and to dribbling the ball around pilons; time divided evenly between kicking with right versus left foot.</td>
</tr>
<tr>
<td>Stand up and walk</td>
<td>Balance LE strength walking</td>
<td>With four standard armchairs placed at four corners of a square, 5 min of repeatedly standing up and walking to the chair directly in front, sitting, then standing up and walking to the chair on the left, etc., progressing from using arms to not using arms, and to decreasing the seat height.</td>
</tr>
</tbody>
</table>
### Appendix (Continued)

<table>
<thead>
<tr>
<th>Task</th>
<th>Target</th>
<th>Description and progression</th>
</tr>
</thead>
<tbody>
<tr>
<td>Obstacle course</td>
<td>Walking balance</td>
<td>Five minutes of stepping over an obstacle, stepping onto, along, and down from an aerobics step, walking over a mat, up a ramp, and returning, progressing by increasing the height and number of obstacles, and from completing the course walking forwards to walking backwards.</td>
</tr>
<tr>
<td>Treadmill</td>
<td>Walking endurance</td>
<td>Ten minutes of walking at a comfortable pace, progressing from using arms to not using arms, by increasing treadmill speed, and by adding an inclination.</td>
</tr>
<tr>
<td>Walk and carry</td>
<td>Walking balance</td>
<td>Five minutes of continuous walking carrying a grocery bag, progressing to carrying a bag in each hand, to increasing the weight of the bag, to carrying a laundry basket, and to stopping on command.</td>
</tr>
<tr>
<td>Speed walk</td>
<td>Walking endurance</td>
<td>Five minutes of continuous walking at maximum speed, progressing to running.</td>
</tr>
<tr>
<td>Walk backwards</td>
<td>Walking</td>
<td>Five minutes of continuously walking backwards, progressing from receiving physical assistance to receiving no assistance.</td>
</tr>
<tr>
<td>Stairs</td>
<td>LE strength</td>
<td>Five minutes of going up and down a flight of stairs, progressing from taking one step at a time to taking alternating steps, from using to not using the handrail, and to achieving a greater number of flights.</td>
</tr>
</tbody>
</table>
Results

• The mobility group members improved their SMWT performance by an average of 35 m more than members of the UE group (95% CI 7, 64).

• Gait speed increases
  – 0.11 m/s_ss (95% CI 0.03, 0.19) and 0.21 m/s_fast (95% CI .12, 0.30) faster than subjects in the UE group

• TUG
  – decreased 1.2 seconds in the intervention group (control increase by 1.7 seconds)

• BBS: No change (2 points to 1 point)

• Later reports indicate an improvement in balance/falls efficacy
Guiding task-oriented gait training after stroke or spinal cord injury by means of a biomechanical gait analysis

Sylvie Nadeau†‡ §, Cyril Duclos†‡ §, Laurent Bouyer§•|| and Carol L. Richards§•||

Abstract: To recover the ability to walk is one of the most important goals of persons recovering from a stroke or spinal cord injury (SCI). While a task-oriented approach to gait training is recommended, randomized controlled trials or meta-analyses comparing different methods of delivering training have failed in general to demonstrate the superiority of one approach over the other. The large variations in the mean outcome gait measures reported in these studies reflect, at least in part, the heterogeneity of the sensorimotor impairments underlying the gait disability as well as variations in the therapeutic response. The purpose of this chapter is to demonstrate that biomechanical gait analysis can reveal information pertinent to the selection of a task-oriented approach to enhance gait training as well as the therapeutic response that clinical evaluations alone cannot provide. We first briefly review locomotor impairments underlying the gait disability after stroke and SCI as well as the effects of selected technological task-oriented gait training interventions. We then give examples that demonstrate the use of gait analysis to pinpoint underlying impairments that can guide the choice of sensorimotor therapy and then immediately identify responders to the intervention. Such an individualized approach should promote therapeutic efficacy while leading over time to the identification of clinical indices to guide therapy when gait analysis is not feasible. Given the requirements of a gait analysis laboratory and the qualified personnel to capture and interpret the data, future studies will need to demonstrate the feasibility of the technological proposed approach and assess the costs and benefits for the health care system.
One therapy will NOT be the answer!
• What is required for successful locomotor rehabilitation?
  – Systematic measurement
  – Intensity
  – Task specific practice
  – Adherence to guiding principles

• What does evidence-based practice tell us about locomotor rehabilitation?

• Where to go from here and what will future generations of therapeutic practice include?
Guideline 1: Maximize weight bearing on the legs
Guideline 2: Afford the sensory experience consistent with walking
Guideline 3: Optimize kinematics for each motor task
Guideline 4: Maximize recovery and independence; minimize compensation
• Systematic
• Intensive
• Progressive
• Repetitive
• Measure outcomes
...plus Plasticity

• Use it or lose it
• Use it and improve it
  • Specificity
• Repetition matters
• Intensity matters
• Time matters
• Salience matters
• Age matters
• Transference
• Interference
Future directions

• Evaluation to yield meaningful, theory-based information to help guide clinical decision making

• Classification to help guide treatment options

• Understand ways to build on base therapy depending on impairments while being consistent with training guidelines.

• What tx, for whom, when, in what combination with other tx?

• Clinic as a laboratory.
Thanks to all of my mentors, collaborators, and funding sources!

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Lindsay Perry, DPT, NCS
Patrick Morgan, MS

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