Chemotherapy-related cognitive impairment (CRCI) was first described in the 1970s, but significant recognition of CRCI did not emerge with consistency until the late 1990s. Estimates of frequency now range from 17%–75%, and evidence suggests that CRCI, or “chemobrain” as it is referred to in the lay literature, is of significant concern to patients. A variety of potentially associated factors have been identified, including age, education level, intelligence, and social support; anxiety, depression, and fatigue; disease site, stage, and comorbidities; treatment regimen, timing, duration, and concomitant therapies; and hormonal levels, cytokine levels, damage to neural progenitor cells, and the presence of the apolipoprotein E 4 allele. Controversy exists as to the most suitable neurocognitive tests to evaluate this sequela of treatment. Neuroimaging techniques are beginning to reveal affected areas of the brain. A neuropsychologist is essential for the assessment, diagnosis, and recommendation of appropriate management strategies for this patient population.

Oncology nurses should be aware of available resources, such as relevant Web sites, support groups, neuropsychologists, and cognitive retraining programs, and provide support for patients concerned about or experiencing CRCI.

At a Glance

- Neuroimaging tests may prove useful in prospective trials to identify areas of the brain affected by standard-dose chemotherapy and provide a rationale for patient self-reports of cognitive impairment despite normal performance on neuro-psychological tests.
- Current neuropsychologic test batteries are criticized for lack of real-life challenges experienced by cancer survivors.
- Neuropsychologists are an important component of the multidisciplinary team needed to provide quality cancer care to patients experiencing treatment-related cognitive changes.

Jamie S. Myers, RN, MN, AOCN®, is a doctoral student in the School of Nursing at the University of Kansas Medical Center in Edwardsville. (Submitted November 2008. Accepted for publication December 31, 2008.)

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Cognitive impairment in patients with cancer has long been acknowledged as a sequel of disease that is primary or metastatic to the central nervous system (CNS), intrathecal chemotherapy for leukemias or CNS tumors, and some high-dose chemotherapy regimens (Silberfarb, 1983). Silberfarb noted in two studies in the 1980s (Silberfarb; Silberfarb et al., 1980) that evidence of cognitive impairment could be associated with commonly used chemotherapeutic agents of the time. He proposed that a high prevalence of subtle and mild cognitive impairment could be assessed in patients receiving chemotherapy. Silberfarb described decreased cognitive function as subtle loss of ability to think abstractly; loss of cognitive flexibility, such as the ability to sequentially order alternating numbers and letters; difficulty with finding the right word; slight forgetfulness; and complaints of mental fatigue, difficulty concentrating, and irritability. Further contributions to the literature about this phenomenon did not become common until late the 1990s.

Much of the CRCI literature has been devoted to retrospective trials for patients with breast cancer because they typically have a good prognosis and survival time allows evaluation of concurrent and long-term sequela (Ahles & Saykin, 2002; Castellon et al., 2004; Inagaki et al., 2007; Jansen, Miaskowski, Dodd, & Dowling, 2005; Klemp, Stanton, Kimler, & Fabian, 2006; Kreukels et al., 2006; Olin, 2001; O’Shaughnessy, 2003; Schagen et al., 1999; Servaes, Verhagen, & Bleijenberg, 2002; Wefel et al., 2004). Ahles and Saykin have published a significant body of literature identifying CRCI as a common sequela to standard-dose chemotherapy, with the recommendation for prospective trials to evaluate causal hypotheses and concomitant factors (Ahles & Saykin, 2001, 2002, 2007; Ahles et al., 2002). A workshop in April 2003 brought together a multidisciplinary group of medical oncologists, radiologists, clinical and experimental psychologists, and patient advocates to compare research results and discuss collaborations. The group deliberated on how to enhance prospective clinical trials to address the scope of the problem; evaluation of cognitive problems; possible mechanisms; and prevention, treatment, and rehabilitation (Tannock, Ahles, Ganz, & Van Dam, 2004). Consensus was achieved that the following were needed.

- Large-scale prospective clinical trials with longitudinal design and appropriate controls to evaluate the probability and magnitude of CRCI, predictive factors, and mechanisms
- Research to identify neuropsychological tests that are sensitive to the subtle changes seen with CRCI and the development and validation of self-report forms
- Expansion of clinical trials to study CRCI in diseases other than breast cancer, particularly including male patients, and to address the influence of hormonal changes
- Priority funding of research to address causal mechanisms, including the development of animal models and the inclusion of imaging techniques

The exact etiology of CRCI is not known, but a variety of etiologies have been proposed, including direct injury to cerebral gray and white matter, microvascular injury (Wefel et al., 2004), DNA damage and oxidative stress (Ahles & Saykin, 2007; Chen, Jungsuwadee, Vore, Butterfield, & St. Clair, 2007), cytokine-induced inflammatory response (Ahles & Saykin, 2007), chemotherapy-induced anemia (Mancuso, Migliorino, De Santis, Saponiero, & De Marinis, 2006; Massa, Madeddu, Lusso, Gramignano, & Mantovani, 2006), and chemotherapy-induced menopause (Jansen, Miaskowski, Dodd, & Dowling, 2005).

Preclinical investigation has highlighted a potential relationship between injury to neural progenitor cells (NPCs), impaired maintenance of white matter integrity, and subsequent cognitive impairment (Dietrich, Han, Yang, Mayer-Proschel, & Noble, 2006; Dietrich, Monje, Wefel, & Meyers, 2008; Han et al., 2008). Dietrich, Han, et al. noted that self-renewing, lineage-committed NPCs and nondividing mature oligodendrocytes (myelin-forming cells) are the most vulnerable cell populations to chemotherapeutic agents. Repetitive exposure to chemotherapeutic agents exceeded cellular repair potential and resulted in long-term suppression of cell division and apoptosis in the subventricular zone, hippocampus, and major white matter tracts of the CNS in animal models.

The presence of the apolipoprotein (APOE) E 4 allele may predispose patients to cognitive impairment (Ahles et al., 2002). APOE is “a complex glycolipoprotein that facilitates the uptake, transport and distribution of lipids” and appears to have a role in neuronal repair after injury (Ahles & Saykin, 2007, p. 198). The E 4 allele is associated with Alzheimer disease and poor recovery from stroke and traumatic brain injury. Some prospective trials to evaluate CRCI now include genetic measurements to assess whether a genetic predisposition to more significant and longer-lasting injury from chemotherapy exists (Ahles & Saykin, 2007). Those prospective trials may help answer the question of whether some patients are genetically predisposed to long-term damage, and the results could have significant impact on treatment options.

Patients with cancer experience multiple concurrent symptoms from the disease and treatment (Miaskowski et al., 2006).
The term “symptom cluster” has been defined to include two to three concurrent, related symptoms (Dodd, MiaoKowski, & Paul, 2001; Kim, McGuire, Tulman, & Barsevick, 2005). Symptom cluster studies have examined relationships among fatigue, pain, sleep disturbances, and depression. Dodd et al. reviewed a number of trials and found that fatigue, pain, and depression were interrelated. Relationships also existed among pain, fatigue, and sleep disturbance (Dodd et al.). According to Barsevick (2007), symptom assessment should include fatigue, sleep disturbance (insomnia), pain, and depression because of evidence of clustering. Meyers (2000) noted that fatigue, pain, and anemia may contribute to cognitive deficits.

The symptom experience known as sickness behavior has been postulated to be related to the release of proinflammatory cytokines as a response to the disease and its treatment (Cleeland et al., 2003). Sickness behavior includes fever, fatigue, lethargy, muscle aches, decreased appetite, decreased ability to concentrate, decreased social interaction, and general behaviors consistent with the conservation of energy (Parnet, Kelley, Bluthe, & Dantzler, 2002; Pollmacher, Haack, Schuld, Reichenberg, & Yirmiya, 2002; Wilson, Finch, & Cohen, 2002). Cognitive impairment has been hypothesized to be a component of sickness behavior from cytokine release (Lee et al., 2004).

**Neuroimaging**

Advances in neuroimaging have provided a number of opportunities to objectively evaluate structural and functional changes in the brain. The techniques are beginning to be used to assess the impact of standard-dose chemotherapy on the brain. The earliest work in this area was conducted in relationship to high-dose chemotherapy associated with bone marrow transplantation in which magnetic resonance (MR) studies for 13 patients were obtained (Brown et al., 1995). The purpose of the study was to determine if white matter changes in this patient population were associated with neurochemical disturbances. Single-voxel proton MR spectroscopy also was obtained in vivo for 12 of 13 patients associated with neurochemical disturbances. Both studies were consistent with the conservation of energy (Parnet, Kelley, Bluthe, & Dantzler, 2002; Pollmacher, Haack, Schuld, Reichenberg, & Yirmiya, 2002; Wilson, Finch, & Cohen, 2002). Cognitive impairment has been hypothesized to be a component of sickness behavior from cytokine release (Lee et al., 2004).
not described (see Table 1). Four of the women had received adjuvant chemotherapy in combination with the anti-estrogenic agent tamoxifen, and four had received chemotherapy alone. Comparisons were made with two breast cancer survivors who were chemotherapy naïve and two healthy participants with no history of breast cancer. The 12 women underwent repeat neuropsychological testing within four days of PET scanning. Brain metabolic activity was assessed in 26 regions of the brain. Results were compared with previously established reference ranges. Significant abnormalities in activity relative to the reference group were seen in the eight women who had received chemotherapy (9% below normal, p < 0.0001 in the superior frontal gyrus of the dorsolateral prefrontal cortex and Broca’s area [see Figure 1] with its contralateral counterpart). Hypometabolism was more severe in the four women who received tamoxifen in combination with chemotherapy. Subsequent investigation was conducted by the same group to evaluate the relationship of cerebral blood flow and metabolism with cognitive function after chemotherapy for breast cancer (Silverman et al., 2007). The sample was comprised of 16 right-handed women with a history of breast cancer treated with chemotherapy within the past 5–10 years; 11 also had received tamoxifen therapy. Comparisons were made to eight right-handed women who had not received chemotherapy, although some had histories of breast cancer. A standard reference group of 10 additional healthy women was included who previously had undergone PET studies. The authors cited previous work in which the Rey-Osterrieth Complex Figure (ROCF) Delayed Recall Task had been used to demonstrate the most significant cognitive deficit (p = 0.0007) in a larger study (n = 72) evaluating neurocognitive performance following chemotherapy for breast cancer survivors. Cognitive testing was conducted within 72 hours of PET scanning and was supervised and statistically assessed by a licensed clinical neuropsychologist. Patients were evaluated by PET imaging during performance of control (resting) and memory tasks (standard

<table>
<thead>
<tr>
<th>TEST</th>
<th>DESCRIPTION</th>
<th>COGNITIVE DOMAINS EVALUATED</th>
</tr>
</thead>
<tbody>
<tr>
<td>California Verbal Learning Test</td>
<td>The respondent must memorize a shopping list and recall the list after a time delay and again after presentation of an alternate list.</td>
<td>Memory</td>
</tr>
<tr>
<td>Functional Assessment of Cancer Therapy–Cognitive Function</td>
<td>Self-report on 33 scaled items (version 3) for cognitive function; includes evaluation of mental acuity, attention and concentration, memory, verbal fluency, functional interference, deficits observed by others, change from previous function, and impact on quality of life.</td>
<td>Self-report of cognitive functioning</td>
</tr>
<tr>
<td>Grooved Pegboard</td>
<td>Uses a board with 25 randomly positioned slots; a set of pegs must be rotated to be inserted correctly into matching slots, one at a time and as quickly and accurately as possible. Dominant and nondominant hands are tested.</td>
<td>Motor function</td>
</tr>
<tr>
<td>Mini-Mental State Examination</td>
<td>A 30-item (10-minute) questionnaire used to screen for dementia; scores are sometimes used as cutoff points for further neuropsychological examination. It includes simple questions related to orientation, repetition of word lists, math problems, language, comprehension, and motor skills.</td>
<td>Memory</td>
</tr>
<tr>
<td>Repeatable Battery for the Assessment of Neuropsychological Status</td>
<td>An abbreviated neuropsychological test battery (20–30 minutes) designed to evaluate a number of cognitive domains; individual tests include list learning, story memory, figure copy, line orientation, picture naming, semantic fluency, list recall, list recognition, and figure recall.</td>
<td>Memory (immediate and delayed), visuospatial ability, language, and attention</td>
</tr>
<tr>
<td>Rey-Osterrieth Complex Figure Test Delayed Recall Task</td>
<td>The respondent must reproduce a complicated line drawing, first by copying and then from memory.</td>
<td>Visuospatial ability, memory, and executive function</td>
</tr>
<tr>
<td>Stroop Interference</td>
<td>The respondent is provided with a sheet or card of words that name a variety of colors; however, the words are printed in a different color ink than the color that the word names. The respondent must substitute an alternative response for a more obvious reaction (i.e., naming the ink color of a word denoting a different color).</td>
<td>Executive function, attention, and concentration</td>
</tr>
<tr>
<td>Trail Making Test A and B</td>
<td>Timed two-part test in which one must draw lines to connect consecutively numbered circles on one work sheet (part A) and then connect the same number of consecutively numbered and lettered circles on another worksheet by alternating between consecutive letters and numbers (part B)</td>
<td>Visual, conceptual, and visuomotor tracking; psychomotor speed; attention and concentration; and processing speed</td>
</tr>
<tr>
<td>Wechsler Adult Intelligence Scale Digit Symbol</td>
<td>Involves a symbol substitution task; consists of pairing numbers to nonsense symbols as quickly as possible</td>
<td>Visuomotor coordination and psychomotor performance</td>
</tr>
<tr>
<td>Wechsler Memory Scale–Revised</td>
<td>Tests immediate and delayed recall from a short paragraph</td>
<td>Memory</td>
</tr>
</tbody>
</table>
word-pair association cognitive tasking protocol). Forty-two brain regions were assessed. The group who had received chemotherapy had significant increase in activity (2.3% increase, p < 0.0005) in the inferior frontal gyrus; untreated patients had a slight increase (p = 0.96). Significant activity also was seen in the treatment group in the contralateral posterior cerebellum near the midline and the superior frontal gyrus (p = 0.01, p = 0.046; respectively). The chemotherapy group performance on the ROCF Delayed Recall Task averaged 3.2 points (13%) lower than the control group. No significant differences were seen in resting metabolism between the groups. Patients who received tamoxifen therapy had significantly lower metabolism in the lentiform nucleus of the basal ganglia (p < 0.01). The results lent support to patients' perceptions of mental slowness and diminished ability to maintain attention, concentrate, and remember things. The three conclusions from the study were (a) altered cortical activation associated with performance of a memory task could be characterized as involving greater recruitment of frontal cortical tissue, (b) chemotherapy-related changes in cerebral activation may be associated with CRCI, and (c) increased frontal activation may be a compensatory response to lower resting metabolism in this area of the brain. The authors acknowledged that the study was limited by the small sample size.

Effects of chemotherapy on hippocampal size also have been evaluated (Yoshikawa et al., 2005). Yoshikawa et al. noted that hippocampal damage has been associated with memory deficits. The authors hypothesized that chemotherapy may damage the hippocampus (see Figure 2), leading to cognitive impairment. The authors evaluated Japanese breast cancer survivors who had (n = 44) or had not (n = 31) received chemotherapy. The participants were at least three years past diagnosis and had completed a full course of chemotherapy treatment. Hippocampal volume was assessed by MRI, and the Wechsler Memory Scale–Revised (WMS-R) assessed delayed recall and retention percentage as surrogate markers of hippocampal function. No significant differences were observed in memory function or hippocampal volume between the two groups, although a slight decrease in attention and concentration for the group who had received chemotherapy was noted. The authors speculated that brain regions other than the hippocampus may be related to memory impairment (such as the prefrontal cortex) and that the timeframe of three years prior to assessment may have allowed time for cognitive recovery. A follow-up study was performed to explore regional brain volume differences at intervals of one and three years following treatment with chemotherapy for breast cancer (Inagaki et al., 2007). Patients surviving 3–15 months after diagnosis were eligible and agreed to participate in the one-year evaluation. Of them, 105 also agreed to participate in the three-year evaluation. Age-matched controls participated at one (n = 55) and three years (n = 37). The WMS-R was used to estimate memory function through the indices of attention and concentration, immediate visual memory, immediate verbal memory, and delayed recall. MRIs were conducted at one and three years. Smaller volumes of the right prefrontal and parahippocampal gyrus were noted in the chemotherapy group at one year, whereas no difference in volume was observed at three years. A subanalysis showed an association between smaller volume and impairment in memory function (attention and concentration and visual memory indices). The authors concluded that structural differences in the superior and prefrontal gyrus may be associated with CRCI and volume changes appear to recover over time.

In a particularly interesting article, monozygotic twins were assessed by functional and structural MRI (Ferguson, McDonald, Saykin, & Ahles, 2007). One twin had received chemotherapy for breast cancer and the other had no history of the disease. The sisters were evaluated in combination with a memory task, and standardized neurocognitive testing was performed. The twins completed self-report tools for cognitive function, anxiety,
and depression. The women were APOE E 4 allele carriers. Both demonstrated white matter hyperintensities on structural MRI, which is known to occur in carriers of the APOE E 4 allele; however, they were significantly pronounced in the twin who had received chemotherapy (in right and left hemispheres). The twin who had received chemotherapy had significantly higher self-reports of cognitive dysfunction. Neither twin demonstrated anxiety or depression, and both scored within the normal range on standardized neurocognitive tests. The twin who had received chemotherapy demonstrated much broader spatial extent of activation in typical working memory circuitry (bifrontal and biparietal regions) but performed with equal accuracy to the untreated twin. The authors attributed the difference in spatial activation and increased cortical activity to the recruitment of a broader neural network to accomplish a task, indicating that compensation was employed. Such analysis may provide additional information regarding the areas of the brain affected by chemotherapy and a rationale for patient self-reports of cognitive dysfunction despite normal performance on standardized neuropsychological tests.

Neuropsychological Testing Challenges

One of the major challenges in the study of CRCI is the selection of appropriate assessment tools. As mentioned previously, the cognitive changes observed in this patient population are subtle. Tests designed to assess gross changes in neurocognitive function associated with severe dementia or head injury are not appropriate for patients experiencing CRCI. Patients who are well educated with high baseline cognitive function may continue to score normally on neurocognitive tests, even though they perceive deficits that interfere with their daily function and QOL (Wefel et al., 2004). The Mini-Mental State Examination (MMSE) has been criticized for those reasons (Meyers & Wefel, 2005). At best, the MMSE may be used as a baseline screen to exclude patients from a prospective trial who have significant cognitive deficits prior to the initiation of therapy.

The battery of neuropsychological tests that would normally be employed to conduct a full cognitive assessment may range in length from four to seven hours (Freeman & Broshek, 2002). Patient burden should be a consideration in determining whether that amount of testing occurs in one or more sessions. An additional challenge exists for patients experiencing the fatigue associated with a cancer diagnosis and treatment (Butt et al., 2008). Several hours of testing may not be practical. In addition, in the context of a clinical trial, the time and expense involved in extensive testing may preclude a complete examination from being included in the protocol (Freeman & Broshek). The search for succinct but reliable clinical tools is ongoing (Freeman & Broshek, 2004). Participants from the multidisciplinary workshop mentioned earlier in this article suggested a two-stage approach to cognitive changes observed with CRCI as well as to develop and validate self-report forms (Tannock et al., 2004). As important as objective tests of cognitive function are, patient perception of cognitive function and the resultant impact on QOL remain important aspects of the assessment process (Minisini et al., 2004).

Consensus has not yet been reached regarding the best neuropsychological tests to employ, but efforts have been made to compare and contrast those available (Freeman & Broshek, 2002). Freeman and Broshek published initial results of a study to evaluate tests selected for sensitivity to mild cognitive impairment in other patient populations. The tests were selected to measure attention and concentration, processing efficiency, verbal and visual memory, executive function, sensorimotor function, visuoconstruction, naming, and verbal fluency. At the time of publication, 17 women had been enrolled. The sample of women had received chemotherapy or currently were receiving chemotherapy following a surgical procedure. Significant deficits were observed in the tests, which were the Repeatable Battery for the Assessment of Neuropsychological Status, Grooved Pegboard Nondominant Hand, and Stroop Interference tests. Freeman and Broshek would like to pare the assessment down to a battery of tests of 30–40 minutes.

Additional challenges of the current process of neuropsychological testing for patients experiencing cognitive impairment revolve around the difficulty in replication of a real-life situation. Typically, neuropsychological testing occurs in a laboratory-like environment that has little overlap with a patient’s everyday experience (Schagen et al., 2002). Current testing procedures are criticized for low ecologic relevance and sterile conditions with minimal distraction. Patients with cancer who self-report CRCI describe an inability to multitask, which is difficult to replicate in a testing situation (Cimprich, So, Ronis, & Trask, 2005).

Role of the Neuropsychologist

Neuropsychologists are doctorally prepared psychologists who specialize in the study of brain behavior relationships. Neuropsychologists are skilled in the administration and interpretation of neuropsychological tests to link behaviors to underlying normal and abnormal brain processes. Neuropsychologists collaborate with multidisciplinary teams in the use of functional MRI techniques (American Psychological Association, 2004). One
The role of the neuropsychologist is essential in the assessment, diagnosis, and counseling needed for patients experiencing cognitive changes resulting from chemotherapy. As more information about which regimens and risk factors associated with CRCI become available, referrals for baseline neuropsychological testing will provide valuable information with which to compare ongoing treatment-related changes and early identification of deficits and implementation of interventions. Freeman and Broshek (2002) stated that “it is critical to have the input of neuropsychologists when choosing methods of assessing neurocognitive functioning” (p. S92). Meyers (2000) stressed the importance of “multidisciplinary assessment of neurocognitive complaints in order to maximize patients’ ability to function at the highest level of independence for the longest period of time” (p. 78). Meyers advocated the use of focused rehabilitation efforts in this patient population. She described the use of cognitive rehabilitation and vocational rehabilitation (strategies usually employed for patients with traumatic brain injuries) as being applicable for patients experiencing CRCI. She also recommended education about expected treatment sequelae, counseling, and support groups to prevent patients experiencing side effects from feelings of isolation. Meyers noted that many oncologists and oncology nurses may be unaware of CRCI and may not speak to patients and families about it.

As the body of literature grows, awareness is increasing. Centers are developing Web sites to facilitate education about available resources (see Figure 3). The need for consulting a neuropsychologist also is being recognized (Grober, 2002). Grober outlined the need for a thorough medical, psychological, developmental, and biographic history in addition to several testing sessions followed by a verbal and written consultation summary and treatment plan. Patients who do not have access to that aspect of the multidisciplinary team experience a negative impact on QOL because of adverse effects on vocational and avocational interests, mood, and self-esteem (Grober).

Costs for neuropsychological evaluation vary based on region, practitioner, and the selection of neuropsychologic tests to include. Anecdotal experience in the southeastern region of the United States indicates a range of $1,000–$1,500 for neuropsychological screening and $1,800–$2,800 for a comprehensive evaluation. Insurance may reimburse the evaluation if a prescription is provided by the referring physician. In these cases, testing was considered to be medical and paid through medical rather than mental health benefits.

**Intervention**

The Memory and Attention Adaptation Training is a brief cognitive behavioral treatment aimed at helping breast cancer survivors manage CRCI (Ferguson, Ahles, et al., 2007). The program was piloted in 29 women, with a mean survival of eight years after chemotherapy. It was comprised of four cognitive behavioral components: education on memory and attention; self-awareness training; self-regulation emphasizing arousal reduction through relaxation training, activity scheduling, and pacing; and cognitive compensatory strategies training. Patients were provided with a workbook, four individual visits (one per month), and four phone contacts (between visits). Visits and phone contacts were conducted to provide support and review compensatory strategies designed to cope with high-risk situations for memory failure. The strategies included covert verbal self-guidance during task performance, verbal rehearsal of auditory information, schedule making, external cuing, and outlining written material. Participants were assessed at baseline, just following completion of chemotherapy, and two and six months later. Measures included the Multiple Ability Self Report Questionnaire, QOL-Cancer Survivors Scale, and a rating of satisfaction with the program (scale of 0–8, with higher scores indicating higher satisfaction). Five neuropsychological tests were employed and improvements were observed in self-report of cognitive function, QOL, and neuropsychological test performance. However, generalizability may be limited because of the high educational level of participants and the possibility of practice effect on the neuropsychological tests. Other interventions being tested include erythropoietin-stimulating agents to correct anemia, neurostimulants, and investigation of neuroprotective agents (Tannock et al., 2004).

**Conclusion**

CRCI is a significant concern to the healthcare community because of the estimated prevalence and impact on patients’ QOL.
As causal factors are as of yet undetermined, much work remains to be done to ascertain the etiologies and risk factors for this problem. Once causal factors are specified, preventative strategies can be developed and applied. Future research should be focused on the determination and development of appropriate neurocognitive testing. Tests should be selected or developed based on sensitivity to the subtle changes observed in patients with cancer and the need to replicate a real-life setting for assessment of patients’ ability to focus on tasks despite competing stimuli. Patient burden and fatigue should be taken into consideration when tools are selected and developed. Assessment of potential confounding and exacerbating factors, such as fatigue, depression, anxiety, pain, hormonal status, anemia, and genetic makeup, should be included. The use of neuroimaging techniques should accompany neuropsychological assessments to further define the areas and function of the brain that are affected.

Oncology nurses are in an excellent position to educate patients, families, and the community about the risks of CRCI and to assess the community for resources such as access to support groups, neuropsychologists, and cognitive retraining programs. Oncology nurses may be instrumental in the development of clinical pathways to identify patients experiencing cognitive changes and facilitate appropriate referrals for neuropsychological testing and intervention.

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Author Contact: Jamie S. Myers, RN, MN, AOCN®, can be reached at jamyers@swbell.net, with copy to editor at CJONEditor@ons.org.

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