D. Cognitive Recovery Post-Stroke Educational Supplement

Robert Teasell MD FRCPC, Andrew McClure, Katherine Salter, Manuel Murie-Fernandez MD

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111 pages
D1. Cognitive Disorders
D1.1 Cognitive Disorders: General Information
D1.1 Cognitive Disorders: General Information

D1.1.1 Prevalence and Natural History

Q1. What is the prevalence and natural history of cognitive impairment post stroke?

Answers
1. Two-thirds of stroke patients experience cognitive impairment or decline following stroke. One-third develop dementia.
2. Almost one-fifth of stroke patients with cognitive impairment post stroke improve.

Discussion
As many as two-thirds of stroke patients experience cognitive impairment or decline following stroke. Approximately one-third develop dementia (Leys et al. 2005). The risk for developing dementia may be 10 times greater among individuals with stroke than those without (Ukraintseva et al. 2006). The risk for cognitive impairment or decline is increased by a history of stroke. While cognitive decline may continue post stroke, approximately 16-20% of patients with cognitive impairment improve (Rasquin et al. 2005, Ballard et al. 2003). While most improvements occur in the first 3 months, recovery may continue for at least the first year post stroke (Kotila et al. 1984). Mortality rates among stroke patients with dementia are 2 to 6 times greater than among stroke patients without dementia (Leys et al. 2005).

D1.1.2 Impact on Rehabilitation Outcomes

Q2. What is the impact of cognitive impairment on rehabilitation outcomes?

Answer
1. Decreased ADLs.
2. Decreased Instrumental ADLs (IADLs).
3. Greater mortality one year post discharge.

Discussion

Although the presence of cognitive impairment may be associated with decreased ADL function, it has been demonstrated that it is not a significant predictor of ADL function at 6 months post stroke (Zinn et al. 2004). However, higher level ADLs or instrumental function may be severely
impacted by the presence of cognitive ability. At 6 months post stroke, the presence of
cognitive impairment has been shown to be associated with and predictive of decreased IADL
function (Zinn et al. 2004).

D1.1.3 Risk of Developing Dementia

Q3. What is the likelihood that stroke survivors will develop dementia?

Answer
1. 2-10X greater likelihood of developing dementia following a stroke.
2. As many as 25% of stroke survivors are diagnosed with dementia 12 months post stroke.

Discussion
It has been suggested that approximately 25% of stroke survivors develop dementia within 12
months of having a stroke (Tatemachi et al. 1993, Sacco et al. 1994, Barba et al. 2000, Inzitari
et al. 1998, Lowery et al. 2002, Desmond et al. 2000, Linden et al. 2004), with older stroke
patients having an even higher risk of developing dementia (Kokman et al. 1996, Tatemachi et
al. 1993, Pohjasvaara et al. 1997). Kokman et al. (1996) reported that stroke survivors have a 2
to 10-fold relative risk of developing dementia, which persists for at least 3 to 5 years. Results
of the Framingham study demonstrated that, over a 10-year period, individuals with baseline
stroke had twice the risk of developing dementia than age and gender-matched controls that at
baseline, had neither a history of dementia nor stroke (OR = 2.0; 95% CI 1.4 to 2.9) (Ivan et al.
2004).

D1.1.4 Distinguishing Between Vascular and Alzheimer’s Dementia

Q4. Describe the difference between Vascular vs. Alzheimer’s Dementia.

Answers
The differences between Vascular and Alzheimer’s Dementia are listed below:
<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Vascular Dementia</th>
<th>Alzheimer's Dementia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Onset</td>
<td>Sudden or gradual</td>
<td>Gradual</td>
</tr>
<tr>
<td>Progression</td>
<td>Slow, stepwise fluctuation</td>
<td>Constant, insidious decline</td>
</tr>
<tr>
<td>Neurological findings</td>
<td>Evidence of focal deficits</td>
<td>Subtle or absent</td>
</tr>
<tr>
<td>Memory</td>
<td>Mildly affected</td>
<td>Early and severe deficit</td>
</tr>
<tr>
<td>Executive function</td>
<td>Early and severe</td>
<td>Late</td>
</tr>
<tr>
<td>Dementia type</td>
<td>Subcortical</td>
<td>Cortical</td>
</tr>
<tr>
<td>Neuroimaging</td>
<td>Infarct of white matter lesions</td>
<td>Normal, hippocampal atrophy</td>
</tr>
<tr>
<td>Gait</td>
<td>Often disturbed early</td>
<td>Usually normal</td>
</tr>
<tr>
<td>Cardiovascular history</td>
<td>TIAs, strokes, vascular risk factors</td>
<td>Less common</td>
</tr>
</tbody>
</table>

**Discussion**

Graham et al. (2004) noted the patients with vascular dementia had greater impairments of semantic memory, executive and attentional functioning as well as deficits of visuospatial and perceptual skills while the Alzheimer’s dementia patients suffered greater impairment of episodic memory.

Sachdev et al. (2004) demonstrated that patterns of impairment in patients with vascular dementia and vascular cognitive impairment were qualitatively similar although they differed quantitatively. Patients with vascular dementia and vascular cognitive impairment both displayed deficits of information processing speed, attention, working memory and praxis-gnosis function, but patients with vascular dementia were significantly more impaired within these cognitive domains. Deficits in abstraction, mental flexibility, information processing speed and working memory distinguished both impaired groups from a group of age-matched controls.

Kalaria and Ballard (2001) described the cognitive syndrome of post-stroke dementia as the following:

- Occurs in up to 30% of stroke patients
- Progresses slowly
- Predominately executive dysfunction
- Subcortical and frontal lobe deficits are less obvious
- Memory and language deficits are less obvious
- Late stage memory and dementia

**D1.1.5 “Gold Standard” for Diagnosis of Post-Stroke Dementia**

**Q5. What is the “gold standard” for the diagnosis of post-stroke dementia?**

**Answer**
1. There is no “gold standard” for the diagnosis of post-stroke dementia.

Discussion
At present there is no “gold standard” for the diagnosis of vascular dementia (Chui 2000). While some use a modified version of the DSM-IV criteria (Ballard 2002) to diagnose vascular dementia, while Roman (2003) used the following criteria:

- Cognitive loss
- Vascular brain lesions identified by brain imaging
- A temporal link between stroke and dementia
- Exclusion of other causes of dementia.

Roman (2003) also adds the onset of dementia must be within 3 months of a symptomatic stroke (this criteria is waived in patients with subacute VaD), and two cognitive domains other than memory be impaired for an appropriate diagnosis. Cognitive domains to be examined include:

1. Memory
2. Praxis
3. Language
4. Orientation
5. Constructional ability
6. Executive control function

The authors suggest that while the first 5 cognitive domains can be assessed with the Mini-Mental State Examination, executive functioning can be examined using the Clock-Drawing Task. It should be noted that the MMSE is insensitive to executive function, an important component of vascular dementia (Royall 2000, Royall et al. 2002).

In a recent report, Hachinski et al. (2006) reported the results of a workshop intended to identify screening methods for the identification of individuals with possible cognitive and behavioural impairment, and to establish minimum datasets for clinical practice and research studies of vascular cognitive impairment. Participants included researchers in clinical diagnosis, epidemiology, neuropsychology, brain imaging, neuropathology, experimental models, biomarkers, genetics and clinical trials. Recommendations were produced for each working group within the following areas: clinical/epidemiology, neuropsychology, imaging, neuropathology, experimental markers, biomarkers, genetics and clinical trials. The 5-minute neuropsychological protocol included selected subsets of the Montreal Cognitive Assessment (5-word memory task – registration, recall, recognition, 6-item orientation and 1-letter phonemic fluency). This could be supplemented with a cube and clock drawing task, a short Trails B test and other brief attention language and abstraction task. Given more time, the original trailmaking test, a semantic fluency test or the MMSE could be added (if administered on a different day or more than one hour following the 5-minute protocol). Inclusion of the MMSE in the abbreviated assessment was rejected as it lacks sufficient assessment of executive function and is relatively insensitive to mild memory impairment (Hachinski et al. 2006).

References

Ballard CG, Rowan E, Stephens S, Kalaria RN, Kenny RA. Prospective follow-up study between 3 and 15 months after stroke. Improvements and decline in cognitive function among dementia-free stroke survivors >75 years of age. Stroke 2003; 34:2440-2445.


D1.2 Assessment of Cognitive Disorders Post-Stroke
D1.2 Assessment of Cognitive Disorders Post-Stroke

D1.2.1 Mini Mental State Examination

Q1. Discuss the Mini Mental State Examination in terms of describing the test and noting its strengths and weaknesses.

Answers
1. Brief screening tool of cognitive impairment.
2. Consists of 11 simple questions or tasks, typically grouped into 7 cognitive domains: orientation to time, orientation to place, registration of three words, attention and calculation, recall of three words, language and visual construction.
3. Total score of 30; score of 23 or less indicates cognitive impairment.
4. Advantages are it is brief, inexpensive, simple to administer and widely accepted.
5. Disadvantages are it is affected by age, education and sociocultural background and may be less sensitive to mild cognitive impairment and right hemispheric strokes.

Discussion

The Mini-Mental State Examination

<table>
<thead>
<tr>
<th>Testing Item</th>
<th>Maximum Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Orientation</td>
<td></td>
</tr>
<tr>
<td>What is the date?</td>
<td>5</td>
</tr>
<tr>
<td>Where are we?</td>
<td>5</td>
</tr>
<tr>
<td>Registration</td>
<td></td>
</tr>
<tr>
<td>Name 3 objects (1 second to say each) and then ask the patient to repeat all 3 after you have said them. Give one point for each correct answer. Continue repeating all 3 objects until the patient learns all 3. Count trials and record.</td>
<td>3</td>
</tr>
<tr>
<td>Attention and Calculation</td>
<td></td>
</tr>
<tr>
<td>Serial 7’s. One point for each correct response. Stop after 5 answers. As an alternative, spell “world” backwards.</td>
<td>5</td>
</tr>
<tr>
<td>Recall</td>
<td></td>
</tr>
<tr>
<td>Ask for the 3 objects named in Registration. Give 1 point for each correct answer.</td>
<td>3</td>
</tr>
<tr>
<td>Language</td>
<td></td>
</tr>
<tr>
<td>Name a pencil and watch</td>
<td>2</td>
</tr>
<tr>
<td>Repeat the following “No ifs, ands, or buts”</td>
<td>1</td>
</tr>
<tr>
<td>Follow a 3-stage command. “Take paper in your right hand, fold it in half, and put it on the floor.”</td>
<td>3</td>
</tr>
<tr>
<td>Read and obey the following: CLOSE YOUR EYES</td>
<td>1</td>
</tr>
<tr>
<td>Write a sentence</td>
<td>1</td>
</tr>
<tr>
<td>Copy a design</td>
<td>1</td>
</tr>
</tbody>
</table>

_/30_
Mini-Mental State Examination

<table>
<thead>
<tr>
<th>Questions</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>What does it measure?</td>
<td>The MMSE is a brief screening tool that provides a quantitative assessment of cognitive impairment (Folstein et al. 1975).</td>
</tr>
<tr>
<td>What is the scale?</td>
<td>The MMSE consists of 11 simple questions or tasks, typically grouped into 7 cognitive domains: orientation to time, orientation to place, registration of three words, attention and calculation, recall of three words, language and visual construction.</td>
</tr>
<tr>
<td>What are the key scores?</td>
<td>The test yields a total score of 30, with a score of 23 or less generally accepted as the cutoff score indicating the presence of cognitive impairment (Dick et al. 1984). Levels of impairment have also been classified as none (24-30); mild (18-24) and severe (0-17) (Tombaugh &amp; McIntyre 1992).</td>
</tr>
<tr>
<td>What are its strengths?</td>
<td>Only requiring 10 minutes to complete, the MMSE is brief, inexpensive and simple to administer. Its widespread use and accepted cut-off scores increase its interpretability.</td>
</tr>
<tr>
<td>What are its limitations?</td>
<td>Low levels of sensitivity have been reported, particularly among individuals with mild cognitive impairment and patients with right-sided strokes (Tombaugh &amp; McIntyre, 1992; de Koning et al. 1998, Dick et al. 1984). The MMSE has been shown to be affected by age, level of education and sociocultural background, which may lead to misclassification (Tombaugh &amp; McIntyre 1992, Bleeker et al. 1988, Lorentz et al. 2002).</td>
</tr>
</tbody>
</table>

Summary

<table>
<thead>
<tr>
<th>Reliability</th>
<th>Validity</th>
<th>Responsiveness</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rigor</td>
<td>Results</td>
<td>Rigor</td>
</tr>
<tr>
<td>+++</td>
<td>+++ (TR)</td>
<td>+++</td>
</tr>
<tr>
<td>+++</td>
<td>++ (IO)</td>
<td></td>
</tr>
<tr>
<td>++</td>
<td>(IC)</td>
<td></td>
</tr>
</tbody>
</table>

NOTE: +++=Excellent; ++=Adequate; +=Poor; n/a = insufficient information; TR = Test re-test; IC = Internal consistency; IO = Interobserver; varied (re floor/ceiling effects; mixed results)

D1.2.2 The Clock-Drawing Test

Q2. Describe the Clock-Drawing Test including its strengths and weaknesses.

Answers
1. Patient asked to draw a clock with numbers and the hand of the clock placed at a requested time.
2. Provides quick assessment of visuospatial and praxis abilities and may detect deficits in attention and executive function.
3. Strengths are it is brief, inexpensive and easy to administer; correlates well with other cognitive screening methods.
4. Weaknesses are it is negatively influenced by increasing age, less education and depression.

**Discussion**

**The Clock-Drawing Test**

<table>
<thead>
<tr>
<th>Questions</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>What does it measure?</strong></td>
<td>The CDT provides a quick assessment of visuospatial and praxis abilities and may detect deficits in both attention and executive dysfunction (Adunsky et al. 2002; Suhr et al. 1998; McDowell &amp; Newell 1996).</td>
</tr>
<tr>
<td><strong>What is the scale?</strong></td>
<td>The CDT involves having the patient draw a clock, place the numbers on the clock in their proper positioning and then place the arms of the clock at a requested time. The task itself is viewed as being highly complex, involving a number of neuropsychological abilities (Suhr et al. 1998).</td>
</tr>
<tr>
<td><strong>What are the key scores?</strong></td>
<td>Numerous scoring systems for the CDT have been suggested, ranging from simple to complex as well as from quantitative to qualitative. In general however, they all evaluate errors and/or distortions in the form of omissions of numbers and errors in their placement such as perseverations, transpositions, and spacing (McDowell &amp; Newell 1996).</td>
</tr>
<tr>
<td><strong>What are its strengths?</strong></td>
<td>The CDT is brief, inexpensive and easy to administer. The CDT may help to create a more complete picture of cognitive function when it is used with other assessment tools (Ruchinskas &amp; Curyto 2003; McDowell &amp; Newell 1996; Suhr &amp; Grace, 1999). Despite different scoring systems, The CDT has demonstrated acceptable levels of reliability and has been shown to correlate highly with other cognitive screening measures. (Scanlan et al. 2002; Ruchinskas and Curyto 2003; McDowell and Newell 1996).</td>
</tr>
<tr>
<td><strong>What are its limitations?</strong></td>
<td>Like most other neuropsychological screening measures, the CDT is negatively influenced by increasing age, reduced education and the presence of depression (Ruchinskas &amp; Curyto 2003; Lorentz et al. 2002). The CDT may also be affected by visual neglect, hemiparesis and motor dyscoordination (Ruchinskas &amp; Curyto 2003). The most effective use of the CDT may be as a supplement to other cognitive assessments rather than as the sole, independent screening device for cognitive impairment (McDowell &amp; Newell 1996). For example, it is an effective supplement to the MMSE and the CAMCOG.</td>
</tr>
</tbody>
</table>

**Summary**

<table>
<thead>
<tr>
<th>Reliability</th>
<th>Validity</th>
<th>Responsiveness</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rigor</td>
<td>Results</td>
<td>Rigor</td>
</tr>
<tr>
<td>++</td>
<td>+++ (TR)</td>
<td>+++</td>
</tr>
</tbody>
</table>
D1.2.3 The Montreal Cognitive Assessment

Q3. Describe the MOCA including its strengths and limitations.

**Answers**
1. Screening tool to detect mild cognitive impairment.
2. Uses tasks such as picture naming, clock drawing and recall to assess the following domains: attention and concentration, executive functions, memory, language, visuoconstructive skills, conceptual thinking, calculations, and orientation.
3. Less than 26/30 indicates cognitive impairment.
4. Strengths are it can detect mild forms of impairment when MMSE is normal.
5. Weaknesses are its reliability and validity have not been fully tested and it has not been evaluated in stroke patients.

**Discussion**

Montreal Cognitive Assessment

<table>
<thead>
<tr>
<th>Questions</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>What does it measure?</td>
<td>Designed as a screening tool to detect mild cognitive impairment (Nasreddine et al. 2005).</td>
</tr>
<tr>
<td>What is the scale?</td>
<td>The MoCA uses tasks such as picture naming, clock drawing and recall to assess the following domains: attention and concentration, executive functions, memory, language, visuoconstructive skills, conceptual thinking, calculations, and orientation.</td>
</tr>
<tr>
<td>What are the key scores?</td>
<td>The MoCA yields a total score out of 30 with scores of 26 or lower indicating the presence of cognitive impairment.</td>
</tr>
<tr>
<td>What are its strengths?</td>
<td>The MoCA is able to detect mild forms of impairment in patients that score in the normal range on other assessment measures (i.e., the MMSE) (Nasreddine et al. 2005).</td>
</tr>
<tr>
<td>What are its limitations?</td>
<td>The MoCA is a relatively new measurement tool; thus, its reliability and validity may not yet be thoroughly tested. It has not been evaluated for use with individuals who have experienced a stroke.</td>
</tr>
</tbody>
</table>
References


D1.3 Treatment of Cognitive Disorders Post Stroke
D1.3 Treatment of Cognitive Disorders Post Stroke

D1.3.1 Medications in Treatment of Vascular Cognitive Impairment

**Q1. Assuming depression has been ruled out, can medications be used in the treatment of vascular cognitive impairment?**

**Answers**

1. Yes.
2. Cholinesterase inhibitors, in particular Donepezil, have been shown to improve cognitive function.
3. Nimodipine, Memantine and Pentoxifyline have all been shown to have some benefit.

**Discussion**

According to Devasenapathy and Hachinsky (2000) the early manifestation of vascular cognitive impairment can be thought of as an important sign of imminent future stroke that requires the same urgent clinical management as symptomatic cerebrovascular disease. Although many medications have been used for the treatment of cognitive impairment, most have only modest and/or clinically irrelevant effects (Chui 2000). Listed below are some of the most commonly used medications:

**Cholinesterase inhibitors:** These medications have mainly been used in the treatment of the Alzheimer’s type of dementia. Although there is much less evidence regarding effectiveness (Craig and Birks 2005), three of the cholinesterase inhibitors (Donepezil, rivastigmine, and galantamine) have also been investigated for use in the treatment of vascular dementia.

**Donepezil:** Use of Donepezil in patients with mild to moderate vascular cognitive impairment is associated with cognitive improvement and global function improvement. (Passmore et al. 2005, Malouf and Birks 2004). Based on the conclusions of the EBRSR (Teasell et al. 2008), there is strong evidence that treatment with donepezil improves cognitive function in patients with vascular dementia.

**Rivastigmine:** Treatment with Rivastigmine is associated with more stable cognitive performance and improved behavioural outcomes among patients with subcortical vascular dementia. (Moeretti et al. 2001, 2002, 2003, 2004). Based on the conclusions of the EBRSR (Teasell et al. 2008), there is limited evidence that treatment with rivastigmine may stabilize cognitive performance and improve behavioural outcomes in patients with subcortical vascular dementia.

**Galantamine:** Treatment with galantamine is associated with improvements in cognitive and functional ability; however these benefits are more apparent for mixed Dementia (Erkinjuntti et al. 2002, 2003). Based on the conclusions of the EBRSR (Teasell et al. 2008), there is moderate evidence that treatment with galantamine is associated with cognitive and functional benefits; however, further study of its effectiveness among patients with vascular dementia is required.
Other drugs which have been studied include Nimodipine, Memantine and Pentoxifyline.

**Nimodipine:** Use of Nimodipine is beneficial for memory (Sze et al. 1998) and may slow cognitive deterioration. It may also improve semantic and phonetic fluency among patients with subcortical vascular dementia (Pantoni et al. 2005). However, Pantoni et al. (2000) was unable to find significant differences between Nimodipine and placebo. Based on the conclusions of the EBRSR (Teasell et al. 2008), there is moderate evidence that treatment with nimodipine is beneficial for memory and may even help to slow cognitive decline among patients with subcortical vascular dementia.

**Memantine:** Among patients with probable vascular dementia, treatment with memantine appears to be beneficial and has been associated with the stabilization and improvement of cognitive outcomes relative to treatment with placebo (Orgogozo et al. 2002, Wilcock et al. 2002). Effects may be larger among patients with greater cognitive impairment (MMSE < 15) or with small vessel disease (Wilcock et al. 2002). Based on the conclusions of the EBRSR (Teasell et al. 2008), there is strong evidence that treatment with memantine is associated with stabilization or improvement of cognitive function.

**Pentoxifyline:** Treatment with pentoxifyline may be beneficial for cognition in patients with multi-infarct dementia (Sha and Callahan 2003). Based on the conclusions of the EBRSR (Teasell et al. 2008), there is strong evidence that treatment with pentoxifyline is associated with cognitive benefits is patients with multi-infarct dementia.

### D1.3.2 Cognitive Rehabilitation

**Q2. Describe Cognitive Rehabilitation. What evidence is there that it is helpful for stroke patients?**

**Answers**

1. Cognitive rehabilitation focuses on: 1) re-establishing previously learned patterns of behavior; 2) learning new compensatory patterns of cognitive activity; 3) using external compensatory mechanisms; 4) enabling adaptation to cognitive disability.
2. Attention training has been shown to be beneficial in mixed populations of TBI with or without stroke patients.
3. Compensatory strategies are useful in remediation of memory deficits.
4. Remediation of executive function and problem solving post stroke has not been studied.

**Discussion**

Cognitive rehabilitation involves “a systematic, functionally oriented service of therapeutic activities that is based on assessment and understanding of the patient’s brain-behavioral deficits” (Cicerone et al. 2000). Various interventions aim to: 1) reinforce, strengthen, or re-establish previously learned patterns of behavior; 2) establish new patterns of cognitive activity through compensatory cognitive mechanisms for impaired neurological systems; 3) establish new patterns of activity through external compensatory mechanisms such as personal orthoses or environmental structuring and support; and 4) enable persons to adapt to their cognitive disability. Accordingly, cognitive rehabilitation directs itself to several areas of cognition such as
attention, concentration, perception, memory, comprehension, communication, reasoning, problem-solving, judgement, initiation, planning, self-monitoring and awareness (Cicerone et al. 2000).

Remediation of Attention Deficits
Post stroke attention training may have a positive effect on specific, targeted outcomes. Cicerone et al. (2000) reviewed 13 studies examining the effectiveness of attention interventions during the acute phase of the rehabilitation of traumatic brain injury (TBI) or stroke. An additional 5 studies examining the remediation of attention deficits following TBI were added in 2005. It should be noted that none of the studies added to the review were specific to the stroke population.

Remediation of Memory Deficits
There is strong evidence that compensatory strategies are effective in improving memory outcomes post brain injury. Strategies include imagery-based training and the use of assistive, electronic devices. It should be noted that relatively few of the study participants had experienced a stroke.

Remediation of Executive Functioning and Problem-Solving
Currently, there are no studies examining the efficacy of remediation of executive functioning and problem solving for stroke patients.

References


Craig D, Birks J. Rivastigmine for vascular cognitive impairment. The Cochrane Database of Systematic Reviews 2005; Art. no. CD004744.pub2.


Malouf R, Birks J. Donepezil for vascular cognitive impairment. The Cochrane Database of Systematic Reviews 2004; Art. No. CD004395.pub2.


**Key Study: Donepezil (anti-cholinesterase) in Vascular Dementia**


<table>
<thead>
<tr>
<th>Author / Year Country PEDro score</th>
<th>Methods</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Black et al. 2003 International 7 (RCT)</td>
<td>603 Patients with probable (70.5%) or possible (29.5%) VaD were randomized to 24 weeks of treatment with donepezil 5 mg/d, donepezil 10 mg/d (5 mg/d for first 28 days), or placebo.</td>
<td>At week 24, both donepezil groups showed significant improvement in cognition versus placebo on the Alzheimer’s Disease Assessment Scale–cognitive subscale. Significant improvements in global function were seen versus placebo at week 24, on the Clinician’s Interview-Based Impression of Change–Plus version only for patients on donepezil 5 mg/d, and on the Sum of the Boxes of the Clinical Dementia Rating only for patients on 10 mg/d. Donepezil-treated patients showed significant benefits in activities of daily living over placebo on the Alzheimer’s Disease Functional Assessment and Change Scale. Withdrawal rates due to adverse events were relatively low (placebo, 11.1%; donepezil 5 mg/d, 11.1%; donepezil 10 mg/d, 21.8%; ( P=0.005 ) versus placebo).</td>
</tr>
</tbody>
</table>

**Comparison of CIBIC*-plus Ratings across Treatment Groups**

(Week 24 Observed Cases)

<table>
<thead>
<tr>
<th>CIBIC*-plus Category</th>
<th>Placebo</th>
<th>Donepezil (5 mg)</th>
<th>Donepezil (10 mg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Marked Improvement</td>
<td>0%</td>
<td>40%</td>
<td>50%</td>
</tr>
<tr>
<td>Moderate Improvement</td>
<td>10%</td>
<td>30%</td>
<td>20%</td>
</tr>
<tr>
<td>Minimal Improvement</td>
<td>20%</td>
<td>20%</td>
<td>10%</td>
</tr>
<tr>
<td>No Change</td>
<td>30%</td>
<td>10%</td>
<td>0%</td>
</tr>
<tr>
<td>Minimal Worsening</td>
<td>0%</td>
<td>0%</td>
<td>0%</td>
</tr>
<tr>
<td>Moderate Worsening</td>
<td>0%</td>
<td>0%</td>
<td>0%</td>
</tr>
<tr>
<td>Marked Worsening</td>
<td>0%</td>
<td>0%</td>
<td>0%</td>
</tr>
</tbody>
</table>

*Clinician’s Interview-Based Impression of Change

**Note:** \( p<0.05 \) for overall Donepezil treatment vs. placebo

**Importance:** Among patients with dementia of the Alzheimer’s type, the use of donepezil has been well studied. The effectiveness of donepezil among patients with vascular dementia has been the subject of 2 large randomized controlled trials of which this is one. Recent meta-analyses of the results from these two RCTs (Passmore et al. 2005, Malouf and Birks 2004) have reported that the use of donepezil in patients with mild to moderate vascular cognitive impairment is associated with significant improvements in cognitive and global function, including improvements in the performance of activities of daily living.
Relevant SREBR Conclusion: There is strong evidence from 2 RCTs that donepezil taken for 24 weeks improves cognitive function in patients with probable or possible vascular dementia.

Related References


Malouf R, Birks J. Donepezil for vascular cognitive impairment. The Cochrane Database of Systematic Reviews 2004; Art. No. CD004395.pub2.


D1.4 Vascular Dementia and Rehabilitation Case Study
Case Study

72 year old male living independently with a very supportive family. He presented to acute neurology with aphasia and hypertensive crisis (264/100 mmHg). The patient remained in the acute care hospital for 8 weeks, undergoing investigations for multiple strokes and suffering several more strokes while in hospital. MRI revealed multiple areas of bilateral subcortical infarctions. Diagnosis was made of multiple embolic strokes from aortic atheroma; risk factors included hypertension and type 2 diabetes from which he had suffered for 10 years. He also had a history of chronic renal dysfunction.

The patient was subsequently admitted to the stroke rehabilitation unit. At the time of admission to the stroke rehabilitation unit, ASA (81 mg daily) had been initiated. A GJ tube had been inserted to assist with feeding. He required maximal assistance to total dependence to manage his ADLs. He was incontinent of bowel and bladder. He had a severe dysarthria and was essentially nonverbal. He required physical restraints when not being directly supervised and frequently refused to attend therapy sessions, although he was not combative or threatening to staff. Mini-Mental Standard Examination (MMSE) score was 13/30 while on rehabilitation.

Q1. Provide a problem list.

Answer
1. Multiple embolic strokes from aortic atheroma
2. Multiple subcortical strokes
3. Hypertension
4. Type II diabetes
5. Chronic renal dysfunction
6. Dysphagia with G-J tube
7. Maximal assist with ADLs
8. Incontinence of bowel and bladder
9. Severe dysarthria
10. Requiring physical restraints
11. MMSE 13/30 – indicative of severe cognitive impairment

Q2. The family wants to know if this is Alzheimer’s dementia. How would you respond?

Answer
1. This is most likely a vascular dementia because of the focal neurological findings, the multiple subcortical infarcts and the step-wise progression.

Q3. What does a MMSE score of 13/30 mean? What other information would you want to know when interpreting the MMSE?

Answers
1. An MMSE of 13/30 suggests severe cognitive impairment.
2. MMSE is influenced by age, educational level and cultural background, all of which can result in misclassification.

Q4. How do you think he will respond to rehabilitation?

Answer
1. The patient has physical focal deficits which may respond to rehabilitation training.
2. Severe cognitive deficits, as indicated by the MMSE and supported by the multiple subcortical strokes would suggest that his response to rehabilitation will be slow and limited because of difficulties with learning.

Case Study (continued)
He remained on the stroke rehabilitation unit for 7 weeks. During that time he made good physical gains. Initially he was ambulating with a walker, albeit not safely, as he tended to
forget to use the brakes and it would often become an obstacle for him. At discharge he was ambulating with no aids.

<table>
<thead>
<tr>
<th></th>
<th>Admission</th>
<th>Discharge</th>
</tr>
</thead>
<tbody>
<tr>
<td>10 meter walk</td>
<td>21 seconds</td>
<td>16 seconds</td>
</tr>
<tr>
<td>2 minute walk test</td>
<td>45 meters</td>
<td>70 meters</td>
</tr>
<tr>
<td>Greatest distance walked</td>
<td>85 meters</td>
<td>135 meters</td>
</tr>
<tr>
<td>before requiring a rest</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Assistance required</td>
<td>Minimal</td>
<td>None</td>
</tr>
<tr>
<td>Berg Balance Score</td>
<td>7/56</td>
<td>47/56</td>
</tr>
<tr>
<td>COVS</td>
<td>53/91</td>
<td>73/91</td>
</tr>
<tr>
<td>Right Leg CMS</td>
<td>3/7</td>
<td>6/7</td>
</tr>
<tr>
<td>Right Foot CMS</td>
<td>3/7</td>
<td>4/7</td>
</tr>
</tbody>
</table>

In hospital, treatment was initiated with Ritalin which appeared to help with short-term memory and initiation. The rehabilitation team met with the family and appraised them of his cognitive impairments including impaired memory, problem-solving, insight, and judgment. The recommendation was made that he receive 24-hour supervision and that he would benefit from a structured and consistent routine.

Q5. Discuss the mechanism of action of Methylphenidate (Ritalin) and when would you give it to a stroke patient with cognitive deficits?

Answer
1. Methylphenidate (0.25 - 0.30 mg/kg bid) is recommended in adults to enhance attentional function and speed of cognitive processing in the adult population.
2. Would tend to treat patients who have difficulty focusing or paying attention.
D1.5 Moderate Cognitive Impairment and Rehabilitation Case Study
D1.5 Moderate Cognitive Impairment and Rehabilitation Case Study

Case Study

74 year old male who suffered a right frontal intracerebral hemorrhage, consistent with congophillic angiopathy. He was admitted to the stroke rehabilitation unit one week after the onset of his intracranial hemorrhage. He presented with an unsteady gait, variable performance, distractibility, perseveration, slow responses with some short-term memory loss. He was able to do basic ADLs with set-up and stand-by assistance. He was continent for bowel and bladder. Due to a decrease in attention and cognition, he required supervision for his transfers and a walker for ambulation. He was ambulating 45 meters with one-person assistance. He was mildly confused with some disorientation at nighttime.

Premorbidly, he had been living independently in his multi-level home with his spouse; he had 3 children, of whom two lived in the same city. He was driving prior to his stroke.

Would become very confused on the rehabilitation unit; was showing signs of sundowning, tendency to wander and needed to be restrained. He was distractible and impulsive at times although he was able to ambulate with gait aids and supervision. His confusion got worse initially after admission to rehabilitation. MMSE performed 2 weeks post stroke was 11/30; he was not oriented to time or place, was not following simple instructions and required hands-on cues to complete simple familiar tasks.

Q1. Provide a problem list for this patient.

Answer
1. Congophillic angiopathy with right frontal intracerebral hematoma.
2. Unsteady gait, variable performance.
3. Distractibility, perseveration, slow responses with some short-term memory loss.
4. Able to do basic ADLs with set-up and stand-by assistance.
5. Required supervision for his transfers and a walker for ambulation; ambulating 45 meters with one-person assistance.
6. Tendency to wander to the point of needing to be restrained.
7. Distractable and impulsive.
8. MMSE 11/30; not oriented to time or place.
9. Not following simple instructions.

Q2. What does the MMSE suggest about this patient?

Answer
1. The MMSE of 11/30 indicates severe cognitive impairment.

Case Study (continued)
The MMSE was repeated again almost 54 days following stroke onset and with improvements in orientation and attention his MMSE was 20/30. The Montreal Cognitive Assessment (MoCA) was administered 76 days post stroke and he was not able to recall 5 words from memory. Abstract thinking, following patterns and visual-motor skills were impaired. He was fully oriented to date and place. Cognitive abilities continued to fluctuate daily and were noticeably affected by his level of fatigue. Although his attention span had increased he was still easily distracted. He continued to have difficulties with the acquisition of new knowledge/skills. He was told not to drive due to cognitive problems. He was discharged home to the care of his wife and family although his wife still had to assist him with some personal care. He was fully ambulatory in his own home.

Q3. What does the MMSE now suggest about this patient?

Answer
1. MMSE of 20/30 suggests the patient has improved but continues to suffer from cognitive impairment.
Q4. Was it appropriate to do a MOCA test in this case and why or why not?

Answer
1. The MOCA is designed for use in individuals with mild cognitive impairment – those that the MMSE could not identify as experiencing cognitive impairment (24/30 or more).
2. The MOCA, although popular remains a screening tool and with the MMSE showing cognitive impairment its use would be considered redundant, particularly in a stroke population where it as of yet remains unproven.
D1.6 Depression and Dementia Case Study
D1.6 Depression and Dementia Case Study

Case Study

An 80 year old female was admitted to the stroke rehabilitation unit 26 days after suffering a right MCA infarct. Prior to the stroke she had been able to ambulate independently with a walker. As a consequence of the right MCA stroke she experienced left hemiplegia, left neglect and was incontinent of bowel and bladder. Her infarct was a large one involving the right temporal, right parietal and right inferoposterior portion of the frontal lobe. Patient was in atrial fibrillation and Coumadin was initiated. She lived with her husband in a retirement home prior to her stroke.

On admission to the stroke rehabilitation unit she still had bowel and bladder incontinence. She could do a pivot transfer with the assist of one and she was wheelchair dependent for mobility. During rehabilitation she exhibited minimal gains, requiring continued assistance with transfers and ADLs. Mobility remained wheelchair dependent.

The patient identified very severe problems with her memory, problem solving and visual perception. She was observed to lose her train of thought in the middle of conversations, had difficulty with problem solving and tasks requiring visual perception. However, her MMSE was 27/30. On testing it was noted that the patient was highly anxious and endorsed many symptoms consistent with depression. Although she did well on formal cognitive testing, the impression on the ward was of severe memory and problem-solving difficulties. The patient refused anti-depressant medications.

Q1. The patient’s spouse is worried that his wife may be suffering from depression. How would you explain this issue in terms of its relationship with cognitive impairment?

Answers

1. Depression is a very important issue that must be kept in mind when you are dealing with cognitive disorders.
2. The presence of depression could affect the results of the cognitive tests (Ruchinskas and Curyto 2003).
3. Depression in patients with amnestic mild cognitive impairment is associated with a risk of developing Alzheimer’s type dementia and cognitive deterioration may proceed at more rapid pace (Modergo and Fernandez 2004).
Q2. The nurse wants to know more about depression and cognitive disorders. Discuss the association between depression and cognitive impairment after stroke.

Answer

1. A significant and independent association between presence of depression and cognitive impairment has been demonstrated in stroke survivors one year following the stroke event (Talelli et al. 2004, Kalaria and Ballard 2001).

2. Brodaty et al. (2007) demonstrated a greater frequency of dementia among stroke patients with depression (27.8%) when compared to patients without depression (17.3%) at three months post-stroke (though this difference was not significance). By 15 months post-stroke 54.2% of patients with depression were diagnosed with dementia vs. 7.1% of non-depressed with significant difference.

3. Murata et al. (2000) concluded that major post-stroke depression leads to cognitive impairment and not vice versa.

Discussion

A recent study reported that the presence of depression in patients with amnestic mild cognitive impairment (MCI) is associated with a risk of developing dementia of the Alzheimer’s type that is twice that of MCI patients without depression (Modrego and Ferrandez 2004). Not only was the risk found to be greater, but cognitive deterioration proceeded at a more rapid pace (Modrego and Ferrandez 2004). Barnes et al. (2006) reported that, among 2220 participants in the Cardiovascular Health Study (Cognition Study), depressive symptoms at baseline were associated with an increased risk for MCI (moderate depression OR = 1.37 95% CI 1.00 – 1.88; moderate to severe depression OR = 2.09 95% CI 1.46 – 2.97) at follow-up 6 years later. While the presence of both small and large infarcts was also associated with increased risk for MCI (OR = 1.47 and 1.67, respectively), this association was independent of depression (Barnes et al. 2006).

It has been reported that the presence of depression is significantly and independently associated with the presence of cognitive impairment in stroke survivors one year following the stroke event (Talelli et al. 2004, Kalaria and Ballard 2001). Indeed, there is considerable evidence that affective disorders are associated with cognitive functioning (Burvill et al. 1995, Dam et al. 1989, Egelko et al. 1989). This phenomenon has been termed the "dementia of depression" or pseudodementia. In 1986, Robinson and colleagues found that patients with major depression after stroke had significantly greater cognitive impairment than patients with minor depression or no mood disturbance. In 2000, Murata and colleagues examined cognitive functioning in 41 patients with, and 135 patients without, major depression in the acute hospital setting and either 3 or 6 months later. The authors noted that at follow-up, patients with major depression and improvement in mood demonstrated significantly greater recovery in cognitive functioning compared to patients with major depression without mood improvements. It is important to note, however, that a patient’s score on the MMSE determined whether or not the patient had cognitive impairment.

In a recent report from the Sydney Stroke Study, Brodaty et al. (2007) demonstrated a greater frequency of dementia among stroke patients with depression (27.8%) when compared to those without depression (17.3%) at 3 months post-stroke, though this difference was not significant (OR = 1.84, 95% CI 0.60-5.67, p=0.29). By 15 months post-stroke, 54.2% of patients with depression were diagnosed with dementia vs. 7.1% of non-depressed patients (OR = 15.36, 95% CI 5.1 – 46.7, p<0.001). However, logistic regression demonstrated that dementia at 3
months was a significant predictor for depression at follow-up (OR = 5.55, 95% CI 1.95 – 15.77, p=0.001) while the reverse was not true. There is evidence from one double-blind, controlled trial of nortriptyline (Kimura et al. 2000) that depression, in combination with other factors, adds to cognitive impairment in stroke patients (Haring 2002). Murata et al. (2000) also concluded that major post-stroke depression leads to cognitive impairment and not vice versa.

Q3. Discuss the difference between dementia and depression-related cognitive impairment.

Answer
1. Depression-related cognitive impairment can sometimes mimic the signs of dementia and is referred to as pseudodementia.
2. Pseudodementia tends to be more sudden onset, more rapid progression, with a previous history of depression, more variable, effort-related cognitive deficits with little nocturnal exacerbation.

Discussion
There is considerable evidence that affective disorders are associated with cognitive functioning (Burvill et al. 1995, Dam et al. 1989, Egelko et al. 1989). This phenomenon has been termed the “dementia of depression” or pseudodementia. Robinson et al. (1986) found that patients with major depression post stroke had significantly greater cognitive impairment than patients with minor depression or no mood disturbance.

Differential Diagnosis of Dementia versus Pseudodementia (Devinsky 2004, p. 291)

<table>
<thead>
<tr>
<th>Feature</th>
<th>Dementia</th>
<th>Pseudodementia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Onset</td>
<td>Often insidious</td>
<td>Usually acute or subacute</td>
</tr>
<tr>
<td>Progression</td>
<td>Usually slow, early changes often missed</td>
<td>Usually rapid</td>
</tr>
<tr>
<td>Symptom duration at presentation</td>
<td>Long</td>
<td>Short</td>
</tr>
<tr>
<td>Psychiatric history or recent life crisis</td>
<td>Uncommon</td>
<td>Common</td>
</tr>
<tr>
<td>Extensive self-report of mental impairment</td>
<td>Uncommon</td>
<td>Common</td>
</tr>
<tr>
<td>Mental status or psychometric testing</td>
<td>Progressive decline</td>
<td>Variable, effort-related</td>
</tr>
<tr>
<td>Memory impairment</td>
<td>Common, most severe for recent events</td>
<td>Common, often selective amnesia, inconsistent deficits over time</td>
</tr>
<tr>
<td>Affective changes</td>
<td>Apathy, shallow emotions</td>
<td>Depression common</td>
</tr>
<tr>
<td>Nocturnal exacerbation of symptoms</td>
<td>Common</td>
<td>Uncommon</td>
</tr>
</tbody>
</table>

References


D1.7 Frontal Lobe Hemorrhage Case Study
D1.7 Frontal Lobe Hemorrhage Case Study

Case Study

A 70 year old single female who suffered a large left frontal intraparenchymal hemorrhage with mass effect was referred to rehabilitation. She had some mild right hand weakness. She was ambulating independently on the acute stroke unit. Her speech was unaffected although she appeared to answer questions reasonably well. However, her MMSE was 18/30 and she seemed to have problems with sequential tasks. She lived alone. She was admitted to the stroke rehabilitation unit and presented with visual and perceptual neglect, mild right hand weakness, decreased sequencing and executive functioning. For ADLs she required minimal assistance due to decreased perception and neglect. She was independent for transfers and mobility with episodes of motor apraxia. She had trouble processing information and high level cognitive skills, including impaired memory, problem-solving, insight and judgement. Route-finding difficulties had been observed.

Q1. Discuss the potential consequences of a left frontal lesion.

Answer
1. Right sided weakness
2. Expressive language difficulties, possibly transcortical motor aphasia
3. Decreased problem-solving, insight and judgement.

Case Study (continued)

On initial rehabilitation assessment, 14 days following her stroke, the Montreal Cognitive Assessment (MoCA) score was 8/30 and Mini Mental State Examination was 18/30. The MOCA was readministered 44 days post stroke and she had increased to a score of 22/30. She demonstrated improvement with her memory but continued to demonstrate impairment for the higher level executive cognitive skills such as problem solving, insight and judgement.

Q2. Discuss the MOCA and whether its use is appropriate in this setting.
**Answer**

1. The MOCA is a screening tool, used for assessing mild cognitive impairment.
2. Using it in this case may be considered redundant as the patient has already been identified as being significantly cognitively impaired on the MMSE (i.e. 18/30).
3. MOCA is a screening tool and is not necessarily designed to assess patients over time.
D1.8 Apraxia
D1.8 Apraxia

Q1. Define apraxia.

Answer
1. Apraxia is a disorder of voluntary movement where one cannot execute a purposeful activity despite the presence of adequate mobility, strength, sensation, coordination and comprehension.

Discussion
Apraxia is common in patients with left hemispheric strokes, especially in lesions involving the left frontal and parietal lobes. Apraxia is a disorder of voluntary movement where one cannot execute willed, purposeful activity despite the presence of adequate mobility, strength, sensation, coordination and comprehension. In other words, patients are unable to perform previously learned tasks and the inability is not explained by weakness, aphasia, or sensory loss (Caplan 1994). Caplan (1994) refers to it as an inability to perform previously learned tasks when such an inability cannot be explained by weakness, aphasia, or sensory loss. The difficulty can be spontaneous and noted during everyday activities (e.g. difficulty with dressing, using utensils, starting the car, turning keys to open doors, and lighting a cigarette). In others the difficulty in performing motor tasks becomes evident when the patient is asked to do something (Caplan 1994).

Q2. Some rehabilitation clinicians attempt to categorize apraxias. Describe a categorization of apraxias and provide examples of each.

Answers
1. Ideomotor Apraxia: Patient can automatically perform a movement (e.g. scratch their nose) but cannot repeat it on command.
2. Ideational Apraxia: Patient cannot identify the purpose of common objects and/or cannot coordinate individual steps into an integrated sequence (e.g. uses toothbrush to comb their hair)
3. Functional Apraxias: Constructional, dressing, gait, oculomotor and orofacial.

Discussion

Types of Apraxias

<table>
<thead>
<tr>
<th>Type</th>
<th>Manifestation</th>
<th>Example</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ideomotor (or production apraxia)</td>
<td>Deficit in the control or programming of the spatial organization, sequencing, or timing of goal-directed movements (Barrett and Foundas)</td>
<td>Patient can automatically perform a movement (e.g. scratch their nose) but cannot repeat it on command.</td>
</tr>
</tbody>
</table>
Deficits affecting action semantics and internal representations of movements.

Patient misuses/cannot identify the purpose of common objects and/or cannot coordinate individual steps into an integrated sequence (e.g., holds toothbrush but doesn’t bring it to their mouth).

**Functional Apraxias**

<table>
<thead>
<tr>
<th>Constructional</th>
<th>Unable to synthesize individual spatial elements into a whole</th>
<th>Patient has difficulty drawing geometric figures.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dressing</td>
<td>Impaired ability to dress oneself despite adequate motor ability.</td>
<td>Patient is unable to orient clothing to the appropriate body location.</td>
</tr>
<tr>
<td>Gait</td>
<td>Impaired ability to walk</td>
<td>Patient has abnormal gait.</td>
</tr>
<tr>
<td>Oculomotor</td>
<td>Abnormal directed eye movement / eyelid opening</td>
<td>Patient has difficulty moving their eyes and may compensate by moving their head.</td>
</tr>
<tr>
<td>Orofacial</td>
<td>Impaired ability to perform actions of the mouth and tongue</td>
<td>Patient has difficulty opening their mouth or protruding their tongue on command.</td>
</tr>
</tbody>
</table>

Apraxia is described according to the body structures used to perform tasks: orofacial, limb, and trunk (Caplan 1994). Patients with orofacial dyspraxia have difficulty whistling, blowing a kiss, and humming when commanded to perform these and similar activities. Although some patients are unable to protrude their tongues on command, the next moment they may spontaneously stick out the tongue while automatically licking the lips (Caplan 1994). Patients with truncal apraxia have difficulty performing whole body commands such as standing, turning and sitting (Caplan 1994). Usually, limb apraxia involves mostly the hands and arms; for instance, patients fail to show how they would salute, hitchhike, flip a coin and wave goodbye (Caplan 1994).

**Q3. How would you test for Apraxia?**

**Answer**

Apraxia can be best tested in the following ways (Caplan 1994):

1. By giving an oral or written command to pretend to do something or pretend to use an object.
2. By using objects placed in front of the patient (e.g., comb, toothbrush, scissors or hammer.
3. By initiating an action (orofacial, limb or trunk) performed by the examiner.
4. For constructional apraxias, copying a geometric shape.
A 65 year old male suffered a left hemispheric stroke, involving the MCA with an infarct involving both the frontal and parietal areas. The patient presented to rehabilitation 2 weeks later with a right hemiplegia and an expressive aphasia. Moreover, the patient demonstrated some bizarre behavior, trying to comb his hair with his toothbrush and trying to eat a bar of soap. On clinical testing he was asked to touch his nose, which he accomplished, followed by single commands to touch his ear (which he did successfully) and his chin (which he also accomplished after some thought). However, when asked to touch his nose, ear and chin in sequence, he quickly became confused touching his chin after some hesitation but not being able to proceed any further. Moreover, when asked to perform a salute, a hitchhiker sign or how he would flip a coin he was not able to but was able to wave goodbye to the interviewer at the end of the interview.

Q4. What is the diagnosis of this unusual behavior?

Answer
1. Patient is suffering from an ideomotor and ideational apraxia.

References


Key Study: Strategy Training for Apraxia Post Stroke


<table>
<thead>
<tr>
<th>Author / Year</th>
<th>Country</th>
<th>PEDro score</th>
<th>Methods</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Donkervoort et al. 2001 Netherlands 8 (RCT)</td>
<td></td>
<td>113 patients with apraxia secondary to left hemisphere stroke were randomly assigned to either strategy training integrated in usual OT or to regular OT. Strategy training involved the use of strategies to compensate for the apraxic impairment during the performance of ADL. Usual OT concentrated on sensory, motor, perceptual and cognitive deficits of the stroke patients and increasing independent functioning in ADL task. Patients underwent 8 weeks of treatment.</td>
<td>After 8 weeks of treatment, strategy training group improved significantly more than controls on ADL observations and on the Barthel ADL. No significant differences between the groups were noted at the 5 month follow-up.</td>
<td></td>
</tr>
</tbody>
</table>

**Outcome Measures for Strategy Training vs. Usual treatment**

![Outcome Measures Graph]

**Importance**: The study by Donkervoort et al. (2001) is the most recent and largest randomized controlled trial to assess the effectiveness of strategy training in the treatment of apraxia following left hemisphere stroke. Further studies are required and assessment of transfer of training effects to untrained activities is recommended.

**Relevant SREBR Conclusion**: There is moderate evidence that strategic training is effective in the treatment of apraxia post-stroke.

**Related References**


D2. Visual Perceptual Disorders
D2.1 Visual Perceptual Disorders: General Information
D2.1 Visual Perceptual Disorders: General Information

Q1. Describe neglect.

Answer
1. Failure to report, respond, or orient to sensory stimuli presented to the side contralateral to the stroke.
2. Unilateral spatial neglect, when severe, is often characterized by the patient colliding with objects on the affected side, ignoring objects in front of them and attending to only one side of the body.
3. Milder neglect involves various degrees of ignoring the affected side when faced with stimulation on the unaffected side (extinction).

Discussion
Unilateral spatial neglect (USN) is one of the disabling features of a stroke, and is defined as a failure to report, respond, or orient to sensory stimuli presented to the side contralateral to the stroke lesion site (Heilman et al. 1985). Many terms are used interchangeably in the literature to describe USN, including unilateral neglect, hemi-inattention, visual neglect and hemi spatial neglect. Clinically, the presence of severe USN is apparent when a patient often collides into his/her surroundings, ignores food on one side of the plate, and attends to only one side of his/her body (Wyness 1985). However, symptoms of USN have to be quite severe for this impairment to be observed easily during the performance of functional activities (Mesulam 2000, Cherney et al. 2001). More subtle forms of USN may go undetected in a hospital setting but are a major concern for client function and safety upon discharge. Mild symptoms of USN become apparent during high-level activities such as driving, working or while interacting with others. Neglect is not a disorder, but rather a complex combination of symptoms that differ from patient to patient. Patients may present with peripersonal neglect (within reaching space), extrapersonal neglect (beyond reaching space), or personal neglect (patient’s body).

Q2. Why is left-side neglect more common than right-sided neglect?

Answer
1. The right hemisphere regulates attention more than the left hemisphere.
2. The left hemisphere is responsible for modulating attention and arousal for the right visual field only, while the right hemisphere is responsible for controlling these processes in both the right and left hemispheres.
3. Hence the right hemisphere is more able to compensate for the left hemisphere, when it suffers a stroke, while the left hemisphere is not able to compensate for the right hemisphere if it is injured in a stroke.

Discussion
Unilateral spatial neglect (USN) is more common in stroke patients with right-sided lesions than left-sided lesions. In the Copenhagen Stroke Study (Pedersen et al. 1997), 42% of individuals with a right-sided lesion were reported to have USN when compared to only 8% of patients with a left-hemispheric lesion. Ringman et al. (2004) in a study of 1,282 stroke patients found that 43% of patients with right-sided lesions experienced neglect when compared to 20% of patients with left-sided lesions (p<0.001). At 3 months post-stroke onset, 17% of patients with right-sided lesions and only 5% of left-sided lesions continued to suffer from USN.

The right hemisphere is thought to regulate attention. Neuroanatomical finding have identified that the left hemisphere is responsible for modulating arousal and attention for the right visual field, whereas the right hemisphere controls these processes in both the right and left visual fields (Feinberg 1990). This may explain why USN is not typical for those with left hemispheric lesions because the intact right hemisphere is capable of compensating for perceptual deficits that result from left hemispheric lesions (Feinberg 1990).

Garmoe et al. (1994, p. 229-230) noted that, “the right parietal lobe is involved in regulating attention to both the right and left spatial hemifields and that, with right parietal damage, the ability to allocate attention to both fields is lost. The left parietal lobe can regulate attention within the right hemifield, but unlike the right parietal lobe, it cannot regulate attention throughout both hemifields. Thus damage to the right parietal lobe leaves the person able to allocate attention to the right hemifield but not to allocate attention shifts between the right and left fields; the net result is a disproportionately high allocation of attention to the right hemifield and a low allocation of attention to the left hemifield.”

**Q3. What is the impact of the left-sided neglect on his functional (rehabilitation) prognosis?**

**Answer**

1. Left-sided neglect tends to have a poorer prognosis in terms of functional recovery on rehabilitation.

**Discussion**

Unilateral spatial neglect has been reported to have a negative impact on functional recovery, length of rehabilitation stay, and the need for assistance post-discharge. While the majority of patients diagnosed with visuospatial inattention post-stroke recover by 3 months, those with severe visuospatial inattention on initial presentation have the worst prognosis (Diamond et al. 2001). The presence of unilateral spatial neglect (USN) has been associated with poorer functional outcome, poorer mobility, longer length of stay in rehabilitation and a greater chance of institutionalization upon discharge from from rehabilitation(Paolucci et al. 2001). Gillen et al. (2005) compared right-sided stroke patients with USN or no USN and matched for severity of functional deficits (FIM scores) at admission to rehabilitation; the presence of USN was associated with longer lengths of stay and slower rates of improvement.

Patients with USN may be more impaired at the beginning of rehabilitation than patients without USN (Katz et al. 1999) and while significant gains may be made throughout the course of rehabilitation, USN patients tend to be more functionally disabled at discharge. The presence of
USN has been identified as a significant predictor of functional dependence in ADLs (Appelros et al. 2002, Katz et al. 1999) and poorer performance in IADLs at 6 months (Katz et al. 1999) and one year post discharge from rehabilitation (Jehkonen et al. 2000). The presence of USN explained 73% of the total variance in IADL at 3 month follow-up, 64% at 6 months and 61% at one-year in 57 subjects post-stroke (Jehkonen et al. 2000). Appelros et al. (2003) reported USN to be a significant predictor of both mortality (OR=2.7) and dependency (OR=4.0) one year after the stroke event. In addition, substantial proportions of individuals (79-82%) with neglect requiring home assistance following discharge (Appelros et al. 2003, Katz et al. 1999) or may be discharged to nursing home care (Appelros et al. 2003, Paolucci et al. 2001).

Q4. **What is meant by the term anosognosia?**

**Answer**
1. Refers to unawareness of loss of an important bodily function, primarily hemiplegia.
2. Involves primarily large right hemispheric strokes which involve the parietal region.

**Discussion**
Caplan (1994) notes that “Anosognosia is a term used to describe unawareness of an important loss of bodily function, most often hemiplegia.” This usually involves large right hemispheric lesions which extend beyond just the parietal region. It may manifest on a continuum form patients denying that there is anything wrong with them to failing to appreciate the importance and significance of the deficit. This will sometimes result in the patient attempting to inappropriately use the affected limb; for instance, the stroke patient who impulsively tries to get up from a chair or bed on the hemiplegic limb and who subsequently falls.

Q5. **In team rounds the physiotherapist complains that a patient has trouble sustaining attention to an activity; that they are impulsive and can’t seem to sustain a task during therapy. What is the term for this deficit and how would you test for it?**

**Answer**
1. Motor impersistence.
2. Tends to occur with right hemispheric strokes.
3. Ask the patient to keep their eyes closed or their arms upraised and they are unable to persist with the activity.

**Discussion**
Motor impersistence tends to be seen with right hemispheric strokes. Caplan (1994) notes that patients with this disorder respond to commands quickly and accurately but impulsively. The action is quickly performed but just as quickly it is terminated. Patients will not persist with the activity even when instructed to do so. For example, patients with motor impersistence will not be able to keep their eyes closed for an extended period of time, even when asked to do so. Motor impersistence can be problematic for stroke rehabilitation. Restlessness, impulsivity and
lack of perseverance leads to failure in everyday tasks requiring sustained attention, which includes many ADL and IADL activities. Motor impersistence does not tend to resolve and patients may not recover resulting in poorer prognosis for recovery (Hier et al. 1983). Motor impersistence can be tested for by asking the patient to stick out their tongue and hold it there, keep the eyes closed or keep the hands outstretched for an extended period of time.

References


D2.2 Visual-Perceptual Disorders: Assessments
D2.2 Visual-Perceptual Disorders: Assessments

**Q1.** What are some of the common tests which can be used to screen for the presence of left-sided neglect?

**Answers**
1. Line bisection test
2. Single letter cancellation test
3. Behaviour inattention test

**Discussion**
There are currently 61 standardized and non-standardized assessment tools available to assess unilateral neglect (Menon and Korner-Bitensky 2004). Line bisection test, Albert’s test, single letter cancellation test, star cancellation test, and Bell’s test are all examples of simple, pencil and paper tests used to detect the presence of USN. All can be administered at the bedside in just a few minutes. However, the patient must be able to follow instructions as well as hold and use a pencil with reasonable accuracy in order to complete these types of tests reliably.

In 2006, the Canadian Stroke Rehabilitation Outcomes Consensus Panel, selected the Line Bisection tool as its preferred standard for the identification of unilateral spatial neglect.

**Q2. Describe the Line Bisection Test including its strengths and weaknesses.**

**Answers**
1. Asked to find the midline on a number of horizontal lines.
2. Test is scored by measuring distance from their line to the actual center of the line.
3. Deviation of 6 mm or more is considered diagnostic of USN.
4. Simple test.
5. Screening tool only.

**Discussion**

**The Line Bisection Test (LBT)**

<table>
<thead>
<tr>
<th>Questions</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>What does it measure?</td>
<td>Designed to detect the presence of unilateral spatial neglect.</td>
</tr>
<tr>
<td>What is the scale?</td>
<td>The LBT consists of 18 horizontal lines drawn on a single piece of paper. Patients are required to place a mark on each line that bisects it into two equal parts.</td>
</tr>
<tr>
<td>What are the key scores?</td>
<td>The test is scored by measuring the distance from the bisection mark to the actual center of the line. A deviation of 6mm or more</td>
</tr>
</tbody>
</table>
is indicative of unilateral spatial neglect. USN may also be suggested if the patient omits two or more lines on one half of the page.

<table>
<thead>
<tr>
<th>What are its strengths?</th>
<th>Simple and inexpensive measure of USN. Does not require formal training to administer.</th>
</tr>
</thead>
<tbody>
<tr>
<td>What are its limitations?</td>
<td>The LBT may not be able to detect USN in as many as 40% of patients with severe USN (Ferber and Karnath 2001). The LBT should only be used as a screening tool as positive results could be indicative of other syndromes, such as hemianopia (Ferber and Karnath 2001).</td>
</tr>
</tbody>
</table>

**Summary**

<table>
<thead>
<tr>
<th>Reliability</th>
<th>Validity</th>
<th>Responsiveness</th>
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<tbody>
<tr>
<td>Rigor</td>
<td>Results</td>
<td>Rigor</td>
</tr>
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</table>

**Q3. Describe the Behavioural Inattention Test including its strengths and weaknesses.**

**Answers**

1. Screens for unilateral visual neglect.
2. Conventional testing (6 subtests) and 9 behavioral tests.
3. Comprehensive test with cutoff scores.
4. Reliable and valid test.
5. Takes a lot of time for therapist and patient.

**Discussion**

**Behavioural Inattention Test (BIT)**

<table>
<thead>
<tr>
<th>Questions</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>What does it measure?</td>
<td>Screens for unilateral visual neglect and provides information relevant to its treatment (Halligan et al. 1991).</td>
</tr>
<tr>
<td>What is the scale?</td>
<td>The BIT is divided into two major sections, each of which has its own set of subtests. The conventional section (BITC) is comprised of the following 6 subtests: line crossing, letter cancellation, star cancellation, figure and shape copying, line bisection, and representational drawing. The behavioural section (BITB) is comprised of the following 9 subtests: pre-scanning, phone dialing, menu reading, article reading, telling and setting the time, coin sorting, address and sentence copying, map navigation, and card sorting.</td>
</tr>
<tr>
<td>What are the key scores?</td>
<td>The BIT yields a total score out of 227 with higher scores indicating greater degrees of neglect. Cutoffs have been established for the total BIT as well as for each of the</td>
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</table>
subsections with scores exceeding the cutoffs leading to a diagnosis of neglect. The cutoff for the total BIT is 196 out of 227, 129 out of 146 for the BITC, and 67 out of 81 for the BITB (reported in Menon and Korner-Bitensky, 2004).

What are its strengths?
The BIT is a comprehensive battery that provides a detailed and ecologically valid assessment of patient functioning (Halligan et al. 1991). A parallel form of the test is available, which allows for re-testing with minimal concern for practice effects. The behavioural subtests can be used to help therapists target tasks that should be given particular attention during treatment.

What are its limitations?
The BIT is both more time consuming and more expensive than most non-battery tests of neglect. Requiring 40 minutes for completion, the BIT is more taxing on patients than individual tests of neglect.

Summary

<table>
<thead>
<tr>
<th>Reliability</th>
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<tbody>
<tr>
<td>Rigor</td>
<td>Results</td>
<td>Rigor</td>
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<tr>
<td>++</td>
<td>+++ (TR)</td>
<td>+++</td>
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<tr>
<td></td>
<td>+++ (IO)</td>
<td>+ (IC)</td>
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</tbody>
</table>

**NOTE:** +++=Excellent; ++=Adequate; + = Poor; n/a = insufficient information; TR = Test re-test; IO = Internal consistency; IC = Interobserver; varied (re floor/ceiling effects; mixed results)

Q4. Is the clock-drawing test a good test for visual neglect?

**Answer**

1. No. CDT is not that sensitive to the diagnosis of visual neglect and is influenced by a number of other cognitive problems such as attention and executive dysfunction.

**Discussion**

The CDT is an extremely brief and very simple tool used in the assessment of cognitive function. The CDT provides a quick assessment of visuospatial and praxis abilities and may reflect both attention and executive dysfunction (Adunsky et al. 2002; Suhr et al. 1998; McDowell & Newell 1996). Performance on the CDT is most related to functions subserved by the right hemisphere (Suhr et al. 1998) and when used with other assessments may help to create a more complete picture of cognitive function. However, performance of the clock drawing task may be affected by other conditions prevalent in the rehabilitation settings such as visual neglect, hemiparesis and motor dyscoordination (Ruchinskas & Curyto, 2003). Individuals with visual neglect, for instance, may omit numbers on one-half of the clock face.

Although this might seem sufficient for identification of neglect, the reported sensitivity of the CDT when used for this purpose appears poor (55.3% - Maeshima et al. 2001; 42% Agrell et al. 1997) when compared to other assessments for neglect including cancellation tests, Albert’s test, and the line bisection test.
References


D2.3 Treatment of Visual-Perceptual Disorders
D2.3 Treatment of Visual-Perceptual Disorders

Q1. Describe rehabilitation interventions for neglect.

Answer
1. Interventions which attempt to improve awareness of or attention to neglected space, i.e. visual scanning retraining.
2. Interventions that improve neglect by targeting deficits with a specific intervention, i.e. prisms (more compensatory).

Discussion
In general, rehabilitation interventions to improve neglect may be classified into those which: 1) attempt to increase the patient’s awareness of or attention to the neglected space and; 2) those which focus on the remediation of deficits of position sense or body orientation (Butter et al. 1990; Pierce and Buxbaum 2002).

Improving Awareness of or Attention to the Neglected Space
Examples of interventions that attempt to improve awareness of or attention to the neglected space include the use visual scanning retraining, arousal or activation strategies and feedback to increase awareness of neglect behaviours (Butter et al. 1990).

Remediation of Deficits of Position Sense or Body Orientation
Interventions that attempt to improve neglect by targeting deficits associated with position sense and spatial representation include the use of prisms, eyepatching and hemispatial glasses, caloric stimulation, optokinetic stimulation, TENS and neck vibration.

Q2. Describe at least two treatments for the treatment of the left neglect.

Answer
1. Visual scanning; training the patient to consistently scan to the involved side (usually the left side).
2. Limb activation therapies, i.e. spatiomotor cueing: involves limb activation +/- application of a sensory stimulation.
4. Feedback strategies: can involve auditory and visual stimuli.
5. Prisms: optical deviation of the visual field to the affected side.
6. Bilateral half-field patches: increase eye movements to contralateral space.

Visual Scanning
Individuals with neglect do not visually scan their whole environment (Weinberg et al. 1977) paying no attention to their left-sided space (Ladavas et al. 1994). Visual scanning involves
training the patient to consistently scan to the left side. There is strong evidence that treatment utilizing primarily enhanced visual scanning techniques improves visual neglect post-stroke with associated improvements in function.

**Limb Activation**
Activation strategies include limb activation +/- the application of a sensory stimulation. Limb activation is based on the concept that any movement on the contralesional side may function as a motor stimulus activating the right hemisphere and improving neglect. There is strong evidence that limb activation therapies improve neglect. For example, Kalra et al. (1997) found that spatiomotor cueing during motor activity was associated with both shorter lengths of stays and less time spent in physiotherapy.

**Sensory Stimulation Interventions**
Increased awareness of the neglected space may be achieved by the application of an external stimulus, which may function as a cue similar to the spatial motor cueing described with motor activation. There was conflicting evidence as to the efficacy of external stimulation in increasing orientation and attention to the neglected space.

**Feedback Strategies**
Feedback strategies are intended to improve awareness of and attention to the neglected space (Pierce and Buxbaum 2002). Typical methods of feedback include auditory and visual feedback. All function to make the patient aware of his/her neglect behaviours and may assist in learning ways to remediate neglect. There is strong evidence that the use of feedback strategies is beneficial in the treatment of neglect.

**Prisms**
Prisms affect spatial representation by causing an optical deviation of the visual field to either the left or the right. One of the most common low vision interventions for stroke induced hemianopia is the incorporation of binocular sector prisms in the person’s habitual spectacle lenses. These may be Fresnel membrane lenses or prisms that are cemented onto the lens surface. The prism is located so that it remains outside the residual field of view when the person is looking straight ahead. When gaze is shifted in the direction of the non-seeing hemifield, the prismatic effect gives a more peripheral view to the side (6-9 degrees) than would be possible without a larger magnitude eye movement. There is strong evidence that treatment with prisms is associated with improvements in visuo-spatial tasks in stroke patients with homonymous hemianopsia and visual neglect. For example, Rossi et al. (1990) found that patients made significant improvements on several visual perception tests after four weeks of treatment with Fresnel prisms, as compared to patients in a non-treatment control group.

**Bilateral Half-Field Patches**
Eyes patches are used to try to alter the processing of visual information by affecting information processing by the brain (Beis et al. 1999). Shulman (1984) noted that in healthy subjects, eye patches should increase eye movements towards the contralateral space. This in turn should encourage the development of voluntary, deliberate control of attention in the short term and the development of automatic shifts of attention over the longer term (Seron et al. 1989). There is moderate evidence that the use of bilateral half-field eye patches improves visual neglect and functional ability.

**Other Treatments**
Other potential treatments include caloric/vestibular stimulation (limited evidence of a transient positive effect), vestibular galvanic stimulation (as effective as caloric stimulation), optokinetic
stimulation (limited evidence improves personal position sense but otherwise little added benefit), trunk rotation therapy (moderate evidence it is ineffective), neck muscle vibration (moderate evidence of benefit when combined with visual exploration therapy), transcutaneous electrical nerve stimulation (TENS) (limited evidence of benefit), dopaminergic medication therapy (limited evidence of benefit) and repetitive transcranial magnetic stimulation (rTMS) (limited evidence of benefit).

References


Key Study: Visual Scanning Training for Right Hemispheric Strokes


<table>
<thead>
<tr>
<th>Author / Year</th>
<th>Country</th>
<th>PEDro score</th>
<th>Methods</th>
<th>Outcome</th>
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<tbody>
<tr>
<td>Weinberg et al. 1977 USA 6 (RCT)</td>
<td></td>
<td>57 unilateral right brain damaged (RBD) patients due to stroke at least 4 weeks after onset of stroke were studied. Patients were randomly assigned to the experimental group receiving 20 hours of testing (1 hour/day for 4 weeks in reading, writing and calculation) or to the control group that received no testing between evaluations. In each group, patients were divided into severe and mild visual perceptual deficits. Both groups received occupational therapy as part of general rehabilitation.</td>
<td>RBD severe experimental patients showed significant improvement on WRAT, paragraph, arithmetic, copying, H-cancellation, C- &amp; E-cancellation, picture completion, digit span, DSS (and confront. RBD mild experimental patients significantly improved on WRAT, H-cancellation, C- &amp; E-cancellation, face matching, digit span and impersistence, p=0.05. RBD severe control patients improved on face counting, picture completion and object assembly. RBD severe control patients demonstrated no significant improvement on any of the outcome measures. Experimental group improved significantly more than the control group, especially patients within the experimental group with more severe deficits.</td>
<td></td>
</tr>
<tr>
<td>Weinberg et al. 1979 USA 6 (RCT)</td>
<td></td>
<td>53 stroke patients, at least 4 weeks post stroke with right unilateral brain damage were randomly assigned to receive either 1-hour treatment, 5 days a week for 4 weeks of occupational and physical therapy (C) or to receive 15 hours of tracking target practice, searching for lights on board, cancellation of stimuli and practice in reading and 5 hours of training in sensory awareness and training in spatial organisation over 4 weeks (SC).</td>
<td>Those with severe brain damage receiving SC demonstrated significant improvement in 24 of 26 psychological test scores. Those with mild brain damage in the experimental group exceeded those in the control group on 3 of 26 measures and those with severe brain damage in the SC group exceeded those in the control group on 15 of the 26 measures.</td>
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</table>
Importance: These early studies by Weinberg et al. demonstrated a dramatic improvement associated with perceptual training (visual scanning, sensory awareness and spatial organization) in individuals with visual perceptual deficits following right-sided stroke. Improvements were most significant for individuals with severe deficits.

Relevant SREBR Conclusion: There is strong evidence that treatment utilizing primarily enhanced visual scanning techniques improves visual neglect post-stroke with associated improvements in function.

Related References


Key Study: Unilateral Neglect Rehab through Trunk Rotation and Scanning


<table>
<thead>
<tr>
<th>Author / Year Country PEDro score</th>
<th>Methods</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wiart et al. 1997 France 4 (RCT)</td>
<td>22 stroke patients with recent stroke of less than 3 months onset who exhibited severe unilateral neglect syndrome with line bisection &gt; 11% of right deviation, line cancellation &gt; 2% of right deviation and line cancellation &gt; 2 left omission (LO) and Bell test of &gt; 6 LO. Patients were randomized to either and experimental or to a control group. Experimental group received 1 hour a day for 20 days of the Bon Saint Come method (use of a device with attached pointer which required trunk rotation to complete scanning tasks) followed by 2 to 3 hours of traditional rehabilitation (1 to 2 hours of PT and 1 hour of OT). Control group received 3 to 4 hours of traditional rehabilitation.</td>
<td>All 4 test results - line bisection, line cancellation, bell test, and change in Functional Independence Measure improved significantly more in the experimental group relative to the control group at 30 and 60 days.</td>
</tr>
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</table>

**Qualitative Outcome of Unilateral Neglect Syndrome**

<table>
<thead>
<tr>
<th>Number of Patients</th>
<th>Experimental Group</th>
<th>Control Group</th>
<th>Experimental Group Only</th>
</tr>
</thead>
<tbody>
<tr>
<td>Study 1</td>
<td>5</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Study 2</td>
<td></td>
<td>6</td>
<td>2</td>
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</table>

**Importance:** Although several observational studies have reported positive effects associated with trunk rotation in the treatment of neglect, the study by Wiart et al. (1997) is the sole RCT to examine the use of trunk rotation as part of a treatment intervention to improve neglect. The Bon Saint Come method, which incorporates trunk rotation with visual scanning, demonstrated positive effects on spatial neglect and functional ability. Given the positive effects associated with trunk rotation, further study of the use of trunk rotation as a treatment intervention is indicated.

**Relevant SREBR Conclusion:** There is moderate evidence that trunk rotation when combined with visual scanning is of benefit in the treatment of spatial neglect.
Related References


Key Study: Perceptual Impairment and its Treatment on Stroke Unit vs. Conventional Unit


<table>
<thead>
<tr>
<th>Author / Year</th>
<th>Country</th>
<th>PEDro score</th>
<th>Methods</th>
<th>Outcome</th>
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<tbody>
<tr>
<td>Lincoln et al. 1997 UK 6 (RCT)</td>
<td></td>
<td></td>
<td>315 stroke patients who were randomly assigned to receive rehabilitation for neglect on the stroke unit or to remain on conventional ward.</td>
<td>Rey Figure Copy score were significantly better for stroke unit patients at 3 months, 6 and 12 months compared to those patients on the conventional ward.</td>
</tr>
</tbody>
</table>

Importance: By far the largest randomized controlled trial to assess the effects of perceptual assessment and rehabilitation provided on a specialized stroke unit on neglect following stroke, Lincoln et al. (1997) reported significant benefits to perceptual abilities assessed at 3 months, 6 months and 12 months post-stroke.
Relevant SREBR Conclusion: There is strong evidence (4 positive, 1 negative and 1 mixed study) that perceptual training interventions improve perceptual functioning.

Related References


Key Study: Visual Neglect’s Impact on Stroke Rehabilitation


<table>
<thead>
<tr>
<th>Author / Year</th>
<th>Country</th>
<th>PEDro score</th>
<th>Methods</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kalra et al. 1997</td>
<td>UK</td>
<td>7 (RCT)</td>
<td>50 stroke patients with partial anterior circulation infarctions and visual neglect identified by a comprehensive assessment were randomly assigned to receive either therapy aimed to restoring normal tone, movement patterns and motor activity or to receive therapy aimed at integrating attentional and motor functions using the limb activation approach.</td>
<td>Significant improvement was noted on body image and cancellation subtest of the (RPAB) Rivermead Perceptual Assessment Battery at 12 weeks in favour of the treatment group receiving spatial cuing via limb activation.</td>
</tr>
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</table>

### Comparison of Patients in Spatiomotor Cueing Group and Conventional Therapy Group

The duration of hospitalization was significantly less in patients assigned to receive spatial cueing during treatment (42 vs. 66 days, p=0.001). In addition, the authors reported that on average, patients in the intervention group spent less time in physiotherapy compared to the control group (17.1 +/- 4.9 hrs vs. 22.6 +/- 8.0 hrs in the control group, p=0.01).

### Outcome and Therapy Use: Patients With or Without Perceptual Deficits

* BADL* at Discharge  
* Length of Hospital Stay (days)  
* Physiotherapy Time per Patient (hr)  
* Occupational Therapy Time per Patient (hr)

*Barthel Index of activities of daily living

*p-values: 0.0001, 0.01, 0.01, 0.01*
**Importance:** The largest of 3 studies examining the effectiveness of limb activation strategies, the study by Kalra et al. (1997) demonstrated that spatiomotor cuing during motor activity resulted in improved performance on assessments of visual neglect, reductions in time spent in physiotherapy and length of hospital stay when compared to patients receiving conventional therapy alone.

**Relevant SREBR Conclusions:** Based on the results of 3 RCTs (2 good quality and 1 fair), there is strong evidence that limb activation therapies improve neglect. However, little information is available with regard to duration of effect or the effect of treatment on functional ability.

**Related References**


Key Study: Limb Activation in Unilateral Neglect


<table>
<thead>
<tr>
<th>Author / Year</th>
<th>Methods</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Robertson et al. 2002</td>
<td>40 patients with right hemisphere strokes were randomly allocated to perceptual training group (PT) or limb activation treatment (LAT) with PT (LAT+PT). The PT group received perceptual training on visuoperceptual puzzles that required scanning to the left. The LAT+PT: received the same training as PT but also had a timer that emitted tone when a left movement was not performed by left wrist, leg or shoulder within a set time period. Both groups received 12 sessions of 45 minutes duration over a 12-week period. Patients were assessed at intake, post-training and 3, 6 and 18 to 24 months post-training.</td>
<td>Time by treatment condition interaction significant for Motricity Index. Improvement up to 24 months in LAT+PT group with little change in PT group over time.</td>
</tr>
</tbody>
</table>

**Importance**: Limb activation is based on the idea that any movement of the contralesional side may function as a motor stimulus activating the right hemisphere and improving neglect. While limb activation appears to have a positive impact on visual neglect, little data is available with regard to the effect of treatment on functional activities or the duration of effect. Robertson et al. (2002) reported improvement (as assessed by the Motricity Index) at 24 months following training via limb activation suggesting durability of treatment.

**Relevant SREBR Conclusions**: Based on the results of 3 RCTs (2 good quality and 1 fair), there is strong evidence that limb activation therapies improve neglect. However, little information is available with regard to duration of effect or the effect of treatment on functional ability.
Related References


D2.4 Case Study: Visual Perceptual Disorders
D2.4 Case Study: Visual Perceptual Disorders

Case Study

A 58 year old male is admitted to the stroke rehabilitation unit with a large right hemispheric stroke secondary to complete occlusion of the internal carotid artery. This stroke involved not only the frontal parietal cortex but extended to involve the temporal cortex as well as the subcortical white matter including the basal ganglia. As a consequence he presents with left hemiplegia, significant left neglect to confrontational testing and a left homonymous hemianopsia.

Q1. Describe how one would assess her neglect.

Answers
1. Initial screening tool (i.e. line bisection test).
2. More comprehensive testing would involve the Behavioural Inattention Test (BIT).

Q2. Assuming she has severe left neglect how would you treat it?

Answers
1. Can use interventions which target awareness of or attention to neglected space, i.e. visual scanning retraining, limb activation treatments.
2. Interventions that improve neglect by targeting deficits with a specific intervention, i.e prisms, hemi-field eye patching (more compensatory).
D2.5 Case Study: Use of Prisms
Case Study

A 56 year old male was admitted to rehabilitation 11 days post stroke. Initially he presented with progressively more severe headache and developed left hemiparesis (arm > leg), ataxia, visual and sensory abnormalities. CT scan demonstrated a right posterior/parietal infarct, a right-sided subdural hemorrhage as well as a right frontal and a left occipital subarachnoid/parenchymal hematoma. MVPT during rehabilitation was 23/36 with an average processing time of 6.6 seconds. Visual assessment demonstrated a left homonymous hemianopsia treated with Fresnel prism lenses. During rehabilitation he improved in his scanning abilities.

Q1. What is the purpose of Prisms placed on eye glasses and do they improve outcomes?

Answers
1. Prisms bend the visual field input and increase visual fields by 5-10 degrees.
2. Associated with improvements in visuospatial tasks in stroke patients with homonymous hemianopsia and neglect.

Discussion
There is strong evidence that treatment with prisms is associated with improvements in visuospatial tasks in stroke patients with homonymous hemianopsia and visual neglect. For example, Rossi et al. (1990) found that patients made significant improvements on several...
visual perception tests after four weeks of treatment with Fresnel prisms, as compared to patients in a non-treatment control group.

Reference

D2.6 Left Homonymous Hemianopsia
D2.6 Left Homonymous Hemianopsia

**Q1. Describe the neuropathways which are affected in homonymous hemianopsia post stroke.**

**Answer**

1. Homonymous hemianopsia can involve the optic tract from the optic chiasma to the lateral geniculate body and the optic radiation from the lateral geniculate body to the occipital visual cortex.

**Discussion**

Homonymous hemianopsia involves the optic tract after it leaves the optic chiasma to the lateral geniculate body. Homonymous hemianopsia post stroke inevitably involves the optic radiation on their way to the occipital visual cortex, located around the calcarine fissure of the occipital lobe.

**Q2. Describe the functional impact of homonymous hemianopsia.**

**Answer**

1. Visual field deficits often are unnoticed by the stroke survivor. Impairment of function is often minor.
2. An inability to drive and difficulties with reading remain the greatest functional consequences.
Case Study

60 year old male underwent CABG bypass surgery at acute care hospital and several days later developed a left posterior cerebral artery territory infarction involving the left thalamus and left perihippocampal gyrus. He was admitted to stroke rehabilitation where he presented with right sided weakness, right ataxia, right neglect, dysphagia, and right homonymous hemianopsia. He was oriented and had some difficulty following motor commands and was slow in processing information. He was independent in his mobility, functional transfers and transitional movements without aids. On admission he required cueing to organize and sequence ADL tasks. He was limited by visual, perceptual and cognitive (memory) deficits but improved at time of discharge from rehabilitation although he still required supervision for community mobility. Initially right upper extremity function was compromised by a mild right hemiparesis, incoordination, ataxia, apraxia, inattention and a right field cut.

Q3. In this case what part of the visual pathway is affected to give a right homonymous hemianopsia or field cut?

Answer
1. The optic radiation extending to the occipital lobes.

Q4. Why would he have memory problems?
1. Posterior cerebral artery territory also includes the medial temporal lobes and the hippocampus and hence memory can be negatively affected.

Q5. What is the typical clinical presentation of a posterior cerebral artery infarction?

Answer
Patients with a posterior cerebral artery infarction present with:
- Homonymous hemianopsia
- Memory loss
- Hemisensory loss
- Alexia without agraphia
D3. Aphasia
D3.1  Aphasia: General Information
Q1. **What is aphasia?**

**Answer**
Aphasia is an impairment of the ability to use language due to localized brain damage. The AHCPR Post-Stroke Rehabilitation Clinical Practice Guidelines defines aphasia as “the loss of ability to communicate orally, through signs, or in writing, or the inability to communicate orally, through signs, or in writing, or the inability to understand such communications; the loss of language usage ability”.

Q2. **Describe a Framework for Classifying Aphasia Post Stroke.**

![Framework diagram]

- Fluent?
  - No
    - Comprehension?
      - Good
        - Repetition?
          - Good
            - Transcortical Motor
          - Poor
            - Broca's
      - Poor
        - Mixed Transcortical
        - Global
  - Yes
    - Comprehension?
      - Good
        - Repetition?
          - Good
            - Transcortical Sensory
          - No
            - Conduction
      - Poor
        - Transcortical Sensory
        - Wernicke's
**Discussion**
Aphasias are classified as cortical, subcortical or mixed.

There are 8 forms of cortical aphasia: Broca’s, transcortical motor, mixed transcortical, and global (all characterized by non-fluent spontaneous speech) as well as Wernicke’s, conduction, transcortical sensory and anomic (all characterized by fluent speech). Two-thirds of aphasia can be classified in this way with the remainder being subcortical or mixed cortical-subcortical aphasias.

Subcortical aphasia are more heterogeneous and more often than not do differ from the classic cortical syndromes although any classic aphasia type may be caused by a subcortical lesion. Subcortical aphasias usually have a good prognosis.

<table>
<thead>
<tr>
<th>Type</th>
<th>Fluency</th>
<th>Comprehension</th>
<th>Repetition</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Broca’s</td>
<td>Nonfluent</td>
<td>Good</td>
<td>Poor</td>
<td>Telegraphic speech, paraphasias</td>
</tr>
<tr>
<td></td>
<td>-</td>
<td>+</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Wernicke’s</td>
<td>Fluent</td>
<td>Poor</td>
<td>Poor</td>
<td>Marked paraphasias and neologism</td>
</tr>
<tr>
<td></td>
<td>+</td>
<td>-</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Transcortical Motor</td>
<td>Nonfluent</td>
<td>Good</td>
<td>Good</td>
<td>Reduced initiation, organization and rate of speech</td>
</tr>
<tr>
<td></td>
<td>-</td>
<td>+</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>Transcortical Sensory</td>
<td>Fluent</td>
<td>Poor</td>
<td>Good</td>
<td>Neologisms, echolalia</td>
</tr>
<tr>
<td></td>
<td>+</td>
<td>-</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>Global</td>
<td>Nonfluent</td>
<td>Poor</td>
<td>Poor</td>
<td>Severely impaired</td>
</tr>
<tr>
<td></td>
<td>-</td>
<td>-</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Conduction</td>
<td>Fluent</td>
<td>Good</td>
<td>Poor</td>
<td>Literal paraphasias with forgetting of words</td>
</tr>
<tr>
<td></td>
<td>+</td>
<td>+</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Anomia</td>
<td>Fluent</td>
<td>Good</td>
<td>Good</td>
<td>Word-finding difficulty</td>
</tr>
<tr>
<td></td>
<td>+</td>
<td>+</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>Mixed Transcortical</td>
<td>Nonfluent</td>
<td>Poor</td>
<td>Good</td>
<td>Echolalia</td>
</tr>
<tr>
<td></td>
<td>-</td>
<td>-</td>
<td>+</td>
<td></td>
</tr>
</tbody>
</table>

### Type of Aphasia

- **Broca’s Aphasia.** Posterior-inferior frontal lobe stroke characterized by nonfluent, effortful speech with preserved comprehension and poor repetition. Associated with marked paraphasias and articulatory errors and often described as telegraphic.

- **Wernicke’s Aphasia.** Posterior part of superior (first) temporal gyrus stroke characterized by fluent speech but poor comprehension and poor repetition. Associated with marked paraphasias and neologisms.
Conduction Aphasia. Stroke of the parietal operculum (arcuate fasciculus) or insula or deep to the suramarginal gyrus marked by disproportional impairment in repeating spoken languages. Literal paraphasias with “targeting” of words (until getting the right one). Accounts for 5-10% of all aphasias.

Global Aphasia. Generally involve the entire MCA region with moderate to severe impairment of language of all language function.

Transcortical Aphasia. Isolation of the perisylvian language cortex causes transcortical aphasia, which is characterized by preserved repetition.

Transcortical Motor Aphasia. Stroke is located in the frontal lobe, anterior or superior to Broca’s area or in the subcortical region deep to Broca’s area. Characterized by nonfluent (reduced rate of speech and limited language output), good comprehension and good repetition.

Transcortical Sensory Aphasia. Watershed stroke isolating the perisylvian speech structures (Broca’s and Wernicke’s areas) from the posterior brain. Characterized by fluent speech (neologisms), poor comprehension and good repetition (possibly echolalia).

Anomic Aphasia. Spontaneous, fluent speech interrupted by word-finding pauses and relatively selective and severe naming impairment.

Q3. Define the following terms: Paraphasias, Neologisms, Telegraphic Speech, Echolalia and Word-Finding Difficulties.

Answers
Paraphasias are incorrect substitutions of words or parts of words. These can be:
- Literal or phonemic paraphasias: similar sounds (e.g., “sound” for “found” or “fen” for “pen”)
- Verbal or semantic paraphasias: word substituted for another form same semantic class (e.g., “fork” for “spoon” or “pen” for “pencil”).

Neologisms are newly coined nonsense words.

Telegraphic speech refers to a form of speech characterized by simple, yet meaningful sentence structures that contain content words but omit grammatical elements.

Echolalia refers to the involuntary repetition of words or phrases which are repeated either immediately after they have been heard or following a delay.
**Word finding difficulties** occur when an individual knows and understands a particular word but has difficulty retrieving it. Some common symptoms associated with word finding difficulties include the use of non-specific replacement words and slowed and hesitant speech.

**Q4. What are the most common types of aphasias seen following a stroke?**

**Answers**

1. The most common types of aphasias are the motor aphasias, in particular Broca’s and anomic aphasias.

**Q5. Describe how you would conduct a language assessment.**

**Answers**

1. Fluency
2. Comprehension
3. Repetition

**Discussion**

1. **Fluency**
   - Is the content empty, circumlocutory? Fluent
   - Are there frequent word-finding pauses? Non-fluent
   - Phonemic or literal paraphasic errors, i.e. “scoon” for “spoon”
   - Semantic paraphasic errors, i.e. “fork” for “spoon”

2. **Comprehension**
   - Yes/No questions: “Am I wearing glasses?”
   - Pointing: “Point to the watch,” “Point to the thing that tells time.”
   - Grammatical: “With the pen touch the glasses.”

3. **Repetition**
   - Multisyllable single words: “Constitutional”
   - Phrases: “Methodist Episcopal”
   - Sentences: “We all went over there”, “Oak trees grow tall”

**Q6. Describe the type of aphasia in each of the following cases.**
Cases and Answers

<table>
<thead>
<tr>
<th>Case Study</th>
<th>Type of Aphasia</th>
</tr>
</thead>
<tbody>
<tr>
<td>72 year old male with left cardioembolic stroke. Language assessment</td>
<td>Broca’s</td>
</tr>
<tr>
<td>revealed labored speech, primarily nouns and verbs, with poor repetition.</td>
<td></td>
</tr>
<tr>
<td>was able to follow simple commands.</td>
<td></td>
</tr>
<tr>
<td>68 year old female with left stroke following cardiac surgery. Language</td>
<td>Transcortical Motor</td>
</tr>
<tr>
<td>assessment revealed slow, labored speech with good</td>
<td></td>
</tr>
<tr>
<td>repetition. Able to follow simple commands.</td>
<td></td>
</tr>
<tr>
<td>59 year old male with left subcortical stroke. Language assessment</td>
<td>Anomia</td>
</tr>
<tr>
<td>revealed word-finding difficulty with good repetition. Able to</td>
<td></td>
</tr>
<tr>
<td>follow simple commands.</td>
<td></td>
</tr>
<tr>
<td>82 year old male with large left stroke. Language assessment reveals the</td>
<td>Global</td>
</tr>
<tr>
<td>patient is unable to follow commands and is unable to speak</td>
<td></td>
</tr>
<tr>
<td>apart from occasional automatic words.</td>
<td></td>
</tr>
<tr>
<td>74 year old female with left stroke related to carotid stenosis. Speech</td>
<td>Transcortical Sensory</td>
</tr>
<tr>
<td>was fluent but there were many errors with many nonsensical words. Patient</td>
<td></td>
</tr>
<tr>
<td>often repeated the questions of the assessor and repetition was done</td>
<td></td>
</tr>
<tr>
<td>well. Not able to consistently follow commands.</td>
<td></td>
</tr>
<tr>
<td>76 year old male with left intracerebral hemorrhage secondary to</td>
<td>Wernicke’s</td>
</tr>
<tr>
<td>congophillic angiopathy. Language assessment revealed normal rate of</td>
<td></td>
</tr>
<tr>
<td>speech but the words did not make sense. Repetition was done poorly and</td>
<td></td>
</tr>
<tr>
<td>the patient was unable to follow simple commands.</td>
<td></td>
</tr>
<tr>
<td>48 year old female with left subcortical/cortical hemorrhage. Speech was</td>
<td>Conduction</td>
</tr>
<tr>
<td>normal and patient was able to follow commands. Repetition was done</td>
<td></td>
</tr>
<tr>
<td>poorly.</td>
<td></td>
</tr>
<tr>
<td>66 year old female with a left stroke following a cardiac arrest. Language</td>
<td>Mixed Transcortical</td>
</tr>
<tr>
<td>assessment revealed very labored speech, primarily one to two word</td>
<td></td>
</tr>
<tr>
<td>responses. She was not able to respond to commands. Repetition was done</td>
<td></td>
</tr>
<tr>
<td>well.</td>
<td></td>
</tr>
</tbody>
</table>
D3.2 Case Study: Broca’s Aphasia
Case Study

A 36 year old woman presents with a moderate size left hemispheric stroke. You assess her 5 days post stroke at which time she has a right hemiparesis, with near complete paresis of the right upper extremity and only partial paresis of the right lower extremity (able to move the hip and knee against gravity but not yet able to dorsiflex the ankle). The patient is able to follow commands with little difficulty but is unable to respond verbally, says only a few words repetitively and is unable to repeat phrases. She is admitted to the rehabilitation unit 12 days after her stroke and at the time her MRI is repeated.

Q1. What impairments does this patient have?

Answers
1. Right hemiparesis.
2. Broca’s or expressive aphasia.

Q2. What type of aphasia is present in this case and describe it?
**Answer**
1. Broca’s or expressive/motor aphasia is caused by left hemispheric damage.
2. Characterized by nonfluent, effortful speech with preserved comprehension.

**Q3. For the patient described above, the doctor looking after the patient questions the value of aphasia therapy. What evidence is there that aphasia therapy is helpful?**

**Answers**
1. Aphasia therapy has been shown to be effective, particular if provided at adequate intensity in the subacute phase of stroke rehabilitation.

**Discussion**
There is a large literature of studies on speech and language therapy in aphasic patients. Robey et al. (1998) analyzed no fewer than 55 studies in his meta-analysis and concluded that there was sufficient evidence showing speech and language therapy had a positive impact on aphasia recovery in the acute phase and a lesser, but still significantly positive, impact during the chronic phase. However, many of the studies in the meta-analysis contained small samples and were of poor quality (non-RCT) studies. Several of the studies included participants who were aphasic due to etiologies other than stroke.

Bhogal et al. (2003) identified 8 RCTs which compared the intensity of speech and language therapy (SLT) delivered by a trained therapist versus a non-trained therapist or a non-SLT control. 4 of the studies showed a positive result in favor of the SLT training while 4 did not. An examination of the intensity of the treatment and mean changes scores showed significant positive treatment effects for those studies which provided a mean of 8.8 hours of therapy per week for 11.2 weeks versus the negative studies which only provided 2 hours of therapy per week for 22.9 weeks. Bhogal et al. (2003) concluded that intense therapy provided over a shorter period of time was effective while less intense therapy provided over a longer period of time was not.

**References**


Key Study: Volunteers Providing Supported Conversation in Aphasia


<table>
<thead>
<tr>
<th>Author / Year</th>
<th>Country</th>
<th>PEDro score</th>
<th>Methods</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kagan et al. 2001</td>
<td>Canada &amp; USA</td>
<td>6 (RCT)</td>
<td>Study included 40 stroke patients with moderate-to-severe aphasia and volunteers at an aphasia centre. Volunteers were randomly assigned to either receive a workshop training session designed to teach them how to acknowledge and reveal the competence of adults with aphasia through supported conversation (SCA) or were assigned to be exposed to aphasia by watching a video that told stories of patients with aphasia and their families. There were also given opportunity to interact with aphasia patients. Patients were randomly assigned to volunteers.</td>
<td>SCA trained volunteers scored higher than controls on rating of acknowledging competence and revealing competence of their aphasic partners. Patients assigned to trained volunteers scored higher on social and message exchange skills than did patients assigned to control volunteers.</td>
</tr>
</tbody>
</table>

**Importance:** Training conversation or communication partners within the aphasic individual’s own setting promotes access to conversation for the aphasic individual. The SCA technique (Kagan et al. 2001) is an effective tool used to teach communication partners skills they can use to promote conversation.

**Relevant SREBR Conclusions:** There is moderate evidence that the technique of training conversation partners, Supported Conversation for Adults with Aphasia (SCA) is associated with enhanced conversational skill for both the trained partner and the individual with aphasia. There is limited evidence, based on several small studies, that training conversation partners is
associated with increased well-being and social participation in addition to positive communication outcomes.

**Related References**


Key Study: Intensity of Aphasia Therapy


<table>
<thead>
<tr>
<th>Author / Year</th>
<th>Canada</th>
<th>No Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>METHODS</td>
<td>A systematic review to explore how the intensity of aphasia therapy (speech and language therapy) is associated with aphasia recovery in stroke patients. Intensity was determined by length (weeks), hours per week, and total hours of therapy. Searched for candidate articles on MEDLINE that were published between 1975 and 2002. Primary outcome measures were the PICA, FCP, and Token Test, and Pearson’s correlation coefficient was used to assess the relationship between intensity and outcome of therapy. Ten studies met the inclusion criteria which established a sample of 864 stroke patients. Hours of therapy per week (p=.001, p=.027), and total hours of therapy (p&lt;.001) were both significantly correlated with improvement on the PICA and Token Test, whereas total length of therapy was found to be inversely correlated (p=.003) with change in PICA scores, suggesting that therapy lasting longer (in weeks) was less intense.</td>
<td></td>
</tr>
</tbody>
</table>

Average Characteristics of Aphasia Therapy Associated with either a Positive or Negative Impact on Recovery (according to the PICA* FCP** or Token Test)

<table>
<thead>
<tr>
<th>Impact of Therapy</th>
<th>Positive</th>
<th>Negative</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hours per Week</td>
<td>8.8</td>
<td>22.9</td>
</tr>
<tr>
<td>Therapy Length (Weeks)</td>
<td>11.2</td>
<td>43.6</td>
</tr>
<tr>
<td>Total Hours of Therapy</td>
<td>98.4</td>
<td>98.4</td>
</tr>
</tbody>
</table>

*Porch Index of Communicative Abilities  **Functional Communication Profile

**Importance:** An examination of intensity of treatment and mean change scores undertaken by Bhogal et al. (2003) showed significant positive treatment effects for a mean of 8.8 hours of therapy per week for 11.2 weeks versus negative studies that provided approximately 2 hours per week for 22.9 weeks. Hours of therapy provided in a week and total number of hours of therapy were significantly correlated with greater improvement on both the PICA and the Token Test while total length of therapy (i.e. time) was inversely correlated with mean change in PICA scores. Bhogal et al (2003) concluded that intense therapy over a short amount of time could improve outcomes of speech and language therapy for stroke patients with aphasia.

**Relevant SREBR Conclusions:** There is conflicting evidence whether speech and language therapy (SLT) is efficacious in treating aphasia following stroke. The failure to identify a
consistent benefit appears to be due, in part, to the low intensity of SLT applied in the negative studies. The positive trials provided very intense therapy over a relative short period of time, whereas, the negative trials provided much less intensive therapy over a longer period of time.

**Related References**


Key Study: Home Treatment of Aphasia by Trained Volunteers


<table>
<thead>
<tr>
<th>Author / Year</th>
<th>Country</th>
<th>PEDro score</th>
<th>Methods</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Marshall et al. 1989 USA 5 (RCT)</td>
<td>This study involved 121 males who were 2 to 12 weeks post onset from a single left hemisphere thrombosis infarct resulting in aphasia. Patients were randomized to receive home therapy treatment given by a wife, friend or relative, treatment by speech-language pathologist or treatment by speech-language pathologist deferred for 12 weeks. Therapy was provided for 8 to 10 hours a week for 12 weeks.</td>
<td>At 12 weeks, the SLP group showed significantly more improvement than deferred group. Improvements noted in home treatment group not differ from SLP group. At 24 weeks deferred treated group caught up to other 2 groups and no significant differences between groups was noted.</td>
<td></td>
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</tbody>
</table>

**Importance:** Language therapy delivered by trained non-professionals (wife, friend or relative) in the home may be just as effective in improving language outcomes as therapy delivered by a trained speech-language professional.

**Relevant SREBR Conclusions:** There is strong evidence that trained volunteers can provide speech and language therapy and achieve similar outcomes to speech-language pathologists. This could serve as an effective adjunct to speech-language pathologists’ treatment.

**Related References**


D3.3 Case Study: Wernicke’s Aphasia
D3.3 Case Study: Wernicke’s Aphasia

Case Study

A 74 year old male was admitted to hospital with a left parietal-temporal intracerebral hemorrhage. He had no motor weakness on the right side, a right upper quadrantanopia. The nurses report that he talked a lot but much of what he said did not make sense and he seemed to make up some words they had never heard before. One nurse noted he used the term “you know” and the word “thing” a lot. Some of them thought that he was confused; others wondered if he had a psychiatric history. When asked “Do helicopters in South America eat their young?” he replied with “I would expect so”.

Q1. What is the name of this patient’s communication disorder?

Answer
1. Wernicke’s or comprehensive aphasia

Q2. What are the defining features of this communication disorder?

Answer
Wernicke’s aphasia is defined by:
- Near normal amount of normal (fluent) speech output.
Comprehension of spoken language is defective although patients may derive meaning from nonverbal cues. 
Lots of empty phrases and circumlocutions. 
Usually many paraphasic errors (phonemic paraphasias or sound-alike and semantic paraphasias or mean-alike words), jargon (nonword sounds or fluent, well-articulated, but incomprehensible speech) and neologisms (newly coined words). 
Frequently use “filler” words, such as thing, it, that, you know. 
Naming is impaired. 
Repetition of spoken language is abnormal. 
Response to nonsense questions asked in an inquisitive manner are inappropriate.

**Q3. What area of the brain is involved?**

**Answer**
1. Left temporal lobe, especially the posterior portion of the superior temporal gyrus.

**Q4. Describe the potential impact of this communication disorder on rehabilitation?**

**Answer**
1. Because Wernicke’s aphasia does impact on the understanding of language, when it is profound it can have a negative impact on learning which is important to rehabilitation.
D3.4 Case Study: Conduction Aphasia
D3.4 Case Study: Conduction Aphasia

Case Study

A 48 year old male was admitted to the stroke rehabilitation program on October 24, 2008 and discharged 6 weeks later. 5 weeks previously he suffered a subarachnoid hemorrhage with right sided weakness. CT scan revealed a bleed in the left putamen and both caudate heads with compression of the ventricles. Etiology of the bleed was felt to be related to hypertension. On admission to rehabilitation he presented with right upper extremity weakness, some mild left lower extremity weakness, apraxia, poor balance, dysarthria with word-finding difficulties and some impulsivity.

Communication assessment revealed verbal output which was fluent but with dysfluencies noted with sound and word repetitions. Frequently verbal expressions contained jargon and literal as well as semantic paraphasias. Auditory compression was judged to be intact for yes/no questions obtained from the Western Aphasia Battery and he was able to follow one and two step commands. Reading comprehension was moderately impaired with multiple step commands but improved. At discharge he did well with sentence formulation to multiple action pictures. Occasionally, during conversational speech at the time of discharge there were circumlocutions, semantic and literal paraphasias and confused language at times.

Q1. What type of communication disorder is present?

Answer
1. Conduction aphasia.

Q2. What are the clinical features of this communication disorder?

Answers
1. Marked by disproportional impairment in repeating spoken languages.
2. Literal paraphasias with “targeting” of words (until getting the right one).
3. Accounts for 5-10% of all aphasias.
Q3. What area of the brain is involved?

Answer
1. Stroke of the parietal operculum (arcuate fasciculus) or insula or deep to the suramarginal gyrus.
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