Innovative Aphasia Intervention: Optimize Treatment Outcomes through Principles of Neuroplasticity, Caregiver Support, and Telepractice

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Learning Modules

- **Module I:** Incorporating Principles of Neuroplasticity in Therapy
- **Module II:** Incorporating Caregiver(s) in Therapy
- **Module III:** Using Telepractice to Increase Treatment Intensity

MODULE I

Incorporating Principles of Neuroplasticity into Therapy
Overview of Module I: Neuroplasticity

- Aphasia Introduction
- Principles of Neuroplasticity
- Translational Research: Repetition Priming Study (Off & Griffin, under review)
- Principles of Neuroplasticity Across Stages of Recovery
- Small Group Activity

Defining Aphasia

*Common Elements:*
- Language-level problem
- Includes receptive and expressive components
- Multimodal deficit of communication
- Caused by CNS dysfunction

*Aphasia is an acquired selective impairment of language modalities and functions resulting from a focal brain lesion in the language-dominant hemisphere that affects the person’s communicative and social functioning, quality of life, and the quality of life of his or her relatives and caregivers.*

(Papathanasiou et al., 2013)

Differential Diagnosis

- Aphasia is NOT a sensory deficit (e.g., hearing, sight, touch)
- Aphasia is NOT a motor deficit (e.g., dysarthria, apraxia)
- Aphasia is NOT a recognition deficit (i.e., agnosia)
- Aphasia is NOT a memory deficit?
- Aphasia is NOT related to confusion
Classifying Aphasic Syndromes

- Classical “Anatomic Disconnection Model” (Geschwind, 1967)
- Goodglass (2001): categorize language deficits under 10 different types (comprehension or production disorders)
- Types of language errors
- Language production and related impairments of spontaneous speech
- Connectionist model: Fluent/Non- Fluent

FLUENT VS. NONFLUENT APHASIA

<table>
<thead>
<tr>
<th>NON-FLUENT APHASIA</th>
<th>FLUENT APHASIA</th>
</tr>
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<tbody>
<tr>
<td>Lesions usually anterior to central sulcus</td>
<td>Lesions usually posterior</td>
</tr>
<tr>
<td>Produce fewer number of words than normal</td>
<td>Patients talk with an easy flow</td>
</tr>
<tr>
<td>Words are often fairly accurate but are difficult to get out.</td>
<td>Difficulty is with word selection</td>
</tr>
<tr>
<td>Numerous pauses in connected speech</td>
<td>Numerous word-finding errors</td>
</tr>
<tr>
<td>Agrammatism is common</td>
<td>Circumlocution is common</td>
</tr>
<tr>
<td></td>
<td>Jargon is common</td>
</tr>
</tbody>
</table>

Classical Aphasia Syndromes (Beeson & Rapcsak, 2006)

**Fluent**
- Anomic
- Conduction
- Transcortical Sensory
- Wernicke

**Non-Fluent**
- Transcortical Motor
- Broca
- Mixed Transcortical
- Global

Unusual Aphasia Syndromes (Potagas, Kasselmis, & Evdokimidis, 2013)

- Subcortical Aphasia
- Crossed Aphasia
- Sign Language Aphasia
WHO International Classification of Functioning, Disability, and Health (ICF) Model

WHO-ICF Impact on Aphasia Assessment & Treatment

- Aphasia effects approximately 25% of all stroke survivors

- A person with aphasia has:
  - Language impairments
  - Communication activity limitations
  - Participation restrictions including professional/vocational, recreational, educational obstacles that impact family and social life

- Assessment and treatment should involve each component of the ICF framework
Evidence-Based Aphasia Treatment

(Worrall, Papathanasiou, & Sherratt, 2012)

Select the best approach for each individual client.
Your decision must be made based upon a strong rationale including:
- Evidence base
- Client/caregiver/family needs
- Your experience and any constraints

Where do I find evidence?
- Cochrane Review
- Academy of Neurologic Communication Disorders and Sciences (www.ancds.org)
- ASHA EBP Compendium & Practice Portal (http://www.asha.org/members/ebp/compendium)
- Literature searches for primary research articles

The Big Picture

• Language is subserved by a highly interactive network, including, but not limited to language areas

• Classic aphasia syndromes do not always coincide with specific brain lesions

• Symptoms of each client do not always coincide with description of classic aphasia types

Activate intact neural connections, promote cortical reorganization, and get around what was damaged by optimizing what is left (undamaged)

Stroke Rehabilitation

*Stroke:* core area of damaged/destroyed neurons with surrounding area also damaged → functional impairments

- Reduce behavioral, motor, and/or cognitive effects of a neurological impairment (Dobkin, 2004)
- Capitalize on neuroplastic processes
**Neuroplasticity** (Papathanasiou, Coppens, Potagas, 2011)

The brain’s capacity to change, allowing the brain to respond to environmental changes or changes in the organism itself (Kolb, 1995)

**Neural Plasticity**: micro level; cellular/network level

**Behavioral Plasticity**: macro level; behavioral/system level

*The existing data strongly suggest that neurons, among other brain cells, possess the remarkable ability to alter their structure and function in response to a variety of internal and external pressures, including behavioral training. We will go so far as to say that neural plasticity is the mechanism by which the brain encodes experience and learns new behaviors. It is also the mechanism by which the damaged brain relearns lost behavior in response to rehabilitation.*

(Kleim & Jones, 2008, S225)

**Neurobiological Approach** (Rayner et al, 2008)

- Neuroplastic processes support
  - Development
  - Learning
  - Maintaining performance while aging
  - Response to brain injury
- Adaptive vs. maladaptive processes
- Differential contribution of neural mechanisms depending on stage of recovery
- Functional reorganization

**GOAL**: maximize neural plasticity that supports functional communication gains

**Timeline & Patterns of Recovery**

- Shock (immediately following injury)
- Rapid recovery (hours to days) – **ACUTE**
  - Reperfusion
  - Physical repair of penumbra cells
  - Begin reorganization (spontaneous)
- Steady improvement (weeks) – **SUBACUTE**
  - Continued spontaneous neural reorganization
  - Added reorganization secondary to rehabilitation
- Decreased rate of improvement (months & years) - **CHRONIC**
Neurobiological Investigations of Recovery


1) Early phase: (0-4 days post CVA) - decreased neural activation in non-damaged speech regions
2) Early Post-Acute phase: (about two weeks post CVA) - increased neuronal activation in the R homologue of L speech region
3) Consolidation (4-12 months): decreased R hem homologue activation and increased activation in intact L speech regions

Rijntjes (2006)

- Phase 1: Depression of entire network
- Phase 2: Up-regulation and over activation of some areas, particularly in the unaffected hemisphere
- Phase 3: Activation decreases; balance achieved b/t hemispheres

Biomechanical & Physiologic Mechanisms of Recovery

Resolution or regression of diaschisis: post-injury protective mechanisms diminish, allowing brain areas to function again

Restoration: Reactivation of brain areas; re-establish neural connectivity as a result of biochemical/physiologic mechanisms

Recruitment: Enlisting brain areas that may not have been used prior to injury for certain functions

Retraining: Brain areas perform novel or additional functions as a result of rehab training

Functional Takeover: Contralateral hemisphere takes over functions "unmasking"

Behavioral Mechanisms of Recovery

Restitution-restoration-reactivation:

Reorganization-reconstruction-substitution:

Relearning:

Facilitation:

Functional substitution-functional reorganization-functional compensation:
**Neurorehabilitation & Neuroplasticity**  
(Kleim & Schwerin, 2010)

<table>
<thead>
<tr>
<th>Behavioral Level</th>
<th>Neurophysiologic Level</th>
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<tbody>
<tr>
<td><strong>Recovery:</strong> Capacity to perform a previously impaired task in the same manner as before the injury</td>
<td><strong>Recovery:</strong> Restoration of the function within an area of the cortex that was initially lost after the injury</td>
</tr>
<tr>
<td><strong>Compensation:</strong> Use of new strategy to perform same task</td>
<td><strong>Compensation:</strong> Different neural tissue takes over the functions lost after injury</td>
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**Principles of Experience-Dependent Plasticity**  
(Kleim & Jones, 2008, S227, table 1)

1. **Use It or Lose It** - Failure to drive specific brain functions can lead to functional degradation.
2. **Use It and Improve It** - Training that drives a specific brain function can lead to an enhancement of that function.
3. **Specificity** - The nature of the training experience dictates the nature of the plasticity.
5. **Intensity Matters** - Induction of plasticity requires sufficient training intensity.
6. **Time Matters** - Different forms of plasticity occur at different times during training.
7. **Salience Matters** - The training experience must be sufficiently salient to induce plasticity.
8. **Age Matters** - Training-induced plasticity occurs more readily in younger brains.
9. **Transfer** - Plasticity in response to one training experience can enhance the acquisition of similar behaviors.
10. **Interference** - Plasticity in response to one experience can interfere with the acquisition of other behaviors.

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**Repetition Matters**  
(Off & Griffin, under review)

**Participants**
- 7 PWA; 1 control
- 6-240 months post-onset
- 47-90 years old

**Training Protocol**
- 40 trained items (20 1-trial/session; 20 4-trial/session)
- 2-3x/week, up to 15 sessions or 5 weeks

**Total Training Sessions (baseline)**

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<thead>
<tr>
<th></th>
<th>P1</th>
<th>P2</th>
<th>P3</th>
<th>P4</th>
<th>P5</th>
<th>P6</th>
<th>P7</th>
<th>Control</th>
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<tbody>
<tr>
<td>P3</td>
<td>16</td>
<td>15</td>
<td>15</td>
<td>15</td>
<td>15</td>
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<td>P6</td>
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<td>12</td>
<td>12</td>
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<td>P7</td>
<td>9</td>
<td>10</td>
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<td>10</td>
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<td>P1</td>
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<td>13</td>
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<td>13</td>
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**Total Naming Attempts (baseline)**

<table>
<thead>
<tr>
<th></th>
<th>Trained Items (1 trial)</th>
<th>Trained Items (4 trials)</th>
</tr>
</thead>
<tbody>
<tr>
<td>P3</td>
<td>600</td>
<td>2400</td>
</tr>
<tr>
<td>P4</td>
<td>520</td>
<td>2080</td>
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<td>340</td>
<td>1440</td>
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<tr>
<td>P7</td>
<td>500</td>
<td>2000</td>
</tr>
<tr>
<td>P1</td>
<td>600</td>
<td>2400</td>
</tr>
<tr>
<td>Control</td>
<td>900</td>
<td>3600</td>
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</table>
Experience-Dependent Training Principles (Kleim & Jones, 2008; Leon et al, 2011)

- **Timing of Treatment Delivery**
  - Intensive intervention early after injury may adversely affect recovery
  - What exactly constitutes the "acute" period for humans? When can we initiate aggressive treatment that is geared to restore impaired functions?

- **Use It or Lose It**
  - "Learned nonuse"

- **Generalization & Transfer**

- **Repetition and Intensity of Treatment**
  - Implications for strength of the response to the intervention
  - Behavioral changes precede neural changes
  - Practice does not necessarily translate to learning/retention
  - Conditions of practice matter

Applying Neuroplasticity to Aphasia Rehabilitation

- **Physical Repair (activation):**
  - enhance temporarily impaired language functions
  - stimulate patient by all means necessary to communicate as appropriately as possible

- **Neuronal adaptation (symptom-specific training):**
  - Relearn degraded linguistic knowledge
  - Reactivate impaired linguistic modalities
  - Establish compensatory linguistic strategies

- **New Learning**
  - Learning mechanisms to complement and maintain, enhance, and transfer linguistic skills previously addressed in therapy

How do you implement these principles?

**Use it or Lose it**

- Continue to practice speech if you want to improve speech
- Maximize opportunities for talking, writing, communicating
- Encourage exploration of communication opportunities at home and in the community

**Use it and Improve it**

- Practice of speech can lead to improvement of speech
- Intensive therapy → All roads lead to verbal production

**Specificity**

- Well-developed speech and language therapy plan to improve speech
- Address specific aspects of communication (e.g., speech, comprehension),
- Address specific communication targets (e.g., core vocabulary)
- Address specific communication situations
Repetition Matters
• Brain plasticity requires a lot of repetition
• Maximize opportunities for production of speech targets

Intensity Matters
• Brain plasticity requires a lot of practice
• Massed practice (vs. distributed) allows for increased intensity, within and across sessions

Time Matters
• Different forms of plasticity occur at different times during training; know

Salience Matters
• Improving your speech MUST be important to you.
• Individualized treatment plans to capitalize on client interest and motivation

Age Matters
• Brain plasticity is better in younger brains. But plasticity is possible at all ages

Transference
• Training one skill can lead to improvements in other similar skills
• Identify areas of need underlying speech production deficits (e.g. auditory comprehension, attention)

Interference
• Sometimes training one thing can hinder learning something else