CHAPTER 7
Mood and Cognition
In Patients Following Stroke
(UPDATE March 2013)

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on Behalf of the Stroke Mood and Cognition
Best Practices Writing Group 2013
# Canadian Best Practice Recommendations for Stroke Care

## Mood and Cognition in Stroke ~ Fourth Edition
(Updated March 2013)

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**CANADIAN BEST PRACTICE RECOMMENDATIONS FOR STROKE CARE**

The Canadian Best Practice Recommendations for Stroke Care are intended to provide up-to-date evidence-based guidelines for the prevention and management of stroke. The goal of disseminating and implementing these recommendations is to reduce practice variations in the care of stroke patients across Canada, and to reduce the gap between knowledge and practice. Recommendations are updated every two years to ensure they continue to reflect contemporary stroke research evidence and leading expert opinion. Each update involves critical review of the current medical and health-related literature, which informs decisions regarding modification of the recommendations and the performance measures used to assess their impact. Every attempt is made to coordinate with other Canadian groups who are developing guidelines that relate to stroke, such as dementia, hypertension, atrial fibrillation and diabetes.

This is the fourth edition of the Canadian Best Practice Recommendations for Stroke Care, which was first released in 2006. The theme of the 2012 – 2013 update is **TAKING ACTION**, and stresses the critical role and responsibility of healthcare providers at every stage of the continuum of care to ensure that best practice recommendations are implemented and adhered to. **TAKING ACTION** will lead to optimal outcomes for each stroke patient by providing the best care within the most appropriate setting. This includes rapid and efficient access to diagnostic services, stroke expertise and medical and surgical interventions, rehabilitation and support for ongoing recovery and community reintegration.

**TAKING ACTION** requires a committed team approach and strong coordination of care across regions and networks, with pre-hospital, acute care, rehabilitation and community-based healthcare providers working together to ensure optimal outcomes for patients and their families, regardless of geographic location.

**TAKING ACTION** applies to patients who have experienced a stroke, their families and informal caregivers. Stroke patients and their families will benefit from active participation in recovery and open communication with their healthcare team. Patients and families are strongly encouraged to participate with the healthcare team in setting the goals they want to achieve during recovery from a stroke. It is important for patients and families to share concerns regarding physical, mood and cognitive issues with their team, which will optimize the care required for recovery in all aspects of health.

**SECTION 2.0  MOOD AND COGNITION IN STROKE OVERVIEW**

**TAKING ACTION** for the management of mood and cognition following stroke is critical. The occurrence of post-stroke depression and changes to cognition (vascular cognitive impairment), even if subtle, affect up to 30% to 60% of patients who have experienced a stroke within the first year after onset. Of equal concern is the large number of family members and informal caregivers who also may experience depressive symptoms in the post-stroke recovery phase. The first steps for healthcare professionals in **TAKING ACTION** for mood and cognition are to understand the frequency of occurrence and build screening for the symptoms of depression and vascular cognitive impairment into regular workflows. Screening should occur through all stages and settings following a stroke, including in acute care, rehabilitation, prevention clinics and outpatient community settings (including primary care, home care and long-term care).

**TAKING ACTION** to address mood and cognition issues involves healthcare providers, policy makers, patients, and caregivers. An essential component of **TAKING ACTION** for mood and cognition issues is to have access to specially trained providers with expertise in mental health and cognition. Ideally, when screening is suggestive of a mood or cognition issue, patients and families should be referred to these clinical experts without delay to facilitate access to appropriate in-depth assessment and management, and to receive support and education for coping and self-management.

Recent reports on the quality of stroke services across Canada and within specific provinces have shown that there is inconsistent screening and monitoring of patients for post-stroke depression and vascular cognitive functioning issues, in both urban and rural settings. Delays in comprehensive assessment and management of mood and cognition issues may result in poor outcomes and slower recovery.
**Highlights of the 2013 Update to the Management of Mood and Cognition Following Stroke**

The 2013 update of the Mood and Cognition Chapter of the Canadian Best Practice Recommendations for Stroke Care reinforces the growing and changing body of research evidence available to better understand the mechanisms involved in post stroke depression and vascular cognitive impairment as well as optimal management. Active screening throughout the continuum of care is emphasized throughout this chapter.

The recommendations and evidence presented in this chapter are applicable to most adults who experience a stroke. This is because the current research evidence is strongest for patients between 40 years and 80 years of age. Given the current state of the evidence, care of each patient with stroke should be individualized to meet the needs of that patient, and therefore some modifications of the recommendations may be required in certain patient populations. These groups may include children with stroke (age newborn to 18 years), young adults who have experienced a stroke (age 19 to 44 years), and the elderly stroke population (over the age of 80 years). The evidence to support unique management needs for these groups is just starting to emerge. Where possible and appropriate, additional recommendations and comments have been included within the recommendations, and supporting sections to address specific considerations for these groups.

Highlights of the 2013 updates to the recommendations for the management of mood and cognition following stroke include:

- Increased guidance on management options for post stroke depression
- Revised definitions for vascular cognitive impairment
- Updates on medication use in vascular cognitive impairment

**Management of Mood and Cognition, 2013 Update Resource Package Includes:**

- Mood and Cognition Stroke Best Practice Recommendations
- Prevention of Stroke Educational Slide Decks
- Tables of Screening and Outcome Measurement Tools for Depression and Vascular Cognitive Impairment
- Tables describing the characteristics of commonly used medications for post stroke depression and vascular cognitive impairment
DEVELOPMENT OF THE CANADIAN BEST PRACTICE RECOMMENDATIONS FOR STROKE CARE

For detailed methodology on the development and dissemination of the Canadian Best Practice Recommendations for Stroke Care please refer to the stroke best practices website at http://www.strokebestpractices.ca/index.php/methods/.

Acknowledgements
The Canadian Stroke Network and the Heart and Stroke Foundation gratefully acknowledges the writing group leader Dr. Gail Eskes and all group members, the external reviewers, all of who volunteered their time and expertise to this project. This chapter was developed in collaboration with the Evidence Based Review in Stroke Rehabilitation group, including Dr. Robert Teasel, Katherine Salter, Norrine Foley and Andrew McClure, and their efforts are greatly appreciated. We thank the Canadian Stroke Performance and Quality Committee for their work in updating and confirming the performance measures that accompany each recommendation. We acknowledge Elena Goubanova and Corrine Davies-Schinkel for their work on meeting coordination and project support; and, we thank Marie-France Saint-Cyr and Jan Carbon for their work on the French translations.

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Citing the Management of Mood and Cognition in Stroke Update 2013


Comments
We invite comments, suggestions, and inquiries on the development and application of the Canadian Best Practice Recommendations for Stroke Care and ongoing updates.

Please forward comments to the Canadian Stroke Network’s Performance and Standards office at bestpractices@canadianstrokcnetwork.ca
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# Canadian Best Practice Recommendations for Stroke Care

## Section 7: Mood and Cognition in Stroke

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### Best Practice Recommendation 7.1
### Identification and Management of Post-Stroke Depression (PSD)

All patients with stroke should be considered to be at high risk for post-stroke depression (PSD), which can occur at any stage of recovery.

*Common risk factors associated with PSD include increasing stroke severity, functional dependence, presence of cognitive impairment, and history of previous depression. Increased functional dependence (e.g., requiring help with activities of daily living) and having a history of pre-stroke depression may be the two most salient risk factors for the development of PSD.*

#### 7.1.1 Screening for Post stroke Depression:

- **i.** All patients with stroke should be screened for depressive symptoms using a validated tool [Evidence Level A]. Refer to reference table 7.1 A for some suggested tools. (Table 7.1A)
- **ii.** Screening should also include evaluation of risk factors for depression, particularly a history of depression. [Evidence Level C]
- **iii.** Screening should take place at various stages throughout the continuum of stroke care [Evidence Level C]. Stages of care may include:
  - a. during acute care stay, particularly if evidence of depression or mood changes are noted;
  - b. following hospital discharge from the emergency department or inpatient setting to an outpatient or community-based healthcare setting;
  - c. throughout rehabilitation within inpatient, outpatient, and home-based settings, according to client progress;
  - d. periodically, following discharge to the community, during follow-up appointments and/or during periodic health assessments with primary care practitioners and consulting specialists.

#### 7.1.2 Assessment for Post stroke Depression:

- **i.** Patients identified as being at risk for depression during screening should be managed by a healthcare professional with expertise in diagnosis and management of depression in stroke patients. If required, a referral should be made to an appropriate mental health specialist (e.g., psychiatrist or psychologist) [Evidence Level C].
- **ii.** Further assessment by the mental healthcare professional may include:
  - a. More in-depth interview for the purpose of assessment and diagnosis based on accepted diagnostic criteria (e.g., Diagnostic and Statistical Manual of Mental Disorders) [Evidence Level B];
  - b. Population-specific assessment measures (e.g., children, elderly, persons with co-morbid neuropsychiatric conditions) [Evidence Level C];
  - c. Determination of appropriate course of treatment and individualized management plan;
  - d. Post-treatment assessment and follow-up as needed.

#### 7.1.3 Treatment and Management Modalities:

**A. Pharmacotherapy**

- **i.** Patients with mild depressive symptoms or those diagnosed with minor depression may initially be managed by “watchful waiting”* (Evidence Level B).
  - Pharmacological treatment should be considered/started if the depression is persistent and interferes with clinical goals, or worsens [Evidence Level B].
- **ii.** Patients diagnosed with a depressive disorder following formal assessment should be considered for a trial of antidepressant medication [Evidence Level A].
- **iii.** No one drug class has been found to be superior for PSD treatment. Side effect profiles, however, suggest that some
selective serotonin reuptake inhibitors may be favoured in this patient population. [Evidence Level A]. Please refer to Pharmacology profile tables (hyperlink)

- Choice of an antidepressant medication will depend upon symptoms of depression, potential known side effects of the medication, particularly in the child or older adult, drug interactions with other current medications and underlying disease conditions. (Hyperlink to Medications Table)

iv. Response to treatment should be monitored regularly by a health professional with expertise in mental healthcare. Monitoring should include evaluation of any changes in the severity of depression, review of potential side effects, and update of ongoing management plans [Evidence Level C].

v. If a good response is achieved, treatment should be continued for a minimum of six months before slowly withdrawing the antidepressant [Evidence Level C].

- Examples of a ‘good response’ may be indicated by positive changes in thoughts and self-perceptions (e.g., hopelessness, worthlessness, guilt), emotional symptoms (e.g., sadness, tearfulness), and improved motivation to carry out daily activities.

vi. Following initial treatment for PSD, patients should continue to be monitored for recurrence of depressive symptoms, as part of ongoing comprehensive stroke management [Evidence Level C]. The involvement and feedback of family and caregivers can be an important component of ongoing monitoring.

*Watchful waiting is defined as a period of time when the patient who displays mild depressive symptoms is monitored closely without additional therapeutic interventions to determine whether the mild depressive symptoms will improve. The timeframe for watchful waiting varies in the literature somewhere between 2 to 4 weeks. It is often described as including suggestions to the patient for self-help strategies and participation in exercise.

B. Non-Pharmacological and Adjunct Treatment

i. There is inadequate evidence at present to support the use of psychotherapy as monotherapy in the treatment of PSD [Evidence Level C].

ii. Treatment for PSD may also include psychotherapy as an adjunct in combination with antidepressants and/or longer-term option to prevent relapse. This approach, while supported by evidence in other populations, requires more research in stroke populations [Evidence Level C].

- Different options that have been explored in small studies have included cognitive behavioural therapy (CBT) and problem solving therapy, although the methodological details of the therapies have not been well described. These therapies could be considered where appropriate at the discretion of the mental health expert [Evidence Level C].

iii. Other approaches to adjunctive treatment of PSD that are emerging, but require more research, include other forms of Repetitive Transcranial Magnetic Stimulation (rTMs), CBT, physical exercise, and acupuncture [Evidence Level C].

C. Other Mood Symptoms (Anxiety)

i. Patients with marked anxiety should be offered psychotherapy [Evidence level B].

- Although evidence is limited in stroke patients, pharmacotherapy may be considered as an adjunct to psychotherapy [Evidence Level C].

D. Post Stroke Emotional Incontinence (PSEI)

i. In cases of severe, persistent or troublesome tearfulness, patients may be given a trial of antidepressant medication [Evidence Level A]. Side effect profiles suggest that some selective serotonin reuptake inhibitors may be preferred over others for this patient population. Please refer to Pharmacology profile Table 7.1B
### 7.1.4 Prevention of Post Stroke Depression

i. Although, emerging data on the use of pharmacotherapy as a preventive intervention for post stroke depression is encouraging, routine use of prophylactic antidepressants is not recommended in post-stroke patients, at this time [Evidence Level A].
   a. Further research is required to define at risk patients, choice of antidepressant agents, optimal timing and duration of intervention [Evidence Level A].

ii. Non-pharmacological, talk-based interventions including problem-solving therapy and motivational interviewing may be used to enhance rehabilitation and prevent depression post stroke [Evidence Level B].

iii. Engaging patients in activities such as exercise or music therapy may also have a beneficial effect on mood post-stroke [Evidence Level C].

### 7.1.5 Ongoing Monitoring, Support and Education

i. Patients should be given information and education about the potential impact of stroke on their mood and that of family and caregivers; patients and families should be provided with the opportunity to talk about the impact of stroke on their lives at all stages of care [Evidence level C]. *(Refer to Chapter 6 for more recommendations on patient education and caregiver support)*

ii. Patients and their caregivers should have their psychosocial and support needs assessed and reviewed on a regular basis (at least annually) as part of long-term stroke management [Evidence level C] by primary care practitioners and consulting specialists.

iii. For patients who experience some degree of communication challenge or deficits following stroke, appropriate strategies for screening of possible PSD should be implemented to ensure adequate assessment and access to appropriate treatment [Evidence Level C].

**Rationale:**

Approximately one-third of all individuals who experience stroke will exhibit symptoms of depression at some time following the stroke event (acute, sub-acute and at long-term follow-up). The majority of cases of post-stroke depression (PSD) may develop in the first 3 months of stroke, and incidence rates of depression tend to decline over time, although research reports have indicated symptoms have emerged up to two years after index stroke. Post-stroke depression (PSD) also was reported in 48% of 71 young stroke patients after at least 1 year of follow-up. PSD may prove to be persistent for as many as one-half of the individuals identified as depressed soon after stroke. Severity of functional limitations, stroke severity, cognitive impairment and a previous history of depression have all been identified as important risk factors for the development of PSD.

PSD is associated with poorer functional recovery, increased risk for dependence, poorer cognitive function and reduction in social participation. In addition, the presence of PSD has been associated with increased risk for mortality. Appropriate identification, diagnosis and treatment of PSD have been associated with improved outcomes.

Families and caregivers of patients who experience a stroke are also at risk for depression, with the reported incidence as high as 30% to 60% of caregivers experiencing depressive symptoms.

**System Implications**

- Education for primary care practitioners and healthcare providers across the continuum of stroke care on recognition, assessment, and management of post-stroke depression.
- Timely access to appropriate mental health specialists as needed who are able to diagnose and evaluate severity of depression, and provide guidance for ongoing management.
- Timely access to specialized therapies to manage post-stroke depression, including medication, counseling and psychotherapy as required.
- Process for ongoing monitoring of any patient with positive screening for depression during screening and assessment process.
- Mechanisms to involve and support caregivers of stroke survivors. Processes should be in place to provide education and ensure that the caregivers' emotional needs are monitored and addressed, ideally through involvement of the primary
Optimization of strategies to prevent the recurrence of stroke.

**Performance Measures:**

1. Proportion of acute stroke patients with documentation indicating initial screening for post-stroke depression was performed (either informally or using a formal screening tool) in the acute care, rehabilitation, long-term care and community settings (e.g., homecare) setting. (Core Indicator)

2. Proportion of acute stroke patients referred for additional assessment or intervention for a suspected diagnosis of depression.

3. Proportion of stroke patients diagnosed with PSD and treated with antidepressants at one month, three months, six months, and one year following the initial stroke event.

**Measurement Notes**

- Recommendation 7.1 and corresponding performance measures apply across the continuum of stroke care and should be considered in the acute, early rehabilitation and longer-term recovery phases and in all healthcare settings.

- When monitoring these performance measures it is important to record when and in what context (continuum of care) the measurement were conducted.

- Data for measurement may be found through primary chart audit. Data quality will be dependent on the quality of documentation by healthcare professionals.

- For patients referred to psychiatry, information may be available through provincial physician billing databases.

- For persons over 65 years old, information on medication prescriptions may be available through provincial and territorial senior drug benefit plan databases.

- For performance measure 3, the issue is not just increasing the use of antidepressants but increasing the number of patients with PSD who are adequately treated, and reducing the number of patients with depression who are untreated (depressive disorder + no antidepressant med) and undertreated (depressive symptoms + antidepressant med). This should be considered in the measurement and analysis plan.

**Implementation Resources and Knowledge Transfer Tools**

- Table 7.1A: Selected Depression Screening Tools
- Table 7.1B: Summary Table for Selected Pharmacotherapy for Post-Stroke Depression
- Stroke Best Practices Teaching Slides: Mood and Cognition Following Stroke
- StrokEngine [http://strokengine.ca/](http://strokengine.ca/)
- HSF 10 warning signs of depression: [http://www.heartandstroke.com/site/c.ikIQLcMWJtE/b.5482531/k.6ACA/Stroke__Mental_changes.htm](http://www.heartandstroke.com/site/c.ikIQLcMWJtE/b.5482531/k.6ACA/Stroke__Mental_changes.htm)
- The Heart & Stroke Living with Stroke™ program [http://www.heartandstroke.on.ca/site/c.pvl3leNWJwE/b.7936265/k.94AE/Living_with_Stroke__Ontario_Locations.htm](http://www.heartandstroke.on.ca/site/c.pvl3leNWJwE/b.7936265/k.94AE/Living_with_Stroke__Ontario_Locations.htm)
- Let's Talk about Stroke [http://www.heartandstroke.com/site/c.ikIQLcMWJtE/b.3882223/k.3FC6/Stroke__Lets_Talk_about_Stroke.htm](http://www.heartandstroke.com/site/c.ikIQLcMWJtE/b.3882223/k.3FC6/Stroke__ Lets_Talk_about_Stroke.htm)
- Life after Stroke website: [http://www.lifeafterstroke.ca](http://www.lifeafterstroke.ca)
Although post-stroke depression (PSD) is a common consequence of stroke, risk factors for the development of PSD have not been clearly delineated. In a systematic review, Hackett and Anderson (2005) included data from a total of 21 studies. Of the many different variables assessed, physical disability, stroke severity and cognitive impairment were most consistently associated with depression. The authors noted that major methodological limitations within the available literature make it difficult to form a definitive conclusion. As part of the DESTRO study, a multicentre observational study of depression in stroke, Paolucci et al. (2005) identified female sex (OR=1.49), previous stroke (OR = 1.55), previous depression (OR=3.97) and severe disability (Modified Rankin Scale score >3, OR = 2.70) as factors likely to facilitate the development of depression following stroke. The risk for post-stroke depression was found to increase exponentially in individuals with more than one risk factor (Paolucci et al. 2005). In young adults with stroke, PSD risk factors are carotid localization of the infarct, severe disability, bad general outcome and absence of return to work (Neau et al., 1992). In a report from the Auckland Regional Community Stroke Study (Hackett and Anderson 2006), the authors described an attempt to create a simple, predictive tool for the identification of individuals most at risk for abnormal mood. Of the factors included in the model, only two were significant predictors of mood; prior treatment for depression (OR = 2.4, 95% CI 1.34 – 3.43) and requiring “much help” with activities of daily living (OR = 2.35, 95% CI 1.33 – 4.14). The ability of the model to predict risk for depression might be increased by the inclusion of other factors such as fatigue and performance of instrumental activities of daily living. However, Van de Port et al. (2007) demonstrated that use of these two predictors (prior treatment for depression and requiring much help with ADLs) in a multivariate model could correctly classify depression in 76% of patients 3 years post stroke.

Examination of the results of multiple meta-analyses (Mitchell and Kakkadasam 2011; Mitchell et al. 2011; Mitchell et al. 2010; Mitchell et al. 2009; Cepoiu et al. 2007) revealed that non-psychiatric physicians, nurses, and therapists demonstrate poor sensitivity relative to gold standard psychiatric interviews and standardized formal rating scales when using clinical observation to identify depression in individuals who have experienced stroke in both inpatient and community-based settings. Use of formal screening tools by direct comparison is associated with significantly greater sensitivity (Lowe et al. 2004). As in the recent CANMAT task force recommendations, it is noted that the use of formal instruments is a key component in the diagnostic process required to promote early detection of depression.

PSD in survivors of childhood stroke is not well studied but appears to be common based on preliminary evidence (Elbers, 2013). Depression and anxiety may also be increased in parents of children with stroke (Goodman 2000; Gordon, 2002).

In 2006, the Canadian Stroke Strategy and Heart and Stroke Foundation of Ontario supported a consensus process to identify a standardized basket of outcome assessment tools that could be used to across the stroke continuum of care. Included in the resulting basket of measures were the following screening tools for the identification of possible depression in individuals with stroke: The Hospital Anxiety and Depression Scale (HADS), the Geriatric Depression Scale (GDS) and the Stroke Aphasic Depression Questionnaire (SADQ-10). A brief description of each of these tools is provided in the accompanying table. Detailed reviews of the GDS, HADS and SAD-Q are available from www.ebrsr.com, www.strokengine.com, and/or in Salter et al. (2007).

Once diagnosed by a healthcare professional with appropriate expertise, the use of pharmacotherapy for the treatment of PSD has been associated with significant benefits in terms of reduction of depressive symptomatology (Chen et al. 2006, Hackett et al. 2008). However, these findings should be considered in light of reports of adverse effects associated with the use of antidepressant medications. While selective serotonin reuptake inhibitors are among the most commonly prescribed pharmacological agents for the treatment of PSD, they may also be associated with increased risk for mortality and stroke (Coupland et al. 2011, Wu et al. 2011). In addition, pharmacodynamic interactions between antidepressants and cardiovascular agents have been noted for several antidepressant classes including SSRIs, which may result in significant adverse events (Tuunainen et al. 2009).

Based on the results of available meta-analyses (Wilson et al. 2009, Hackett et al. 2008), there is insufficient evidence to support the use of talk-based, cognitive behavioural therapies (CBT) as monotherapy for the treatment of PSD. However, talk-based...
therapy, such as problem-solving therapy, when used in combination with pharmacotherapy may be an effective means to reduce symptoms of depression (Mitchell et al. 2009, Alexopoulos et al. 2012, Lincoln and Flanagan 2003). The use of other, non-pharmacological strategies, such as light therapy (Tuuainen et al. 2009, Sondergaard et al. 2006), physical exercise (Graven et al. 2011) and music therapy (Jun et al. 2012, Sarkamo et al. 2008), acupuncture (Zhang et al. 2010) have all been demonstrated to have a positive effect on mood within the population of individuals with stroke.

For individuals who, following screening and appropriate follow-up assessment, experience mild symptoms of depression, watchful waiting may be the most appropriate strategy. While there is no direct evidence of the effectiveness of this approach as an intervention within the stroke population, it has been used with good results as part of interventions, such as stepped care management (van’t Veer-Tazelaar et al. 2009, Dozeman et al. 2012), undertaken in populations of older individuals. A single randomized controlled trial examined the use of pharmacotherapy for individuals with mild depression and found a significant reduction in depressive symptomatology associated with the use of antidepressant therapy; however, study participants were not drawn from the stroke population (Williams et al. 2000).

Given the high prevalence of PSD and the negative consequences associated with it, there has been increasing attention paid to strategies for its prevention. The most commonly studied strategy has been universal pharmacologic prophylaxis, although discussions have expanded to include selected or indicated prophylaxis, problem-solving therapy (Robinson et al. 2008) and motivational interviewing (Watkins et al. 2007, 2011). Results of recent meta-analyses have provided encouraging results in favour of pharmacotherapy for the prevention of post-stroke depression (Salter et al. 2012, Yi et al. 2010, Chen et al. 2007); however, little is known about optimal timing and duration of intervention, or the best approach (e.g. universal, selected or indicated) to take in offering such a strategy.

**Post Stroke Emotional Incontinence (PSEI)**

Reported frequency of PSEI ranges from 11%-35% depending upon the criteria used to define the condition and time elapsed since stroke onset (House et al. 1989, Kim and Choi-Kwon 2000). While there has been an association reported between PSEI and PSD, more individuals with emotionalism do not have significant or diagnosable depression (Kim and Choi-Kwon 2000, Tang et al. 2004). In a recent Cochrane review (Hackett et al. 2010), pooled analyses were reported for data gathered from 5 randomized controlled trials examining antidepressant therapy for PSEI. Large treatment effects in terms of emotionalism, reduced tearfulness, clinical impressions of change and Lability Scale scores were reported in favour of antidepressant treatment when compared to control conditions. Given methodological limitations, the authors conclude that the existing literature is suggestive, but not definitive, evidence that pharmacotherapy is effective in the treatment of PSEI.

**Anxiety Following Stroke**

Anxiety following stroke occurs more often in women than in men. In the Perth Community Stroke Study, it was reported that 20% of women who experienced stroke developed symptoms of anxiety following the event while, in the same sample, only 9% of men experienced post-stroke anxiety (Burvill et al. 1995). However, individuals who experienced post-stroke anxiety, often reported having anxiety or depression at the time of the stroke event (Burvill et al. 1995). Individuals with generalized anxiety disorder (GAD) after stroke may often experience co-morbid depression. Castillo et al. (1995) reported that, in a sample of individuals with post-stroke GAD approximately 75% were also depressed. Despite the prevalence of post-stroke anxiety, very few studies have evaluated the effectiveness of potential treatments. A recent Cochrane review (Campbell Burton et al., 2011) identified only 2 randomised trials suggesting a positive effect associated with the provision of pharmacotherapy with or without the addition of psychotherapy. However, further research in this area is certainly indicated in this population.

Hyperlink to Evidence Tables:
1. Post-Stroke Depression Screening and Assessment
2. Post-Stroke Depression Non-Pharmacological Therapy
3. Post-Stroke Depression Pharmacological Therapy
### Table 7.1A  Summary of Select Screening Tools for Use in Post-Stroke Depression (PSD)

<table>
<thead>
<tr>
<th>Assessment Tool and Link</th>
<th># of Items</th>
<th>Response Format</th>
<th>Total Score</th>
<th>Stroke-specific reliability/validity</th>
<th>Interpretation of Scores*</th>
<th>Sensitivity/Specificity for PSD</th>
</tr>
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<tbody>
<tr>
<td><strong>Recommended First Line Tools</strong></td>
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<tr>
<td>Geriatric Depression Scale (GDS)</td>
<td>30</td>
<td>Self-report Yes/No responses</td>
<td>0-30</td>
<td><strong>Reliability:</strong> Though thoroughly evaluated in populations of elderly individuals, relatively little has been done specific to individuals with stroke. Agrell and Dehlin (1989) reported high internal consistency (α=0.90) as did Sivrioglu et al. (2009) (α=0.88). <strong>Concurrent Validity:</strong> Agrell and Dehlin (1989) reported good correlations between GDS scores and scores on self-report and observational depression assessment scales. <strong>Discriminative Validity:</strong> Sivrioglu et al. (2009) demonstrated significant differences in GDS scores between groups of depressed vs. non-depressed participants (p&lt;0.001).</td>
<td>Normal = 0 – 10, scores ≥11 indicate presence of depression; 11-20 = mild depression, 21-30 = moderate to severe depression (McDowell et al. 1996)</td>
<td>Many studies have examined the relative sensitivity and specificity of the GDS – most have reported sensitivity and specificity values &gt; 80% (Stiles and McGarrahan (1998). Within the stroke population, Johnson et al. (1995) using a cut-off of 10/11, Johnson et al. (1995) reported sensitivity = 85%, specificity = 66% and a misclassification rate of 29%. More recently, using DSM-IV-TR as the criterion for diagnosis, Sivrioglu et al. (2009) reported sensitivity = 69% &amp; specificity = 75% for using a cutoff point of 10/11, and sensitivity = 66% and specificity = 79% for a cut off of 11/12.</td>
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<tr>
<td>French</td>
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<tr>
<td>Hospital Anxiety and Depression Scale (HADS)</td>
<td>14 (2 x 7-item sub-scales)</td>
<td>Self-report Multiple choice response options graded on a 4 pt scale</td>
<td>0-42 (0-21 for each subscale)</td>
<td><strong>Reliability:</strong> Visser et al (1995) reported test retest reliability (0.87); reported internal consistency reliability for the depression portion of the HADS has been &gt;0.70 (Johnston et al. 2000, Aben et al. 2002); most recently Sagen et al (2009) reported α=0.83. <strong>Construct validity:</strong> Reported satisfactory on confirmatory factor analysis (Johnston et al. 2000). <strong>Discriminative validity:</strong> HADS-D and HADS-A scores obtained by stroke patients differed significantly from controls (p&lt;0.001) (Visser et al. 1995).</td>
<td>Scale authors recommended either 8/9 (high sensitivity) or 10/11 (high specificity) be used to identify the presence of depression using the depression subscale of the HADS (Zigmond and Sniaith 1983). Alternate cut-off points have been evaluated for the post stroke population.</td>
<td>Aben et al. (2002) reported sensitivity of 72.5% and specificity of 78.9% for the HADS-D, using a cut-off score of ≥7. For the total scale, using a cut-off of ≥11, sensitivity and specificity were 86.8% and 69.9% respectively. Johnson et al. (1995) used a cut-off of 4/5 for the HADS-D and demonstrated a sensitivity of 93% and specificity of 44% while O-Rourke et al. (1998) reported sensitivity of 80% and specificity of 79% using the same cut-off point as Aben et al. More recently, Sagen et al. (2009) reported sensitivity and specificity for the HADS-total (relative to the DSM-IV) of 90% and 83% (cut off of ≥11), 79% and 85% (cut off of ≥12) respectively. For the HADS-D, sensitivity = 79% and specificity = 82% (cut</td>
</tr>
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</table>
### Patient Health Questionnaire - 9 (PHQ-9)

**http://strokengine.ca/assess/module_phq9_intro-en.html**  
**http://www.phqscreensers.com/**  

| Patient Health Questionnaire - 9 (PHQ-9) | 9 | Multiple choice response options, 4pt scale | 0-27 | Reliability: Interrater reliability = 0.98, test re-test = 0.75 and internal consistency = 0.79 (de Man-van Ginkel et al. 2012). Concurrent validity: PHQ-9 was significantly correlated with GDS-15 scores (r=0.8, p<0.01) (de Man-van Ginkel et al. 2012). Scores ≥10 (sensitivity=80%, specificity=78%) for identification of PSD 6-8 weeks post stroke (deMan van Ginkel et al. 2012). | A single study evaluated the sensitivity and specificity of the PHQ-9 for both major depression and any depression against a structured clinical interview in a subgroup of outpatients with stroke who endorsed either 2 or more symptoms on the PHQ-9 or either of the PHQ-2 items at study baseline (Williams et al. 2005). The authors reported sensitivity of 91% and specificity of 89% for major depression as well as sensitivity of 78% and specificity of 96% for any depression associated with a cut-off score ≥10. These numbers may, however, have been influenced by the pre-screening (using items from the PHQ-9) and formal assessment of selected individuals only. De Man-vanGinkel et al. (2012) also reported the results of a validation study that evaluated the PHQ-9 against the results of a composite international diagnostic interview for the DSM-IV conducted with 164 individuals with stroke (outpatients approximately 6-8 weeks post stroke). Similar to Williams et al., the authors reported that the accuracy of the PHQ-9 was best using a cutoff of ≥10 with a sensitivity of 80% and specificity of 78%. Using the PHQ-9 in patients pre-screened with the PHQ-2 increased the accuracy of identification (sensitivity = 87%) (de man-van Ginkel et al. 2012). |

### Additional Tools for Consideration

| Beck Depression Inventory (BDI) | 21 | Self-report Multiple-choice response set graded for severity | 0-63 | Reliability: Aben et al. (2002) confirmed high internal consistency reliability of the BDI in a population of individuals with stroke. Outside of the stroke population estimates of internal consistency tend to exceed 0.80 (Beck et al. 1988) Predictive validity: BDI scores are predictive of functional recovery and Threshold for presence of depression = 10; 10 – 18 = mild depression, 19 – 29 = moderate depression, 30 – 63 = severe depression (Beck et al. 1988) | ROC analysis completed by Lincoln et al. (2003) suggests that the accepted cut-off point indicative of presence of depression might be too low – recommends 15/16 to optimize sensitivity; however specificity is reduced relative to the DSM-III-R. Aben et al. (2002) reported the standard cut-off points to be acceptable for used for individuals with stroke. |

**http://strokengine.ca/assess/module_bdi_intro-en.html**  
**http://www.pearsonassessments.com/HAIWEB/Cultures/en-us/Productdetail.htm?Pid=015s**
### Center for Epidemiological Studies Depression Scale (CES-D)

| http://cesd-r.com/ | 20 | Self-report 4-pt scale | 0-60 |

**Reliability:** Internal consistency reliability has been reported ranging from 0.64-0.86 (Agrell & Dehlin 1989, Toedter et al. 1995). Reported item-to-total correlations ranged from 0.39-0.75 (Shinar et al. 1986).

**Concurrent validity:** Results of the CES-D used to assess individuals with stroke have correlated significantly with results of other standardized self-report and observational depression assessment tools (Agrell and Dehlin 1989, Shinar et al. 1986, Parikh et al. 1988).

**Presence of depression = ≥16** (McDowell et al. 1996)

Using the suggested cut-off score, Shinar et al. and Parikh et al. reported sensitivity of 73% and 86%, and specificity of 100% and 90% respectively (relative to the DSM-III-R).

### Tools to Consider for Aphasic Patients

**Stroke Aphasic Depression Questionnaire-10 (SADQ-10)**

| http://strokengine.ca/assess/module_sadq_intro-en.html | http://www.nottingham.ac.uk/iwho/documents/sadq-h.pdf | 10 | Observer rating of observed behaviour 4-point scale | 30 |

**Reliability:** Using carers of individuals with aphasia to complete follow-up assessments, 4-week test-retest reliability was reported to be 0.69 for the SADQ-10 (Sutcliffe and Lincoln 1998). Internal consistency has been reported as $\alpha = 0.80$ (Sutcliffe and Lincoln 1998, Lincoln and Sutcliffe 2000).

**Construct validity:** Results of factor analysis suggested that the SADQ-10 items may be unidimensional (Sutcliffe and Lincoln 1998)

**Concurrent validity:** SADQ-10 scores have been positively associated with scores on the HADS-D, HADS-A, Wakefield Depression Inventory (Sutcliffe and Lincoln 1998), and the GDS-15 (Leeds et al. 2004), though correlations with healthcare professional ratings have varied (Lincoln and Sutcliffe 2000).

**Scores ≥15 may represent presence of depression** (Leeds et al. 2004).

Using the suggest cut-off score of ≥15, Leeds et al. (2004) reported sensitivity = 70% and specificity = 77% in a group of stroke rehabilitation inpatients. Based around cut-offs used for the HADS, Bennett et al. (2006) identified a cut-off of 17/18 on the SADQ-H (sensitivity= 100% and specificity=81%), and an optimum cut-off of 5/6 on the SADQ-H 10 (sensitivity = 100% and specificity = 78%).
### Aphasia Depression Rating Scale (ADRS)

- **Observer rating**: Based on interview & observation. Rating scale varies per item.
- **Reliability**: Test retest reported to be 0.89 by scale authors. Interobserver reliability = 0.89 (Benaim et al. 2004).
- **Concurrent validity**: ADRS scores were correlated with CAS ratings and with results of HRSD (Benaim et al. 2004).
- **Scores of ≥ 9 are used to indicate the presence of depression** (Benaim et al. 2004).
- **Reliability**: Test retest reported to be 0.89 by scale authors. Interobserver reliability = 0.89 (Benaim et al. 2004).
- **Concurrent validity**: ADRS scores were correlated with CAS ratings and with results of HRSD (Benaim et al. 2004).

### Children’s Depression Inventory (CDI)

- **27 Self-report 3 pt scale**
- **Scores of ≥ 19 have been identified as representing the 90th percentile within a general population of children in grades 3-9 (Smucker et al. 1986).**

### Kidscreen 52 (Generic HRQL measure)

- **52 Self-report 5 pt scale**
- **Scores for each dimension are calculated as T-values (mean=50, SD=10).**
- **Higher scores indicate higher Health-Related Quality of Life and well-being.**

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- It should be emphasized that a score indicating depression on a screening tool is not equivalent to a diagnosis of depression. Rather, a positive score indicates the need for further follow-up and assessment.
- **more detailed review of these screening tools may be obtained via the ebrsr.com, strokengine.com or in Salter et al. (2007)**
### Table 7.1B  Summary of Select Medications for Post-Stroke Depression

*Coming Soon*
References for Identification and Management of Post-Stroke Depression


7.2 Vascular Cognitive Impairment and Dementia Recommendations

2012 Definition of Vascular Cognitive Impairment:

Vascular Cognitive Impairment (VCI) includes the cognitive and behavioural disorders associated with cerebrovascular disease and risk factors, from mild cognitive deficits to frank dementia. VCI is a syndrome with cognitive impairment affecting at least one cognitive domain (e.g., attention, memory, language, perception or executive function) and with evidence of clinical stroke or subclinical vascular brain injury. VCI encompasses a large range of cognitive deficits, from relatively mild cognitive impairment of vascular origin (VaMCI) to Vascular Dementia (VaD), the most severe form of VCI. VCI also plays an important role in patients with Alzheimer’s disease (AD) pathology who have coexisting vascular lesions.

Cognitive deficits: The pattern of cognitive deficits in VCI may encompass all cognitive domains, including focal stroke syndromes, but there is likely to be an underlay or preponderance of attention and executive function deficits, such as slowed information processing, impairments in the ability to maintain task set or shift from one task to another and deficits in the ability to hold and manipulate information (e.g., working memory).

Vascular pathology: Cognitive impairment can result from a range of vascular pathology, including multiple cortical infarcts, multiple subcortical infarcts, covert (“silent”) infarcts, strategic infarcts, small-vessel disease with white matter lesions and lacunae, and brain hemorrhage.

Diagnosis criteria for vascular cognitive impairment following stroke has been defined by Gorelick et al (2011) and can be found in Table 7.2A (Hyperlink).

Best Practice Recommendation 7.2
Vascular Cognitive Impairment and Dementia

All patients with vascular risk factors and those with clinically evident stroke or transient ischemic attack should be considered at increased risk for vascular cognitive impairment (VCI), particularly those patients with cognitive, perceptual or functional changes that are clinically evident or reported during history taking.

7.2.1 Screening and Assessment

i. Patients with significant vascular risk factors for VCI, such as hypertension, diabetes, transient ischemic attack or clinical stroke, neuroimaging findings of covert stroke or white matter disease, hypertension-associated damage to other target organs, atrial fibrillation, other cardiac disease, and/or sleep apnea should be considered for VCI screening [Evidence Level A].

ii. Screening for VCI should be conducted using a validated screening tool, such as the Montreal Cognitive Assessment test [Evidence Level C]. Refer to Table 7.2B under Implementation Resources for information about selected tools and their psychometric properties. (Table 7.2A)

iii. Screening to investigate a person’s cognitive status should address arousal, alertness, attention, orientation, memory, language, agnosia, visual-spatial/perceptual function, praxis, and executive function. Executive function screening may include assessment of initiation, insight, planning and organization, judgment, problem solving, abstract reasoning, and social cognition [Evidence Level C].

iv. Post-stroke patients with suspected cognitive impairment should also be screened for depression, given that depression has been found to contribute to vascular cognitive impairment. A validated screening tool for depression should be used [Evidence Level A].

Refer to recommendation 7.1 for additional information

v. Patients who demonstrate cognitive impairments in the screening process should be managed by a healthcare professional with expertise in the assessment and management of neurocognitive functioning.* This assessment
should include cognition, perception and/or function as appropriate to guide comprehensive management [Evidence Level B]. If required, a referral should be made to an appropriate cognitive specialist [Evidence Level C].

- Additional assessments should be undertaken to determine: the nature and severity of cognitive impairments, as well as the presence of remaining cognitive abilities and strengths;
- The impact of deficits on function and safety in activities of daily living and instrumental activities of daily living, and occupational and school functioning should also be assessed.
- The results of these assessments should be used to guide selection and implementation of appropriate remedial, compensatory and/or adaptive intervention strategies according to client-centred goals and current or anticipated living environment (e.g., to help with discharge planning). [Evidence Level B].

*Experts in neurocognitive assessment may include a neuropsychologist, psychologist, occupational therapist, speech-language pathologist, clinical nurse specialist, psychiatrist, physiatrist, geriatrician, neurologist, and developmental pediatricians. Experts require specific qualifications to administer many of the identified assessments.*

### 7.2.2 Timing of Screening and Assessments

- All patients considered at high risk for cognitive impairment should be assessed periodically throughout the stages of care as indicated by the severity of clinical presentation, history and/or imaging abnormalities to identify cognitive, perceptual deficits, depression, delirium and/or changes in function [Evidence Level C].
- Stages of care across the continuum may include:
  - during presentation to emergency when cognitive, perceptual or functional concerns are noted;
  - during acute care stay, particularly if cognitive, perceptual or functional concerns, or evidence of delirium is noted;
  - throughout rehabilitation within inpatient, outpatient, and home-based settings, according to client progress;
  - following hospital discharge from the emergency department or inpatient setting to an outpatient or community-based healthcare setting.
- While assessment at different stages of care is important for guiding diagnosis and management, it is also important to be aware of the potential impact of multiple assessments on both the validity of the results as well as on the patient (e.g., test fatigue, practice effects) [Evidence Level B].
- Effects of age must also be considered, particularly in children with stroke where outcomes will evolve in parallel with development and deficits may not be fully realized until many years later [Evidence Level C].

### 7.2.3 Management of Vascular Cognitive Impairment

- Vascular risk factors (e.g., hypertension, atrial fibrillation) should be managed aggressively to achieve optimal control of the pathology underlying cognitive impairment following a stroke or TIA [Evidence Level A].
  Refer to Chapter 2, Prevention of Stroke, for additional information
- Interventions should be tailored according to the following considerations:
  - Goals should be patient-centred and sensitive to the values and expectations of patient, family and caregivers [Evidence Level B]
  - Goals should be developed in the context of both the cognitive impairments as well as patients’ intact cognitive abilities, with the aim to facilitate resumption of desired activities and participation (e.g., self-care, home management, leisure, social roles, driving, volunteer participation, financial management, return to work) [Evidence Level B].
  **NOTE:** Issues such as intensity and dose of therapy, stage of treatment, and impact of severity of deficits can modify effectiveness of therapy, and require more research.
- Evidence for interventions for cognitive impairment is growing, although more research is required. Interventions with the patient can be broadly classified as either compensatory strategy training, or direct remediation/cognitive
skill training. These approaches are not mutually exclusive, and, depending upon the impairments and goals, may be offered together.

NOTE: It should be noted, however that if the level of impairment has reached the moderate dementia stage, interventions may be more focused on providing education and support for the caregiver in addition to, or in lieu of, cognitive rehabilitation with the patient.

a. **Compensatory Strategy training** focuses on teaching strategies to address impairments and is often directed at specific functional limitations in activities of daily living to promote independence. Compensatory strategies can include learning to use external devices (e.g., memory notebooks or alarms), adapting the external environment (e.g., additional social supports or reorganization of living space), and/or learning to use internal mental operations or processes (e.g., problem-solving techniques) that enhance the impaired cognitive domain. Certain types of strategy training have been shown to be effective for improving attention, memory, language, praxis and executive function domains [Evidence Level B].

b. **Direct remediation/cognitive skill training** focuses on providing intensive specific training to directly improve the impaired cognitive domain. Computer-based training has been shown to be effective in improving attention and working memory impairments as well as language impairments [Evidence Level B]. The impact of direct skill training on achievement of goals at the activities and participation levels of functioning requires more research.

c. **New developments** in cognitive intervention that may be of potential benefit include repetitive transcranial magnetic stimulation or direct stimulation, the use of virtual reality environments or simulations, and application of constraint-induced therapy for the impaired cognitive domain. These strategies require more research before recommendations can be developed on their use.

iv. Patients with cognitive impairment and evidence of changes in mood (e.g., depression, anxiety), or behavioural changes on screening should be referred and managed by an appropriate mental healthcare professional [Evidence Level B]. Refer to recommendation 7.1 for additional information.

### 7.2.4 Pharmacotherapy for Vascular Cognitive Impairment

i. Patients with evidence of vascular cognitive impairment should be managed by a physician with expertise in vascular cognitive impairment for further assessment and recommendations regarding pharmacotherapy [Evidence Level C].

ii. Cholinesterase inhibitors should be considered for management of vascular cognitive impairment diagnosed using the National Institute of Neurological Disorders and Stroke (NINDS) – Association Internationale pour la Recherche et l’Enseignement en Neurosciences (AIREN) diagnostic criteria [Evidence Level B].

   a. There is fair evidence of small magnitude benefits for donepezil in cognitive and functional outcomes, with less robust benefits on global measures [Evidence Level B]. Donepezil can be considered as a treatment option for vascular dementia. More research is needed on the benefits of donepezil for vascular cognitive impairment.

   b. There is fair evidence of small magnitude benefits for galantamine on cognition function and behaviour in mixed Alzheimer and cerebrovascular disease. Galantamine can be considered a treatment option for mixed Alzheimer and cerebrovascular disease [Evidence Level B].

#### Rationale

Vascular cognitive impairment affects up to 60 percent of stroke survivors and is associated with poorer recovery and decreased function in activities of daily living and instrumental activities of daily living. Patients may require long-term, ongoing intervention and rehabilitation (Teasel et al, 2009; Madureira et al 2001). Cognitive abilities in the areas of executive function, attention and memory appear important in predicting functional status at discharge. In addition, cognitive impairment can be chronic and progressive after stroke; post-stroke dementia is estimated to occur in 26 percent of stroke patients by three months (95% CI 3% in age-matched controls) and adversely affects recovery. Cognitive impairment increases long-term dependence and is associated with increased mortality rates (61 percent versus 25 percent) (Black, 2007).
Cognitive impairment due to covert vascular pathology is also increasing. Covert strokes, usually lacunes, are common (23 percent of community elderly) and are associated with cognitive decline, dementia, and stroke. Evidence is emerging that demonstrates that for every clinically evident stroke, there may be up to ten “covert” strokes. Signs of covert stroke are often manifested as signs and symptoms of cognitive impairment. Intracerebral small-vessel disease is a disorder that is on the rise with the aging of the population, leading to an increase in the need for long-term care support services.

Emotional and related behavioural changes are known to occur following stroke, with specific behaviours linked to the affected hemisphere and stroke location. Disorders such as apathy, anxiety, labile laughing and crying, and other behaviours such as disinhibition, denial, indifference, overt sadness, and aggressiveness can occur in the early days following stroke. Some of these behaviours may be related to later development of depression; however, prospective studies of mood changes during and immediately after stroke are limited as yet.

In most population studies, vascular dementia is the second most common cause of dementia, after Alzheimer disease. The combination of Alzheimer disease and vascular disease results in the commonest substrate of dementia in the elderly. A single macroscopic hemispheric infarct is sufficient to cause dementia in people with intermediate Alzheimer pathology.

System Implications

- Public education to increase awareness that cognitive changes may be considered as manifestations of vascular disease and stroke.
- Public education to increase awareness of untreated or uncontrolled hypertension and other vascular risk factors and their relationship to cognitive changes and dementia.
- Professional education to increase awareness among family physicians that patients with vascular risk factors, if not treated, will be at high risk for cognitive deficits.
- Professional education across specialties (e.g., nephrology, ophthalmology, family medicine) to increase awareness that patients with small-vessel disease should be investigated for stroke risk factors and cognitive impairment.
- Access to interprofessional teams with the expertise to appropriately manage patients with vascular cognitive impairment across the continuum of stroke care and in the community.
- Continuing professional education to ensure proficiency in assessment administration, interpretation and management of patients demonstrating post stroke and vascular cognitive impairment or at risk of vascular cognitive impairment.

Performance Measures

1. Percentage of patients with stroke or stroke risk factors who undergo a brief cognitive screening at each transition point along the continuum of stroke care (i.e., acute inpatient care, inpatient rehabilitation, outpatient clinics and programs, home-based services, and stroke prevention clinics) and in the community following inpatient discharge and at any time when there is a suspected change in cognitive status. (Core indicator)
2. Percentage of patients with stroke who are referred for more in-depth cognitive or neuropsychological assessment throughout the continuum of stroke care (for example, during inpatient care, inpatient rehabilitation, outpatient and ambulatory clinics or programs (stroke prevention clinics) and/ or following inpatient discharge to the community).
3. Percentage of change in Montreal Cognitive Assessment (MoCA) scores at baseline and three months, six months and one year following initiation of therapy.
4. Percentage improvement in control of high blood pressure and other vascular risk factors in patients with vascular cognitive impairment.

Measurement Notes

- Recommendation 7.2 and corresponding performance measures apply across the continuum of stroke care and should be considered in acute inpatient care, inpatient rehabilitation, outpatient clinics, home-based services, and stroke prevention clinics and/or following inpatient discharge to the community.
- When using these performance measures it is important to record when and in what context (continuum of care) the measurements were conducted. Data for measurement may be found through primary chart audit. Data quality will be dependent on the quality of documentation by healthcare professionals.
This is a new area and will require a great deal of education for healthcare professionals especially in the area of documentation.

**Implementation Resources and Knowledge Transfer Tools**

- Table 7.2B: Select Tools for the Screening and Initial Assessment for Vascular Cognitive Impairment in Stroke Patients
- CSN Algorithm for Screening and Assessment of VCI (in development).
- Vascular Harmonization Guidelines [http://stroke.ahajournals.org/content/37/9/2220.full](http://stroke.ahajournals.org/content/37/9/2220.full)
- HSF Centre for Stroke Recovery [http://centreforstroke.recovery.ca/](http://centreforstroke.recovery.ca/)
- AHA/ASA Scientific Statement on Vascular Contributions to Cognitive Impairment and Dementia [http://stroke.ahajournals.org/content/early/2011/07/21/STR.0b013e3182299496](http://stroke.ahajournals.org/content/early/2011/07/21/STR.0b013e3182299496)
- Stroke Best Practices Teaching Slides: Mood and Cognition Following Stroke
- StrokEngine [http://strokengine.ca/](http://strokengine.ca/)
- HSF 10 warning signs of depression: [http://www.heartandstroke.com/site/c.ikIQLcMWJtE/b.5482531/k.6ACA/Stroke__Mental_changes.htm](http://www.heartandstroke.com/site/c.ikIQLcMWJtE/b.5482531/k.6ACA/Stroke__Mental_changes.htm)
- The Heart & Stroke Living with Stroke™ program [http://www.heartandstroke.on.ca/site/c.pvl3eNWJwe/b.7936265/k.94AE/Living_with_Stroke__Ontario_Locations.htm](http://www.heartandstroke.on.ca/site/c.pvl3eNWJwe/b.7936265/k.94AE/Living_with_Stroke__Ontario_Locations.htm)
- Let's Talk about Stroke [http://www.heartandstroke.com/site/c.ikIQLcMWJtE/b.3882223/k.3FC6/Stroke__Lets_Talk_about_Stroke.htm](http://www.heartandstroke.com/site/c.ikIQLcMWJtE/b.3882223/k.3FC6/Stroke__Lets_Talk_about_Stroke.htm)
- Life after Stroke website: [http://www.lifeafterstroke.ca](http://www.lifeafterstroke.ca)
- HSF Ontario Tips and Tools: [http://www.heartandstroke.on.ca/site/c.pvl3eNWJwe/_.6194819/k.FEB1/Tips_and_Tools__2010.htm](http://www.heartandstroke.on.ca/site/c.pvl3eNWJwe/_.6194819/k.FEB1/Tips_and_Tools__2010.htm)
- Coping with stress [http://www.heartandstroke.com/site/c.ikIQLcMWJtE/b.5590155/k.3B4A/Heart_disease__Coping_with_stress.htm](http://www.heartandstroke.com/site/c.ikIQLcMWJtE/b.5590155/k.3B4A/Heart_disease__Coping_with_stress.htm)

**Summary of the Evidence (Update 2013)**

It has been estimated that 5% of all people over the age of 65 years, in Canada, have evidence of vascular cognitive impairment (Rockwood et al. 2000). However, in individuals who have experienced stroke, reported prevalence rates tend to be much greater, ranging from 15 – 20% in various clinical settings to 39%, 35%, 30% and 32% at 3 months, one year, 2 years and 3 years post stroke, respectively, as reported by Patel et al. (2003). While the risk for cognitive impairment is greater following stroke and, certainly, not all individuals with cognitive impairment have dementia, post-stroke cognitive impairment is associated with an increased risk for dementia.

Pendlebury and Rothwell (2009) conducted a systematic review and meta-analysis of 73 published studies examining prevalence and predictors of dementia in individuals with stroke. Overall, pooled prevalence of pre-stroke dementia was 14.4% in hospital-based cohorts (n=22) and 9.1% in community-based studies (n=8). Prevalence of post-stroke dementia ranged from 7.4% in population-based studies of individuals with first-ever stroke and no existing dementia to 41.3% in hospital-based studies of...
individuals with recurrent stroke (both with and without existing dementia). Rates of dementia were at least doubled following recurrent stroke when compared to first-ever stroke and were higher in hospital-based than in community-based studies. At 3-6 months, post-stroke incidence of dementia was approximately 20%; this increased linearly at a rate of 3.0% in hospital-based studies of either first or recurrent stroke. Incidence rates were lower in population-based studies of first-ever stroke and when cases with recurrent stroke were excluded.

In the review by Pendlebury and Rothwell (2009), multivariate analyses were identified in 19 studies. From these 19 studies, the most commonly reported independent predictors of post-stroke dementia were older age, lower education level, previous stroke, diabetes, atrial fibrillation, existing cognitive impairment and stroke severity. In summary, Pendlebury and Rothwell (2009) suggest that approximately 10% of patients have existing dementia at the time of stroke. An additional 10% develop new dementia shortly after a first-ever stroke while more than one-third of patients may experience dementia following a recurrent stroke. Recurrent stroke was identified as an important, and commonly cited, predictor of dementia. Gorelick (1997, 2004) reported on the risk factors for vascular dementia in 4 categories: demographic (older age, male sex, lower level of education), atherosclerotic (hypertension, smoking, myocardial infarction, diabetes, hyperlipidemia), genetic (cerebral autosomal dominant arteriopathy with subcortical infarct, leukoencephalopathy, apolipoprotein) and stroke-related (volume of cerebral tissue loss, evidence of bilateral cerebral infarction, strategic infarction, white matter disease).

Cognitive impairment following stroke has been associated with reduction in functional ability, and increased risk for depression (ebrsr.com). Individuals with cognitive impairments may require more therapy over longer periods of time (Zinn et al. 2004). In addition, the presence of executive dysfunction may have a negative impact on engagement in rehabilitation (Skidmore et al. 2010). A review by Leys et al. (2005) reported that higher rates of mortality have also been found among patients with post stroke dementia in both community-based and hospital-based studies. Overall, mortality rates are reported to be 2 to 6 times higher among individuals with post stroke dementia after adjusting for demographic factors, associated cardiac disease, stroke severity and stroke recurrence (Leys et al. 2005).

At present, there is no ‘gold standard’ for the diagnosis of vascular dementia. However, it has been recommended that given the anticipated presence of executive dysfunction in cases of vascular cognitive impairment post stroke, any assessment used to detect the presence of VCI be appropriate to the assessment of executive function. As part of the development of the Canadian Stroke Network vascular cognitive impairment harmonization standards, Hachinski et al. (2006) recommended use of a 5-minute neuropsychological protocol for the identification of VCI that included selected subtests 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 tasks. Given more time, the original Trail-making 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 Mini Mental Status Examination in the initial 5-minute protocol was rejected, as it does not provide adequate assessment of executive function and is insensitive to mild memory impairment (Hachinski et al. 2006).

Evidence for cognitive rehabilitation interventions within the stroke population is not strong in many areas. In general, interventions may be considered to have one of two objectives: 1) to reinforce or re-establish previous behavioural skills or function (to remediate) or 2) to establish new patterns of activity or behaviour through compensatory mechanisms that may be either internal or external to the individual (Cicerone 2000, 2005). A meta-analytic examination of the 2 systematic reviews of cognitive rehabilitation interventions conducted by Cicerone et al. (2000, 2005) and updated in 2011, reported effect sizes for cognitive interventions in attention, visuospatial, language, memory and comprehensive training. Overall, cognitive rehabilitation interventions were associated with small, but significant treatment effects (ES=0.30). Overall treatment effect was moderated by treatment domain, etiology of injury (e.g. TBI vs. stroke) and time since injury. For studies of individuals with stroke, the reported pooled effect size associated with cognitive rehabilitation was slightly larger (ES=0.40) than the overall effect size; however, it should be noted that these were primarily in the areas of language and visuospatial deficits (Cicerone et al. 2005). Studies of attention or executive function, memory or comprehensive cognitive function were focused more often on individuals with TBI or other brain injury. Although generally positive results have been reported for studies examining the use of attention training, memory strategy training, use of mnemonic devices (e.g. personal pagers), and problem-solving training for study participants who have experienced brain injury (including stroke) evidence from randomized controlled trials specific to the stroke population is limited (Cicerone et al, 2011; ebrsr.com).

**Pharmacotherapy**

Cholinergic agents have been used in the treatment of dementia of the Alzheimer’s type. Three such agents, donepezil, rivastigmine and galantamine, have also been investigated for use in the treatment of vascular dementia. Donepezil, a selective
Acetylcholinesterase inhibitor, has been the subject of 3 large randomized controlled trials (Black et al. 2003, Wilkinson et al. 2003, Roman et al. 2010). A meta-analysis of the first 2 trials demonstrated significant improvements in cognitive and global function, including improvements in the performance of activities of daily living associated with use of donepezil in the treatment of patients with mild to moderate vascular dementia (Passmore et al. 2005). The most recent trial also reported significant improvement in cognitive outcomes associated with treatment (Roman et al. 2010).

Galantamine is an acetylcholinesterase inhibitor that has been shown to be of benefit in terms of cognition, behaviour and the performance of activities of daily living when used in the treatment of Alzheimer’s dementia (Erkinjuntti et al. 2002, 2003); however the benefit associated with treatment was most obvious in a subgroup of individuals with mixed dementia (Alzheimer’s Disease plus cerebrovascular disease) (Erkinjuntti et al. 2002).

Children and Vascular Cognitive Impairment:

Cognitive outcomes for children with stroke must be considered differently. Covert VCI is not a common issue except in select disease states (moyamoya, sickle-cell, small vessel vasculitis). Most importantly, cognitive outcomes must be considered within the context of constantly evolving neurodevelopment. Thus, outcomes must use developmentally and age-appropriate outcome measures. Deficits in cognition and higher brain functions may not be evident until the relevant stage of development is reached, whereby young children “grow into” their deficits over time. [Westmacott et al. 2007a; Westmacott et al. 2009]

About 1 in 3 children with stroke have cognitive deficits at outcome, limiting academic, social, and independent functional success [Friefeld et al. 2004; Nass and Trauner 2004](Carr L 2011). Adverse cognitive outcomes from childhood cerebral sinus venous thrombosis (CSVT) [Wasay et al. 2008;deVeber et al. 2001;Moharir et al. 2010;Berfelto et al. 2010] and hemorrhagic stroke [Blom et al. 2003;Meyer-Heim and Boltshauser 2003] are also common. Overall cognitive function including intelligence, verbal ability, working memory, and processing speed are lower than average in children with stroke (Westmacott, 2009), [Hetherington et al. 2005; McLinden et al. 2007] [Max 2004; Lansing et al. 2004].

Post-stroke psychiatric/behavioural disorders also appear to be common in childhood stroke (Max, 2002; Elbers 2013). Difficulties with social and behavioral development are also increased in children after stroke with secondary impact on parental mental health (De Schryver, 2002; Goodman, 2000). Limited studies suggest that social function is commonly impaired following stroke in childhood [Mosch et al. 2005]. Rates of potentially treatable Attention Deficit Hyperactivity Disorder (ADHD) are increased [Max et al. 2002;Max et al. 2003;Max et al. 2004], particularly with lesions involving the putamen [Teicher et al. 2000].

Contradicting the idea that the immature brain may be more plastic with a greater capacity for recovery, a younger age at stroke may be associated with worse cognitive and behavioural outcomes. (Westmacott, 2009; Everts, 2008) [Stiles 2000;Lansing, Max, Delis, Fox, Lancaster, Manes, and Schatz2004;Westmacott et al. 2007b]. There is also evidence for late emergence of cognitive deficits after perinatal stroke, with IQ measured in the preschool period higher than that measured later (Westmacott, 2011). About one third of childhood stroke survivors require specialized education (Delsing, 2001). Regular, age-appropriate neuropsychological evaluations should be considered in all at-risk children to determine educational and support needs.
Appendix 7.2A: Diagnostic Criteria for Vascular Cognitive Impairment and Dementia

(Gorelick et al, 2011)

1. The term VCI characterizes all forms of cognitive deficits from Vascular Dementia (VaD) to Mild Cognitive Impairment (MCI) of vascular origin

2. These criteria cannot be used for subjects who have an active diagnosis of drug or alcohol abuse/dependence. Subjects must be free of any type of substance for at least 3 months.

3. These criteria cannot be used for subjects with delirium.

Dementia

1. The diagnosis of dementia should be based on a decline in cognitive function from a prior baseline and a deficit in performance in 2 cognitive domains that are of sufficient severity to affect the subject’s activities of daily living.

2. The diagnosis of dementia must be based on cognitive testing, and a minimum of 4 cognitive domains should be assessed: executive/attention, memory, language, and visuospatial functions.

3. The deficits in activities of daily living are independent of the motor/sensory sequelae of the vascular event.

Probable Vascular Dementia (VaD)

1. There is cognitive impairment and imaging evidence of cerebrovascular disease and
   a. There is a clear temporal relationship between a vascular event (eg, clinical stroke) and onset of cognitive deficits, or
   b. There is a clear relationship in the severity and pattern of cognitive impairment and the presence of diffuse, subcortical cerebrovascular disease pathology (eg, as in CADASIL).

2. There is no history of gradually progressive cognitive deficits before or after the stroke that suggests the presence of a nonvascular neurodegenerative disorder.

Possible Vascular Dementia (VaD)

There is cognitive impairment and imaging evidence of cerebrovascular disease but

1. There is no clear relationship (temporal, severity, or cognitive pattern) between the vascular disease (eg, silent infarcts, subcortical small-vessel disease) and the cognitive impairment.

2. There is insufficient information for the diagnosis of VaD (eg, clinical symptoms suggest the presence of vascular disease, but no CT/MRI studies are available).

3. Severity of aphasia precludes proper cognitive assessment. However, patients with documented evidence of normal cognitive function (eg, annual cognitive evaluations) before the clinical event that caused aphasia could be classified as having probable VaD.

4. There is evidence of other neurodegenerative diseases or conditions in addition to cerebrovascular disease that may affect cognition, such as
   a. A history of other neurodegenerative disorders (eg, Parkinson disease, progressive supranuclear palsy, dementia with Lewy bodies);
   b. The presence of Alzheimer disease biology is confirmed by biomarkers (eg, PET, CSF, amyloid ligands) or genetic studies (eg, PS1 mutation); or
   c. A history of active cancer or psychiatric or metabolic disorders that may affect cognitive function.
Vascular Mild Cognitive Impairment (VaMCI)

1. VaMCI includes the 4 subtypes proposed for the classification of MCI: amnestic, amnestic plus other domains, nonamnestic single domain, and nonamnestic multiple domain.

2. The classification of VaMCI must be based on cognitive testing, and a minimum of 4 cognitive domains should be assessed: executive/attention, memory, language, and visuospatial functions. The classification should be based on an assumption of decline in cognitive function from a prior baseline and impairment in at least 1 cognitive domain.

3. Instrumental activities of daily living could be normal or mildly impaired, independent of the presence of motor/sensory symptoms.

Probable Vascular Mild Cognitive Impairment (VaMCI)

1. There is cognitive impairment and imaging evidence of cerebrovascular disease and
   a. There is a clear temporal relationship between a vascular event (eg, clinical stroke) and onset of cognitive deficits, or
   b. There is a clear relationship in the severity and pattern of cognitive impairment and the presence of diffuse, subcortical cerebrovascular disease pathology (eg, as in CADASIL).

2. There is no history of gradually progressive cognitive deficits before or after the stroke that suggests the presence of a nonvascular neurodegenerative disorder.

Possible Vascular Mild Cognitive Impairment (VaMCI)

There is cognitive impairment and imaging evidence of cerebrovascular disease but

1. There is no clear relationship (temporal, severity, or cognitive pattern) between the vascular disease (eg, silent infarcts, subcortical small-vessel disease) and onset of cognitive deficits.

2. There is insufficient information for the diagnosis of VaMCI (eg, clinical symptoms suggest the presence of vascular disease, but no CT/MRI studies are available).

3. Severity of aphasia precludes proper cognitive assessment. However, patients with documented evidence of normal cognitive function (eg, annual cognitive evaluations) before the clinical event that caused aphasia could be classified as having probable VaMCI.

4. There is evidence of other neurodegenerative diseases or conditions in addition to cerebrovascular disease that may affect cognition, such as
   a. A history of other neurodegenerative disorders (eg, Parkinson disease, progressive supranuclear palsy, dementia with Lewy bodies);
   b. The presence of Alzheimer disease biology is confirmed by biomarkers (eg, PET, CSF, amyloid ligands) or genetic studies (eg, PS1 mutation); or
   c. A history of active cancer or psychiatric or metabolic disorders that may affect cognitive function.

Unstable Vascular Mild Cognitive Impairment (VaMCI)

1. Subjects with the diagnosis of probable or possible VaMCI whose symptoms revert to normal should be classified as having “unstable VaMCI.”

Notes: VCI indicates vascular cognitive impairment; VaD, vascular dementia; MCI, mild cognitive impairment; CADASIL, cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy; CT/MRI, computed tomography/magnetic resonance imaging; PET, positron emission tomography; CSF, cerebrospinal fluid; and VaMCI, vascular mild cognitive impairment.
Canadian Best Practice Recommendations for Stroke Care
Update 2012 - 2013

Section 7: Mood and Cognition in Stroke


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Table 7.2 B  Select Tools for the Screening and Initial Assessment for Vascular Cognitive Impairment in Stroke Patients

<table>
<thead>
<tr>
<th>Assessment Tool and Reference</th>
<th>Purpose</th>
<th>Content &amp; Population</th>
<th>Length of Test</th>
<th>Reliability</th>
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<tr>
<td><strong>Montreal Cognitive Assessment Tool (MoCA)</strong></td>
<td>Measures Mild Cognitive Impairment</td>
<td>The items of the MoCA examine attention and concentration, executive functions, memory, language, visuococonstructional skills, conceptual thinking, calculations, and orientation. Population: Can be used in patients with stroke and any individual who is experiencing memory difficulties but scores within the normal range on the MMSE.</td>
<td>5-10 minutes</td>
<td>Only 1 study has examined the internal consistency of the MoCA and reported excellent levels of internal consistency. Only 1 study has examined the test-retest reliability of the MoCA, and reported excellent test-retest</td>
<td>Criterion: Concurrent. Excellent correlations with the Mini Mental State Examination (MMSE) have been reported. Construct: Known groups. One study reported that the MoCA can distinguish between patients with mild cognitive impairment and healthy controls.</td>
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<td><strong>NINDS-CSN Harmonization VCI Neuropsychology Protocols</strong></td>
<td>Designed to measure vascular cognitive impairment in stroke patients</td>
<td>The 60 minute assessment tests: executive/activation function, visuospatial, language/lexical retrieval, memory and learning, and neuropsychiatric/depressive symptoms. The 30 minute assessment tests a subset of the 60 minute assessment including: semantic and phonemic fluency, Digit Symbol-Coding and the revised Hopkins Verbal Learning Test, in addition to the CES-D and</td>
<td>60, 30, or 5 minute versions available</td>
<td>NA</td>
<td>One group has tested the validity in ischemic stroke patients. 1. All three protocol scores are significantly lower than in patients than in matched controls (F statistics range from 15.7 to 50.5; all p values &lt; .000; eta2 values range from .14 to .31). 2. ROC analyses shows the 60M Executive subtest to be the most sensitive and specific, followed by the Memory, Language, and Spatial subtests (AUC values: .86, .75, .70, .67, respectively). 3. WMH volumes show the most consistent relationship between regional imaging and protocol scores, when compared to Stroke Volume and Brain Parenchymal Fraction scores.</td>
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<td>Neuropsychiatric Inventory. The 5 minute protocol consists of selected subtests from the Montreal Cognitive Assessment, including a 5-word immediate and delayed memory test, a 6-item orientation task and a 1-letter phonemic fluency test (the letter F).</td>
<td>Population: Stroke patients</td>
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<td>Additional Screening and Assessment Tools for Vascular Cognitive Impairment and Dementia</td>
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<td>Cognitive- Functional Independence Measure (Cognitive- FIM)</td>
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<td>There are 5 cognitive items: comprehension, expression, social interaction, problem solving, and memory.</td>
<td>30-45 minutes</td>
<td>In a review of 11 studies, Ottenbacher et al., 1996 reported a mean inter-observer reliability value of 0.95; a median test-retest reliability of 0.95 and a median equivalence reliability (across versions) of 0.92. The FIM scores were found to predict amount of home care required; admission scores predict FIM discharge scores; placement after discharge; functional gain; length of stay; depression; ability to return to work following stroke or traumatic brain injury.</td>
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<td><a href="http://strokengine.ca/assess/module_fim_intro-en.html">http://strokengine.ca/assess/module_fim_intro-en.html</a></td>
<td></td>
<td>Population: It has been tested for use in patients with stroke, traumatic brain injury, spinal cord injury, multiple sclerosis, and elderly individuals undergoing inpatient rehabilitation and has been used with children as young as 7 years old.</td>
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<td>Reliability was higher for items in the motor domain than for those in the social/cognitive domain. Internal consistency: - alpha of 0.93 – 0.95 reported at admission vs. discharge (Dodd et al. 1993); alpha = 0.88 to 0.91(Hsueh et al. 2002); Hobart et al. (2001) reported item-to-total correlations ranging from 0.53 to 0.87 for FIM total, 0.60 for FIM motor and 0.63 cognitive FIM – mean inter-item correlations were 0.51 for FIM, 0.56 – 0.91 for motor FIM and 0.72 – 0.80 for cognitive FIM, alpha = 0.95, 0.95 and 0.89 for Cognitive-FIM; Content: The FIM was created based on the results of a literature review of published and unpublished measures and expert panels and was then piloted in 11 centers. The Delphi method was applied, using rehabilitation expert opinion to establish the inclusiveness and appropriateness of the items. The FIM scores were found to predict amount of home care required; admission scores predict FIM discharge scores; placement after discharge; functional gain; length of stay; depression; ability to return to work following stroke or traumatic brain injury. Construct: FIM scores discriminated between groups based on spinal cord injury and stroke severity, and the presence of comorbid illness both at admission and discharge. It has also been found to distinguish between patients with or without neglect and with or without aphasia at both admission and discharge. Concurrent. The Cognition-FIM was found to have an excellent correlation with the DRS; an adequate correlation with the Montebello Rehabilitation Factor Score (MRFS) (efficacy); and a poor correlation with the MRFS (efficiency).</td>
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<td>daily living.</td>
<td>FIM, motor FIM and cognitive FIM respectively.</td>
<td>Convergent/Discriminant. The total FIM was found to demonstrate an excellent correlation with the Office of Population Censuses and Surveys Disability Scales disability scores; an adequate correlation with the London Handicap Scale and the Wechsler Adult Intelligence Test-verbal IQ test; and a poor correlation with the SF-36 Physical and Mental components, and the General Health Questionnaire. The Cognition-FIM was found to demonstrate an excellent correlation with the Mini-Mental State Examination (MMSE); an adequate correlation with the Lowenstein Occupational Therapy Cognitive Assessment (LOTCA), Office of Population Censuses and Surveys Disability scores, and the revised Wechsler Adult Intelligence Test-verbal IQ; and a poor correlation with the London Handicap Scale, SF-36 Physical and Mental components, and the General Health Questionnaire. Ecological: The Cognition-FIM demonstrated adequate correlations with the OT-APST.</td>
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<td>The CAMCOG is a standardized assessment instrument for diagnosis and grading of dementia</td>
<td>The CAMCOG consists of 67 items. It is divided into 8 subscales: orientation, language (comprehension and expression), memory (remote, recent and learning), attention, praxis, calculation, abstraction and perception. Population: The CAMCOG can be used with, but is not limited to clients with stroke. 20 to 30 minutes</td>
<td>No studies have examined the internal consistency of the CAMCOG in clients with stroke. No studies have examined the reliability of the CAMCOG in clients with stroke. Predictive Validity. 6 studies examined the predictive validity of the CAMCOG and reported that the CAMCOG can be predicted by age, the R-CAMCOG, the Mini-Mental State Examination and cognitive and emotional impairments. Additionally, the CAMCOG was an excellent predictor of dementia 3 to 9 months post-stroke. However, the CAMCOG was not able to predict QOL in clients with stroke and is not predicted by the Functional Independence Measure. Known Groups: 2 studies examined known groups validity of the CAMCOG and reported that the CAMCOG is able to distinguish between clients with or without dementia as well as aphasia severity in clients with stroke. Convergent validity: 1 study examined the convergent validity of the CAMCOG in clients with stroke and reported excellent correlations between the CAMCOG and the R-CAMCOG and the Mini-Mental State Examination shortly after and 1 year post-stroke. Correlations between the CAMCOG and the Functional Independence Measure range from adequate after stroke to poor at 1 year post-stroke. 1 study examined the convergent validity of the CAMCOG-R and reported excellent correlations between the...</td>
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<td>The CAMCOG can be obtained by purchasing the entire CAMDEX from the Cambridge University Department of Psychiatry <a href="http://strokengine.ca/assess/module_camcog_intro-en.html">http://strokengine.ca/assess/module_camcog_intro-en.html</a></td>
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<td><strong>Frontal Assessment Battery</strong></td>
<td>The FAB is a brief tool that can be used at the bedside or in a clinic setting to assist in discriminating between dementias with a frontal dysexecutive phenotype and Dementia of Alzheimer’s Type (DAT).</td>
<td>Tests: conceptualization, mental flexibility, programming, sensitivity to interference, inhibitory control, and environmental autonomy</td>
<td>Approximately 10 minutes</td>
<td>Chinese FAB: The CFAB had low to good correlation with various executive measures: MDRS I/P (r = 0.63, p &lt; 0.001), number of category completed (r = 0.45, p &lt; 0.001), and number of perseverative errors (r = −0.37, p &lt; 0.01) of WCST. Among the executive measures, only number of category completed had significant but small contribution (6.5%, p = 0.001) to the variance of CFAB. A short version of CFAB using three items yielded higher overall classification accuracy (86.6%) than that of CFAB full version (80.6%) and MMSE (77.6%). In another test, which compared the Chinese FAB to the Mattis Dementia Rating Scale Initiation/Perseveration subset: Both tests showed comparably good ability in Receiver Operating Characteristic curves analysis (AUCMDRS I/P = 0.887; AUC FAB = 0.854, p = .833) in discriminating between controls and patients and correctly classified over 78% of subjects. Verbal fluency and motor programming contributed most to the discriminating power in the two tests.</td>
<td>Chinese FAB: Internal consistency (alpha = 0.77), test-retest reliability (rho = 0.89, p &lt; 0.001), and interrater reliability (rho = 0.85, p &lt; 0.001) of CFAB were good.</td>
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<td>Assessment Tool and Reference</td>
<td>Purpose</td>
<td>Content &amp; Population</td>
<td>Length of Test</td>
<td>Reliability</td>
<td>Validity</td>
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<td><strong>Kettle Test</strong> &lt;br&gt; A preliminary version of the Kettle Test manual can be obtained from: <a href="http://strokengine.ca/assess/module_kt_inddepth-en.html">http://strokengine.ca/assess/module_kt_inddepth-en.html</a> <a href="http://www.rehabmeasures.org/Lists/RehabMeasures/DispForm.aspx?ID=939">http://www.rehabmeasures.org/Lists/RehabMeasures/DispForm.aspx?ID=939</a></td>
<td>The Kettle Test measures cognitive skills in a functional context.</td>
<td>The task of preparing two hot beverages is broken down into 13 discrete steps that can be evaluated. Population: Clients with stroke who were living independently in the community prior to stroke</td>
<td>5-20 minutes</td>
<td>No studies have examined the internal consistency of the Kettle Test. No studies have examined the test-retest reliability of the Kettle Test. No studies have examined the intra-rater reliability of the Kettle Test. 1 study examined the inter-rater reliability of the Kettle Test and reported excellent inter-rater reliability.</td>
<td>Convergent: 1 study reported excellent correlation with the Functional Independence Measure (FIM) Cognitive scale and adequate correlation with the Mini-Mental Status Examination (MMSE), Clock Drawing Test and the Behavioural Inattention Test (BIT) Star Cancellation subtest. Known groups: The Kettle Test was able to discriminate clients with stroke from healthy controls.</td>
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<td><strong>Mini-Mental State Exam (MMSE)</strong> &lt;br&gt; <a href="http://strokengine.ca/assess/module_mmse_intro-en.html">http://strokengine.ca/assess/module_mmse_intro-en.html</a> <a href="http://www.mhpcn.ca/uploads/MMSE.1276128605.pdf">http://www.mhpcn.ca/uploads/MMSE.1276128605.pdf</a></td>
<td>Screens for cognitive impairment Population While originally used to detect dementia within a psychiatric setting, its use is now widespread and is available with an attached table that enables patient-specific norms.</td>
<td>The MMSE consists of 11 simple questions or tasks that look at various functions including: arithmetic, memory and orientation.</td>
<td>approx. 10 minutes</td>
<td>Out of 9 studies examining the internal consistency of the MMSE, 3 reported poor internal consistency, 1 reported adequate internal consistency, 2 reported poor to excellent internal consistency, 2 reported excellent internal consistency, 1 reported excellent internal consistency in patients with Alzheimer's Disease and poor internal consistency in patients with cognitive impairment. Out of 6 studies examining the test-retest reliability of the MMSE, 2 studies reported excellent test-retest, 1 reported adequate test-retest, 1 reported adequate to excellent test-retest, 1 reported poor to adequate test-retest, 1 reported poor test-retest. Out of 3 studies examining the inter-rater reliability of the MMSE, 1 reported excellent inter-rater, 2 reported adequate inter-rater.</td>
<td>Criterion: The MMSE can discriminate between patients with Alzheimer's Disease and frontotemporal dementia; can discriminate between patients with left- and right-hemispheric stroke. Construct: Concurrent. MMSE had a poor correlation with the Mattis Dementia Rating Scale; poor to excellent correlations with the Wechsler Adult Intelligence Test; adequate correlation with the Functional Independence Measure; significant correlations with the Montgomery Asberg Depression Rating Scale and the Zung Depression Scale. Predictive. MMSE scores found to be predictive of functional improvement in patients with stroke following rehabilitation; discharge destination; developing functional dependence at a 3-year follow-up interval; ambulatory level; length of hospital stay such that for patients with moderate dementia; death. Floor/Ceiling effects: Folstein, Folsten, and McHugh (1998) reported that the MMSE demonstrates marked ceiling effects in younger intact individuals and marked floor effects in individuals with moderate to severe cognitive impairment.</td>
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<td><strong>Repeatable Battery for the Assessment of Neuropsychological Status (RBANS)</strong></td>
<td>brief neurocognitive battery with four alternate forms.</td>
<td>The content of the RBANS consists of neurocognitive test paradigms including tests</td>
<td>25 min</td>
<td>NA in a stroke population</td>
<td>We present a rare case of stroke in a 22-year-old psychiatric patient, who received neuropsychological evaluations before and after sustaining a right middle cerebral artery (MCA) stroke. The RBANS demonstrated...</td>
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<td><a href="http://www.rbans.com">http://www.rbans.com</a></td>
<td>measuring immediate and delayed memory, attention, language, and visuospatial skills</td>
<td>for: immediate memory, visuospatial/constructional, language, attention, and delayed memory. Population: Not specific</td>
<td></td>
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<td>sensitivity to post-stroke changes despite pre-stroke cognitive impairments and a complex psychiatric overlay, with the Visuospatial/Constructional index being one of the most sensitive indicators of right hemisphere dysfunction. Line Orientation fell from normal to defective levels; these findings were associated with decline in related standard neuropsychological measures.</td>
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Table 7.2 C  Summary of Select Medications for Vascular Cognitive Impairment

*Coming Soon*
Reference List for Vascular Cognitive Impairment


Erkinjuntti T, Skoog I, Lane R, Andrews C. Potential long-term effects of rivastigmine on disease progression may be linked to drug effects on vascular changes in Alzheimer brains. Int J Clin Pract 2003;57(9):756-760.

Ferro JM, Crespo M. Young adult stroke: neuropsychological dysfunction and recovery. Stroke 1988;19:982-6


Hoffmann M. Higher cortical function deficits after stroke: an analysis of 1,000 patients from a dedicated cognitive stroke registry. Neurorhabil Neural Repair 2001;15:113-27


Paediatric References For Vascular Cognitive Impairment Following Stroke


Nass RD, Trauner D. Social and affective impairments are important recovery after acquired stroke in childhood. CNS Spectr 2004; 9: 420-434.


Westmacott R, Barry V, MacGregor D, deVeber GA. Age at stroke and involvement of cortex modulate cognitive outcome in children with basal ganglia infarcts. 2007b.
