Neuropsychol Rev (2010) 20:52–85
DOI 10.1007/s11065-009-9125-y

REVIEW

Rehabilitation Treatments for Adults with Behavioral and Psychosocial Disorders Following Acquired Brain Injury: A Systematic Review

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Received: 24 November 2008 / Accepted: 21 December 2009 / Published online: 9 February 2010

Abstract This review was aimed at systematically investigating the treatment efficacy and clinical effectiveness of neurobehavioral rehabilitation programs for adults with acquired brain injury and making evidence-based recommendations for the adoption of these rehabilitation trainings. Using a variety of search procedures, 63 studies were identified and reviewed using a set of questions about research methods, treatments, results and outcomes for the 1,094 participants. The 63 studies included treatments falling into three general categories: approaches based on applied behavior analysis, interventions based on cognitive-behavior therapy (CBT), and comprehensive-holistic rehabilitation programs (CHRPs). Considerable heterogeneity exists in the reviewed literature among treatment methods and within reported sample subjects. Despite the variety of methodological concerns, results indicate that the greatest overall improvement in psychosocial functioning is achieved by CHRP that can be considered a treatment standard for adults with behavioral and psychosocial disorders following acquired brain injury. Both approaches based on applied behavior analysis and CBT can be said to be evidence-based treatment options. However, findings raise questions about the role of uncontrolled factors in determining treatment effects and suggest the need for rigorous inclusion/exclusion criteria, with greater specification of theoretical basis, design, and contents of treatments for both interdisciplinary-comprehensive approaches and single-case methodologies.

Keywords Acquired brain injury · Neurobehavioral disorders · Psychosocial disorders · Evidence-based rehabilitation · Review

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Introduction

Acquired brain injury (ABI) is defined as a cerebral damage that occurs after birth and is not related to congenital disorders, developmental disabilities, or processes that progressively damage the brain. Many individuals with ABI, particularly of traumatic aetiology, exhibit a variety of alterations, especially in the areas of cognition, mood, and behavior, even in the presence of generally good somatic/functional recovery (Adolphs 2003). Although cognitive deficits and behavioral disorders tend to decrease spontaneously during recovery, they may have a significant impact on community integration and psychosocial adjustment (Milders et al. 2003; McCabe et al. 2007). Moreover, the persistence and magnitude of social and behavioral problems after ABI is a frequent cause of a non-compliant condition and may be a serious obstacle to the treatment of associated cognitive or functional deficits (Sohlberg and Mateer 2001; Wood and McMillan 2001).

Neurobehavioral and emotional disorders can be divided into two main categories based on the clinical features of their symptoms (Ducharme 1999; Wood and McMillan 2001): (1) internalizing symptoms indicate a defective-type behavior not primarily related to a psychogenic background of dysphoric mood disorder such as depression or demoralization, including apathy, initiation impairment, carelessness, reduced self-confidence or self-esteem, frustration, and social withdrawal (Finset and Andersson 2000; Rao et al. 2007); (2) externalizing symptoms synthesize an impulse control disorder with a non-compliant and oppositional attitude, including impulsivity, irritability, aggression, excitement, disinhibition, confabulation, and sexually inappropriate activities (Wood and McMillan 2001).

A large body of literature supports the critical role of the frontal lobes in social cognition and social behavior, but there is a debate regarding the exact nature of this relationship, and of the higher-level organization and execution of complex thoughts and behavior (Alvarez and Emory 2006). A number of findings highlight the complex interaction among cognitive abilities, self-monitoring of social skills, awareness of social rules and boundaries, and behavioral or emotional control (Anderson et al. 1999; Blair and Cipolotti 2000; Grafman 2007). According to various authors (e.g., Sbordone 2000) three major frontal-subcortical circuits (dorsolateral, ventromedial and orbitofrontal) are involved in these interactions.

The dorsolateral prefrontal cortex (which projects primarily to the dorsolateral head of the caudate nucleus) has a critical role in the temporal organization of behavior (Fuster 1989); it allows the individual to elaborate sequences of voluntary actions important for motor programming, verbal fluency and the use of strategies (e.g., Milner and Petrides 1984). Patients with localized damage to the dorsolateral prefrontal cortex show reduced cognitive flexibility, a tendency toward perseveration and reduced verbal fluency; they may appear "pseudo-depressed" (Blumer and Benson 1975) due to the presence of apathy, lack of planning, difficulty in developing new ideas and strategies. The ventromedial circuit, which involves the anterior cingulate and projects primarily to the nucleus accumbens, is important for motivation and initiative. Lesions to this region, often associated with the supplementary motor area, produce akinesia, apathy, mutism and decreased social interaction (Sbordone 2000). While unilateral lesions tend to produce temporary disorders, deficits are more lasting in the case of bilateral damage (Fesenmeier et al. 1990). The orbitofrontal cortex (which projects to the ventromedial caudate nucleus) acts as a filter for sensory information, inhibiting less important/appropriate stimuli in favor of those required to reach a current goal (Malloy et al. 1993). Patients with orbitofrontal damage appear to have disinhibited reasoning and behavior, they are easy to distract and unable to suppress automatic responses. Particularly in the case of bilateral damage, dramatic personality changes can occur characterized by lack of insight and antisocial behavior (e.g., Eslinger and Damasio 1985; Blumer and Benson 1975). To explain these dramatic personality changes, Damasio et al. (1991) proposed the somatic marker hypothesis (see also Damasio 1996; for a critical evaluation of this theory see Dunn et al. 2006) according to which the orbitofrontal cortex allows associating complex situations with corresponding emotional states to regulate decision-making. Markers are somatic as they relate to body-state structure and regulation even when they do not properly arise in the body but rather in the brain's representation of the body (Damasio 1996). Besides the site and nature of the brain damage, a number of other factors interact to determine the probability of behavioral, emotional and psychosocial disorders in individuals with ABI (Wood and McMillan 2001). These include premorbid behavior, personality traits and skill level, and cognitive and physical sequelae of the injury. It is this amalgam of factors that often results in decreased everyday life competencies, impeding the effective performance of daily activities and access to desired social activity.

Given the large number of behavioral and emotional disturbances resulting from various kinds of brain damage, one might expect behavioral and psychosocial intervention to be a major component of neuropsychological rehabilitation programs. However, apart from comprehensive-holistic approaches, the rehabilitation of inappropriate social behavior still seems to be considered supplementary to other neuropsychological interventions rather than a core part of the treatment program. This is particularly true with respect to the rehabilitation of specific cognitive deficits (e.g., disorders of language, attention, memory). The greater concern with cognitive deficits is not only testified by the amount of scientific research in this field but also by the growing number of evidence-based reviews that systematically evaluate interventions in the rehabilitation of cognitive deficits (Carney et al. 1999; Cicerone et al. 2000, 2005; Sohlberg et al. 2003; Cappa et al. 2003, 2005; Rees et al. 2007). Further, a meta-analytic re-examination of the literature (Rohling et al. 2009) has substantiated some, though not all, of the claims of effectiveness originally identified by the original reviews (Cicerone et al. 2000, 2005).

Regarding non-pharmacological approaches for neurobehavioral disorders, few published reviews have examined the effectiveness of treatments. For example, Malec and Basford (1996) compared outcomes of adult post-acute brain injury rehabilitation with natural recovery after brain injury and appraised potential outcome predictors and the effectiveness of specific behavioral interventions (including applied behavior analysis and cognitive-behavior therapy) as well as cognitive and vocational treatments for intensive comprehensive-holistic day treatment programs. Although the reviewed studies were mostly uncontrolled, benefits

were documented in many brain-injured participants, including increased independent living status. Regarding neurobehavioral treatments as integral interventions in comprehensive-holistic approaches, the reduction of severe behavioral disturbances supported treatment effectiveness based on applied behavior analysis. These were primarily single-case controlled studies. Malec and Basford (1996) recommended developing standardized measures of potential confounding variables, such as pre-treatment functional status, self-awareness level, pre-injury psychosocial functioning, and other individual characteristics that may determine treatment selection or outcome.

With specific regard to behavioral interventions, a recent review by Ylvisaker et al. (2007) examined the treatment efficacy of methods for behavioral disorders mainly connected with traumatic brain injury. The study was restricted to 172 experimental participants, including children and adults. The reviewed interventions fell into two traditional applied behavior analysis approaches (i.e., contingency management procedures and positive behavior interventions). The results revealed methodological concerns that undermined certainty about the clinical effectiveness of these interventions.

The present review study will complement the above mentioned review on traditional applied behavior analysis approaches (Ylvisaker et al. 2007) by extending the examination of the efficacy and effectiveness arising from treatments based on cognitive-behavioral therapy and comprehensive-holistic approaches. For the purpose of this review, existing interventions for individuals with behavioral disorders following acquired brain injury were placed in one of three general approach categories depending on their core features: theoretical and methodological bases, final goals of the interventions, and characteristics of the environmental structure within which the treatment took place.

Approaches Based on Applied Behavior Analysis

These interventions are clinical applications of learning theory (respondent and operant conditioning) and are based on procedures emphasizing, but not restricted to, the management and modification of specific maladaptive behavior by manipulating antecedents or consequences. Treatment programs are usually time-limited, individualized and problem-focused in the sense that only one or few behaviors are targeted for intervention. Environmental structuring, stimulus-response analysis, systematic monitoring, and planned contingency management constitute the treatment requirements for successful interventions (McGlynn 1990; Ducharme 1999). A trusting and safe therapeutic alliance is viewed as an essential ingredient, but not as the main vehicle of change.

Traditional contingency management procedures (CMPs) represent a moderating approach based on early application of the learning theory to skill development. The operant principle is that behavior increases or decreases in frequency as a result of positive and negative consequences, such as verbal praise, token-economies, extinction/time-out and response-cost procedures. These approaches can be useful as initial intervention choices mainly in the acute phase of recovery to manage severe behavioral episodes possibly associated with conditions, such as agitation and non-compliant and disruptive behavior. CMP can also be used as a prelude to remedial frameworks in the post-acute environment when maladaptive behavior continues to occur with high frequency, intensity or duration, thus making the use of skill-teaching approaches and other forms of rehabilitation extremely difficult (Ducharme 1999). Several studies have used a token-economy system to increase compliance with therapeutic activities and treatment of socially inappropriate behavior or inadequate self-care skills. In addition to positive reinforcement using tokens, punishment procedures may be used in the form of fines.

The central theme in another remedial approach—the positive behavior intervention (PBI)—is the proactive prevention of negative behavior and systematic facilitation of repertoires of positive behavior that render the negative behavior irrelevant (Ducharme 2000; Carr et al. 2002). PBI emphasize the management and modification of behavior by manipulating antecedents, including both immediate and remote setting events. In a planned environmental structure, several different procedures are essential. These include fading, shaping, feedback, prompting, stress inoculation training, redirection and planned assurance of positive communication. They are often used together with observational learning (modelling), and are based on systematic analysis of the variables responsible for maintaining the problem behavior. All these procedures are focused on skill acquisition rather than problem behavior reduction, and learning or relearning strategies for self-managing the environment are the main goal of treatment (Ducharme 2000). For example, rather than trying to stop patients from engaging in their disruptive behavior and inadvertently reinforcing it with attention, the therapist and the other staff professionals completely ignore disruptive outbursts and systematically reward the patient with special attention for positive and cooperative behavior. Patients should also learn to reward themselves whenever they are successful in expressing new and adequate reactions to crucial situations.

Interventions Based on Cognitive-Behavior Therapy (CBT)

These interventions (which include education, cognitive restructuring, self-monitoring, self-talk training, and sup-

porting communication) are distinguished in this review because some differences in treatment protocols were found in the research literature. Although the studies using CBT have a behavioral orientation, the approach highlights internal setting events (e.g., emotional discomfort, loneliness, low self-esteem) by integrated application of neuropsychological and psychodynamic theories as opposed to the artificial rewards often associated with traditional behavioral interventions (Dobson 2000; Leichsenring et al. 2006). While the latter rely on environmental antecedents and consequences of behavior, CBT approaches emphasize the therapeutic relationship as an active part of treatment. The assumption is that cognitive functions, emotions, and behavior are strictly interconnected (Langer and Padrone 1992; Chittum et al. 1996). By establishing and maintaining a therapeutic relationship, as well as reinforcing alternatives to disruptive behavior, the main goals of CBT applied to people with ABI are the following: (1) to recognize illness-perpetuating behavior, (2) to change dysfunctional thought patterns, (3) to increase the use of effective coping strategies, (4) to reduce levels of stress, (5) to teach skills for preventing a relapse into emotional distress, and (6) to help subjects cope with feelings of loss related to decreased functioning (Giles and Manchester 2006).

Comprehensive-holistic Approaches

The comprehensive-holistic rehabilitation programs (CHRPs) are typically designed to develop alternative or compensatory behaviors rather than to restore the underlying dysfunctional cognitive systems per se, which is the main goal of traditional remedial interventions (Malec and Basford 1996). CHRP generally is appropriated in the post-acute environment to teach adaptive skills replacing maladaptive psychosocial patterns, facilitating the establishment of a therapeutic alliance and enhancing cooperation and motivation to address rehabilitation goals. Subjects learn to self-manage difficult everyday situations without external control by others. Positive changes in behavior acquired with this type of intervention are more likely to generalize outside the rehabilitation environment than those achieved with approaches that do not involve skill acquisition. Most comprehensive-holistic interventions are based on a theoretical approach that recognizes the contribution of both neuropsychological and psychological elements in treating maladaptive psychosocial functioning. Moreover, in the CHRP the milieu itself is recognized as an active therapeutic factor in integrated cognitive, behavioral, affective and positive psychosocial changes. Hence residential, planned, environmental structuring is necessary if a comprehensive-holistic approach is to be useful. The rationale of standard milieu-oriented rehabilitation programs is to help patients improve their level of insight, realism, emotional adjustment and acceptance of brain-injury-related deficits. The main focus is on life-style change satisfactory to the individual and important others. Integral to CHRP are the individual and group interventions aimed at creating therapeutic alliance and issues related to the cognitive, behavioral, psychosocial, and affective components of recovery. Considering the overlapping of cognitive and psychological disorders in maladaptive psychosocial functioning, the key components can be summarized as follows: (1) integrated trans-disciplinary staff roles, (2) psychosocial and emotional adjustment as main treatment goals, with the most important final step that of regaining a level of community functioning and productivity, (3) developing a positive working alliance with patients and families, (4) active participation of family members who have an integral role in planning and monitoring the individualized rehabilitation programs, (5) systematic feedback on group activities based on video-taped records, (6) formal staff meetings four times a week, (7) dedicated vocational and/or independent living trials (Christensen and Uzzel 1994; Klonoff 1997).

The present review is concerned with the treatment efficacy and clinical effectiveness of any of the above mentioned intervention programs for adults with maladaptive behavior, emotional disorders, or reduced psychosocial functioning after moderate-to-severe acquired brain injury. In addition to traumatic brain injury, other types of non-progressive brain damage were also included: anoxia, cerebrovascular event (subarachnoid haemorrhage, stroke), rupture of a cerebral artery aneurysm, viral encephalitis, and brain tumour. Overall, the aim was to evaluate the effectiveness of these interventions and to provide evidence-based recommendations for rehabilitation practice.

Method

Study Selection

The following databases were searched using combinations of the terms *'acquired brain injury', 'traumatic brain injury', 'brain injury', 'behavior disorders', 'behavioral problems', 'emotional disorders', 'psychosocial problems', 'treatment', 'intervention', 'rehabilitation', 'therapy'*: PubMed/Medline (1970–2008), PsycINFO (1970–2008), EMBASE (1980–2008), CINAHL (1982–2008). Studies cited in review articles or in selected study articles that were not identified through the original literature search strategy were also included. Studies in which participants met our definition for ABI and which involved the evaluation of a behavioral treatment with measurable outcomes were selected. Once an article was selected for

full review, it was rated by two blind, independent reviewers (authors RC and MZ) who classified study design, level of research and other review data. Disagreements between reviewers were resolved by discussion or by deferring to a third reviewer (author PZ).

Articles were excluded if: (1) they were case studies without quantitative data; (2) they had theoretical focus or were descriptive reports of treatment approaches; (3) they had an epidemiological focus; (4) they were non-peer-reviewed papers, expert or consensus opinions, abstracts of presentations or book chapters; (5) participants were younger than 16; (6) interventions did not concern behavioral or emotional disorders or psychosocial problems; (7) they dealt primarily with mild or mild-to-moderate severity level of ABI; (8) they were based exclusively on specific skills training, occupational therapy, vocational rehabilitation, or job coaching; (9) they described pharmacological interventions or alternative medicine approaches (such as acupuncture, music and art therapy).

Evidence Levels

Level of evidence was determined by referring to the neurological management guidelines of the European Federation of Neurological Societies (Hughes et al. 2001) and the rating systems used in previous reviews on cognitive rehabilitation (Carney et al. 1999; Cicerone et al. 2000; Cappa et al. 2003). Statistically homogeneous or well-designed, prospective, randomized controlled trials (RCTs) were considered Class I evidence. Well-designed case-control studies or non-RCTs were considered Class II evidence. RCTs with clinically irrelevant design flaws were considered Class II+evidence. Case series and case reports of one or more single cases with adequate quantification and analysis of results were considered Class III evidence.

Treatment efficacy is concerned with how successful a particular treatment will be for a given patient. It refers to internal validity, that is, the strength of an intervention as assessed under highly controlled conditions such as those found in experimental research trials (Rowland and Goss 2000; Chambless and Ollendick 2001).

The clinical effectiveness of behavioral changes (or the external validity of a treatment) is concerned with their practical significance and social validity. It does not just refer to whether gains are generalized to a variety of natural tasks and settings, but also whether they are maintained over time in vocational, educational, or socially meaningful daily living environments (Rowland and Goss 2000; Chambless and Ollendick 2001).

Clinical recommendations were made by considering the relative strength of the evidence, from the best supported Practice Standard and Practice Guideline to the less evidenced Practice Option (see Table 1 for definitions). The reference for each rated article and a summary of the information collected from each study are provided in the Appendix.

Results

The search identified 793 published articles for potential inclusion. In the screening process, only the 63 papers summarized in the Appendix fulfilled the criteria for inclusion. Overall, 1,094 participants had received treatment for neurobehavioral and psychosocial problems following acquired brain injury.

Clinical Features of the Participants

Primary Diagnosis

In 33 studies reporting results on a total of 293 patients, the primary diagnosis was traumatic brain injury (TBI). Three studies (54 participants) reported strokes (Lincoln et al. 1997; Lincoln and Flannaghan 2003; Gracey et al. 2007) and three single-case studies reported encephalitis (McMillan et al. 1990; Alderman et al. 1995; Dewar and Gracey 2007). In most cases (a total of 744 participants), mixed aetiologies other than TBI or stroke were reported. They included the following brain injury categories: anoxia, subarachnoid

Table 1 Definitions of the three-levels of clinical recommendations

Practice standard	Practice guideline	Practice option
High degree of clinical certainty based on at least one well-designed Class I study with an adequate sample, with support from very strong Class II evidence that directly addresses the effectiveness of the treatment in question, providing substantive evidence of effectiveness to support a recommendation that the treatment be specifically considered for people with acquired neurobehavioral impairments.	Moderate clinical certainty based on one or more Class I studies with methodological limitations, or well-designed Class II studies with adequate samples, that directly address the effectiveness of the treatment in question, providing evidence of probable effectiveness to support a recommendation that the treatment be specifically considered for people with acquired neurobehavioral impairments.	Unclear or conflicting clinical certainty based on Class II or Class III studies, providing evidence of possible effectiveness to support a recommendation that the treatment be specifically considered for people with acquired neurobehavioral impairments.

haemorrhage, rupture of a cerebral artery aneurysm, brain tumour, and acquired hydrocephalus.

Brain Injury Severity

Consistent with the exclusion/inclusion criteria, almost all individuals had severe injuries. Nevertheless, criteria for injury severity were most often described anecdotally and not specified by clinical descriptions such as duration of the acute period of confusion (unconsciousness and post-traumatic amnesia), coma score and neuroimaging records. In seven group studies (Ownsworth et al. 2000; Malec 2001; Ownsworth and McFarland 2004; Niemeier et al. 2005; Anson and Ponsford 2006b), 272 participants were described with variable injury severity; however they were in a predominantly moderate-to-severe range, thus matching the inclusion criteria for severity.

Pathophysiological Features

Information on the site-of-lesion was included in only 28 out of 63 studies (i.e., 44.4%) and referred nearly always to a narrative description of the lesion, based on routine CT or MRI scans. Based on this information, frontal lobe injuries predominated; in a number of cases brain stem involvement was also reported. When participants were well described most had sensory-motor disabilities or cognitive and executive system impairments (e.g., attention and/or memory problems, poor planning and problem-solving) that were present in various combinations with behavioral and psychosocial disorders. However, in many cases there was insufficient information to document associated psychiatric impairments. Only four studies (Zencius et al. 1989b; Rothwell et al. 1999; Giles et al. 2005; Cicerone et al. 2008) specified premorbid conditions for 78 subjects that could have introduced a confounding bias (e.g., substance abuse, attempted suicide, mania, and other psychiatric disorders).

Behavioral Issues

Approximately two-thirds of the participants with specified behavioral problems had predominantly externalizing symptoms such as irritability, impulsiveness, aggressive and disinhibited behavior or sexually inappropriate activities. Only nine studies described patients (n=150) with predominantly internalizing disorders. All had depressive-type symptoms (such as social withdrawal, reduced self-esteem, anger, anxiety, and denial) associated with a broad range of injury severity, ranging from mild-to-severe. All treatments were based on cognitive-behavior therapy (CBT) except for one comprehensive-holistic rehabilitation program (CHRP) delivered to 11 participants with TBI (see Table 2 for details).

Table 2 Results and outcome for treatments of predominantly internalizing disorders (depressive-type symptoms)

Reference/class	Treated individuals	Treatment type/duration	Results
Lincoln and Flannaghan (2003) Class I	34 Strokes; mean age 65 yrs	CBT/3 months	No significant differences between groups in patients' mood, independence in IADLs, handicap, or satisfaction with care
Ruff and Niemann (1990)/Class II	12 TBIs; mean age 28 yrs	CBT/8 weeks	No significant differences between groups by KAS
Medd and Tate (2000)/Class II+	8 ABIs; mean age 35 yrs	CBT/5–8 weeks	Decreased anger but no transfer to self-esteem, anxiety, depression, and self-awareness
Anson and Ponsford (2006b)/Class II+	31 TBIs; mean age 38 yrs	CBT/10 weeks	Not clear anti-depressant effects; no significant changes in depression, anxiety, self-esteem and psychosocial function
Lincoln et al. (1997)/Class III	19 Strokes; mean age 67 yrs	CBT/5–12 sessions	Beneficial treatment effects in four subjects; some benefit in six subjects, and no benefit in nine subjects
Walker et al. (2005)/Class III	11 TBIs; mean age 30 yrs	CHRP/18 months	Equivocal because of not clear anti-depressant effects, and light changes on self-rated depression, anxiety, stress, general well-being, and family ratings on overall difficulties
Anson and Ponsford (2006a)/Class III	33 TBIs; mean age 38 yrs	CBT/10 sessions	Significant variance in percentage change of depression. Better outcomes associated with greater self-awareness
Gracey et al. (2007)/Class III	1 Stroke 42 y/o	CBT/10 weeks	Reduced anxiety and depression symptoms, and frequency of avoidant behaviors but slight increasing at follow-up
Dewar and Gracey (2007)/Class III	1 Encephalitis 43 y/o	CBT/24 weeks	Equivocal because of not clear anti-depressant effects; anxiety and depressive symptoms remained elevated with anxiety levels increased at follow-up

TBI Traumatic Brain Injury, *CBT* Cognitive-Behavior Therapy, *CHRP* Comprehensive-Holistic Rehabilitation Program, *KAS* Katz Adjustment Scale, *IADL* Instrumental Activities of Daily Living

Chronicity

This was defined as the length of time from injury-onset to initiation of treatment. Chronicity varied from studies in which participants were within 1–4 weeks (a total of 133 participants) to more than 5 years post-onset, with most individuals within 1–4 years from injury (351 participants). Several group studies reported wide variability in chronicity: four group studies (150 participants) reported chronicity ranging from 1 month to 7, 10 or 30 years injury-to-onset. Six group studies (100 participants) reported chronicity ranging from 6–12 months to 10, 12, 21 or 36 years since injury.

Treatment Type

As shown in Table 3, 33 studies reported results for a total of 151 participants treated primarily with applied behavior analysis: 11 studies (40 participants) used traditional CMP procedures, four PBI (10 participants) and 18 studies (101 participants) used a combination of these two approaches. Most papers (31 studies) reported results of Class III single-subject studies or case series without experimental controls. Two RCTs-Class I studies were rated (Carnevale et al. 2002, 2006); in both cases, a combination of CMP and PBI procedures were used on a total of 23 participants with ABI of mixed aetiology; no substantial changes for treated disorders (aggressive and disinhibited behavior) were detected.

Cognitive-behavior therapy (CBT) not included in a comprehensive-holistic rehabilitation program was reported in 13 papers involving 201 participants. Four-Class I and Class II studies treated a total of 85 individuals with behavioral disorders following TBI or brain injury of mixed aetiology; no substantial changes or mixed results were reported (see Table 4).

As shown in Table 5, 17 studies reporting treatments based on a comprehensive-holistic rehabilitation program (CHRP) involving a total of 742 individuals with brain injuries of mixed aetiology compared with 113 control subjects. Two RCT-Class I studies were rated. Significant treatment effects (improved community functioning, perceived self-efficacy and quality of life) which were maintained at a 6-month follow-up were reported for 34 subjects with mild-to-severe traumatic brain injury (Cicerone et al. 2008). Otherwise a 67-sample of military personnel with moderate-to-severe traumatic brain injury compared with a parallel group (53-blinded individuals) that received telephone counselling at home did not report any treatment effect in quality of life, return to employment, or fitness for duty at 1 year post-treatment (Salazar et al. 2000). Two non-RCT-Class II studies reported improvements in life functioning, psychosocial competence, emotional adjustment and

Table 3 Results and outcome for treatments based on applied behavior analysis

Treatment type	Class	Studies	Participants		Results and outcome
			Treated individuals	Controls	
		$n=33$	$n=151$	$n=45$	
Contingency Management Procedures (CMP)	III	11	40	0	Positive behavioral changes for seven treated individuals (five studies)
					Mixed results for 33 subjects (six studies)
Positive Behavior Interventions (PBI)	III	4	10	0	Positive behavioral changes for five treated individuals (three studies)
					No substantial changes for five subjects with traumatic brain injury (one study)
Combined CMP and PBI	I	2	23	45	No substantial changes for 23 treated individuals with injuries of mixed aetiology (two studies)
	III	16	78	0	Positive behavioral changes for 27 treated individuals (nine studies)
					Mixed results for 10 treated individuals (five studies)
					No substantial changes or equivocal results for 41 treated individuals with injuries of mixed aetiology (two studies)

Table 4 Results and outcome for treatments based on cognitive-behavior therapy

Treatment type	Class	Studies	Participants		Results and outcome
			Treated individuals	Controls	
		n=13	*n*=201	*n*=120	
Cognitive-Behavior Therapy (CBT)	I	1	34	84	No substantial changes for subjects with stroke (one study)
	II+	2	39	24	Mixed results for 31 subjects with traumatic brain injury (one study)
					No substantial changes for 8 subjects with brain injury of mixed aetiology (one study)
	II	1	12	12	No substantial changes for 12 subjects with traumatic brain injury (one study)
	III	9	116	0	Positive behavioral changes for 63 subjects with brain injury of mixed aetiology (three studies)
					Mixed results for 21 subjects with traumatic brain injuries or strokes (three studies)
					No substantial changes or equivocal results because of added neuroleptics for 32 subjects with brain injury of mixed aetiology (three studies)

Table 5 Results and outcome for treatments based on a Comprehensive-Holistic Rehabilitation Program (CHRP)

Class	Studies	Participants		Results and outcome
		Treated individuals	Controls	
	n=17	*n*=742	*n*=113	
I	2	101	87	Significant treatment effects (improved community functioning, perceived self-efficacy and quality of life) for 34 subjects with traumatic brain injury (one study)
				No treatment effect at 1 year post-treatment for 67 individuals with traumatic brain injury (Military personnel) in quality of life, return to employment, or fitness for duty (one study)
II	2	36	26	Positive results (improvements in life functioning, psychosocial competence, emotional adjustment and employment) for 36 treated subjects with brain injury of mixed aetiology compared with 26 controls (two studies)
III	13	605	0	Positive results for 212 subjects with injury of mixed aetiology (five studies)
				Mixed results for 382 subjects with injury of mixed aetiology (seven studies)
				Light changes and equivocal results because of added neuroleptics for 11 subjects with traumatic brain injury (one study)

employment for 36 treated subjects with brain injury of mixed aetiology compared with 26 controls (Prigatano et al. 1984; Fryer and Haffey 1987).

Duration, Frequency and Intensity of Interventions

Treatment duration ranged widely, from a minimum of 2 weeks to a 10-year period reported in one single-case study in which cognitive-behavior therapy carried out at home (Williams et al. 2003). One study did not specify the treatment duration (Malec and Moessner 2000); however it was concerned with a comprehensive-holistic rehabilitation program (CHRP) then a treatment duration of at least 3-to-6 months should be supposed. Six studies reported interventions ranging from 10–126 sessions rather than days or weeks. Most interventions lasted for at least 1 month and generally ranged from 1 to 6 months (42 studies); nevertheless, five single-case studies reported treatment duration of less than 30 days in which primarily CMP or PBI were used.

Information regarding frequency and intensity of interventions was omitted from approximately two-thirds of the studies, and when specific information was provided substantial variability was observed, ranging from intensive treatments of several hours a day for several months (this was the case in comprehensive-holistic approaches) to no more than 30 min twice a week for 1 month.

Intervention Setting

One study did not specify the intervention setting (Medd and Tate 2000). Most intervention settings included residential rehabilitation services (acute, post-acute, and long-term care facilities) and inpatient or outpatient-medical day programs. Community-based rehabilitation or comprehensive day treatment was precisely the intervention setting of comprehensive-holistic rehabilitation programs (CHRPs). Family homes, educational environment, and other natural settings were reported by a minority of studies for a total of 76 participants. Individual session was the intervention setting reported by 35 studies: all but six cognitive-behavior therapies (CBTs) were based on applied behavior analysis (CMP and PBI).

Outcome Measures

Decreasing the intensity and frequency of specific externalizing behaviors (physical and verbal aggression, uncontrolled and disruptive behavior, self-injury or sexually inappropriate behavior) was the primary outcome measure applied in studies which reported CMP, PBI or combined treatments. Productivity level, standardized functional scales and customized rating scales or self-rated questionnaires were generally used as primary or secondary outcome measures to analyze behavioral and psychosocial changes in the studies reporting CBT interventions and comprehensive-holistic approaches (CHRP) (see the legend of the Appendix).

Maintenance Over Time and Generalization/Social Validity of Treatment Effects

Only 23 of the 63 reviewed studies included quantitative or anecdotal follow-up reports. The length of time from discharge/end of treatment ranged widely, from a minimum of 1 or 2 months for the participants in three single-case reports to at least 1 year (14 studies involving 509 participants). In the remaining seven studies (involving 87 participants), the follow-up ranged from 3 to 6 months. Most studies reported at least some maintenance of the gains recorded at discharge/end of treatment. Nevertheless, mixed, equivocal, or no maintenance of treatment effects over time was reported in 6 out 14 studies with at least 1-year follow-up.

Countries of Origin of the Reviewed Studies

With respect to the countries of origin, 34 studies were conducted in the United States, 22 in three European countries: 17 in the United Kingdom, four in Denmark, and one in The Netherlands. Seven studies originated from Oceania: six from Australia and one from New Zealand.

Evidence-based Recommendations

Overall, 63 studies of treatments delivered to 1,094 adults for challenging behavior and psychosocial problems following moderate-to-severe acquired brain injury were selected and rated to provide evidence-based recommendations.

Treatment Efficacy

The present review highlights omissions and methodological weaknesses concerning (1) frequency and intensity of interventions; (2) systematic measures of target behavior before, during and after treatment; (3) clear information about pre-injury skill levels and pre-existing psychopathological conditions; (4) detailed description of patients' clinical picture (such as severity of neurological impairment and profile of associated cognitive deficits); (5) rigorous control of medications and pharmacological treatments often administered as an adjunct to neuropsychological rehabilitation. Consequently, although the reviewed studies document benefits, it is difficult to establish how successful a particular treatment will be for a patient due to the heterogeneity of aetiologies and range of severity associat-

ed with ABI. Moreover, often behavioral changes and psychosocial improvements cannot be clearly attributed to a specific intervention, and the effects of the natural course of healing or other confounding factors were not separated out. This is especially true when behavioral treatment occurs within the first year of injury.

Clinical Effectiveness of Behavioral and Psychosocial Interventions

Only 23 of the 63 reviewed studies included quantitative or (more often) anecdotal follow-up reports. Moreover, mixed, equivocal, or no maintenance of beneficial effects was reported in 6 of the 14 studies with at least a 1-year follow-up, that is, approximately half of the patients receiving treatment failed either to generalize or to maintain long-lasting behavioral or psychosocial improvements.

Notwithstanding the above mentioned considerations, following we provide separate evidence-based recommendations for the different interventions considered.

Approaches Based on Applied Behavior Analysis

The results of the present analysis (summarized in Table 3) indicate that most interventions based on CMP, PBI, or a combination of both, demonstrated improvement in target behaviors (i.e., internal validity or efficacy); however, the absence of Class I and Class II studies suggests that approaches based on applied behavior analysis for post-injury behavior disorders should be viewed as Practice Options and prevented us from reaching stronger recommendations. This conclusion is consistent with that of Ylvisaker et al. (2007) based on a smaller sample of participants. Conclusions that treatments based on CMP and PBI are only evidence-based practice options. It does not mean that these treatments are not useful for the management and modification of maladaptive behavior. From a clinical point of view, the positive results (i.e., reduction of frequency and intensity of target behavior disorders) reported by most single case-Class III studies on individuals with predominantly impulse control disorders and non-compliant or oppositional attitudes (including impulsivity, irritability, aggression, disinhibition, and confabulation) are of great interest. This highlights the importance that improved methodologies have to be considered in future studies using these approaches.

Cognitive-behavior Therapy

Difficulties in determining the level of evidence for the effectiveness of contingency management procedures and positive behavior interventions also apply to CBT. Most of the reviewed studies based on this approach demonstrated improvements (i.e., internal validity or efficacy) in at least one dependent variable. However, due to the unsuccessful or equivocal outcomes reported in RCT-Class I and cohort-Class II studies (Ruff and Niemann 1990; Medd and Tate 2000; Lincoln and Flannaghan 2003) combined with the negative or equivocal results raised from uncontrolled-Class III studies (Ownsworth and McFarland 2004; Swan and Alderman 2004; Dewar and Gracey 2007), it appears that interventions promoting internalizing of self-regulation strategies through self-instruction or self-monitoring and integrated treatment of individualized cognitive and interpersonal therapies should be considered as Practice Options for persons with impaired executive functioning and emotional self-regulation following TBI or stroke (see Table 4). These conclusions are similar to those reached in the reviews by Cicerone et al. (2000, 2005).

Comprehensive-holistic Rehabilitation Programs

One RCT-Class I and two Class II studies reported significant treatment effects with CHRP on a total of 70 patients (Prigatano et al. 1984; Fryer and Haffey 1987; Geurtsen et al. 2008) (Table 5). These results provide evidence that CHRP can be recommended as practice standard for the treatment of people with acquired neurobehavioral impairments and psychosocial problems. However, it should be noted that one RCT-Class I study reviewed here (Salazar et al. 2000) provided no strong clinical effectiveness for this type of treatment approach (further comments on this study will be presented below).

A Comparison of the Different Types of Treatment

On the basis of these evidence-based recommendations, a definite statement on the relative effectiveness of the different types of treatment programs in relationship to patients with different aetiology and severity appears premature given the series of issues that need to be addressed. First, only a small number of reported treatments were administered to individuals whose brain damage had a different aetiology than TBI. Further, 5 out of 10 RCT-Class I and Class II controlled group studies examining participants with a pure aetiology referred to CHRP or CBT interventions applied to individuals with TBI, and only one RCT-Class I study reported data for CBT applied to subjects with depressive symptoms after stroke. This prevents us from reaching definite conclusions based on patients' aetiology.

Discussion

Overall, the present review synthesizes for the first time findings of a large body of studies dealing with more

than 1,000 patients with ABI and underscores the considerable potentiality of developing neuropsychological rehabilitation programs for behavioral and psychosocial disorders. It must be stressed that behavioral disturbances constitute a fundamental problem in the management of patients with ABI; indeed, effective intervention may have far-reaching effects on the interpersonal relations and daily living activities of patients. Therefore, it seems important that treatment of behavioral disturbances be given a central place in rehabilitation programs, not just an ancillary role in cognitive and motor treatments. At the same time, the review highlights a series of important limitations of the studies available thus far that seriously limit the recommendations that can be offered on the basis of empirical evidence. The following issues spell out the challenges remaining for researchers and clinicians in the field of neurobehavioral and psychosocial rehabilitation:

(a) There is considerable heterogeneity in the reviewed studies regarding individual variables, particularly in studies with large samples. This variability arises from demographic characteristics of participants, etiology and site of brain damage, time since injury, severity of target behavior, severity of associated cognitive impairments and general clinical picture, confounding bias of prior treatment history, pre-existing psychiatric illness, and associated pharmacological therapies. The goal of future studies should be to make a better clinical and pathological definition of individuals receiving treatment for neurobehavioral disorders based on detailed descriptions of demographic and clinical conditions. As suggested by Carney et al. (1999), one solution might be to limit the range of clinical severity, chronicity, premorbid factors and any other source of individual differences so that each study can specifically describe the category of individuals being treated and evaluated.

(b) Most studies omitted information on the site of the brain injury or limited themselves to routine neuroradiological examinations (i.e., CT or MRI). However, it is well established that this type of analysis is insufficient for adequately describing the nature of damage particularly with traumatic brain injuries, which represent most cases with behavioral and psychosocial disorders. These patients may show focal cortical contusions resulting from inertial forces causing localized damage in ventral and polar frontal and anterior temporal areas where the brain is confined by bony ridges of the inner skull. However, the major and most devastating consequence of the lesion is commonly represented by diffuse axonal injury (DAI), i.e., widespread disruption of axons occurring during abrupt acceleration or deceleration (Graham 1996). Importantly, DAI is largely undetected by standard CT or MRI examinations; however, a number of MRI sequences sensitive to DAI have been developed. For example, T2*-weighted gradient-recalled echo (GRE) sequences detect field in homogeneities due to paramagnetic blood breakdown products, such as deoxyhemoglobin, methemoglobin, and hemosiderin (Scheid et al. 2003). Effective results have also been reported using turbo Proton Echo Planar Spectroscopic Imaging (t-PEPSI), a very fast sequence that is particularly suited to uncooperative and medically unstable patients (Giugni et al. 2005). Recently, great interest has been shown in diffusion tensor imaging (DTI), an MRI modality that measures the microscopic molecular motion of water in order to investigate white matter microstructure. DTI is a potentially important technique for identifying in vivo white matter microstructural alterations following TBI (Garnett et al. 2001). It has not yet been used in the context of rehabilitation trials; however, Sidaros et al. (2008) hold that DTI repeated during rehabilitation may prove particularly effective in capturing the relationship between long-term clinical outcome and mechanisms of neuroplasticity and repair during recovery from TBI. Also functional imaging studies (based on SPECT, PET or fMRI) may contribute important information on brain-behavior relationships, particularly if tasks appropriate for detecting the behavioral changes are developed (for a review see Levine et al. 2006). For example, Schmitz et al. (2006) used fMRI to test patients with TBI on a task involving self-appraisal of one's traits and abilities. Increased activation associated with self-evaluation was reported in the anterior cingulate, right anterior temporal pole, and the precuneus. The authors concluded that post-injury level of self-referential insight is related to a neural network that includes the medial and right dorsal prefrontal cortices. However, only a few studies have tackled rehabilitation issues with functional neuroimaging (for a review see Strangman et al. 2008) and they have been limited to motor and cognitive deficits in stroke patients. Overall, until now research on the rehabilitation treatments of persons with behavioral disorders following ABI has given little attention to the neural bases of recovery. However, recent advances in neuroimaging (including DTI) will make it possible for rehabilitation studies to directly consider the role of brain structures in recovery after treatment.

(c) Only three studies outlined a variety of premorbid conditions that could have introduced a bias. However, differentiating between maladaptive social competency as an outcome of premorbid psychopathology and

post-injury residual neuropsychological damage is complicated, and it is often difficult to make a correct differential diagnosis (Gagnon 2006). A broad range of factors can play a role in maintaining problem behavior after brain injury: the extent and nature of the brain damage, premorbid personality traits, behavior and skill level, and the physical, cognitive and emotional sequelae of the injury. Thus, there are many sources of diagnostic confusion. For example, much of the cognitive and behavioral phenomenology typically encountered in ABIs (e.g., difficulty with social communication skills, poor quality of intellectual functions and apathy) closely resembles developmental disabilities or other psychopathological disorders of different etiology. Moreover, premorbid psychopathological traits and neuropsychologically based personality changes may coexist, influencing each other thus creating comorbidity. In this case, it may be difficult for the clinician to determine which traits belong to the basic character of the person and which maladaptive behaviors are consequences of ABI.

(d) Information regarding the treatment dosage (i.e., frequency and intensity of interventions) was omitted from approximately two-thirds of the studies. Even when information was provided, great variability was observed ranging from intensive treatments of several hours a day to no more than 30 min twice a week. Frequency and intensity of intervention may not be relevant in environmental approaches with multidisciplinary professionals and/or family members trained to implement the intervention throughout the day; this was the case in many of the 63 reviewed studies. Nevertheless, knowing the frequency and intensity of the intervention as well as its duration could contribute to determining treatment effects.

(e) Only nine studies examined individuals with predominantly internalizing disorders. In all cases consisting of depressive-type symptoms and with a great variability of injury severity, ranging from mild-to-severe injuries (Table 2). Thus, the psychogenic background of their dysphoric mood disorder might have confounded the participants' clinical conditions. All treatments were based on cognitive-behavior therapy except for one comprehensive-holistic program: all but one reported predominantly equivocal or insubstantial beneficial treatment effects. Based on these findings, some issues need to be discussed. The first is why intervention studies have targeted external rather than internal psychopathology, given that persons with ABI are particularly prone to defective or depressive-type problems (Finset and Andersson 2000; Rao et al. 2007). Second, the effectiveness of rehabilitation approaches based on cognitive-behavior therapy, particularly if applied to internal psychopathologies and depressive-type symptoms, needs to be further investigated. Indeed, cognitive-behavior therapy has beneficial results in many specific psychiatric disorders of primary/psychogenic nature, but the rates of positive outcomes are still not satisfactory for diagnostic categories such as social phobia or generalized anxiety disorder (for a review see Leichsenring et al. 2006).

(f) Evidence-based medicine is rooted in the belief that the strongest evidence is based on RCTs, which control for biases better than other methodologies. Nevertheless, Kennedy and Turkstra (2006) noted that even though the RCT framework has been adopted by behavioral therapists it was originally designed for clinical drug trials, many features of which are not appropriate, practical, or relevant in behavioral intervention research. Moreover, RCTs were initially designed to measure change by using a restricted number of primary outcome measures. Thus, due to the wide individual variability among clinical features following an ABI (with the consequent need to include multiple outcome measures) Kennedy and Turkstra (2006) concluded that a single-case rather than an RCT approach might be useful to gather evidence for effective interventions. In fact, behavioral treatments applied during individually tailored interventions should provide the best available advice, especially in an inpatient/residential rehabilitation environment. However, there is a problem with external validity. Nevertheless, sophisticated single-subject experimental designs, which use multiple baseline measures or counterbalanced designs that allow attributing observed effects specifically to the treatment or the component of treatment of interest, should be encouraged. Also noteworthy are Prigatano's (1999, 2003) criticisms. He noted that review studies often focus on methodology more than on the phenomena under investigation, and that if not properly used RCT designs may actually lead to misleading findings. In his commentary, the author discusses the study by Salazar et al. (2000)—one of the only five RCTs-*Class I* studies rated in the present review—as an example of what he defines as the "*dogma that randomized controlled studies provide the most convincing evidence regarding the efficiency of an intervention*". Prigatano (2003) stated the following reasons for the putatively misleading conclusions of Salazar et al. (2000) on the ineffectiveness of MORP in TBI individuals: (1) the program was designed for postacute rather than acute patients (within the first month from injury in this study); (2) the working alliance between the patient and the

rehabilitation team as a predictive value for those benefiting from such treatment was neither mentioned nor studied; (3) the unusually high rate of return to work (90%) may indicate that patients were much less severely impaired than those reported in other studies. Prigatano's main conclusions were twofold: (1) a potentially useful program (MORP) has been applied inappropriately; (2) careful clinical observation of patients who benefit from rehabilitation programs compared with those who fail is the most important method for advancing knowledge and aiding clinical practice in this field. Prigatano's opinion is synthesized by his statement "*first-class evidence is based on first-class observation, not on randomized controlled studies*".

(g) Unlike medical interventions, behavioral treatments for individuals with ABI should always be primarily aimed at promoting meaningful participation in social, work and leisure activities (Sohlberg and Mateer 2001). However, this does not mean that interventions focused on reducing specific challenging behaviors (rather than promoting social participation) could not constitute a unique and reasonable goal in individuals with severe clinical conditions and very little chance of returning to school or work or living independently. Apart from research reporting a comprehensive-holistic approach or a community-based intervention, few studies have evaluated maintenance over time and generalization of treatment effects to everyday functioning and natural community environments. The absence of follow-up reports in 40 out of the 63 reviewed studies, with only 14 studies reporting the maintenance of treatment effects for at least 1 year, is a substantial shortcoming of this evidence base. Future efforts to validate the clinical utility of rehabilitation treatments for behavioral and psychosocial disorders resulting from ABI should include systematic and valid outcome measures that reflect levels of everyday functioning, community integration and quality of life of the treated persons. The use of reliable outcome measures might allow evaluating the effect size of a treatment (Cohen 1988) with regard to symptoms, social adjustment and other outcome criteria, as has been done for other neuropsychological domains (Rohling et al. 2009).

Conclusions

Because outcomes of persons with ABI could be the result of a combination of factors other than treatment, including type and severity of brain injury, recovery level, premorbid characteristics and subjects' social conditions, it remains difficult to determine the role of these uncontrolled factors in treatment effects. For ethical and practical reasons, often the methodology in rehabilitation for neurobehavioral and psychosocial disorders does not fulfill the ideal of the double-blind, randomized control study. Nevertheless, better-controlled research is needed, with rigorous inclusion/exclusion criteria and greater specification of the theoretical basis, design and contents of treatments for interdisciplinary or traditional remedial interventions and also for cognitive-behavior therapy. In this regard, Malec and Basford's (1996) indications for increasing the quality of studies should be taken into account: (1) using randomly assigned waiting-list controls (rather than no-treatment controls) could represent a more ethically accepted option; (2) carrying out large, multicenter studies would allow gathering adequate data to support estimates of benefits and cost-effectiveness; (3) performing research based on experimental designs that contrast different combinations of treatment procedures should be incremented.

However, as pointed out by many researchers in the field of clinical psychology and psychotherapy, the conflict between evidence-based medicine and behavioral/psychosocial rehabilitation and the controversies over *efficacy*, *evidence-based practice*, and *empirically supported psychotherapies* cannot be overcome only by methodological advances or increasing pluralism in research. Indeed, they are related to wider issues about the importance of the professional judgment, the basis of knowledge and the very nature of the phenomena under investigation or the treatments adopted (e.g., Chambless and Ollendick 2001; Bower 2003; Leichsenring and Leibing 2007).

Finally, as outlined by McGlynn (1990) and Prigatano (2003), but rarely discussed in the rehabilitation literature, the quality of the therapeutic relationship plays an important role in every type of non-pharmacological treatment. A patient who does not trust or respect the rehabilitation therapist will be unlikely to cooperate and thus will not benefit as much as a patient who has a positive attitude toward the working relationship. This issue must be faced to evaluate the effectiveness of rehabilitation programs tailored for individuals with behavioral and psychosocial disorders following acquired brain injury.

Acknowledgments The research was carried out under the auspices of the Società Italiana di Neuropsicologia (Italian Society of Neuropsychology), a non-profit scientific organization.

Disclosure The authors state no conflicts of interest. The authors received no government funding pertinent to this review. The research was not funded by any corporation or by any external party directly interested in the results.

Appendix

Table 6 Table of evidence (references are listed in a chronological order)

Reference	Country/ design/class/ *support*	Treated individuals	Time since injury	Treatment		Dependent variables (measures)	Results and outcome
				Duration (intensity)	Type/setting pharmacological therapy		
Prigatano et al. (1984)	USA Non-RCT (17 untreated TBI subjects) Class II	18 TBIs; mean age 26 yrs Neuropathology: cerebral and/or brain stem contusions; 12 subjects with aphasia, dysarthria, or residual paresis. Behavioral issues: signs of general psychopathology by KAS.	6–81 months (mean 21.6 months)	6 months (6 h/die for 4-day/wk)	CHRP Combined individual and group setting	Cognitive-psychosocial competence, emotional adjustment, employment (KAS)	*Positive results.* Intervention group showed trend towards better emotional adjustment and higher incidence of employment (50%) compared with untreated group (36%).
Eames and Wood (1985)	UK Case series (6–39 month follow-up) Class III	24 ABIs; mean age 30.8 yrs; range 17–64 yrs Neuropathology: 18 severe brain stem damage; 8 mixed pictures of forebrain damage; 5 anoxia or diffuse insult. Behavioral issues: manipulation, aggression, sexual behavior, self-injury, irritability, odd behavior, reduced motivation.	1–126 months	Mean 6 months; range 3–25 months	CMP Individual setting	Placement (behavior and ADL ratings, caregiver questionnaires)	*Mixed results.* At discharge: placements/ ADL improved in 2/3 s; no improvement in 1/4. At follow-up: sexual behavior, aggression and other targeted behavior often increased; ADL remained constant; mixed results in activities, community participation and perception of improvement.
Fordyce and Roueche (1986)	USA Retrospective case series Class III	28 ABIs; age not reported (adults) Neuropathology: not reported. Group 1 (n=11) with initial high awareness; Group 2 (n=9) with initial low awareness and improved at discharge; Group 3 (n=8) with initial low awareness and not improved at discharge.	Not reported	6 months (6 h/die for 4-day/wk)	CHRP Combined individual and group setting	Awareness and vocational outcome; emotional distress (self-reports, staff and family reports)	*Mixed results.* Group 1: reduced emotional distress: Group 3: increased emotional distress. No group difference in vocational outcome, but trend for Group 2 to have better outcome than Group 3.

Table 6 (continued)

Reference	Country/ design/class/ *support*	Treated individuals	Time since injury	Treatment		Dependent variables (measures)	Results and outcome
				Duration (intensity)	Type/setting pharmacological therapy		
		Behavioral issues: emotional distress, reduced self-awareness.					
Ben-Yishay et al. (1987)	USA Case series (3-yr follow-up) Class III	94 ABIs; age 18–55 yrs Neuropathology: not reported.Specific behavioral issues not reported.	Not reported (long-term since injury)	At least 6 months plus 3–9 months of supervised trials	CHRP Combined individual and group setting	Levels of productivity (ERS)	*Mixed results.* At discharge: 84% classified as able to engage in productive activity, with 63% at a competitive level. At follow-up: no stable results over 3 years, with an emerged tendency for more subjects to be rated as unemployable.
Fryer and Haffey (1987)	USA Non-RCT (9 untreated ABI subjects) and a 1-yr follow-up Class II	18 ABIs; age not reported (adults) Neuropathology: not reported. Specific behavioral issues not reported.	Not reported (long-term since injury)	At least 6 months	CHRP Combined individual and group setting	Cognitive and psychosocial competence; employment	*Positive results.* At discharge: significant reduction in disability status for the subjects receiving treatment. At follow-up: 50% were engaged in their preinjury role-related activities.
Giles et al. (1988)	UK Case study Class III	1 TBI 27 y/o Neuropathology: extensive depressed right-sided fracture; evacuated right-temporal haematoma; right parietal contusion. Behavioral issues: talkativeness, attention-seeking verbal aggression, inappropriate response to frustration/criticism.	2 years	1 month (30 min/wk)	CMP Individual setting	Number of utterances per minute across different types of conversations	*Mixed results.* Decreased utterance across any type of conversations, but small improvements in semi-structured conversations.
Godfrey and Knight (1988)	New Zealand Case study	1 TBI 25 y/o Neuropathology: right	2 years	5 months	Combined CMP and	Psychosocial functioning,	*Mixed results.* At discharge: significant

	(1-yr follow-up) Class III	frontal and temporal damage. Behavioral issues: aggressive and sexually inappropriate behavior; poor social and adaptive skills.			PBIIndividual setting	adaptive and social behavior (ABS)	improvement in adaptive measures and social functioning. At follow-up: maladaptive behavior still present.
Hegel (1988)	USA Case study Class III	1 TBI 18 y/o Neuropathology: visual-perceptual abnormalities; hemiplegia; atrophied skeletal muscles. Behavioral issues: disinhibition, apathy.	2 months	26 days	CMP Individual setting	Disruptive vocalizations during therapy as a measure of compliance; % of goals met	*Positive results.* Disruptive vocalization decreased to zero; compliance increased; increased % of goals met.
Zencius et al. (1989a)	USA Case study Class III	2 TBIs 16 y/o Neuropathology: P1: hemiparesis, left visual difficulties; P2: bifrontal and left temporal contusions; hemiparesis. Behavioral issues: impulsivity, refusal to attend therapy. P1: history of substance abuse.	P1: 6 years P2: 2 years	1 month	Combined CMP and PBI Individual setting	Frequency of profanity in therapy sessions	*Positive results.* Profanity reduced to near zero in all treatment sessions and settings.
Zencius et al. (1989b)	USA Case study Class III	2 TBIs 24 y/o P1: history of substance abuse. Neuropathology: P1: left hemiparesis; P2: right hemiparesis, balance deficits. Behavioral issues: P1: inertia, impulsivity, unauthorized breaks at work; P2: non-compliance at physical therapy.	P1: 6 years P2: 2 years	P1: 16 days P2: 42 days	Combined CMP and PBI Individual setting	P1: number and duration of unauthorized breaks at work P2: % of time attending therapies	*Positive results.* P1: unauthorized breaks reduced to zero. P2: 100% increased attending of therapies.
McMillan et al. (1990)	UK Case study (3-month follow-up) Class III	1 Encephalitis 38 y/o Neuropathology: right frontal and temporal damage. Behavioral issues: violent	8 months	10 weeks for aggression; 27 weeks for hypersexuality	CMP Individual setting Neuroleptic therapy	Frequency of targeted behavioral disorders	*Mixed results.* At discharge: aggression reduced to zero; hypersexual behavior reduced to less than 1/die;

Table 6 (continued)

Reference	Country/ design/class/ *support*	Treated individuals	Time since injury	Treatment		Dependent variables (measures)	Results and outcome
				Duration (intensity)	Type/setting pharmacological therapy		
		behavior, sexual disinhibition, social inappropriateness, lack of hygiene routine, hyperphagic tendency.					hygiene required less prompting. At follow-up: social inappropriateness still noted.
O'Reilly et al. (1990)	USA Case study (2-month follow-up) Class III	4 TBIs; 20, 37, 18, 19 y/o Neuropathology: neuromotor deficits. Specific behavioral issues not reported, but all were safety risks in living environment because of deficient home hazard prevention skills.	P1: 8 years P2: 1 year P3: 3 years P4: 1 year	2–4 weeks (5–50 min/wk)	Combined CMP and PBI Individual setting	% of completed task (Checklists of hazardous items)	*Mixed results.* At discharge: all participants improved. At follow-up: only 2/4 maintained skills.
Ruff and Niemann (1990)	USA Non-RCT (12 matched controls) Class II *Private foundation*	12 TBIs; mean age 28 yrs Neuropathology: not reported. Behavioral issues: psychoticism, withdrawn depression.	1–7 years	8 weeks (160 h distributed into 4 modules)	CBT Group setting	Self-ratings and family reports (KAS)	*No substantial results.* In both treated and untreated groups depression was decreased by KAS.
Zencius et al. (1990)	USA Case study Class III	3 TBIs; 19, 32, 24 y/o Neuropathology: P1: left hemiparesis, dysarthria; P2: frontal damage; P3: massive subdural haematoma. Behavioral issues: P1: disinhibited sexual behavior, aggression, suicidal gestures, runaways; P2: alcohol abuse, impulsivity, inappropriate sexual behavior, episode of criminal conduct; P3: verbal abusiveness, inappropriate touching, impulsivity.	Not reported	P1: 2 months P2: 2 weeks P3: 4 weeks	Combined CMP and PBI Individual setting	P1: % of 30 min intervals of appropriate sexual activity P2: frequency of sexual exhibition P3: frequency of inappropriate touching	*Positive results.* Inappropriate sexual activity decreased to zero or at least acceptable levels while appropriate interaction increased.

Alderman (1991)	UK Case study (5-week follow-up) Class III	1 TBI 24 y/o Neuropathology: right hemisphere damage; left hemiplegia. Behavioral issues: disinhibited sexual behavior, urinal incontinence, prolonged periods of shouting.	6 years	3 weeks	CMP Individual setting	Frequency of shouting	*Positive results.* At discharge: significant reduction of shouting. At follow-up: maintained results.
Mills et al. (1992)	USA Case series (18-month follow-up) Class III	42 TBIs; mean age 29 yrs Neuropathology: mixed or diffuse damage. Specific behavioral issues not reported.	Mean 50.3 months	6 weeks; mean 64 days (6 h/die for 5-day/wk)	CHRP Combined individual and group setting	Community independence and social functioning: status in home, community, leisure and vocational functions	*Positive results.* At discharge: significant improvement in community independence and social functioning: status in home (87.5%), community (87.5%), leisure (90%), vocational (90%). At follow-up: gains continued to be maintained.
Christensen et al. (1992)	Denmark Case series (1–2 yr follow-up) Class III *Danish Health and Social Services*	46 ABIs; mean age 30 yrs; Neuropathology: not reported. Specific behavioral issues not reported (general psychosocial dysfunction).	Mean 2.9 years	4 months (6 h/die for 4-day/wk)	CHRP Combined individual and group setting	Independence-use of home assistance and health services; return to productive work; engagement in leisure activities; psychosocial functioning (Staff and family questionnaires)	*Mixed results.* At discharge: reduction in help received; increased productive activities; unchanged engagement in leisure activities and degree of social involvement. At follow-up: no significant further improvement; increased engagement in leisure activities and degree of social involvement.
Malec et al. (1993)	USA Case series (1-yr follow-up) Class III	29 ABIs; mean age 33 yrs Neuropathology: not reported. Specific behavioral issues not reported; reduced emotional, behavioral and psychosocial competence by PAI.	Mean 1463 days	At least 3 months (5.5 h/die)	CHRP Combined individual and group setting	Independent living (PAI, GAS)	*Positive results.* At discharge: 93% were living with no supervision compared with 59% on admission. At follow-up: 86% were living independently; 31% were unemployed

Table 6 (continued)

Reference	Country/ design/class/ *support*	Treated individuals	Time since injury	Treatment		Dependent variables (measures)	Results and outcome
				Duration (intensity)	Type/setting pharmacological therapy		
							compared to 76% on admission.
Teasdale et al. (1993)	Denmark Case series (1-yr follow-up) Class III	36 ABIs; mean age 31.5 yrs, Neuropathology: not reported. Specific behavioral issues not reported.	2–3 years	4.5 months (6 h/die for 4-day/wk)	CHRP Combined individual and group setting	Independence-use of home assistance and health services; employment-nature/time; leisure activities-nature/time (Staff and family questionnaires)	*Mixed results.* At discharge: reduction in help received; slight increase in productive activities substantially stable at follow-up; unchanged engagement in leisure activities and degree of social involvement. At follow-up: increased engagement in leisure activities and degree of social involvement.
Kennedy (1994)	USA Case study Class III	1 TBI 20 y/o Neuropathology: quadriparesis. Specific behavioral issues not reported.	5 years	40 sessions	PBI Individual setting Anticonvulsivant therapy	Compliance, correctness of task performances, problem behavior, social affect (MRS)	*Positive results.* Decreased problem behavior and improved compliance. MRS: 89% improved responses and social affect.
Youngson and Alderman (1994)	UK Case study Class III	1 TBI 34 y/o Neuropathology: left frontal damage. Behavioral issues: perseveration, frequent-excessive requests to use the toilet, inappropriate sexual talk.	3 years	21 sessions	Combined CMP and PBI Individual setting	Time in the community without verbalizing the need to urinate	*Mixed results.* Increased time without requests to urinate, but non significant or partial improvement of other target behavior.
Alderman et al. (1995)	UK Case study (15-month follow-up) Class III	1 Encephalitis 21 y/o Neuropathology: diffuse and bilateral temporal damage. Behavioral issues: disinhibited, verbally	1 year	126 sessions (days)	CMP Individual setting	Frequency of uncontrolled verbal output	*Mixed results.* At discharge: reduced verbal outputs. At follow-up: no transfer to community settings.

		aggressive and repetitive behavior.					
Feeney and Ylvisaker (1995)	USA Case study (1-yr follow-up) Class III	3 TBIs; 18–19 y/o Neuropathology: P1: diffuse and left frontal, parietal and temporal damage; P2: diffuse and bilateral frontal damage; P3: diffuse and left frontal-temporal damage. Behavioral issues: physical aggression, disruptive behavior, non-compliance.	P1: 5 years P2: 3 years P3: 17 years	3 weeks	PBI Individual setting	Frequency and intensity of aggressive behavior; amount of completed activities (ABC)	*Positive results.* At discharge: frequency and intensity of challenging behavior decreased; amount of completed activities increased. At follow-up: all had high school diploma; 2 subjects were working with support and 1 without support.
Teasdale and Caetano (1995)	Denmark Cohort study (1-yr follow-up) Class III	30 ABIs; age 15–55 yrs Neuropathology: not reported. Behavioral issues: obsessive-compulsive and psychoticism as most marked degree of psychopathology.	Mean 3 years	4 months (4-day/wk)	CHRP Combined individual and group setting Anticonvulsivant therapy for most subjects	Subjectively perceived distress (SCL-90-R)	*Mixed results.* At discharge: significant reduction in level of psychopathology in 6 out of 10 SCL-90-R scales. At follow-up: no significant further improvement.
Carnevale (1996)	USA Case series Class III *U.S. Dept. of Education*	11 ABIs; mean age 33.5 yrs Neuropathology: not reported. Behavioral issues: verbal/physical aggression, self-injury; lack of productive activity.	1.5–21 years (mean 10 yrs)	3 years	Combined CMP and PBI Individual setting	Target behavior selected by caregivers	*Positive results.* 82% reported reduction in target behavior and/or increased positive behavior.
Treadwell and Page (1996)	USA Case study Class III	1 TBI; 37 y/o Neuropathology: not reported. Behavioral issues: aggression, self-injury, property destruction, non-compliance.	13 years	15 days (25 sessions)	Combined CMP and PBI Individual setting	Percentage of interval without target behavior	*Positive results.* Decreased frequency of target behavior to near zero and increasing compliance.
Alderman and Knight (1997)	UK Case study Class III	3 ABIs; 58, 35, 33 y/o Neuropathology: P1: right occipital damage; hemiparesis; P2: extensive subarachnoid haemorrhage and infarctions; P3:	P1: 2 years P2: 3 years P3: 7 years	P1: 7 weeks P2: 8 weeks P3: 8 weeks	CMP Individual setting	Frequency of disruptive behavior during hygiene routine	*Positive results.* Target behavior reduced to acceptable levels; increased independence and expectations.

Table 6 (continued)

Reference	Country/ design/class/ *support*	Treated individuals	Time since injury	Treatment		Dependent variables (measures)	Results and outcome
				Duration (intensity)	Type/setting pharmacological therapy		
		quadriparesis. Behavioral issues: throwing, shouting, sexual comments, swearing, verbal abuse.					
Lincoln et al. (1997)	UK Case series Class III *Medical Research Council*	19 Strokes; mean age 67 yrs; range 31–89 yrs Neuropathology: not reported. Behavioral issues: depressive symptoms.	8–109 weeks (mean 43 wks)	3 months (3 to 15 sessions)	CBT Individual setting	Depressive symptoms; functional independence (BDI, HADS)	*Mixed results.* No significant change in functional abilities; beneficial treatment effects in 4 subjects, some benefit in 6 subjects, and no benefit in 9 subjects.
Persel et al. (1997)	USA Case study Class III	1 TBI 40 y/o Neuropathology: not reported. Behavioral issues: aggressive behavior.	13 years	13 weeks	Combined CMP and PBI Individual setting	Frequency of aggression and self-injurious behavior	*Mixed results.* 97% reduced physical aggression and self-injury, but increase during reversal phase.
Slifer et al. (1997)	USA Case study Class III	3 TBIs; 16, 17, 16 y/o Neuropathology: not reported. Behavioral issues: verbal/ physical aggression, grabbing, throwing, refusal to participate in therapy.	P1: 16 days P2: 13 days P3: 65 days	P1: 50 days P2: 25 days P3: 76 days	CMP Individual setting	% therapy sessions attended; % intervals without disruptive behavior	*Mixed results.* 100% increase in therapy attending; disruptive behavior reduced to near zero, but demands systematically increased.
Aeschleman and Imes (1999)	USA Case study Class III	5 TBIs; 20, 24, 27, 30, 39 y/o Neuropathology: P1: left hemisphere damage; P2: diffuse anoxia; P3: frontal and posterior-parietal damage; P4: not reported; P5: brain stem injury. Behavioral issues: verbal/ physical aggression, destructive behavior.	16 months to 12 years	10 weeks (20 sessions of 50 min 3 times/wk)	PBI Individual setting	Impulsive aggression: verbal, gestural, physical (Role-play probes; Counsellor questionnaire)	*No substantial results.* Slight reduction in impulsive behavior. Role-play probes improved, but judged to lack validity.

Alderman et al. (1999)	UK Case study Class III	3 ABIs; 34, 45, 40 y/o Neuropathology: not reported. Behavioral issues: aggression, disinhibited sexual behavior.	P1: 4 months P2: 3 years P3: 2 years	P1: 5 weeks P2: 6 weeks P3: 60 weeks	Combined CMP and PBI Individual setting Neuroleptic therapy	Frequency and intensity of aggressive behavior (OAS-MNR)	*Mixed results.* P1: aggression reduced to zero in 4 weeks. P2: frequency slightly reduced and intensity not reduced. P3: minimal results until expectations were dramatically reduced.
Rothwell et al. (1999)	USA Case study Class III	2 ABIs; 31, 42 y/o Neuropathology: P1: subdural temporal-parietal haematoma; P2: right temporal haematoma; left hemiplegia and neglect. Behavioral issues: physical aggression, abusive shouting.	P1: 20 months P2: 24 months	P1: 3 months P2: 17 weeks	Combined CMP and PBI Individual setting	Frequency of physical aggression	*Positive results.* Frequency of physical aggression reduced to zero. P2: returned to home with no reported problems.
Schlund and Pace (1999)	USA Case study Class III	3 TBIs; 27, 33, 48 y/o History of substance abuse in 2 cases. Neuropathology: not reported. Behavioral issues: physical/verbal aggression, non-compliance, inappropriate sexual behavior.	P1: 4 years P2: 9 years P3: 4 years	P1: 18 weeks P2: 8 weeks P3: 56 weeks	Combined CMP and PBI Individual setting Neuroleptic therapy	Frequency of challenging behavior P1: pseudoseizures P2: inappropriate sexual behavior P3: non-compliance	*Positive results.* All target behavior reduced in frequency.
Hegel and Ferguson (2000)	USA Case study Class III	1 TBI 28 y/o Neuropathology: ataxia, speech apraxia. Behavioral issues: verbal/physical aggression.	10 years	50 days	CMP Individual setting	Frequency of aggressive behavior	*Positive results.* Aggressive behavior reduced to near zero.
Malec and Moessner (2000)	USA Case series Class III	62 ABIs; mean age 35 yrs Neuropathology: intracranial haematoma in 20 subjects. Specific behavioral issues not reported: array of cognitive, emotional, behavioral and functional impairments by MPAI.	45 days to 30 years (median 679 days)	Not reported	CHRP Combined individual and group setting	Functional, cognitive, and emotional adjustment (MPAI)	*Mixed results.* Impaired awareness and distress diminished after program; better awareness and less distress predicted positive behavioral outcomes but not vocational outcomes.

Table 6 (continued)

Reference	Country/ design/class/ *support*	Treated individuals	Time since injury	Treatment		Dependent variables (measures)	Results and outcome
				Duration (intensity)	Type/setting pharmacological therapy		
Medd and Tate (2000)	Australia RCT (8 controls/ waitlist) Class II+	8 ABIs; mean age 35 yrs Neuropathology: not reported. Behavioral issues: anger, depression, anxiety, low self-esteem.	Not reported	5–8 weeks (1 session/ wk; mean 6 sessions)	CBT Individual setting	Anger management; self-esteem; depression-anxiety (STAAES, SEI, HADS)	*No substantial results.* Anger: both groups decreased pre with respect to post treatment (experimental group greater decrease); no transfer to self-esteem, anxiety, depression, self-awareness.
Ownsworth et al. (2000)	Australia Case series (6-month follow-up) Class III *BIA of Queensland*	21 ABIs; mean age 33.5 yrs Mild-to-severe injury Neuropathology: 4 bilateral frontal damage; 6 right frontal damage; 3 left frontal damage; 3 frontal-parietal damage; 3 diffuse (multiple) damage; 2 brainstem/occipital damage. Behavioral issues: low level of self-control/self-regulation skills, reduced self-awareness, psychosocial problems.	1–36 years (mean 8.6 yrs)	16 weeks	CHRP Group setting	Psychosocial functioning, self-control, and self-awareness (SADI, HIBS, SRSI, SIP)	*Positive results.* At discharge: significant improvement in emergent-anticipatory awareness and strategy use, improved psychosocial functioning. Follow-up: gains maintained.
Yodi et al. (2000)	USA Case study Class III	1 TBI 48 y/o Neuropathology: subarachnoid haemorrhage and contusions. Behavioral issues: verbal/ physical aggression, elopement behavior.	Not reported (several months post-injury)	3.5 months	Combined CMP and PBI Individual setting	Frequency of elopement, medication refusal, verbal and physical aggression	*No substantial results.* Variable/unstable decreasing in frequency of all target behavior.
Salazar et al. (2000)	USA RCT (53 controls/ telephone counselling at home) and	67 TBIs; mean age 26 yrs; Military personnel MRI cerebral damage: 51 haematoma or contusion; 95 shear injury.	Within the first month from injury	6 months (6 h/die for 4-day/wk)	CHRP Combined individual and group setting	Quality of life, return to military duty and level of fitness for duty (KAS)	*No substantial results.* At 1 year post-treatment: no significant difference between groups in quality of life, return to employment (90% of

	1-yr follow-up Class I *Defense and Veterans HIP and Dept. of Veterans Affairs*	Behavioral issues: aggressive behavior, major depression or generalized anxiety.					treated group with respect to 94% of untreated group) or in fitness for duty (73% of treated group with respect to 66% of untreated group).
Malec (2001)	USA Case series (1-yr follow-up) Class III *TBIMS and NIDR Research*	96 ABIs; mean age 34 yrs Mild-to-severe injury. Neuropathology: not reported; physical disabilities. Specific behavioral issues not reported: global cognitive impairment with reduced self-awareness, poor social activities, by MPAI, GAS, VIS.	Mean 5 years	190 days	CHRP Combined individual and group setting	Living and vocational independence (MPAI, GAS, VIS)	*Positive results.* At discharge: significant goal achievement on GAS and improvement on MPAI. At 1 year follow-up: increased societal participation: 72% living independently; 39% working independently; 10% in transitional placements, and 18% in supported or volunteer work.
Watson et al. (2001)	UK Case study Class III	1 TBI 35 y/o; gunshot wound Neuropathology: right frontal and temporal lobotomies; epilepsy and left hemiplegia. Behavioral issues: physical/verbal aggression.	10 years	2 years	CMP Individual setting	Frequency of aggressive behavior	*Positive results.* Aggressive behavior reduced to zero.
Carnevale et al. (2002)	USA RCT study (10 controls/ baseline and 8 controls/ family education) Class I *NIDR Research*	9 ABIs; mean age 43 yrs Neuropathology: not reported. Behavioral issues: aggressive and disinhibited behavior.	Mean 8 years	8 weeks (2 h/wk)	Combined CMP and PBI Individual setting	Caregiver burden (MBI, QRS)	*No substantial results.* No significant differences between groups on MBI or QRS.
Knight et al. (2002)	UK Case study Class III	3 ABIs; 19, 44, 53 y/o Neuropathology: P1: left frontal and right temporal damage; P2: right frontal-temporal craniotomy; P3: right hemisphere haemorrhage.	P1: 5 years P2: 5 years P3: 13 years	P1: 36 weeks P2: 12 weeks P3: 8 weeks	CMP Individual setting	Frequency of target behavior	*Mixed results.* All target behavior reduced, but reduction not related to self-awareness; self-monitoring not necessary for reinforcement learning.

Table 6 (continued)

Reference	Country/ design/class/ *support*	Treated individuals	Time since injury	Treatment		Dependent variables (measures)	Results and outcome
				Duration (intensity)	Type/setting pharmacological therapy		
		Behavioral issues: aggression, sexual disinhibition, unawareness.					
Alderman (2003)	UK Case study Class III	2 TBIs; age not reported (adults) Neuropathology: dysexecutive syndrome. Behavioral issues: irritability, verbal/physical aggression.	P1: not reported P2: 4 years	P1: 22 weeks P2: 20 weeks	Combined CMP and PBI Individual setting	Frequency of aggressive behavior	*Positive results.* P1: aggression reduced to zero. P2: decrease in verbal and physical aggression.
Dixon et al. (2003)	USA Case study Class III	1 TBI 21 y/o Neuropathology: not reported. Behavioral issues: refusing to attend-participate in physical therapy sessions (left arm kept closed and wrapped around torso).	Not reported	120 sessions	Combined CMP and PBI Individual setting	Self-control: shift to choice of larger delayed reinforcer over smaller immediate reinforcer	*Positive results.* Improved self-regulation of behavior with increasing choice of delayed reinforcer.
Ebanks and Fisher (2003)	USA Case study Class III *Dept. of Health and Human Services*	1 TBI 19 y/o Neuropathology: not reported; mental retardation and pervasive developmental disorder secondary to TBI. Behavioral issues: self-injurious behavior, aggression, property destruction.	Not reported	20 sessions (10 min/session)	PBI Individual setting	Escape-motivated aggressive and self-injurious behavior	*Positive results.* Frequency of aggressive and self-injurious behavior reduced to near zero.
Lincoln and Flannaghan (2003)	UK RCT study (43 subjects/ placebo intervention; 41 subjects/ standard care)	34 Strokes; mean age 65 yrs Neuropathology: not reported. Behavioral issues: depressive disorder by SCAN.	Within the first month from injury	3 months (10 1-hour sessions)	CBT Individual setting	Internalizing disorders-depressive symptoms; ratings of satisfaction with care (BDI, WDI, ADLS-E, LHS)	*No substantial results.* No significant differences between groups in patients' mood, independence in IADLs, handicap, or satisfaction with care.

	Class I *Stroke Association*						
Willis and La Vigna (2003)	USA Case study Class III	1 TBI 20 y/o Neuropathology: bilateral frontal and right occipital damage. Behavioral issues: physical/verbal aggression, property destruction, elopement, inappropriate interaction.	3 years	10 years	CBT Individual setting	Frequency of disruptive behavior	*Mixed results.* Property destruction eliminated, but aggression continued to escalate with medication and staff changes.
Williams et al. (2003)	UK Case study (6-month follow-up) Class III	1 TBI; age not reported (adult) Neuropathology: not reported. Behavioral issues: obsessive-compulsive disorder, health anxiety, avoidance, negative self-beliefs and other maladaptive behavior.	2 years	8 months	CBT Individual setting	Anxiety, obsessive-compulsive behaviors, avoidance, negative beliefs (M-OCDS, HADS)	*Positive results.* At discharge: improvement on HADS and M-OCDS. At follow-up: maintained progress.
Ownsworth and McFarland (2004)	Australia Case series (6-month follow-up) Class III *NHMR Council*	28 ABIs; mean age 36 yrs, range 22–60 yrs Mild-to-severe injury. Neuropathology: 16 right, left or bilateral frontal damage; 3 frontal-parietal damage; 1 frontal-temporal damage; 4 diffuse damage; 3 brainstem/occipital damage; 1 no evidence of CT/MRI abnormality. Behavioral issues: denial, reduced volition, dysfunction of strategic behavior and psychosocial functioning.	1–36 years (mean 7.4 yrs)	16 weeks (90 min/wk)	CBT Group setting	Awareness, strategic behavior (SRSI, SIP)	*Equivocal results.* At discharge: clinical improvement in level of awareness in subjects with lower level of volition or reduced problem-solving skills. At follow-up: subjects with higher level of personality-related denial were less likely to improve skills in recognising and anticipating their difficulties.
Swan and Alderman (2004)	UK Case study Class III	3 TBIs; 23, 27, 43 y/o Neuropathology: P1: brain stem haemorrhages; hemiparesis; P2: diffuse	P1: 17 months P2: 1 month P3: 11 months	P1: 9 weeks P2: 9 weeks P3: 52 weeks	CBT Individual setting Neuroleptic therapy for 2/3 subjects	Frequency of aggressive behavior; level of expectation and	*Equivocal results.* Not clear neuroleptic therapy effect. OAS-MNR: decreasing

Table 6 (continued)

Reference	Country/ design/class/ *support*	Treated individuals	Time since injury	Treatment		Dependent variables (measures)	Results and outcome
				Duration (intensity)	Type/setting pharmacological therapy		
		oedema; P3: frontal lobe gliosis. Behavioral issues: verbal/ physical aggression, self-injurious behavior.				demand in the environment (OAS-MNR, NES)	of aggression related to the level of expectation/ demand.
Giles et al. (2005)	USA Case series Class III	40 ABIs; mean age 46 yrs Pre-existing psychiatric disorders in most subjects Neuropathology: not reported. Behavioral issues: physical violence, inappropriate sexual activities, non-compliance.	Not reported (long-term since injury)	20 months	Combined CMP and PBI Combined individual and group setting Neuroleptic therapy for most subjects	Frequency of violent behavior-assaults and other grossly disorganized behavior (OAS-MNR)	*Equivocal results.* Reduced frequency of client-to-client incidents, but increased frequency of client-to-staff assaults.
Niemeier et al. (2005)	USA Case series Class III	29 ABIs; mean age 45 yrs Mild-to-severe injury Neuropathology: not reported. Specific behavioral issues not reported.	2–43 days (mean 15.7 days)	10 sessions (30 min/ 3-time/wk)	CBT Group setting	Interaction skills, independence; learning and awareness resources (FIM-SI, DRS, FSLA)	*Positive results.* FSLA: 85% of the information/awareness taught in the intervention; significant and positive correlation with DRS and FIM-SI.
Walker et al. (2005)	Australia Case series Class III	11 TBIs; mean age 30 yrs, range 19–47 yrs Neuropathology: not reported; executive dysfunctions. Behavioral issues: depression, anxiety.	6 months to 21 years (mean 6 yrs)	18 months I Stage: 9 months II Stage: 9 days (camping course) III Stage: 4 months (7 meetings)	CHRP Combined individual and group setting Neuroleptic therapy	Depression, anxiety, stress; percentage of achieved goals, family burden (DASS, GWBS, EBIQ)	*Equivocal results.* Not clear neuroleptic therapy effect. Achievement of over 80% of the participants' identified goals, but little change on self-ratings of depression, anxiety, stress, general well-being, and family ratings of overall difficulties.
Anson and Ponsford (2006a)	Australia Case series Class III	33 TBIs; mean age 38 yrs Mild-to-severe injury Neuropathology: not reported.	46 days to 7 years	10 sessions (30 min 3-time/wk)	CBT Group setting	Depression, anxiety, self-esteem (HADS, CSA, RSES, SIP, STAEI,	*Positive results.* Significant variance in percentage change of depression; better

		Behavioral issues: depression, anxiety, reduced psychosocial competence, self-esteem, self-awareness, coping strategies.				PCRS, SADI)	outcomes associated with greater self-awareness.
Anson and Ponsford (2006b)	Australia RCT (16 subjects/waitlist) Class II+	31 TBIs; mean age 38 yrs Mild-to-severe injury. Neuropathology: not reported. Behavioral issues: depressive symptoms, denial, reduced social competence, coping, and self-esteem.	46 days to 7 years	5 weeks (twice-a-week)	CBT Group setting Antidepressant medication for 26% of subjects	Skill acquisitions, depressive symptoms (CSA, HADS, RSES, SIP, subjective ratings)	*Mixed results.* Unclear anti-depressant effects. Improvement of subjective understanding of emotional issues and adaptive coping. No significant change in depression, anxiety, self-esteem and psychosocial function.
Carnevale et al. (2006)	USA RCT (13 subjects/ baseline; 14 subjects/family education) Class I *U.S. Dept. of Education*	14 ABIs; mean age 40.5 yrs Neuropathology: not reported. Behavioral issues: aggressive or disinhibited behavior.	Mean 7.6 years	8 weeks (2 h/wk)	Combined CMP and PBI Individual setting	Frequency of aggressive and disinhibited behavior (NSBM)	*No substantial results.* Slight reduction in target behavior.
Schoenberger et al. (2006)	Denmark Case series Class III *German DAAD and Danish CIRIUS*	86 ABIs; mean age 43 yrs Neuropathology: 34 bifrontal, right frontal or right hemisphere damage; 52 other localizations. Specific behavioral issues not reported (reduced compliance and self-awareness).	Mean 1 year	At least 4 months (4 day/wk)	CHRP Combined individual and group setting	Compliance, working alliance, awareness (WAI)	*Mixed results.* WAI: significant improvement of working alliance for therapist scales, but for none of the patient scales; no significant variation in awareness and compliance ratings between pre-post intervention.
Gracey et al. (2007)	UK Case study (6-month follow-up) Class III	1 Stroke 42 y/o Neuropathology: cerebral convexities, left frontal regions, left side pontine lesions; seizures; cognitive dysfunctions (attention	20 months	10 weekly sessions	CBT Individual setting Anticonvulsivant therapy	Frequency of panic attacks, coping behaviors and social participation (HADS, PRS)	*Mixed results.* At discharge: improvements in anxiety and depression symptoms; reduced frequency of avoidant behaviors with

Table 6 (continued)

Reference	Country/ design/class/ *support*	Treated individuals	Time since injury	Treatment		Dependent variables (measures)	Results and outcome
				Duration (intensity)	Type/setting pharmacological therapy		
		dysfunction and memory difficulties). Behavioral issues: depression and anxiety with phobic disorders and pseudoseizures; reduced ability to manage social situations.					improved levels of social participation. At follow-up: HADS ratings slightly increased; increased frequency of 2 specific avoidant behaviors.
Dewar and Gracey (2007)	UK Case study (6-week follow-up) Class III	1 Encephalitis 43 y/o Neuropathology: right mesial temporal lobe and left temporal lobe damages extended into basal ganglia, insula and inferior frontal lobe; cognitive dysfunctions (prosopagnosia, autobiographical memory problems, impaired planning and abstraction). Behavioral issues: generalised anxiety disorder (affective distress, hopelessness, loss of interest), altered sense of identity.	16 months	24 weeks	CBT Combined individual and group setting Antidepressant therapy	Affective distress, independent living skills, self-advocacy (BAI, BDI, SCQ)	*Equivocal results.* Not clear antidepressant therapy effect. At discharge: increased self-esteem and sense of identity, but below the norm; improved acceptance; anxiety and depressive symptoms remained elevated by BDI and BAI. At follow-up: improved independent living skills; anxiety levels increased by BAI.
Cicerone et al. (2008)	USA RCT (34 control subjects/ standard neurorehabilitation) and 6-months follow-up Class I *NIDR Research*	34 TBIs; mean age 38.7 yrs Mild-to-severe injury. History of psychiatric illness (6%) or substance abuse (10%). Neuropathology: not reported; cognitive dysfunctions. Specific behavioral issues not reported (decreased	Mean 49.6 months	16 weeks (15-h/wk)	CHRP Combined individual and group setting	cognitive functioning, perceived self-efficacy, life satisfaction, employment (CIQ, PQOL, VIS)	*Positive results.* At discharge: cognitive functioning showed equal improvement under both treatment conditions but significant greater improvements for treated subjects on CIQ (ES=0.59), PQOL (ES=0.30), VIS, perceived

		community functioning and quality of life).					self-efficacy and employment (47% vs 21%). At follow-up: treated subjects maintained improvements without the need for continued rehabilitation.
Geurtsen et al. (2008)	The Netherlands Case series (1-yr follow-up) Class III *BIO Kinderre-validatie and Johanna Kinderfonds*	24 ABIs; mean age 28.5 yrs, range 17–51 yrs Neuropathology: not reported; impaired illness awareness, executive problems. Behavioral issues: 33% with alcohol and drug problems; emotional disturbances and reduced labour/work integration by GAF.	Mean 5.4 years (range 0.5–31.4 yrs)	Mean 198.9 days (range 112–382 days with at least 254 h distributed into 3 modules)	CHRP Combined individual and group setting	Emotional well-being, depressive complaints, quality of life, community integration, employability, independent living (CIQ, CES-D, Euro-QOL, ERS)	*Positive results.* At discharge: overall significant improvements on CIQ, CES-D, Euro-QOL, ERS, depression, independent living; non significant increasing for employability; all participants were no longer abusing drugs and alcohol. At follow-up: maintained most of the observed improvements.

Brain injury: *ABI* Acquired Brain Injury, *TBI* Traumatic Brain Injury

Rehabilitation treatments: *CBT* Cognitive-Behavior Therapy, *CHRP* Comprehensive-Holistic Rehabilitation Program, *CMP* Contingency Management Procedure, *PBI* Positive Behavior Procedure

Functional measures: *ABC* Aberrant Behavior Checklist, *ABS* Adaptive Behavior Scale, *ADLS-E* Activities of Daily Living Scale-Extended, *BAI* Beck Anxiety Inventory, *BDI* Beck Depression Inventory, *CES-D* Centre for Epidemiological Studies-Depression, *CSA* Coping Scale for Adults, *DASS* Depression, Anxiety and Stress Scale, *DRS* Disability Rating Scale, *EBIQ* European Brain Injury Questionnaire, *ERS* Employability Rating Scale, *Euro-QOL* European-Quality of Life, *FIM-SI* Functional Independence Measure-Social Interaction, *FSLA* First-Steps Learning Assessment, *GAF* Global Assessment of Functioning, *GAS* Goal Attainment Scale, *GWBS* The General Well-Being Schedule, *HADS* Hospital Anxiety and Depression Scale, *HIBS* Head-Injury Behavior Scale, *KAS* Katz Adjustment Scale, *LHS* London Handicap Scale, *MBI* Maslach Burnout Inventory, *M-OCDS* Maudsley-Obsessive Compulsive Disorder Scale, *MPAI* Mayo-Portland Adaptability Inventory, *MRS* Motivation Rating Scale, *NES* Neurobehavioral Expectation Scale, *NSBM* Natural Setting Behavior Management, *OAS-MNR* Overt Aggression Scale-MNR, *PAI* Portland Adaptability Inventory, *PCRS* Patient Competency Rating Scale, *PRS* Panic Rating Scale, *PQOL* Perceived Quality of Life, *QTS* Questionnaire on Resources and Stress, *RSES* Rosenberg Self-Esteem Scale, *SADI* Self-Awareness Deficits Interview, *SCAN* Schedules for Clinical Assessment in Neuropsychiatry, *SCL-90-R* Symptom Check-List-90-Revised, *SCQ* Self-Concept Questionnaire, *SEI* Self-Esteem Inventory, *SIP* Sickness Impact Profile, *SRSI* Self-Regulation Skills Interview, *STAAES* State-Trait Anger & Anger Expression Scale, *STAEI* State-Trait Anger Expression Inventory, *VIS* Vocational Independence Scale, *WAI* Working Alliance Inventory, *WDI* Walkefield Depression Inventory

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