5. Rehabilitation of Cognitive Impairment Post Stroke

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5.1 The Nature of Cognitive Impairment Post Stroke

Cognitive impairments are common after stroke. At three months, 1, 2 and 3 years post stroke, the prevalence rates of cognitive impairment were 39%, 35%, 30% and 32% respectively (Pater et al. 2003).

Cognitive impairments are generally divided into various domains which include:

- Attention
- Memory
- Executive function
- Perception and praxis
- Language

<table>
<thead>
<tr>
<th>Attention</th>
<th>• Focus attention, sustained attention, selective attention, divided attention</th>
</tr>
</thead>
<tbody>
<tr>
<td>Memory</td>
<td>• Visual memory, auditory memory, working memory, episodic memory, semantic memory, working memory, procedural memory</td>
</tr>
<tr>
<td>Executive Function</td>
<td>• Initiation, processing speed, problem solving, planning</td>
</tr>
<tr>
<td>Perception, praxis</td>
<td>• Visuo-spatial, visuo-perceptual, Unilateral neglect, inattention, apraxia, agnosia, prosopagnosia</td>
</tr>
<tr>
<td>Language</td>
<td>• Aphasia: Broca’s, Wernicke’s, transcortical motor/sensory or mixed, conductive, global</td>
</tr>
</tbody>
</table>

5.2 Vascular Cognitive Impairment

5.2.1 Definition of Vascular Cognitive Impairment (VCI)

- VCI encompasses a wide range of cognitive deficits, from relatively Mild Cognitive Impairment of Vascular Origin (VaMCI) to Vascular Dementia (VaD), the most severe form of VCI.
- VCI is a syndrome with cognitive impairment affecting at least one cognitive domain (e.g., attention, memory, executive function, perception or language) with evidence of clinical stroke or subclinical vascular brain injury.
5.2.2 Characteristics of Cognitive Deficits in VCI

• The pattern of cognitive deficits in VCI may include variable degree of deficits in any of the cognitive domains, including focal stroke syndromes.
• Cumming et al. (2013) notes that, “It is now thought that stroke tends to have greater deleterious impact on attention and executive function than on memory”.
• In a community-based comparison of stroke patients with population controls, stroke patients were more frequently impaired than controls in spatial ability, executive function, attention and language but were not more impaired in orientation or memory (Srikanth et al. 2003, Cumming et al. 2013).
• Cumming et al. (2013) noted that, “Cognitive slowing is a common complaint after stroke, and a majority of patients exhibit marked slowness of information processing (Hochstenbach et al. 1998; Rasquin et al. 2005). Processing speed is clinically relevant, as it makes an independent contribution to functional outcome after stroke (Barker-Collo et al. 2010) and is independently predictive of dependency in stroke survivors (Narasimhalu et al. 2011)”.
• Cumming et al. (2013) also noted that, “It is possible that attention and executive deficits appear to predominate after stroke these domains are more often tested using time-sensitive tasks (e.g., Trail-Making and verbal fluency) than the domains of memory or language”.

5.2.3 Vascular Pathology in VCI

• Cognitive impairment seen in VCI result from a range of vascular pathology, including multiple cortical infarcts, multiple subcortical infarcts, “silent” infarcts, small-vessel disease with white matter lesions (leukoaraiosis) and lacunae, and brain hemorrhage.
• Cumming et al. (2013) notes that, “There is a broad distinction between focal damage, which can lead to selective cognitive impairments, and diffuse neuronal dysfunction, which produces a more uniform profile of mental slowing, memory problems and executive deficits (de Haan et al. 2006) ... Diffuse dysfunction typically results from underlying sub-clinical cerebrovascular disease, such as white matter disease, or an accumulation of small infarcts as in small-vessel disease (Pantoni 2010). Over the four years following a stroke, higher load of white matter hyperintensities (WMHs) is strongly associated with dementia and cognitive decline (Dufouil et al. 2009). Stroke patients with white matter lesions and silent infarcts were worse on cognitive tasks at baseline and two-year follow-up than those without this damage (Rasquin et al. 2005)”.

5.2.4 Prevalence of Dementia Post-Stroke

• As many as two-thirds of stroke patients go on to experience cognitive impairment or decline following stroke.
• Risk of developing dementia may be 10x greater among individuals with stroke than those without stroke.
• Presence of white matter changes (leukoaraiosis) is related to development of dementia. The LADIS study (2011) reported that severe white matter changes pose a 3-fold risk of developing dementia independent of age and sex.
• Independent predictors of post-stroke dementia were older age, lower education, history of stroke, diabetes, atrial fibrillation, stroke severity and existing cognitive impairment. (Pendlebury and Rothwell 2009) Additionally, the above risk factors as well as cerebral amyloid angiopathy, low physical activity, HTN, both hyper- and hypoglycemia, smoking, and carotid and
intracranial atherosclerosis have been correlated with an increased risk of VCI (Farooq and Gorelick 2013; Pendlebury and Rothwell 2009).

- The above risk factors as well as cerebral amyloid angiopathy, low physical activity, HTN, both hyper- and hypoglycemia, smoking, and carotid and intracranial atherosclerosis have been correlated with increased risk of VCI (Farooq and Gorelick 2013).
- 10% have existing dementia at the time of stroke, 10% develop dementia after first-ever stroke, and approximately one-third of patients experience dementia following recurrent stroke (Pendlebury and Rothwell 2009).

5.2.5 Impact of Vascular Cognitive Impairment

- VCI affects functional abilities.
- VCI is often associated with depression.
- Can result in higher mortality rate, 2-6 times higher among those with post-stroke dementia, after adjusting for stroke severity, stroke recurrence, co-morbid cardiac disease and demographic factors (Leys et al. 2005).

5.2.6 Diagnosis of Vascular Cognitive Impairment

The clinical diagnosis of VCI are based on clinical assessment of the cognitive domains comprise of attention, executive function, memory, visuospatial function and language.

**Classification of VaMCI and VaDementia**

<table>
<thead>
<tr>
<th></th>
<th>Mild Vascular Cognitive Impairment (VaMCI)</th>
<th>Vascular Dementia (VaD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>The classification of VaMCI and VaD must be based on cognitive testing. A minimum of 4 domains should be assessed: executive/attention, memory, visuospatial function, language</td>
<td>Decline in cognitive function from prior baseline and impairment performance in at least 1 cognitive domain.</td>
<td>Decline in cognitive function from prior baseline and impairment performance in at least 2 cognitive domains.</td>
</tr>
<tr>
<td>Function</td>
<td>Instrumental ADLs may be normal or mildly impaired; independent of motor and sensory deficits.</td>
<td>ADLs sufficiently severely impaired; independent of motor and sensory deficits.</td>
</tr>
<tr>
<td>Other related categories</td>
<td>Probable VaMCI* Possible VaMCI* Unstable VaMCI*</td>
<td>Probable VaD* Possible VaD*</td>
</tr>
</tbody>
</table>

*Detailed diagnostic criteria: Refer to Gorelick et al. (2011)*
5.2.7 Vascular Dementia

- Vascular dementia is the second most common cause of dementia after Alzheimer’s disease.
- Often confused with Alzheimer’s Dementia, and may even co-exist.

The distinguishing features of vascular dementia versus Alzheimer’s disease are as below.

**Vascular Dementia vs. Alzheimer’s Dementia**

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Vascular Dementia</th>
<th>Alzheimer’s Disease</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>Infarcts or white matter lesions (leukoraiosis)</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 (including covert/silent stroke), vascular risk factors</td>
<td>Less common</td>
</tr>
</tbody>
</table>

**Memory Function Post Stroke**

- Studies have shown that patients with vascular dementia had superior long-term memory but suffered from more frontal executive impairment when compared to Alzheimer’s patients (Looi and Sachdev 1999).
- However, memory is not necessarily spared after a stroke or in patients with a diagnosis of vascular cognitive impairment.
- Cumming et al. (2013) notes that, “The presence of sub-cortical infarcts in older people has been associated with lower episodic, semantic, and working memory performance (Schneider et al. 2007). Memory deficits, though, appear to be less prevalent than deficits in other cognitive domains, and when they do occur, they are likely to have a different genesis to those seen in Alzheimer patients, Recognition memory, which tests retention of information without effortful search and retrieval, may be less affected than non-cued recall after stroke (Hochstenbach et al. 1998; Sachdev et al. 2004), suggesting that the underlying cause may be less amnestic and more executive”.

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5.3 Screening and Assessment of Cognitive Impairment Post Stroke

5.3.1 Mini-Mental Screening Evaluation

- Brief screening tool of cognitive impairment.

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 cut-off 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 and 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; does not require training. 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). Lacks an evaluation of executive function. 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, Bleecker et al. 1988, Lorentz et al. 2002).</td>
</tr>
</tbody>
</table>

Improving the MMSE

- Suggested solutions to the MMSE’s poor sensitivity rates includes the use of age-specific norms (Bleecker et al. 1988) and the addition of a clock-drawing task to the test (Suhr & Grace, 1999).
- Clock-drawing tests themselves have been assessed as acceptable to patients, easily scored and less affected by education, age and other non-dementia variables than other very brief measures of cognitive impairment (Lorentz et al. 2002) and would have little effect on the simplicity and accessibility of the test.
5.3.2 Clock-Drawing Test (CDT)

<table>
<thead>
<tr>
<th>Questions</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>What does it measure?</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>What is the scale?</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>What are the key scores?</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>What are its strengths?</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>What are its limitations?</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 discoordination (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>
5.3.3 Montreal Cognitive Assessment (MoCA)

- Screening tool to detect mild cognitive impairment.
- Less than 26/30 indicates cognitive impairment.
- Strengths are, it can detect mild forms of impairment when MMSE is normal.
- Availability of alternate MoCA to reduce learning bias, and MoCA version for visually impaired individuals, who omit trail task, cube copying, clock drawing and picture naming.
- Available in multi-languages, freely accessible.

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, visuoconstructional 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.</td>
</tr>
</tbody>
</table>

5.3.4 Neuropsychological Testing

The accepted “gold standard” for assessment of cognitive impairment is a battery of neuropsychological tests which covers various domains, with domain-specific deficits being determined using normative data (Cumming et al. 2013).
5.4 Cognitive Recovery

- While cognitive decline may continue post stroke, approximately 16-20% of patients with cognitive impairment improve.
- While most improvements occur in the first 3 months, recovery may continue for at least the first year post-stroke.
- There appears to be domain-specific trend in the prevalence and the temporal evolution of the stroke-related cognitive impairment (Hurford et al. 2013).
- Attention and speed was the most impaired domain at < 1 month after stroke, but had the greatest trend for decreasing impairment, from 72.4% acutely to 37.9% after 3 months (p<0.01).
- Perceptual skill impairment showed a high prevalence of impairment in the acute stage (29.5% impaired, 95% CI: 21.8 to 38.1) but a strikingly lower prevalence of impairment at 1 month (9.5%, 95%CI: 2.7 to 22.6) and 3 months (8.1%, 95% CI: 1.7 to 21.9; p=0.002).
- Cumming et al. notes that, “At one year poststroke, a majority of patients still had attention deficits, while deficits in language and memory were more likely to have resolved (14)”.
- Mortality rates among stroke patients with dementia are 2-6x greater than those without.

5.5 Management for Vascular Cognitive Impairment

5.5.1 Non-Pharmacological Management

**General Management Strategy** (Farooq and Gorelick 2013)

- Focus on managing modifiable risk factors for stroke.
- Life style: smoking cessation, moderate alcohol intake, healthy diet, weight control and physical activity.
- Medical: hypertension, hyperglycemia, hyperlipidemia, smoking and atrial fibrillation.

5.5.2 Pharmacotherapy for Vascular Cognitive Impairment

The main aims of pharmacological management in vascular cognitive impairment are:

- Disease modifying - to prevent further decline in cognitive function, reduce white matter changes and stroke recurrence.
- Symptomatic management - to improve current level of cognitive function.

a) Disease-Modifying Pharmacological Management in VCI

**Aspirin**

- Moderate evidence, based on a single pilot RCT, that ASA is effective in stabilizing +/- improving cognitive outcomes in patient with dementia.
ASA in Multi-Infarct Dementia

**STUDY**

- Randomized 70 patients with multi-infarct dementia to 325 mg ASA per day vs. usual care.
- Mean length of F/U was 15.6 months for treatment and 14.9 months for control groups.
- ASA treated patients experienced fewer TIAs +/- strokes with lower mortality although numbers too small for meaningful statistical analysis.

Perindopril and Indapamide (PROGRESS)

**STUDY**
Effects of blood pressure lowering with perindopril and indapamide therapy on dementia and cognitive decline in patients with cerebrovascular disease. The Perindopril Protection against Recurrent Stroke Study (PROGRESS). Arch Int Med 2003; 163(9):1069-75.

- PROGRESS was a RCT (N=6105) people with prior stroke or TIA.
- Participants were assigned to either active treatment (perindopril for all participants and indapamide for those with neither an indication for nor a contraindication to a diuretic) or matching placebo(s).
- The primary outcomes for these analyses were dementia.
- During a mean follow-up of 3.9 years, dementia was documented in 193 (6.3%) of the 3051 randomized participants in the actively treated group and 217 (7.1%) of the 3054 randomized participants in the placebo group (relative risk reduction, 12% [95% confidence interval, -8% to 28%]; P =.2).
- Cognitive decline occurred in 9.1% of the actively treated group and 11.0% of the placebo group (risk reduction, 19% [95% confidence interval, 4% to 32%]; p<0.01).
- The risks of the composite outcomes of dementia with recurrent stroke and of cognitive decline with recurrent stroke were reduced by 34% (95% CI: 3% to 55%, p =.03) and 45% (95% CI: 21% to 61%) p<0.001, respectively, with no clear effect on either dementia or cognitive decline in the absence of recurrent stroke (using DSM-IV criteria) and cognitive decline (a decline of 3 or more points in the Mini-Mental State Examination score).

b) Symptomatic Pharmacological Management in VCI

**Cholinesterase Inhibitors**

- Cholinergic agents have been used in the treatment of Alzheimer’s dementia where their role is well-established.
- Three reversible acetylcholinesterase inhibitors, donepezil, rivastigmine, and galantamine, have been investigated in the treatment of vascular dementia. Donepezil and galantamine can be helpful in VaD or mixed Alzheimer’s disease and cerebrovascular disease. Limited evidence for treatment with rivastigmine.
**Donepezil in Vascular Dementia**

- Selective acetylcholinesterase inhibitor well studied in treatment of mild to moderate Alzheimer’s dementia.
- Effectiveness among patients with vascular dementia in 2 large RCTs (Black et al. 2003; Wilkinson et al. 2003).

**STUDY**

**Black S, Roman GC, Geldmacher DS, Salloway S, Hecker J, Burns A, Perdomo C, Kumar D, Pratt R.**


- 603 patients with probable (70.5%) or possible (29.5%) VaD randomized to 24 weeks of Donepezil 5mg/day or 5mg/day x28days then 10mg/day or placebo.
- Groups receiving Donepezil showed significant improvement in cognition vs placebo.
- Withdrawal due to adverse reactions was 11.1% in 5mg/d, 11.1% in placebo and 21.8% in 10mg/day (p=0.005).

**STUDY**


- 616 patients with probable (76%) or possible (24%) VaD randomized to Donepezil 5mg/day or 5mg/day x 28d then 10 mg/day or placebo x 24 weeks.
- Both Donepezil groups showed significant improvements in cognition outcomes vs. placebo.
- Withdrawal due to adverse events: placebo - 8.8%, donezepril - 5mg/day 10.1%, 10mg/day - 16.3%.

- Strong evidence, based on 2 RCTs, donepezil taken for 24 weeks improves cognitive function in patients with probable or possible vascular dementia (Black et al. 2003; Wilkinson et al. 2003).

**Rivastigmine in Vascular Dementia**

- In non-randomized, open-label clinical studies, there have been benefits associated with Rivastigmine among patients with subcortical VaD.
- Limited evidence treatment with Rivastigmine is associated with more stable cognitive performance and improved behavioural outcomes among patients with subcortical vascular dementia.

**Galantamine in Vascular Dementia**

- Moderate evidence based on a single RCT of excellent quality, galantamine associated with improvements in cognitive and functional ability.
STUDY

- Studied 592 patients with probable vascular dementia or mixed dementia randomized to 24 mg/day galantamine or matching placebo x 6 months.
- Galantamine associated with improvements in cognitive and functional ability.
- However, benefits more clearly demonstrated among patients with mixed dementia than vascular dementia.

Nimotidine in Vascular Dementia

- Calcium-channel blocker that readily crosses BBB.
- Vasoactive effect and may improve blood flow to hypoperfused areas.
- Recent meta-analysis treatment with nimotidine with vascular dementia is associated with non-significant improvements in global function and ADLs when compared to placebo.
- Well tolerated with few side-effects.

Other Drugs in Vascular Dementia

- Strong evidence Memantidine (NMDA receptor antagonist) associated with stabilization or improvement of cognitive function.
- Strong evidence Pentoxifylline (increases cerebral blood flow) associated with cognitive benefit in patients with multi-infarct dementia.
- Moderate evidence Milnacipran (SNRI used in Rx depression) has positive effects on cognition in stroke patients with depression.

Methylphenidate (Ritalin)

- Methylphenidate (0.25 - 0.30 mg/kg bid) is recommended in adults to enhance attention and speed of cognitive processing in the adult population.
- To consider in treating patients with difficulty in attention and focus.

5.6 Depression and Cognitive Disorders

5.6.1 Impact of Depression on Cognitive Disorders

- Depression is an important issue which must be considered when managing cognitive disorders.
- Depression may affect the results of the cognitive tests (Ruchinskas & Curyto 2003).
- Depression in patients with amnestic mild cognitive impairment is associated with risk of developing Alzheimer’s type dementia and cognitive deterioration may proceed at a more rapid pace (Modrego & Ferrández 2004).
- A significant and independent association between presence of depression and cognitive impairment has been demonstrated in stroke survivors one year following the stroke event (Kalaria & Ballard 2001; Talelli et al. 2004).
• Brodaty et al. (2007) have 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.

5.6.2 Pseudo Dementia

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

Dementia vs. Pseudodementia

<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</td>
<td>Usually rapid</td>
</tr>
<tr>
<td></td>
<td>often missed</td>
<td></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>

5.7 Cognitive Rehabilitation Post Stroke

Interventions for cognitive rehabilitation are broadly classified as:

1. Direct remediation/cognitive skill training.
2. Compensatory strategy training.

Cognitive rehabilitation interventions aim to:

1. Reinforce, strengthen or re-establish previously learned patterns of behavior.
2. Establish new patterns of cognitive activity through internal compensatory cognitive mechanisms for impaired neurological systems.
3. Establish new patterns of activity through **external compensatory mechanism** such as external aids, or environmental structuring and support.

4. **Enable persons to adapt** to their cognitive disability.

Evidence on cognitive rehabilitation:

- Many studies of cognitive rehabilitation were performed in a heterogeneous population consisting of stroke and traumatic brain injury. Overall, cognitive rehabilitation interventions were associated with small but significant treatment effects.
- Most recent review by Cicerone et al. (2011) reported that existing evidence supports visual spatial rehabilitation, interventions for aphasia and apraxia.
- Recommendations for Practice Standards are as below (Cicerone et al. 2011): Most recent review by Cicerone et al. (2011) reported that existing evidence supports visual spatial rehabilitation, interventions for aphasia and apraxia.
- Recommendations for Practice Standards are as below (Cicerone et al. 2011):
  - For right hemispheric stroke: Visual scanning for left visual neglect.
  - For left hemispheric stroke: Cognitive-linguistic interventions for aphasia, specific gestural or strategy training for apraxia.

### 5.8 Cognitive Rehabilitation for Attention, Memory, Executive Function

#### 5.8.1 Remediation of Attention

- Most interventions relied on drills and practice used within stimulus-response paradigm.
- Gains made during speeded tasks are less durable than gains made via non-speeded tasks.
- Greater benefit observed from attention training on complex tasks requiring selective or divided attention when compared to attention training on basic tasks of reaction time or vigilance.
- **Moderate** evidence that visual attention retraining can help with on-road driving test performance but no other attention testing.
- Moderate evidence that attention process training may improve attention deficits post stroke. **Limited** evidence that computer-assisted training of attention may improve performance of specific attention tasks.

**STUDY**


- 84 hemispheric stroke patients (<6 months) who wanted to return to driving randomized to training of visual processing speed, divided attention and selective attention vs traditional computerized visuoperceptual retraining for 20 sessions each.
- No significant differences except 2X improvement in rate of success on on-road driving test.
**STUDY**


- 78 acute stroke patients with attention deficits identified by neuropsychological assessment. Participants were randomly allocated to standard care plus 30 hours of Attention Process Training (APT) or standard care alone. APT training consisted of 1 hour sessions provided for a total of 4 weeks. The primary outcome was Integrated Visual Auditory Continuous Performance Test Full-Scale Attention Quotient (IVA-CPT).

- Patients in the intervention group performed significantly better on the primary outcome, as compared to patients in the control group (p < 0.05). No other significant differences were reported between the two groups.

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5.8.2 Remediation of Memory Deficits

- 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.
- Relatively few of the study participants had suffered a stroke.

5.8.3 Remediation of Executive Functioning and Problem Solving

- Executive functioning defined as “those integrative cognitive processes that determine goal directed and purposeful behaviour and are superordinate in the orderly execution of daily life functions” (Cicerone et al. 2000).
- Functions affected include: ability to formulate goals; to initiate behaviour; to anticipate the consequences of actions; to plan and organize behavior according to spatial, temporal, topical, or logical sequences; and to monitor and adapt behavior to fit a particular task or context (Cicerone et al. 2000).
- Cicerone et al. (2011) studied 17 studies on executive function involving patients with TBI. There were no stroke patients in the studies included in the review (Chung et al. 2013) also concluded that there is insufficient evidence on cognitive rehabilitation for executive function post stroke.

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**STUDY**


DOI: 10.1002/14651858.CD008391.pub2.

- Nineteen studies (907 participants) met the inclusion criteria for this review. 13 studies (770 participants) were included in meta-analyses (417 traumatic brain injury, 304 strokes, 49 other acquired brain injuries) reducing to 660 participants once non-included intervention groups were removed from three and four group studies.
- The authors identified insufficient high-quality evidence to reach any generalised conclusions about the effect of cognitive rehabilitation on executive function, or other secondary outcome measures.
5.8.4 Alternative Therapies for Attention, Memory, Executive Function

- Various alternative modes of intervention for cognitive rehabilitation has been tried and include acupuncture, TENS, rTMS, tDCS, virtual reality and simulation with inadequate evidence.
- These interventions require more research.

5.9 Cognitive Rehabilitation for Apraxia

5.9.1 Apraxia

- Disorder of voluntary movement where one cannot execute willed purposeful activity despite the presence of adequate mobility, strength, sensation, coordination, and comprehension.
- Occur in up to 30% of strokes in acute phase.
- Thought to results from loss of motor engrams or a disconnection between praxis system (Greene 2005).
- Anatomy correlations:
  - Left parietal or frontal premotor area are most commonly associated with apraxia.
  - Orofacial apraxia usually associated with insular and left inferior frontal lesion.
  - Other areas: right parietal lobe, temporal and even subcortical regions.
- While apraxia usually improves over time, spatiotemporal errors in imitation and tool use may persist.

5.9.2 Types of Apraxia

- Praxis requires:
  - Conceptual system for action- semantic knowledge of tool functions and actions (eg. the purpose of a screwdriver).
  - Production system for action - actual sensorimotor action programs needed to carry out tasks (e.g. ability to move a limb in the correct direction).
- Subdivisions of apraxia can be confusing. Many classifications on apraxia have been proposed. Traditional subdivisions of apraxia, particularly ideomotor and ideational apraxia are often used in an inconsistent manner (Greene 2005).

Classification of Apraxia (Mendez et al. 2012)

**Ideomotor Apraxia – Parietal Variant:** Disruption of movement formulas in the inferior parietal lobule with impaired pantomime, imitation and gesture recognition.

**Ideomotor Apraxia – Disconnection Variant:** Disruption of motor programs in the supplemental motor or interhemispheric connections with impaired pantomime, imitation of gestures and spatiotemporal errors. Movement formulas are preserved so patients are able to recognize and identify gestures.

**Dissociation Apraxia** – Errors when the movement is evoked by stimuli in one specific modality I.e., disconnection between lingual areas and movement formulas resulting in impaired pantomime but normal imitation of gestures or object use. Other types include verbal dissociation apraxia and visual dissociation apraxia.
Conceptual Apraxia – Errors in content of action, i.e., tool identification, use, object knowledge. Unable to point to name or identify a tool with its function is discussed or recall the actions associated with a specific tool or object.

Limb-Kinetic Apraxia – Inability to perform precise coordinated individual finger movements.

Ideational Apraxia – Can perform separate component tasks but cannot coordinate all steps for an integrated sequence.

Constructional and Dressing Apraxia – Not true apraxias. Usually involvement of right parietal lobe lesion impacting on visual spatial function.

5.9.3 Testing for Apraxia

- There are various methods to test for apraxia.
- It is important to test all input modalities.

Test to Measure Upper Limb Apraxia (TULIA)

- Test to Measure Upper Limb Apraxia (TULIA) is one method of determining upper limb apraxia through the qualitative and quantitative assessment of gesture production.
- In contrast to previous publications on apraxic assessment, the reliability and validity of TULIA was thoroughly investigated (Vanbellingen et al. 2010).
- The TULIA consists of subtests for the imitation and pantomime of non-symbolic (“put your index finger on top of your nose”), intransitive (“wave goodbye”) and transitive (“show me how to use a hammer”) gestures.
- Discrimination (differentiating between well- and poorly-performed tasks) and recognition (indicating which object corresponds to a pantomimed gesture) tasks are also often tested for a full apraxia evaluation (Vanbellingen et al. 2010).

Mendez et al. (2012) outlined the following tests:

Ideomotor intransitive actions: (Alternate sides)

- Pantomime: Ask to perform tasks like salute, wave goodbye, the peace sign.
- Imitation: Ask to copy examiners actions - examiner gestures salute, peace sign.
- Gesture Knowledge: Patient asked to identify the function or purpose of the action demonstrated.

Ideomotor transitive actions: (Alternate sides)

- Pantomime: Ask to perform tasks like comb hair, brush teeth, use hammer.
- Imitation: Ask to copy examiners action – examiner gestures combing hair, brushing teeth.
- Gesture Knowledge: Patient asked to identify the function or purpose of the action performed by the examiner.
- Conceptual Knowledge: Patient asked to identify, demonstrate use or explain use of a tool shown by the examiner.
**Ideational Apraxia: (Sequential Actions)**

- Pantomime: Examiner ask patient to show how they would prepare a letter for mailing, a sandwich with imaginary objects.
- This process of performing transitive and intransitive actions continues in assessment of trunk, Lower limb and orofacial apraxia.

### 5.9.4 Treatment of Apraxia

- There is **strong** evidence that strategic training (compensatory strategies) is effective in treatment of apraxias post-stroke.
- Not an area where a great deal of research has been done.

**STUDY**


- 113 patients with apraxia due to LH stroke randomly assigned to strategy training (involving use of compensatory strategies) integrated into usual OT vs usual OT.
- At 8 weeks treatment group improved more than controls on ADLs and Barthel Index scores.
- At 20 weeks no differences noted between groups.

### 5.10 Rehabilitation for Perceptual Disorders Post-Stroke

- Titus et al. (1991) defined perceptual performance as “the ability to organize, process, and interpret incoming visual information, tactile-kinesthetic information, or both, and to act appropriately on the basis of the information received”.

#### 5.10.1 Unilateral Spatial Neglect

- Defined as a failure to report, respond, or orient to sensory stimuli presented to the side contralateral to the stroke lesion.
- USN is found in about 23% of stroke patients.
- More common in patients with Right sided lesions (42%) than Left sided lesions (8%) and is more persistent with Right sided strokes.
- Recovery of USN common; most recovery occurs in 1st 6 months and later recovery less common
- USN associated with negative prognosis for functional outcome, poorer mobility, longer LOS in rehab, and slower rates of improvement; tend to be more functionally disabled at discharge (Wee & Hopman 2008).
- Unilateral spatial neglect can be classified as **egocentric or allocentric**:

  **a) Egocentric neglect:** Neglect of the body or personal space, tendency to neglect the opposite side of the lesion, in reference to the midline the body.
b) **Allocentric neglect**: Can be peripersonal or extrapersonal.
- Peripersonal space refers to space within the patient’s normal reach.
- Extrapersonal refers to object/environment beyond the patient’s normal reach.
- In allocentric neglect, the neglect is to the contralesional side of each object/environment in the peri/extrapersonal space.

- More obvious forms of neglect involve colliding with environment on involved side, ignoring food on one side of plate, and attending to only one side of body.
- More subtle forms are more common, more apparent during high levels of activity such as driving, work, or interacting with others.
- Milder neglect involves various degrees of ignoring the affected side when faced with stimulation on the unaffected side (extinction).

### 5.10.2 Why is Left Sided Neglect More Common than Right Sided Neglect?

- Neuroanatomical studies found that the left hemisphere is responsible for modulating attention and arousal for the right visual field only the right hemisphere is responsible for modulating attention and arousal in both the right and left hemispheres.
- With left hemispheric stroke, the right hemisphere is able to compensate resulting in a lower incidence of neglect. With a right hemispheric stroke, the left hemisphere is not able to fully compensate resulting in neglect.

**Figure. Regulation of Attention by Cerebral Hemispheres**

![Image of cerebral hemispheres showing normal visual field, left sided neglect, and no neglect with different lesion locations.](www.ebrsr.com)
5.10.3 Common Screening and Assessment Tests for Unilateral Neglect

- Screening and assessment tests for neglect can be performed via pen and paper test and by observation of behavioural/activity, or a combination of both.
- It is important to note that a single test may detect a specific type of neglect, thus a battery of tests are often more sensitive that single test.

a) Pen and Paper Tests

Line Bisection Test
- Asked to find the midline on a number of horizontal lines

<table>
<thead>
<tr>
<th>Questions</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>What does it measure?</strong></td>
<td>Designed to detect the presence of unilateral spatial neglect.</td>
</tr>
<tr>
<td><strong>What is the scale?</strong></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><strong>What are the key scores?</strong></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 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.</td>
</tr>
<tr>
<td><strong>What are its strengths?</strong></td>
<td>Simple and inexpensive measure of USN.</td>
</tr>
<tr>
<td><strong>What are its limitations?</strong></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>
Cancellation Test

- Several versions of cancellation tests are available.
- Requires patient to cancel/mark target items printed on a paper placed directly in front of them.
- Cancellation can either be: 1) single target items (no distractors): Line cancellation/crossing test; 2) target item with distractors: Bells Test, star cancellation, alphabet.

b) Behaviour/Activity Observation

Comb and Razor Test

- The Comb and Razor Test screens for unilateral spatial neglect (USN) in the client's personal space by assessing their performance in functional activities, such as using a comb or applying makeup.
- Catherine Bergego Scale involves direct observation of spontaneous (i.e., self-initiated) behaviors in 10 everyday activities.

c) Combination of Pen and Paper Test with Observation of Behaviors/Activity

Behavioural Inattention Test (BIT)

- Screens for unilateral visual neglect.
- Conventional testing (6 subtests) and 9 behavioral tests.
**Behavioural Inattention Test**

<table>
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<tr>
<th>Questions</th>
<th>Answer</th>
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</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 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).</td>
</tr>
<tr>
<td>What are its strengths?</td>
<td>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.</td>
</tr>
<tr>
<td>What are its limitations?</td>
<td>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.</td>
</tr>
</tbody>
</table>

**Clock-drawing Test for Visual Neglect**

- 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 rehabilitation settings such as visual neglect, hemiparesis and motor dyscoordination (Ruchinskas & Curyto, 2003)
- The reported sensitivity of the CDT when used to detect neglect 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 line bisection.
- Although visual neglect can be apparent on Clock Drawing Test, it is not sensitive in diagnosing visual neglect and is influenced by a number of other cognitive problems such as executive function.
5.10.4 Treatments of Spatial Neglect

Treatments of neglect can be divided into a compensatory or remedial approach:

**Remedial**

- Remedial training aims for direct restoration of function.
- It focuses on training the patient to voluntarily compensate for their deficits.
- Require full cooperation from the patient, patient must be aware of the deficit.
- Top down focus on the level of disability, and not impairment.
- Example is visual scanning or visual/mental imagery.

**Compensatory Approach**

- Compensatory approaches involve adapting the external environment.
- Bottom-up focus to increase a patient’s perception of space.
- Commonly uses sensory stimulation.
- Does not require the patient to be aware of the deficit.
- Focus on the level of impairment, e.g. altered perception.
- Examples:
  - Prisms adaptation
  - Limb activation therapy
  - Feedback training
  - Neck muscle vibration
  - Trunk rotation
  - Eyepatching and Hemispatial Glasses
  - Caloric Stimulation
  - Optokinetic Stimulation
  - TENS and Neck Vibration

**Remedial Treatments in Unilateral Spatial Neglect**

a) **Visual Scanning**

- Patients with neglect often don’t visually scan their whole environment, thus paying no attention to their left-sided space.
- Visual scanning involves teaching patients to look to left side in a consistent manner.
- **Strong** evidence that treatment utilizing enhanced visual scanning techniques improves visual neglect with associated improvements in function (Weinberg et al. 1977, 1979).
STUDY
- 53 RH stroke patients >4 weeks post onset randomly assigned to either 20 hours/4 weeks of specific neglect training (NT) vs 20 hours OT/PT over 4 weeks.
- 53 RH stroke patients >4 weeks post onset randomly assigned to either 20 hours/4 weeks of specific neglect training (NT) vs 20 hours OT/PT over 4 weeks.
- Severe NT group improved on 24 of 26 psychology test scores; Mild NT > control on 3 of 26 scores while severe NT > control on 15 of 26 scores.

STUDY
- 59 right hemispheric stroke patients onset 2-6 months randomized to neglect Rx 5 hours/week x 8 weeks and then general cognitive Rx 3 hours/week x 8 weeks in cross-over design.
- Improvement noted in both immediate and delayed treatment group.

b) Computer-Based Scanning in Neglect
- Computer versions of tasks associated with visual scanning have been developed and their use evaluated.
- Computer training offers a means to supplement costly therapy with massed practice.
- Moderate evidence (based on 1 RCT) computer-based visual scanning does not remediate visual neglect.

c) Virtual Reality Therapy for Neglect
- Several studies of virtual reality – promising but to date has been very expensive.
- Nintendo Wii and other gaming systems offers a potentially cheap alternative and is gaining in popularity on rehab units.
- Limited evidence virtual reality training may help to improve awareness of neglected space.
Compensatory Approach in Unilateral Spatial Neglect

a) Prisms Adaptation for Neglect

- Prisms affect spatial representation by causing an optical deviation of the visual field.
- Prisms tend to shift the visual field input and increase visual fields by 5-10 degrees.
- Outside of visual field when looking straight ahead but when gaze is shifted to the affected side increases visual field.
- Strong evidence prism treatment associated with increases in visual perception scores in stroke patients with homonymous hemianopsia and visual neglect; however, it was not associated with improvement in ADL scores (Rossi et al. 1990).

STUDY

- 39 stroke patients with homonymous hemianopsia or unilateral spatial neglect randomly assigned to receive 4 weeks of treatment with Fresnel prisms or to control group.
- At baseline, both groups had similar MVPT response behavioural scores in the affected visual field (53.2 vs. 47.7).
- By the end of 4 weeks, experimental group improved significantly (p<0.01) relative to baseline and control group.
- Patients in the prism group also made significant improvements on the Line Bisection Test, the Line Cancellation Task, the Harrington Flocks Visual Field Screener, and the Tangent Screen Examination.

b) Limb Activation Strategies

- Intended to increase orientation and attention to neglected hemi-space.
- A motor or externally-applied sensory stimulus to the affected side attempts to “activate” the right hemisphere.
- Includes limb activation (better studied) as well as application of a sensory stimulus (lesser studied).
- Strong evidence that limb activation therapies improve neglect (Robertson et al. 2002).
- However, little information available with regard to duration of effect or effect of treatment on functional ability.

STUDY

- 50 stroke patients with visual neglect were randomized to spatiomotor cueing during motor activity (integrating attentional and motor functions using limb activation approach) vs. conventional therapy (aimed at restoring normal tone, movement patterns and motor activity).
- Intervention group had significantly shorter length of hospital stay (42 vs. 66 days, p=0.001), less time in physiotherapy and improved neglect scores at 12 weeks.
STUDY

- 40 right hemispheric stroke patients randomly allocated to perceptual training (PT) (puzzles requiring scanning) or limb activation with PT (LA+PT) (added alarm when Lt arm movement absent over set time).
- Improvement up to 24 months in LA+PT with little improvement in PT group.

c) Sensory Feedback Strategies for Neglect

- Feedback strategies are intended to improve awareness and attention to neglected space
- Include auditory and visual feedback.
- Make patient aware of his/her neglect behaviors and may assist in learning ways to remediate neglect.
- **Strong** evidence feedback strategies are beneficial in treatment of neglect.
- More study required to establish the degree to which treatment effects generalize to other behaviors and to determine the durability of effect.

d) Eye Patching and Hemispatial Glasses

- Eye patching of the eye ipsilateral to lesion (right eye for left neglect) causes the patient to attend more to the unpatched side.
- **Moderate** evidence that monocular, opaque patching to improve neglect produces inconsistent results (Beis et al. 1999; Walker et al. 1996).
- **Moderate** evidence that bilateral half-field eye patches improves visual neglect and functional ability (Ianes et al. 2012; Tsang et al. 2009; Zeloni et al. 2002).

e) Other Treatments

- Caloric, vestibular, and optokinetic stimulation provide either no improvement or temporary improvements of neglect and are not current treatments.
- Limited evidence that trunk rotation to the side affected by neglect helps to compensate and aid for visual scanning.
- Limited evidence somatosensory stimulation (vibration) therapy to left posterior neck muscles reduces left neglect.
- Limited evidence TENS treatments improve neglect post-stroke.
- Limited evidence repetitive transcranial magnetic stimulation may improve spatial neglect.
5.11 Rehabilitation for Aphasia

5.11.1 Definition of Aphasia

- The loss of ability to communicate orally, through signs, or in writing, or the inability to understand such communications.
- Impairment of language as a result of focal brain damage to the language dominant hemisphere.
- Expression is most often affected.

5.11.2 Aphasia: Relation to Handedness

- 93% of the population is right hand dominant.
- Right hand dominant individuals – 99% use left hemisphere for language.
- Left hand dominant individuals – 70% use left hemisphere, 15% use right hemisphere and 15% use both hemispheres for language function.

5.11.3 Classification of Aphasia

<table>
<thead>
<tr>
<th></th>
<th>Expression or Fluency**</th>
<th>Comprehension* or Understanding</th>
</tr>
</thead>
<tbody>
<tr>
<td>Broca’s (motor or expressive)</td>
<td>Poor</td>
<td>Good</td>
</tr>
<tr>
<td>Wernicke’s (receptive)</td>
<td>Good</td>
<td>Poor</td>
</tr>
<tr>
<td>Global</td>
<td>Poor</td>
<td>Poor</td>
</tr>
</tbody>
</table>

* Includes reading
** Includes writing

Flowchart:

1. Fluent?
   - No
     - Comprehension?
       - Good
         - Repetition?
           - Good
             - Transcortical Motor
           - Poor
             - Broca’s
       - Poor
         - Mixed Transcortical
2. Yes
   - Comprehension?
     - Good
       - Repetition?
         - Yes
           - Wernicke’s
         - No
           - Conduction
     - Poor
       - Transcortical Sensory
**Paraphasias**

- 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 from same semantic class (e.g., “fork” for “spoon” or “pen” for “pencil”).

**Broca’s Aphasia**

- Motor aphasia.
- Problems with verbal output; understanding remains intact.
- Nonfluent, hesitant, labored, and paraphasic; speaking vocabulary and confrontation naming is severely impaired.
- Writing is similarly affected.
- 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.

**Anomic Aphasia**

- Mild motor aphasia.
- Problem with verbal output; understanding remains intact.
- Word-finding difficulties or mild articulatory errors (often called verbal apraxia).

**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.
Wernicke’s Aphasia

- Sensory aphasia.
- Problem with input or understanding of language.
- Fluent speech with severe comprehension deficit, poor repetition and often unintelligible jargon; reading is similarly affected.
- Posterior part of superior (first) temporal gyrus stroke characterized by fluent speech but poor comprehension and poor repetition.
- Associated with marked paraphasias and neologisms.

Wernicke’s Area

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).
Conduction Aphasia

- Stroke of the parietal operculum (arcuate fasciculus) or insula or deep to the supramarginal gyrus characterized by disproportional impairment in repeating spoken language.
- Literal paraphasias with “targeting” of words (until getting the right one).
Global Aphasia

- Motor and sensory aphasia.
- Problem with input (understanding of language) and output (verbal and writing).
- No communication even with gestures and no speech or only stereotypical repetitive utterances.
- Reading and writing affected.
- Often not good rehabilitation candidates because of difficulty with understanding.
- Generally involve the entire MCA region with moderate to severe impairment of language of all language function.

5.11.4 Therapy of Aphasia Post-Stroke

- Many studies of speech and language therapy post-stroke.
- Robey (1998), in a meta-analysis, found SLT had a significant impact acutely and a lesser but still significant impact chronically.
- However, many of the studies were of poor quality – small samples and not randomized trials.

Non-Pharmacological Interventions for Aphasia

a) Intensity of SLT on Aphasia

- Bhogal et al. (2003) identified 8 RCTs comparing intensity of SLT delivered by trained therapist vs. non-SLT control.
- 4 RCTs showed a positive impact of speech and language therapy and 4 did not. The association of therapy intensity and therapy effectiveness was examined.
- Positive RCTs provided a mean of 8.8 hours per week for an average of 11.2 weeks; Total therapy hours were 98.4 hours.
- Negative trials provided a mean of 2 hours per week for an average of 22.9 weeks; Total therapy hours were 43.6 hours.
- More intensive therapy over a shorter period of time efficacious.
c) **Trained Volunteers in Aphasia Training**

- 5 RCTs have looked at trained volunteers providing the SLT and have found they can achieve similar outcomes.
- Many serve as an important supplement to scarce SLT resources.

**STUDY**

- 121 aphasic males 2-12 months post onset.
- Randomized to home therapy treatment given by a wife, friend or relative, treatment by SLP or treatment by SLP deferred for 12 weeks.
- Therapy provided 8-10 weeks x 12 weeks.
- At 12 weeks SLP Rx better than deferred but not significantly different from home therapy.
- Deferred group caught up after 12 more weeks.
- Trained volunteers can provide an effective adjunct to SLP treatment.

d) **Group Therapy for Aphasia Post-Stroke**

- Group therapy for aphasic patients is a potential means to maximize limited language resources and thus encourage social interactions (Wertz et al. 1981; Elman & Bernstein-Ellis 1999).

**STUDY**

- 67 male aphasic stroke patients, 4 weeks post-onset.
- Randomized to 4 hours/week of individualized SLT vs. 4 hours/week of group therapy.
- Patients who received individualized therapy did better with writing.

**STUDY**

- 24 chronic aphasics were randomized to 4 months of 5 hours per week group therapy versus control.
- Patients in treatment group did significantly better at 2 and 4 months.

- Moderate evidence (based on 1 RCT of fair quality) that group aphasia therapy results in improvement of chronic aphasia.
• Moderate evidence (based on 1 RCT of good quality) that group therapy results in less improvement in graphic (writing) elements of aphasia when compared to individualized therapy.
• Limited evidence participation in group therapy results in improved communication.

d) Training Conversation / Communication Partners

• Conversation is important in social participation and plays an important role in many social functions such as establishing and maintaining relationships, sharing ideas and opinions, and making plans.
• Training conversation or communication partners within the aphasic’s social setting can promote opportunities for restored access to conversation.
• A systematic review by Simmons-Mackie et al. (2010) noted that communication partner training is effective in improving communication activities and/or participation of the communication partner. Communication partner training is also probably effective in improving communication activities and/or participation of persons with chronic aphasia when they are interacting with trained communication partners.

STUDY
• York-Durham Aphasia Center’s community-based program.
• 35 patients and 12 family members showed significant improvements in measures of well-being over course of treatment.
• Suggests that community-based aphasia therapy improves well-being of patients and families.

STUDY
• 40 volunteers randomly assigned to supported conversation training vs video regarding stories of aphasics and their families then assigned to stroke patients with moderate to severe aphasia.
• SCA trained volunteers associated with enhanced conversation skills for both trained partner and aphasic individual (moderate evidence).
• Limited evidence SCA trained conversation partners may result in improved access to conversation and increased social participation.

e) Computer-Based Treatment

• Computer-based aphasia therapy is appealing as a means of massed practice increasing intensity of therapy (Doesborgh et al. 2004; Katz & Wertz 1997).
STUDY
- 18 aphasic patients post stroke received intensive impairment-based interventions.
- Randomly assigned to 10-11 hours treatment with computer program vs. no treatment.
- Improvement in impairment-based language skills but not improvement in functional communication skills.

STUDY
- RCT examined 55 aphasic patients at least one year post-stroke onset.
- Received 3 hours/week x 6 months of computer reading, computer stimulation or control.
- Computer reading > stimulation > control.

f) Constraint-Induced (CI) Aphasia Therapy
- Chronic aphasic patients use communication channels that are most accessible and which require the least amount of effort, such as gesturing and drawing, or using communicative utterances that they know they can produce with ease.

Constraint Induced aphasia therapy is based on 3 principles:

1. Intensive practice for short intervals.
2. Constraints are used to force the patient to perform actions that are being avoided.
3. Therapy focuses on action relevant to everyday life.

STUDY
- RCT of 17 patients compared CI therapy 3 hours per day x 2 weeks vs. conventional therapy 3 hours per day x 4 weeks.
- CI patients showed greater improvement.
- Moderate evidence forced-use aphasia therapy results in greater language performance in chronic aphasics over short period of time.
Pharmacological Intervention for Aphasia Recovery

- **Piracetam** (a drug not available in Canada) in 4 RCTs has significant impact on aphasia recovery.
- **Bromocriptine** in 2 RCTs does not have a significant impact on aphasia therapy.
- **Amphetamines** in 1 small RCT (Walker-Batson et al. 2001) improved aphasia recovery when combined with language therapy.
- **Donepezil** in 1 RCT has a positive effect on global language function but only during active treatment and may not extend to everyday communication.

5.11.5 Efficacy of Aphasia Therapy

Aphasia therapy is efficacious:

1. When sufficiently intensive.
2. Provided by SLPs or trained volunteers.
3. Perhaps in a group setting.
4. Perhaps with a CI approach.
5. Perhaps with computer-based programs.
6. Perhaps when combined with amphetamines.
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