Clinical Policy Bulletin: Cognitive Rehabilitation

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Policy

Note: Coverage of outpatient cognitive rehabilitation is subject to applicable benefit plan terms and limitations for physical and occupational therapy (see CPB 0250 - Occupational Therapy Services and CPB 0325 - Physical Therapy Services). Please check benefit plan descriptions for details.

I. Aetna considers cognitive rehabilitation as adjunctive treatment of cognitive deficits (e.g., attention, language, memory, reasoning, executive functions, problem solving, and visual processing) medically necessary when all of the following are met:

   A. Neuropsychological testing has been performed and neuropsychological results will be used in treatment-planning and directing rehabilitation strategies, and
   B. The cognitive deficits have been acquired as a result of neurologic impairment due to traumatic brain injury, brain surgery, stroke, or encephalopathy, and
   C. The member has been seen and evaluated by a neuropsychiatrist or neuropsychologist, and
   D. The member is expected to make significant cognitive improvement (e.g., member is not in a vegetative or custodial state).

Note: Cognitive rehabilitation may be performed by an occupational therapist, physical therapist, speech/language pathologist, neuropsychologist, or a physician.

Note: According a review article on cognitive rehabilitation (Ciceron et al, 2000), rehabilitation for visuo-spatial deficits generally entails 20 1-hour sessions delivered over the course of 4 weeks. For language and communication deficits, patients usually receive 8 hours of weekly therapy, beginning at 4 weeks post-onset and continuing up to 48 weeks post-onset. Courses of cognitive rehabilitation substantially longer than these durations may be reviewed for medical necessity.
II. Aetna considers cognitive rehabilitation experimental and investigational for all other indications, such as the treatment of epilepsy/seizure disorders, mental retardation, cerebral palsy, dementia (e.g., from Alzheimer's disease, HIV-infection*, or Parkinson's disease), cognitive decline in multiple sclerosis and chronic obstructive pulmonary disease, Wernicke encephalopathy, and behavioral/psychiatric disorders such as attention-deficit/hyperactivity disorder, depression, schizophrenia, social phobia, substance abuse disorders, and pervasive developmental disorders including autism, as it has not been proven to be effective for these indications.

*Note: Cognitive rehabilitation is considered medically necessary for encephalopathy due to HIV when medical necessity criteria in section I above are met.

III. Aetna considers coma stimulation, also known as the "Responsiveness Program" (cognitive remediation of comatose persons), coma arousal program/therapy, sensory stimulation, and multi-sensory stimulation programs, experimental and investigational for coma and persistent vegetative state because its effectiveness has not been established.

Background

Cognitive rehabilitation offers retraining in the ability to think, use judgment, and make decisions. The focus is on correcting deficits in memory, concentration and attention, perception, learning, planning, sequencing, and judgment. A neuropsychologist, aided by other specialists (e.g., occupational therapists, speech and language pathologists) may be asked to evaluate the level and kind of cognitive dysfunction following traumatic brain injury (TBI), and they may reassess the individual over time to measure recovery.

The goals of cognitive rehabilitation are to enhance the person's capacity to process and interpret information and to improve the person's ability to function in all aspects of family and community life. Restorative training focuses on improving a specific cognitive function, whereas compensatory training focuses on adapting to the presence of a cognitive deficit. Compensatory approaches may have restorative effects at certain times. Some cognitive rehabilitation programs rely on a single strategy (e.g., computer-assisted cognitive training), while others use an integrated or inter-disciplinary approach. A single strategy program can target either an isolated cognitive function or multiple functions concurrently.

Although the interventions falling under the rubric of cognitive rehabilitation are heterogeneous, a Consensus Panel convened by the National Institutes of Health noted that these interventions share certain characteristics in that they are structured, systematic, goal-directed, and individualized and they involve learning, practice, social contact, and a relevant context.

A report of a consensus conference sponsored by the National Institute of Child Health and Human Development (NIH, 1999) concluded that, despite many descriptions of specific strategies, programs, and interventions, limited data on the
effectiveness of cognitive rehabilitation programs are available because of heterogeneity of subjects, interventions, and outcomes studied. Outcome measures present a special problem, since some studies use global "macro"-level measures (e.g., return to work), while others use "intermediate" measures (e.g., improved memory). These studies also have been limited by small sample size, failure to control for spontaneous recovery, and the unspecified effects of social contact. Nevertheless, a number of programs have been described and evaluated. Despite these limitations in evidence, the consensus conference report concluded: "Evidence supports the use of certain cognitive and behavioral rehabilitation strategies for individuals with TBI [traumatic brain injury] in particular circumstances. These interventions share certain characteristics in that they are structured, systematic, goal-directed, and individualized and they involve learning, practice, social contact, and a relevant context."

Cognitive exercises, including computer-assisted strategies, have been used to improve specific neuropsychological processes, predominantly attention, memory, and executive skills. A NIH Consensus Statement notes that both randomized controlled studies and case reports have documented the success of these interventions using intermediate outcome measures. Certain studies using global outcome measures also support the use of computer-assisted exercises in cognitive rehabilitation. An AHCPR Evidence Report/Technology Assessment concluded that there is some evidence that compensatory cognitive rehabilitation reduces anxiety and improves self-concept and relationships for people with TBI.

Compensatory devices, such as memory books and electronic paging systems, are used both to improve particular cognitive functions and to compensate for specific deficits. Training to use these devices requires structured, sequenced, and repetitive practice. According to a NIH Consensus Statement, the efficacy of these interventions has been demonstrated.

Interventions in cognitive rehabilitation are being developed and have only recently been subjected to the scientific inquiry. The efficacy of cognitive rehabilitation so far has been measured by its objective influence on function and the subjective value of these changes to the individual. An NIH Consensus Conference Report (NIH, 1999) stated: "It is important to recognize that a great deal of the scientific evidence to support the use of these approaches derives from relatively limited studies that should be replicated in larger, more definitive clinical trials."

Amato et al (2006) stated that despite its frequency and high functional impact, very little is known about effective strategies for managing cognitive impairment in patients with multiple sclerosis (MS). Disease-modifying drugs may prevent or reduce the progression of cognitive dysfunction by containing the development of new cerebral lesions. Available evidence has provided inconsistent findings, with neuropsychological effects documented only in 1 trial. Moreover, pilot studies have tested symptomatic therapies for fatigue, a frequent symptom in MS, which may share a common physiopathological substrate with cognitive dysfunction. Small studies with amantadine, pemoline, 4-aminopyridine and 3-4 aminopyridine have provided mainly negative results. Acetylcholinesterase inhibitors used to treat Alzheimer's disease (e.g., donepezil, rivastigmine, and galantamine) have recently been tested in other cognitive disorders, including MS. The majority of pilot trials with acetylcholinesterase inhibitors in MS have provided promising
results, and the donepezil study recently published by Krupp and colleagues represented a major development in this field. As for non-pharmacological interventions based on cognitive rehabilitation, few studies have used an experimental approach and, in general, results have been disappointingly negative. The authors noted that further research is clearly needed in this area.

In an evidenced-based review of cognitive rehabilitation for persons with MS, O'Brien et al (2008) concluded that cognitive rehabilitation in MS is in its relative infancy. More methodologically rigorous research is needed to determine the effectiveness of various cognitive rehabilitation interventions.

A Cochrane systematic evidence review by Thomas et al (2006) found "some evidence of effectiveness" of cognitive rehabilitation on cognitive outcomes in persons with MS who have cognitive impairments, although the authors found that "this was difficult to interpret because of the large number of outcome measures used".

A systematic evidence review by the BlueCross BlueShield Association Technology Evaluation Center (BCBCA, 2008) concluded that cognitive rehabilitation for traumatic brain injury does not meet the TEC criteria. An important weakness in the literature on cognitive rehabilitation is that many clinical trials report impacts of cognitive rehabilitation on cognitive tests rather than on health outcomes. The assessment stated that "[d]emonstration of the effectiveness of cognitive rehabilitation ... requires prospective, randomized designs that employ validated measures of health outcomes."

Coma stimulation refers to clinical intervention related to cognitive rehabilitation by attempting to improve or increase the rate of recovery and arousal of the comatose patient through increasing sensorimotor input. It has been suggested that increasing baseline stimulation to critical brain structures, including the reticular activating system in particular, promotes arousal and recovery of these patients. Suggestive findings of such approaches include reports of increased arousal and improvement in findings on electroencephalograms in prolonged vegetative states following dorsal column stimulation and improvement of the comatose patient's condition. However, there are no published studies that confirm the overall efficacy of such approaches in altering the recovery patterns of comatose patients. A Cochrane systematic evidence review (Lombardi et al, 2002) concluded that "there is no reliable evidence to support the effectiveness of multisensory programmes in patients in coma or vegetative state."

A guideline of "Guidance on diagnosis and management of the vegetative state" from the Royal College of Physicians (2003) concluded that "there is no evidence that constant stimulation of someone who is in a vegetative state can bring about improvement in the long-term outcome."

In a randomized, controlled study, Incalzi et al (2008) examined the effects of cognitive training in patients with hypoxemic chronic obstructive pulmonary disease (COPD). This study consisted of 105 COPD patients with at rest (n = 36) or effort (n = 69) hypoxemia and free from concurrent dementing diseases. Neuropsychological assessment included a screening test, the Mini Mental State Examination (MMSE), and a standardized confirmatory battery of neuropsychological tests, the Mental Deterioration Battery (MDB). After baseline
assessment, patients were randomized to receive standardized multi-dimensional care (standardization of pharmacological therapy, health education, selection of inhalers according to patient's ability, respiratory rehabilitation, nutritional counseling, oxygen therapy, and control visits) with \( n = 53 \) or without \( n = 52 \) cognitive training aimed at stimulating attention, learning, and logical-deductive thinking. Cognitive performance was re-assessed after 1.5, 4, and 6 months. The analysis of variance for repeated measures (ANOVA) having the group membership (study versus control) as grouping factor was used to assess changes in cognitive performance. Both intervention and control groups showed no significant changes in cognitive performance except for a trend toward improvement in verbal fluency and verbal memory, but cognitive intervention had no significant effect. The authors concluded that cognitive training seems ineffective in COPD.

Brissart and colleagues (2011) noted that cognitive impairment is now well-known in MS. However, few rehabilitation interventions are proposed or really efficient. These investigators presented a review of cognitive rehabilitation intervention research conducted in people with MS, regarding different findings about episodic memory, working memory, attention and executive function disorders in MS. A search of Medline (yield 20 papers) and of PsychInfo (yield 1 article), using combinations of the following terms: cognitive rehabilitation, multiple sclerosis, cognitive therapy, neuropsychological rehabilitation, in the title or in the abstract, from 1960 to March 2010, excluding animal studies. Episodic memory rehabilitation studies appear to be promising. Programs on working memory, attention and executive functions are in the very early phases. The authors concluded that results are encouraging and allow specific recommendations for future research regarding (i) inclusion criteria, often not defined, (ii) a specific baseline adapted to the program of rehabilitation, (iii) a control measure regarding program efficiency, and (iv) a role for the psychologist (presence and advice during the program).

Note: Cognitive rehabilitation should not be confused with cognitive behavior therapy. Cognitive behavior therapy (also known as cognitive therapy) is a form of psychotherapy that emphasizes the role of thought patterns in moods and behaviors.

Georgiopoulos et al (2010) performed a systematic review of the proposed medical or surgical treatments in patients in chronic vegetative state (VS) or minimally conscious state (MCS), as well as of their mechanisms of action and limitations. For this review, these researchers have agreed to include patients in VS or MCS having persisted for over 6 months in post-traumatic cases, and over 3 months in non-traumatic cases, before the time of intervention. Searches were independently conducted by 2 investigators between May 2009 and September 2009 in the following databases: Medline, Web of Science and the Cochrane Library. The electronic search was complemented by cross-checking the references of all relevant articles. Overall, a total of 16 papers were eligible for this systematic review. According to the 16 eligible studies, medical management by dopaminergic agents (levodopa, amantadine), zolpidem and median nerve stimulation, or surgical management by deep brain stimulation, extra-dural cortical stimulation, spinal cord stimulation as well as intra-thecal baclofen have shown to improve the level of consciousness in certain cases. The authors concluded that
the treatments proposed for disorders of consciousness have not yet gained the level of “evidence-based treatments”. Moreover, the studies to-date had led to inconclusiveness. They stated that the published therapeutic responses must be substantiated by further clinical studies of sound methodology.

The American Academy of Neurology’s practice parameters on “Assessment and management of patients in the persistent vegetative state” (AAN, 2006) did not mention the use of coma stimulation as a treatment modality. Also, the American Occupational Therapy Association’s practice guideline on “Adults with traumatic brain injury” (Golisz, 2009) made no recommendation regarding the use of sensory stimulation or coma arousal programs. Furthermore, the National Institute of Neurological Disorders and Stroke’s “Coma information page” (NINDS, 2012) did not mention the use of coma stimulation as a therapeutic option.

Cognitive enhancement therapy (CET) is a performance-based, comprehensive, developmental approach to the rehabilitation of social cognitive and neurocognitive deficits. Participants work at recovery through structured group and computer exercises. Cognitive enhancement therapy is designed as a recovery phase intervention for symptomatically stable persons with severe mental illness, who nonetheless remain socially and vocationally disabled. This approach is the culmination of more than 30 years of clinical experience and research in schizophrenia treatment. Overall, CET attempts to increase mental stamina, active information processing, and the spontaneous negotiation of un-rehearsed social challenges. It does so with a focus on enhancing perspective taking, social context appraisal, and other components of social cognition.


There are 3 basic components in CET: (i) Computer exercises to enhance cognitive skills, (ii) A psycho-educational group where interactive work is done through lectures, homework and group exercises. This understanding facilitates a personal process of adjusting to disability and to help participants eventually become more socialized into meaningful adult roles that they identify as goals in their recovery plan, and (iii) 1-on-1 coaching.

Gard et al (2009) noted that a burgeoning area of research has focused on motivational deficits in schizophrenia, producing hypotheses about the role that motivation plays in the well-known relationship between neuro-cognition and functional outcome. However, little work has examined the role of motivation in more complex models of outcome that include social cognition, despite the increased understanding of the critical role of social cognition in community functioning in schizophrenia, and despite new basic science findings on the association between social cognitive and reward processing in neural systems in humans. Using path analysis, these researchers directly contrasted whether motivation (i) causally influences known social cognitive deficits in schizophrenia, leading to poor outcome, or (ii) mediates the relationship between social cognitive deficits and outcome in this illness. A total of 91 patients with schizophrenia or schizo-affective disorder completed interview-based measures of motivation and functional outcome as well as standardized measures of neuro-cognition and social cognition in a cross-sectional design. In line with recent research, motivation appears to mediate the relationship between neuro-cognition, social cognition and functional outcome. A model with motivation as a causal factor
resulted in poor fit indicating that motivation does not appear to precede neurocognition. The authors concluded that findings in the present study indicated that motivation plays a significant and mediating role between neuro-cognition, social cognition, and functional outcome. Potential psychosocial treatment implications were discussed, especially those that emphasize social cognitive and motivational enhancement.

Titov et al (2010) (i) replicated an earlier trial showing that a self-guided Internet treatment for social phobia is efficacious, and (ii) examined if the addition of self-guided motivational enhancement strategies improves completion rates and clinical outcomes. Randomized controlled trial (RCT) of self-guided Internet-based cognitive behavioral treatment (iCBT), or iCBT plus self-guided motivational enhancement strategies (iCBT+MS), was conducted. An intention-to-treat and last observation carried forward model was used for data analyses. The participants consisted of 108 volunteers with social phobia. The iCBT intervention consisted of 2 online lessons about symptoms and treatment of anxiety disorders and 6 lessons about management of social phobia (the Shyness program) with complex automated reminders. The motivational intervention was based on traditional techniques including understanding and exploring ambivalence about change using a cost-benefit analysis, developing and resolving discrepancy between values and symptoms, and enhancing self-efficacy for change. The main outcome measures were the Social Interaction Anxiety Scale and Social Phobia Scale. More iCBT+MS group participants completed the 8 lessons than iCBT group participants (75 % versus 56 %, respectively), but there were no between-group differences in outcome measures at post-treatment or at 3 month follow up. Large mean within-groups effect sizes (Cohen's d) for the 2 social phobia measures were found for both the iCBT and iCBT+ MS groups (1.1 and 0.95, respectively), which were sustained at 3 month follow-up (1.06 and 1.07, respectively). Both iCBT and iCBT+MS group participants reported that the procedures were highly acceptable. The authors concluded that both self-guided versions of the Shyness program were reliably effective, confirming that people with social phobia may significantly benefit from a highly structured self-guided intervention. Moreover, they stated that the addition of motivational techniques increased completion rates but did not improve clinical outcomes or acceptability.

In a 2-year, randomized-controlled trial with annual structural magnetic resonance imaging and cognitive assessments, Eack and colleagues (2010) examined differential changes in brain morphology in early schizophrenia during cognitive rehabilitation versus supportive therapy. A total of 53 symptomatically stable, but cognitively disabled outpatients in the early course of schizophrenia or schizoaffective disorder were included in this study. Cognitive enhancement therapy is an integrated approach to the remediation of cognitive impairments in schizophrenia that utilizes computer-assisted neurocognitive training and group-based social-cognitive exercises. Enriched supportive therapy is an illness management approach that provides psycho-education and teaches applied coping strategies. Broad areas of frontal and temporal gray matter change were analyzed using longitudinal voxel-based morphometry methods employing mixed-effects models, followed by volumetric analyses of regions demonstrating significant differential changes between treatment groups. Patients receiving CET demonstrated significantly greater preservation of gray matter volume over the course of 2 years in the left hippocampus, para-hippocampal gyrus, and fusiform
gyrus, and significantly greater gray matter increases in the left amygdala (all corrected p < 0.040), compared with those receiving enriched supportive therapy. Less gray matter loss in the left para-hippocampal and fusiform gyrus, and greater gray matter increases in the left amygdala were significantly related to improved cognition and mediated the beneficial cognitive effects of CET. The authors concluded that CET may offer neurobiologic protective and enhancing effects in early schizophrenia that are associated with improved long-term cognitive outcomes.

Moreover, the authors stated that “Despite the beneficial effects of CET on brain morphology demonstrated in this study, these findings need to be interpreted in the context of a number of important limitations. Although morphometric findings support a neuroprotective effect of CET against the gray matter loss seen during the early course of schizophrenia, and in the case of the amygdala, even increase in gray matter, in the absence of functional neuroimaging data the pathophysiological significance of these results for brain function is not clear. Overall structural changes in regional brain volumes were not large, but were reliably detectable, and may reflect functional changes. That we observed significant relations between increased gray matter and cognitive improvement, and that the effects of CET on gray matter change were significant mediators of CET effects on cognition, would suggest that brain functions sub-serving neurocognition and social cognition have been improved. Nonetheless, functional neuroimaging data are needed to better understand the effects of CET on brain function. An integration of morphometric and fMRI studies could be particularly informative in this regard. It is also interesting to note that CET effects on brain regions commonly implicated in neurocognitive dysfunction in schizophrenia were quite modest. For example, no effects were seen in the dorsolateral prefrontal cortex, and only modest effects were observed in the anterior cingulate and hippocampus, which were not associated with neurocognitive change. Although gray matter change in the anterior cingulate and hippocampus might be more strongly related to individual neuropsychological tests, this pattern of findings parallels, to some degree, the cognitive effects observed in this trial of early course patients. In this population, we have observed much stronger effects on social cognition and noted a relative preservation of some general cognitive functions (particularly processing speed) among this sample. The absence of morphometric findings could reflect the better preserved neurocognitive capacity of early course patients. It is also possible that the effects of CET on brain regions implicated in neurocognitive impairment cannot be detected at a morphometric level, but that the primary effects of this approach on frontal brain regions is toward a normalization of functioning. To date, many studies have documented frontal hypofunction in schizophrenia, and if cognitive improvement occurs in the disorder it is also likely to be the result of improved brain function. As a consequence, while this study provides important information on the potential neuroanatomical effects of cognitive rehabilitation in early schizophrenia, future studies are clearly needed to continue to characterize the effects of CET on a variety of other neurobiologic parameters. It is important to remember, however, that significant relations were observed between changes in medial-temporal regions and neurocognition, as well as social cognition, suggesting the relevance of gray matter change in these regions to neurocognitive functioning. However, associations between gray matter and cognitive change were exploratory and not
corrected for multiple inference testing, as such these results need to be interpreted with caution until confirmatory replications are available.

This study is also limited by the absence of an appropriately matched group of healthy individuals who could provide data on normative brain development in early adulthood. Although a large body of evidence has accumulated in schizophrenia research indicating a progressive loss of gray matter from the earliest phases of the disorder, healthy individuals also demonstrate some gray matter loss in early adulthood. However, loss appears to be greatest in the frontal cortex, not the subcortical regions demonstrating the most cognitive change in this study, which remain relatively stable or continue to grow after childhood.

In summary, this investigation suggests that CET, a comprehensive cognitive rehabilitation approach, can protect against gray matter loss and may even support gray matter growth in medial-temporal areas of the brain in service of cognitive enhancement among early course schizophrenia patients. Although replication and further neurobiologic characterization is needed, these findings support the potential for cognitive rehabilitative approaches to positively affect the brain in schizophrenia. Further studies are needed to examine the durability of these effects on the brain, as Hogarty and colleagues and Wexler and Bell have both shown that cognitive rehabilitation can continue to confer benefits to schizophrenia patients even after the completion of treatment. Studies of neuronal mechanisms underlying brain change, such as possible effects of cognitive remediation on dopaminergic function, brain derived neurotrophic factor, as well as the genomic underpinnings of response to cognitive remediation are also needed.

Jak and colleagues (2013) noted that cognitive enhancement strategies have gained recent popularity and have the potential to benefit clinical and non-clinical populations. As technology advances and the number of cognitively healthy adults seeking methods of improving or preserving cognitive functioning grows, the role of electronic (e.g., computer- and video game-based) cognitive training becomes more relevant and warrants greater scientific scrutiny. This paper served as a critical review of empirical evaluations of publicly available electronic cognitive training programs. Many studies have found that electronic training approaches resulted in significant improvements in trained cognitive tasks. Fewer studies have demonstrated improvements in untrained tasks within the trained cognitive domain, non-trained cognitive domains, or on measures of everyday function. Successful cognitive training programs will elicit effects that generalize to untrained, practical tasks for extended periods of time. Unfortunately, many studies of electronic cognitive training programs were hindered by methodological limitations such as lack of an adequate control group, long-term follow-up and ecologically valid outcome measures. Despite these limitations, evidence suggested that computerized cognitive training has the potential to positively impact one’s sense of social connectivity and self-efficacy.

UpToDate reviews on “Mindfulness based cognitive therapy as maintenance treatment for unipolar major depression” (Segel, 2013), “Treatment of co-occurring schizophrenia and substance use disorder” (Campbell et al, 2013), and “Psychosocial interventions for schizophrenia” (Bustillo and Weil, 2013) did not mention the use of cognitive enhancement therapy as a management tool.
There is a clinical trial on “Cognitive Enhancement Therapy for Early-Stage Schizophrenia”; however, the recruitment status of this study is unknown because the information has not been verified recently (last verified August 2008). Also, there is currently a clinical trial on “Brain Imaging, Cognitive Enhancement and Early Schizophrenia (BICEPS)” that is not yet open for participant recruitment (last verified March 2012). Furthermore, there is a clinical trial on “Cognitive Enhancement Therapy for Adults with Autism Spectrum Disorders” that is currently recruiting participants (last verified January 2012).


Bahar-Fuchs et al (2013) noted that cognitive impairments, and particularly memory deficits, are a defining feature of the early stages of Alzheimer's disease (AD) and vascular dementia. Interventions that target these cognitive deficits and the associated difficulties with activities of daily living are the subject of ever-growing interest. Cognitive training and cognitive rehabilitation (CR) are specific forms of non-pharmacological intervention to address cognitive and non-cognitive outcomes. These researchers systematically evaluated the evidence for these forms of intervention in people with mild AD or vascular dementia. Randomized controlled trials, published in English, comparing CR or cognitive training interventions with control conditions and reporting relevant outcomes for the person with dementia or the family caregiver (or both), were considered for inclusion. A total of 11 RCTs reporting cognitive training interventions were included in the review. A large number of measures were used in the different studies, and meta-analysis could be conducted for several primary and secondary outcomes of interest. Several outcomes were not measured in any of the studies. Overall estimates of the treatment effect were calculated by using a fixed-effects model, and statistical heterogeneity was measured by using a standard chi-squared statistic. One RCT of CR was identified, allowing the examination of effect sizes, but no meta-analysis could be conducted. Cognitive training was not associated with positive or negative effects in relation to any of the reported outcomes. The overall quality of the trials was low-to-moderate. The single RCT of CR found promising results in relation to some patient and caregiver outcomes and was generally of high quality. The available evidence regarding cognitive training remains limited, and the quality of the evidence needs to improve. However, there is still no indication of any significant benefits from cognitive training. Trial reports indicated that some gains resulting from intervention may not be captured adequately by available standardized outcome measures. The authors concluded that the results of the single RCT of CR showed promise but are preliminary in nature. They stated that further well-designed studies of cognitive training and CR are needed to provide more definitive evidence.

In a Cochrane review, Bowen et al (2013) examined if CR improves functional independence, neglect (as measured using standardized assessments), destination on discharge, falls, balance, depression/anxiety and quality of life in stroke patients with neglect measured immediately post-intervention and at longer-term follow-up; and determined which types of interventions are effective and whether CR is more effective than standard care or an attention control. These investigators searched the Cochrane Stroke Group Trials Register (last searched June 2012), MEDLINE (1966 to June 2011), EMBASE (1980 to June 2011),
CINAHL (1983 to June 2011), PsycINFO (1974 to June 2011), UK National Research Register (June 2011). They hand-searched relevant journals (up to 1998), screened reference lists, and tracked citations using SCISEARCH. They included RCTs of CR specifically aimed at spatial neglect. They excluded studies of general stroke rehabilitation and studies with mixed participant groups, unless more than 75% of their samples were stroke patients or separate stroke data were available. Two review authors independently selected studies, extracted data, and assessed study quality. For subgroup analyses, review authors independently categorized the approach underlying the cognitive intervention as either ‘top-down’ (interventions that encourage awareness of the disability and potential compensatory strategies) or ‘bottom-up’ (interventions directed at the impairment but not requiring awareness or behavioral change, e.g. wearing prisms or patches). They included 23 RCTs with 628 participants (adding 11 new RCTs involving 322 new participants for this update). Only 11 studies were assessed to have adequate allocation concealment, and only 4 studies to have a low risk of bias in all categories assessed. Most studies measured outcomes using standardized neglect assessments: 15 studies measured effect on activities of daily living (ADL) immediately after the end of the intervention period, but only 6 reported persisting effects on ADL. One study (30 participants) reported discharge destination and 1 study (8 participants) reported the number of falls. Eighteen of the 23 included RCTs compared CR with any control intervention (placebo, attention or no treatment). Meta-analyses demonstrated no statistically significant effect of CR, compared with control, for persisting effects on either ADL (5 studies, 143 participants) or standardized neglect assessments (8 studies, 172 participants), or for immediate effects on ADL (10 studies, 343 participants). In contrast, these investigators found a statistically significant effect in favor of CR compared with control, for immediate effects on standardized neglect assessments (16 studies, 437 participants, standardized mean difference (SMD) 0.35, 95% confidence interval [CI]: 0.09 to 0.62). However, sensitivity analyses including only studies of high methodological quality removed evidence of a significant effect of CR. Additionally, 5 of the 23 included RCTs compared one CR intervention with another. These included 3 studies comparing a visual scanning intervention with another CR intervention, and 2 studies (3 comparison groups) comparing a visual scanning intervention plus another CR intervention with a visual scanning intervention alone. Only 2 small studies reported a measure of functional disability and there was considerable heterogeneity within these subgroups (I² > 40%) when they pooled standardized neglect assessment data, limiting the ability to draw generalized conclusions. Subgroup analyses exploring the effect of having an attention control demonstrated some evidence of a statistically significant difference between those comparing rehabilitation with attention control and those with another control or no treatment group, for immediate effects on standardized neglect assessments (test for subgroup differences, p = 0.04). The authors concluded that the effectiveness of CR interventions for reducing the disabling effects of neglect and increasing independence remains unproven. As a consequence, no rehabilitation approach can be supported or refuted based on current evidence from RCTs. However, there is some very limited evidence that CR may have an immediate beneficial effect on tests of neglect. This emerging evidence justifies further clinical trials of CR for neglect. However, future studies need to have appropriate high quality...
methodological design and reporting, to examine persisting effects of treatment and to include an attention control comparator.

In a Cochrane review, Loetscher and Lincoln (2013) examined if (i) stroke survivors receiving attentional treatment show better outcomes in their attentional functions than those given no treatment or treatment as usual, and (ii) stroke survivors receiving attentional treatment techniques have a better functional recovery, in terms of independence in ADL, mood and quality of life, than those given no treatment or treatment as usual. These investigators searched the Cochrane Stroke Group Trials Register (October 2012), Cochrane Central Register of Controlled Trials (CENTRAL) (The Cochrane Library October 2012), MEDLINE (1948 to October 2012), EMBASE (1947 to October 2012), CINAHL (1981 to October 2012), PsycINFO (1806 to October 2012), PsycBITE and REHABDATA (searched October 2012) and ongoing trials registers. They screened reference lists and tracked citations using Scopus. They included RCTs of CR for impairments of attention for people with stroke. The primary outcome was measures of global attentional functions, and secondary outcomes were measures of attention domains, functional abilities, mood and quality of life. Two review authors independently selected trials, extracted data and assessed trial quality. They included 6 RCTs with 223 participants. All 6 RCTs compared CR with a usual care control. Meta-analyses demonstrated no statistically significant effect of CR for persisting effects on global measures of attention (2 studies, 99 participants; SMD 0.16, 95% CI: -0.23 to 0.56; p = 0.41), standardized attention assessments (2 studies, 99 participants; p ≥ 0.08) or functional outcomes (2 studies, 99 participants; p ≥ 0.15). In contrast, a statistically significant effect was found in favor of CR when compared with control for immediate effects on measures of divided attention (4 studies, 165 participants; SMD 0.67, 95% CI: 0.35 to 0.98; p < 0.0001) but no significant effects on global attention (2 studies, 53 participants; p = 0.06), other attentional domains (6 studies, 223 participants; p ≥ 0.16) or functional outcomes (3 studies, 109 participants; p ≥ 0.21). Thus, there was limited evidence that CR may improve some aspects of attention in the short-term, but there was insufficient evidence to support or refute the persisting effects of CR on attention, or on functional outcomes in either the short- or long-term. The authors concluded that the effectiveness of CR remains unconfirmed. The results suggested there may be a short-term effect on attentional abilities, but future studies need to assess the persisting effects and measure attentional skills in daily life. Trials also need to have higher methodological quality and better reporting.

In a Cochrane review, Chung et al (2013) examined the effects of CR on executive dysfunction for adults with stroke or other non-progressive acquired brain injuries. They searched the Cochrane Stroke Group Trials Register (August 2012), the Cochrane Central Register of Controlled Trials (The Cochrane Library, August 2012), MEDLINE (1950 to August 2012), EMBASE (1980 to August 2012), CINAHL (1982 to August 2012), PsycINFO (1806 to August 2012), AMED (1985 to August 2012) and 11 additional databases. They also searched reference lists and trials registers, hand-searched journals and conference proceedings, and contacted experts. They included randomized trials in adults after non-progressive acquired brain injury, where the intervention was specifically targeted at improving cognition including separable executive function data (restorative interventions), where the intervention was aimed at training participants in methods to
compensate for lost executive function (compensative interventions) or where the intervention involved the training in the use of an adaptive technique for improving independence with ADL (adaptive interventions). The primary outcome was global executive function and the secondary outcomes were specific components of executive function, working memory, ADL, extended ADL, quality of life and participation in vocational activities. They included studies in which the comparison intervention was no treatment, a placebo intervention (i.e., a rehabilitation intervention that should not impact on executive function), standard care or another cognitive rehabilitation intervention. Two review authors independently screened abstracts, extracted data and appraised trials. They undertook an assessment of methodological quality for allocation concealment, blinding of outcome assessors, method of dealing with missing data and other potential sources of bias. A total of 19 studies (907 participants) met the inclusion criteria for this review; these included 13 studies (770 participants) in meta-analyses (417 traumatic brain injury, 304 stroke, 49 other acquired brain injury) reducing to 660 participants once non-included intervention groups were removed from 3 and 4 group studies. These researchers were unable to obtain data from the remaining 6 studies. Three studies (134 participants) compared CR with sensorimotor therapy. None reported the primary outcome; data from 1 study was available relating to secondary outcomes including concept formation and ADL.

Six studies (333 participants) compared CR with no treatment or placebo. None reported the primary outcome; data from 4 studies demonstrated no statistically significant effect of CR on secondary outcomes. Ten studies (448 participants) compared 2 different CR approaches. Two studies (82 participants) reported the primary outcome; no statistically significant effect was found. Data from 8 studies demonstrated no statistically significant effect on the secondary outcomes. These researchers explored the effect of restorative interventions (10 studies, 468 participants) and compensative interventions (4 studies, 128 participants) and found no statistically significant effect compared with other interventions. The authors concluded that they identified insufficient high-quality evidence to reach any generalized conclusions about the effect of CR on executive function, or other secondary outcome measures. They stated that further high-quality research comparing CR with no intervention, placebo or sensorimotor interventions is recommended.

Farina et al (2015) reviewed the modalities of CR, outcome end-points, and the levels of evidence of efficacy of different interventions. A systematic research in PubMed, Psychinfo, and SCOPUS was performed assessing the articles written in the entire period covered by these databases till December 2013. Articles in English, Spanish or French were evaluated. A manual research evaluated the references of all of the articles. The experimental studies were classified according to the level of evidence of efficacy, using a standardized Italian method (SPREAD, 2007), adopting the criteria reported by Cicerone et al (2000, 2011). A total of 18 papers were classified into 2 reviews, 4 papers dealing with the principles and efficacy of CR in epilepsy, a methodological paper, a single-case report, a multiple-case report, and 9 experimental papers. Most studies involved patients with temporal lobe epilepsy. Different types of CR were used to treat patients with epilepsy. A holistic rehabilitation approach was more useful than selective interventions to treat memory and attention disturbances. The authors concluded that CR may be a useful tool to treat cognitive impairment in patients
with epilepsy. However, the modalities of treatment and outcome end-points are important concerns of clinical care and research. They stated that controlled studies are needed to determine the efficacy of rehabilitation in well-defined groups of patients with epilepsy.

Appendix

Aetna requires that cognitive therapy and other rehabilitation be provided in accordance with an ongoing, written plan of care created by the therapist. The purpose of the written plan of care is to assist in determining medical necessity and should include the following:

The written plan of care should be sufficient to determine the medical necessity of treatment, including:

- The diagnosis along with the date of onset of the condition;
- A reasonable estimate of when the goals will be reached;
- Long-term and short-term goals that are specific, quantitative and objective;
- Cognitive therapy evaluation;
- The frequency and duration of treatment; and
- The specific techniques to be used in treatment.

The plan of care should be ongoing, (i.e., updated as the patient's condition changes), and treatment should demonstrate reasonable expectation of improvement. Cognitive therapy is considered medically necessary only if there is a reasonable expectation that cognitive therapy will achieve measurable improvement in the patient's condition in a reasonable and predictable period of time.

The therapist should re-evaluate the patient regularly (this is typically done on a monthly basis) and document the progress toward the goals of cognitive therapy in the patient's clinical record. The treatment goals and subsequent documentation of treatment results should specifically demonstrate that cognitive therapy services are contributing to such improvement.

CPT Codes / HCPCS Codes / ICD-9 Codes

CPT codes covered if selection criteria are met:

<table>
<thead>
<tr>
<th>Code</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>97532</td>
<td>Development of cognitive skills to improve attention, memory, problem solving (includes compensatory training), direct (one-on-one) patient contact, each 15 minutes</td>
</tr>
<tr>
<td>97537</td>
<td>Community/work reintegration training (e.g., shopping, transportation, money management, avocational activities and/or work environment/modification analysis, work task analysis, use of assistive technology device/adaptive equipment), direct one-on-one contact by provider, each 15 minutes</td>
</tr>
</tbody>
</table>

Other CPT codes related to the CPB:
Neuropsychological testing (e.g., Halstead-Reitan Neuropsychological Battery, Wechsler Memory Scales and Wisconsin Card Sorting Test), per hour of the psychologist's or physician's time, both face-to-face time administering tests to the patient and time interpreting these test results and preparing the report

Neuropsychological testing (e.g., Halstead-Reitan Neuropsychological Battery, Wechsler Memory Scales and Wisconsin Card Sorting Test), with qualified health care professional interpretation and report, administered by technician, per hour of technician time, face-to-face

Neuropsychological testing (e.g., Wisconsin Card Sorting Test), administered by a computer with qualified health care professional interpretation and report

HCPCS codes not covered for indications listed in the CPB:

S9056 Coma stimulation per diem

ICD-9 codes covered if selection criteria are met:

310.2 Postconcussion syndrome

348.1 Anoxic brain damage

348.30 - 348.39 Encephalopathy, not elsewhere classified

349.82 Toxic encephalopathy

430 - 434.91 Subarachnoid hemorrhage, intracerebral hemorrhage, other and unspecified intracranial hemorrhage, occlusion and stenosis of precerebral arteries, and occlusion of cerebral arteries

436 Acute, but ill-defined cerebrovascular disease

437.0 - 437.2 Cerebral atherosclerosis, other generalized ischemic cerebrovascular disease, and hypertensive encephalopathy

437.4 - 438.12 Cerebral arteritis, moyamoya disease, nonpyogenic thrombosis of intracranial venous sinus, transient global amnesia, other and unspecified cerebrovascular disease, and late effects of cerebrovascular disease including cognitive deficits, speech and language deficits, aphasia and dysphasia

851.00 - 854.19 Cerebral laceration and contusion, subarachnoid, subdural, and extradural hemorrhage, following injury, other and unspecified intracranial hemorrhage following injury, and intracranial injury of other and unspecified nature
905.0 Late effect of fracture of skull and face bones
907.0 Late effect of intracranial injury without mention of skull fracture

**ICD-9 codes not covered for indications listed in the CPB (not all-inclusive):**

- 042 Human immunodeficiency virus [HIV] disease
- 265.1 Other and unspecified manifestations of thiamine deficiency [Wernicke encephalopathy]
- 290.0 - 319 Mental disorders
- 331.0 Alzheimer's disease
- 332.0 Paralysis agitans
- 340 Multiple sclerosis
- 343.0 - 343.9 Infantile cerebral palsy
- 490 - 496 Chronic obstructive pulmonary disease and allied conditions
- 780.01 Coma
- 780.03 Persistent vegetative state
- 783.40 Lack of normal physiological development, unspecified
- 799.51 - 799.55 Signs and symptoms involving cognition
- 799.59 Other signs and symptoms involving cognition

**The above policy is based on the following references:**


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