Journal of Clinical and Experimental Neuropsychology, 27:977–1021, 2005

ISSN: 1380-3395
DOI: 10.1080/13803390490919245

Psychology Press
Taylor & Francis Group

The Effect of Moderate to Severe Traumatic Brain Injury (TBI) on Different Aspects of Memory: A Selective Review

ELI VAKIL

Department of Psychology, and the Leslie and Susan Gonda (Goldschmied) Multidisciplinary Brain Research Center, Bar-Ilan University, Ramat-Gan, Israel

Deficient learning and memory are frequently reported as a consequence of traumatic brain injury (TBI). Because of the diffuse nature of the injury, patients with TBI are not the ideal group for studying brain-behavior relations. Nevertheless, characterization of the memory breakdown following TBI could contribute to the assessment and rehabilitation of this patient population. It is well documented that memory is not a unitary system. Accordingly, in this article I review studies that have investigated the long-term effect of moderate to severe TBI on different memory aspects, including explicit and implicit tests of memory. This review demonstrates that TBI affects a large range of memory aspects. One of the conclusions is that the memory impairment observed in TBI patients could be viewed, at least to some degree, as a consequence of a more general cognitive deficit. Thus, unlike patients suffering from global amnesia, memory in patients with TBI is not selectively impaired. Nevertheless, it is possible to detect a subgroup of patients that do meet the criteria of amnesia. However, the most common vulnerable memory processes following TBI very much resemble the memory deficits reported in patients following frontal lobe damage, e.g., difficulties in applying active or effortful strategy in the learning or retrieval process. The suggested similarity between patients with TBI and those suffering from frontal lobe injury should be viewed cautiously; considering the nature of TBI, patients suffering from such injuries are not a homogeneous group. In view of this limitation, the future challenge in this field will be to identify subgroups of patients, either a priori according to a range of factors such as severity of injury, or a posteriori based on their specific memory deficit characteristics. Such a research approach has the potential of explaining much of the variability in findings reported in the literature on the effect of TBI on memory.

The prevalence of traumatic brain injury (TBI)* in industrialized countries is very high (Kraus, 1993). According to the National Center for Injury Prevention and Control (1999), it is estimated at 2% of the population. TBI frequently leads to widespread diffuse injury, and thus patients suffering from TBI are not the ideal group for studying brain-behavior relations. In light of this, it is quite surprising to find a wealth of research on the effect of TBI on memory. Memory is one of the most (if not the most) common and disabling impairments

Received 13 May 2004; accepted 1 September 2004.

This work was supported by the Paula Rich Multidisciplinary Center in Mind, Brain and Behavior, Bar-Ilan University, Ramat-Gan, Israel. The author thanks Mr. Judah Koller for his help with the preparation of the tables.

*TBI is used in this review because it is a broader term than the alternatives (e.g., closed-head injury - CHI).

Address correspondence to Eli Vakil PhD, Department of Psychology, Bar-Ilan University, Ramat-Gan, 52900, Israel. E-mail: vakile@mail.biu.ac.il

caused by TBI. A considerable number of papers continue to appear, attesting to sustained interest in the effect of TBI on memory. The prevalence of these patients in the population and their availability in rehabilitation centers probably make them an attractive research population.

Several review papers and textbook chapters on the effect of TBI on memory were published in the past (Baddeley, Harris, Sunderland, Watts, & Wilson, 1987; Goldstein & Levin, 1995a; Levin, 1989a, 1989b; Levin, Benton, & Grossman, 1982; Richardson, 2000; Schacter & Crovitz, 1977). By using new tasks and paradigms (e.g., explicit and implicit tasks), newer studies address questions not discussed in earlier reviews. Furthermore, in light of the considerable increase in general neuropsychological knowledge of memory, previous findings about TBI may be reinterpreted. Thus, there is a need for an updated review of the literature in an attempt to organize and systematically compare the results of various studies on the effect of TBI on a large range of theoretically defined memory aspects. This would serve both clinicians as well as researchers interested in the consequences of TBI on memory. A better understanding of the nature of memory impairment following TBI could contribute primarily to the assessment and rehabilitation of this patient population, and could further the understanding of memory processes in general.

The studies on memory deficit following TBI can be broadly divided into two subgroups, one that is more clinically driven and the other that is more theoretically driven. In the former group of studies, patients were typically assessed with standard memory tests (e.g., Wechsler Memory Scale - WMS: Wechsler, 1945), usually as part of a larger neuropsychological battery administered routinely to the patients. By their nature, the standard memory tests provide a range of memory measures that is very informative for clinical purposes. In the latter group of studies, patients were tested with well-controlled experimental paradigms, usually addressing a very focused question, and as such provided information on a specific memory aspect (e.g., release from proactive interference: Wickens, Born, & Allen, 1963). Obviously, the clinically driven studies could make a theoretical contribution and vice versa—the theoretically driven studies could have important clinical implications. To provide a more comprehensive understanding of the nature of memory deficit following TBI, an attempt was made in this review to integrate the different types of studies. Hence, the findings were classified into sections reflecting theoretically driven memory categories. These categories are well defined and frequently addressed in the literature on memory and amnesia, as well as in the literature on memory and TBI. Therefore, a particular study, usually clinically driven, might be reported in different sections, each time highlighting a different aspect of the findings.

This is a selective survey of the literature. It concentrates mostly on the long-term neuropsychology of memory impairment following TBI. The studies reviewed are only in English and focus mainly on groups of adults with moderate to severe TBI. The characterization of severity of TBI is based primarily on three measures: Glasgow Coma Scale (GCS), Loss of consciousness, and Post Traumatic Amnesia (PTA). In moderate TBI the GCS is 9–12, length of coma is between 20 minutes and 36 hours, and PTA is 1–7 days. In severe TBI the GCS is 3–8, length of coma is more than 36 hours, and PTA is more than 7 days (Williamson, Scott, & Adams, 1996). Levin, Goldstein, High, and Eisenberg (1988) would consider severity of injury as moderate even when GCS is "greater than 12 with at least one of the following deficits: neurological deficit, computed tomographic and/or surgical findings indicating an intracranial lesion and/or cerebral swelling, or depressed skull fracture with dural laceration"

(p. 1295) (for a similar definition of 'severe TBI' see Frankowski, Annegers, & Whitman, 1985).

There are several reasons which lead to the decision to focus the review on studies of patients with moderate to severe TBI. First, because the diagnosis of these patients is usually clearer than in mild TBI and there are fewer questions of differential diagnosis. Second, memory impairment in this group occurs frequently and is expected to be more pronounced than in milder injuries in which it is not always clear whether a memory deficit exists at all. This rules out studies with patients with heterogeneous severity of TBI (e.g., Numan, Sweet, & Ranganath, 2000), or studies in which the inclusion and exclusion criteria were not sufficiently clear to indicate that patients with mild TBI were not included. However, some studies that were included incorporated a subgroup of mildly injured patients in addition to a moderate or severe group, in order to assess the relations between severity of injury and memory (e.g., Deshpande, Millis, Reeder, Fuerst, & Ricker, 1996). Additional studies were included in which the authors defined their patients' injury as moderate or severe, even if some of the severity measures (i.e., GCS, length of coma, & PTA) are missing. Studies conducted while patients were still in the PTA stage were not included in this survey. For the most part, case reports are omitted in this review, since they characteristically present very interesting and often unique findings that are not necessarily representative of results with larger groups. In an attempt to focus on the genuine effect of TBI on memory, studies dealing with malingering, or the effects of drugs on memory, were not discussed in the present review.

Following a brief description of the characteristics of TBI, findings on memory impairment in patients with TBI are divided into three main categories: Explicit, implicit, and source-context memory. Each category is subdivided into different sections representing the memory aspects most frequently studied in that category. In the discussion section that follows, several important issues that emerged from the present review are addressed. Among these issues is the question of the resemblance between memory deficits following TBI to global amnesia on the one hand, and memory impairment following frontal lobe damage, on the other hand. Finally, based on the literature survey, some recommendations are made about how to increase consistency of the reports on memory impairment following TBI.

Neuropathology of TBI

TBI frequently leads to widespread, diffuse axonal injury (Ommaya & Gennarelli, 1974), in which the frontal and temporal lobes were found to be the most vulnerable cortical areas (Adams, 1975). In addition, head injury could lead to contusions, cerebral edema, ischemia, and hemorrhages (Bigler, 1990). Abnormalities on MRI have been frequently found in the mesial temporal and lateral frontal lobes in addition to ventricular enlargement (Crosson, Sartor, Jenny, Nabors, & Moberg 1993). Changes in ventricular size and other measures of white matter are commonly reported (Anderson & Bigler, 1995; Levin, Meyers, Grossman, & Sarwar, 1981). Furthermore, ventricle enlargement was found to be the best indicator of severity of injury and outcome, as measured by the Glasgow Outcome Scale (Henry-Feugeas et al., 2000). Hippocampal atrophy was also observed following severe TBI, possibly due to apoxia and/or high intra-cranial pressure (Bigler, Johnson, Anderson, & Blatter, 1996; Kotapka, Graham, Adams, & Gennarelli, 1992).

Functional neuroimaging studies are consistent with the structural neuroimaging findings reported above. Fontaine, Azouvi, Remy, Bussel, and Samson (1999) measured brain activity with Positron Emission Tomography (PET) in patients with TBI *at rest* in

addition to a series of tests of attention, speed of information processing, memory, and executive functions. Performance of patients on the latter two was closely associated with decreased cortical metabolism in the prefrontal cortex and the cingulate gyrus. Unlike Fontaine et al. who used the PET at rest, Ricker et al. (2001) used a [O-15]- water PET in an *active* functional neuroimaging paradigm to test five patients who sustained severe TBI. Bifrontal activation was observed in the control group and in the patients with TBI when performing a recognition task. However, during free recall, as compared to controls, patients with TBI showed reduced activation in the frontal lobe and increased activation in posterior brain regions. The authors interpreted these results as possibly indicating the patient group's use of a different strategy (phonological) than that of the controls (executive-organization) during free recall. Levine et al. (2002) also used the PET in an *active* functional neuroimaging paradigm. They compared brain activity of patients with TBI to that of the controls, *during* a memory retrieval task. The brain areas activated (i.e., frontal, temporal, and parietal) in patients with TBI during a cued recall task were similar to those of the controls. However, the patients showed additional activated areas that were not activated in the controls. The authors interpreted the results as reflecting either a cortical disinhibition, or as an indication of a compensatory effort. Diffusion Tensor Imaging (DTI) of the brain is a new MR imaging technique. This technique was found to be more sensitive than other imaging methods to diffuse axonal injury following TBI (Neil, Miller, Mukherjee, & Huppi, 2002; Rugg-Gunn, Symms, Barker, Greenwood, & Duncan, 2001). In a recent study a significant correlation was found between changes in the white matter measured with DTI and acute GCS and Rankin scores at discharge (Huisman et al., 2004).

Memory Functioning Following TBI

Several indices of TBI are used in the literature as severity of injury measures. These indices include: length of coma, GCS (Teasdale & Jennett, 1974), PTA duration (Russell & Nathan, 1946), time after onset of the injury, and extent of lesion. Based on a review of the literature, Levin (1989b) concluded that there is a moderate relationship between these measures of severity of injury and memory performance of patients with TBI. Brooks (1974a) reported a correlation between length of PTA following TBI and memory impairment. Interestingly, this relation was stronger for the older patients. Bennett-Levy (1984) reported that permanent memory impairment was found only in patients whose PTA exceeded three weeks, but not in other patients, although they were considered to be severely injured (see also Schacter & Crovitz, 1977).

Memory impairment is one of the most significant residual deficits following TBI (Levin, 1989a) and is among the most frequent complaints heard from patients and their relatives (Arcia & Gualtieri, 1993; Oddy, Coughlan, Tyerman, & Jenkins, 1985). It has also been found to be slower to recover than other cognitive functions (Lezak, 1979). Some improvement is observed from six months to one year (Kersel, Marsh, Havill, & Sleigh, 2001) and between six months and two years (Lannoo, Colardyns, Jannes, & De Soete, 2001). Nevertheless, deficient learning and memory were detected in patients with severe TBI even ten years post-injury (Zec et al., 2001). Furthermore, Brooks, McKinlay, Symington, Beattie, and Campsie (1987) reported that memory (and reduced speed of processing) are highly related to unemployment, seven years post-injury. Possibly as a result of all the above observations, memory impairment is the most widely investigated cognitive domain in patients who have sustained TBI (Goldstein & Levin, 1995a).

The memory literature clearly demonstrates that memory is not a unitary system, but consists of different systems composed of different cognitive sub-processes (Squire &

Zola-Morgan, 1991). Accordingly, in this article I review studies that have investigated the long-term effect of moderate to severe TBI on the different memory aspects.

Over the last two decades it has become clearer that memory can be assessed explicitly and implicitly. In explicit memory tasks, such as recall and recognition, the person is explicitly asked to retrieve particular information. The facilitatory (or inhibitory) effect of performance due to previous exposure to the particular information is viewed as an implicit memory measure (Schacter, 1987). The memory studies with patients following moderate to severe TBI are reported in the next three sections: explicit, implicit, and context-source memory. Each of these sections is further divided into subcategories reflecting specific memory domains.

Explicit Memory

Working Memory. According to the model of working memory proposed by Baddeley and Hitch (1974), working memory is composed of three components: the central executive plus two subsidiary slave systems, the visuo-spatial sketchpad and the phonological loop. The former manipulates visual and spatial material, and the latter manipulates auditory or language-based material. This model has been modified recently (Baddeley, 2000) with the addition of a fourth component, "episodic buffer." To the best of my knowledge, there are not as yet any studies with patients after TBI that have utilized the new model.

Several studies have tested the working memory of individuals post-TBI using digit span from the Wechsler Adults Intelligence Scale–Revised (WAIS-R; Wechsler, 1981), which is composed of two sub-tests, digits forward and digits backward. Haut, Petros, Frank, and Lamberty (1990b) suggested that the former reflects memory span and the latter requires manipulation of information in short-term memory. Using Baddeley and Hitch's (1974) model, Isaacs and Vargh-Khdem (1989) suggested that digits forward taps the phonological loop and digits backward taps the central executive. Brooks (1975) reported that short-term memory, as measured by digits forward and backward, is preserved following TBI. However, later studies reported that while digit span forward is intact, digit span backward is impaired in patients after TBI (Brooks, 1976; Haut et al., 1990b). Levin, Grossman, and Kelly (1976) found that short-term visual memory was impaired in patients with TBI. McDowell, Whyte, and D'Esposito (1997) and Stablum, Leonardi, Mazzoldi, Umilta, and Morra (1994) interpreted the difficulty of patients with TBI in a dual-task paradigm to reflect dysfunction of the central executive system. Azouvi, Jokic, Van der Linden, Marlier and Russel (1996) found that patients with severe TBI were impaired in the random generation task, tapping the central executive, even when speed of processing was controlled statistically. Haut et al. (1990b) used the Sternberg's paradigm in order to test the speed and accuracy of short-term memory scanning. In this task a set of two, four, or six digits appeared on the computer screen, and then participants are asked whether a particular number was a member of the set just presented. It was found that the overall response time was longer for the patients after TBI than for the controls. This result indicates that the patients need more time to scan short-term memory than controls. In addition it was found that scanning time increased disproportionately as the memory load increased (i.e., set size, 2, 4, & 6). Christodoulou et al. (2001) found in an fMRI study that performance of patients with TBI was impaired as compared with that of controls, on a working memory task (a modified version of the paced auditory serial addition task—PASAT). In general, the same brain regions were activated in patients and healthy participants (i.e., frontal, temporal, and parietal lobes) during the performance of a working memory task. However, activation in patients with TBI was more bilateral. The

right hemisphere in particular was more activated relative to controls. In addition, in areas normally activated by healthy individuals, the patient group demonstrated more dispersed activation. The authors explained these changes of brain activity in patients with TBI as reflecting a need to recruit additional cerebral resources to cope with the demands of the task. Curtiss, Vanderploeg, Spencer, and Salazar (2001) used trial 1 of list A and list B of the California Verbal Learning Test - CVLT (Delis, Kramer, Kaplan, & Ober, 1987), and digit span forward from the WMS-R (Wechsler, 1987) to calculate a working memory span index. The residual of digit span forward minus backward was used as a central executive index. The authors report that these two indexes are not sensitive to TBI. Recency effect (i.e., recall of words from the end of the list), assumed to reflect working memory capacity, was found preserved in patients with TBI (Brooks, 1975).

The impression from the studies reviewed in this section is that tasks that require manipulation of stimuli, and as such probably tap the central executive component of working memory (e.g., digit backward, PASAT, & Sternberg's paradigm), are more sensitive to the effects of TBI than tasks that probably tap the phonological loop (e.g., digit forward & recency effect). This impression needs to be tested empirically.

Immediate Memory. Immediate memory does not represent a pure memory process because it is usually tested with material exceeding working memory span such as the first trial of Rey Auditory Verbal Learning Test – AVLT (Lezak, 1995; Rey, 1964; Vakil & Blachstein, 1997), or the immediate test of the Logical Memory subtest from the WMS-R. These memory tests involve long as well as short term memory processes, as demonstrated by the serial position effect (Glanzer & Cunitz, 1966). Nevertheless, since patients with TBI are frequently tested with such memory tests, verbal and visual immediate memory tests are reported in the following section. (*Note: The focus in this section is on immediate memory test following a single presentation of the material. Nevertheless, where immediate memory is only one component of the study, other memory aspects such as multitrial learning or delayed memory tests are reported as well*).

Verbal. Baddeley et al. (1987) found that individuals suffering from TBI performed more poorly than controls on paired-associate, cued-recall, recall, and recognition tasks. Zec et al. (2001) tested patients with very severe TBI at an average of ten years post-injury, on a variety of memory tests (i.e., WMS-R, Rey AVLT, & the Selective Reminding Test). These patients were consistently impaired on all memory tests, even compared to the spinal cord injury control group. For example, the Index Scores (the norm is, $M = 100$, $SD = 15$) for patients with TBI on the WMS-R were: Verbal Memory — 77.9; Visual Memory — 82.6; General Memory — 76.9; Delayed Recall — 74.3. Patients following TBI had impaired memory, particularly on the Logical Memory and Paired-associate Learning subtests of the WMS (Brooks, 1976). Bennett-Levy (1984) also reported that the immediate and delayed recall in Logical Memory were significantly impaired in patients following TBI. Haut, Petros, and Frank, (1990a) and Haut, Petros, and Frank (1991a) reported that although they had a steeper forgetting rate than controls (recall declined as a function of the severity of injury), patients with TBI were as sensitive as controls to the semantic meaning of the units of information in the Logical Memory subtest of the WMS-R. Using a similar procedure, Vakil, Arbell, Gozlan, Hoofien, and Blachstein (1992) also reported a steeper forgetting rate for patients with TBI compared to that of controls. However, inconsistent with the results by Haut et al. (1990a; 1991a), patients with TBI have difficulty selectively retrieving the more important elements of the story after a long delay. A possible reason for the discrepancy between these studies is that the time delay used by

Vakil et al. was much longer than that used by Haut et al. (1991a), Vakil et al. tested their participants three times: immediately, after 40 minutes and after a one-day delay, whereas Haut et al. (1991a) tested only twice, immediately and delay (as part of the standard administration of the WMS-R which is about 30-40 minutes). Supporting this interpretation, Haut et al. (1990a) indicate that patients "were unable to differentiate medium- from low-importance units after a delay, even though initial recall showed differentiation at each level, as controls did at both immediate and delayed recall" (p. 285). Curtiss et al. (2001) used the CVLT and the WMS-R Digit Span subtest to generate seven theoretically driven learning and memory indexes: working memory span, central executive functions, long-term memory encoding, consolidation, retention, retrieval, and control abilities. Using the cluster analysis procedure, researchers identified different subgroups of patients with TBI, characterized by particular deficits in consolidation, retention, and retrieval processes. None of the groups showed a specific deficit in the functioning of any component of working memory. Kersel et al. (2001) tested a group of patients with severe TBI six months and one-year post injury on an auditory verbal learning test. Over 50% of the patients were impaired on all trials of the test at both six months and one-year post injury. Interestingly, improvement was observed from the first to the second testing session on most trials of the test, with the exception of the retention and delayed recall trials, which did not improve from six months to one-year post injury.

Visual. Although overall TBI affects both hemispheres equally (Mapou, 1992), the impression received while surveying the literature is that verbal memory was evaluated more thoroughly than visual memory. A possible reason for this situation is the relevance of verbal learning and memory to education and academic performance. Another possible reason is that verbal learning and memory tests are more available and more frequently used in neuropsychological batteries. Nevertheless, visual memory was assessed in patients following TBI in several studies. Brooks (1974a, 1976) tested patients with TBI with several visual memory tests (i.e., geometric design, Rey complex figure, Kimura's figures) and found them all to be sensitive to brain injury. The Recognition test of Kimura's figures was also reported by Brooker and George (1984) to be impaired in patients with severe TBI. Hannay, Levin, and Grossman (1979) used eight categories of line drawings (e.g., flowers and birds) and also found them sensitive to severe and moderate TBI. Patients following severe TBI had difficulties in associating words, either as names or possessions, with unfamiliar faces (Milders, 1998). The visual memory index derived from the WMS-R was reported by Reid and Kelly (1993) and Zec et al. (2001) to be impaired in patients with TBI as compared to controls. Shum, Harris, and O'Gorman (2000) performed an interesting experiment with TBI, involving a number of memory measures: (1) The Shum Visual Learning Test composed of complex Chinese characters; (2) The Rey AVLT verbal memory test; and (3) Spatial memory using an electronic maze. The advantage of the Shum Visual Learning Test is that it enables the testing of immediate and delayed recognition of visual patterns, while measuring learning effect and interference. Based on the results obtained from the Shum Visual Learning Test, patients were found to be impaired as compared to controls on the learning rate and the overall number of visual patterns correctly recognized. However, the groups did not differ on their vulnerability to retroactive interference or on delayed retention. Patients' spatial memory, as measured with the electronic maze, was not reliably different from that of controls. Patients following TBI (8 out of 12) demonstrated poor spatial memory when tested with a computer generated "virtual arena maze" (Skelton, Bukach, Laurance, Thomas, & Jacobs, 2000). Interestingly, performance on this task was significantly associated with

difficulties in day to day "way finding" reported by the patients. In sum, quite consistently, verbal and visual immediate memory are both sensitive to moderate to severe TBI. More research is required in order draw more conclusive findings about immediate visual memory.

Learning Rate. Repeated trials of information presentation enable the assessment of learning rate. The tests frequently used to assess the learning rate of verbal material in patients with TBI are the Rey AVLT (Lezak, 1995; Rey, 1964; Vakil & Blachstein, 1997), CVLT (Delis et al., 1987) and the Selective Reminding Test (Buschke & Fuld, 1974). Learning rate as measured by the Rey AVLT was significantly slower compared to that of controls (Blachstein, Vakil, & Hoofien, 1993; Geffen, Butterworth, Forrester, & Geffen, 1994; Zec et al., 2001). Shum et al. (2000), using the same test, did not find a reliable difference between the learning rate of patients with TBI and that of normal controls. However, the overall number of words recalled in the first five trials of the task was significantly higher for the control group than for the patient group. There is no obvious explanation (e.g., sample size or patient selection) for the inconsistency between Shum et al.s' results and the results reported above. Reasons that may contribute to inconsistent findings with survivors of TBI are examined in the Discussion section.

The learning rate for patients with TBI was also found to be slower than that of controls when measured with the CVLT (Haut & Shutty, 1992; Novack, Kofoed, & Crosson, 1995, but see Vanderploeg, Crowell, & Curtiss, 2001). Gardner and Vrbancic (1998), using a regression model with the CVLT scores, found that the General Verbal Learning factor (consists of total trials 1–5) was the factor that could differentiate best between individuals suffering from moderate/severe TBI and normal controls. A steeper learning rate for controls compared to that of patients after TBI was detected with the Selective Reminding Test (Levin, Grossman, Rose, & Teasdale, 1979; Zec et al., 2001). Patients with TBI learned fewer words and at a slower rate than the non-injured when words were presented either verbally or visually, or when presented in both modalities simultaneously (Constantinidou & Neils, 1995; Constantinidou, Neils, Bouman, & Lee, 1996). DeLuca, Schultheis, Madigan, Christodoulou, and Averill (2000) compared controls and patients with TBI on initial acquisition on a verbal list-learning task. Patients with TBI required more learning trials than did the controls in order to reach the same criterion. Those patients who were able to meet the learning criterion did not differ from controls on delayed recall and recognition tested at 30- and 90-minutes. Based on these results, the authors concluded that memory impairment following TBI is "primarily attributable to deficiencies in the initial acquisition of verbal information rather than in retrieval failure" (p. 1331). Among the few studies that tested the learning rate of visual patterns using the Shum Visual Learning Test, patients' learning rate was slower than that of control individuals (Shum et al., 2000).

Levin et al. (1979) analyzed the nature of the deficient learning rate seen in patients with TBI. Applying the Selective Reminding Test, they found inconsistent word recall that is mostly turnover of words. Analyzing the same task, Paniak, Shore, and Rourke (1989) reported that recall by patients after TBI was sporadic and disorganized. Similarly, using the Rey AVLT, Blachstein et al. (1993) found that compared to patients with TBI, the control group's steeper learning curve was a product of both a significantly greater increment of words in each trial and a significantly smaller number of words omitted from trial to trial. This learning pattern of the patients leads to a higher rate of turnover of words. The researchers in these two studies interpreted the results as reflecting an inefficient organization and learning strategy.

Cluster analysis procedures with CVLT scores were used in several studies in order to identify subtypes of patients after TBI that are characterized by different learning and memory patterns. Haut and Shutty (1992) identified three subgroups of these patients: non-impaired, mildly impaired, and significantly impaired learning rate. Millis and Ricker (1994) claim that the subgroups detected by Haut and Shutty differ quantitatively from each other, but not qualitatively in terms of a distinctive pattern of learning and memory. Using five CVLT variables based on the learning pattern (identified in a previous factor analysis study), Millis and Ricker detected five subgroups of patients with TBI (only four were interpretable): active, passive, disorganized, and deficient. In a later study with a different group of patients with TBI, Deshpande et al. (1996) essentially replicated the previous findings of Millis and Ricker (see Curtiss et al., 2001, for discussion of the similarities and differences between the subgroups identified by them and by Millis and Ricker). In an attempt to rectify some of the sampling and methodological difficulties in the previous clustering studies, Demery, Pedraza, and Hanlon (2002) evaluated a large patient sample (n = 160) including mild, moderate, and severe TBI. Two subgroups with distinct learning profiles were reliably detected in this study, one within the normal range and the other with moderate-to-severe impairment. Learning rate of patients with TBI did not differ from that of the control participants when measured with cued recall rather than free recall (Vakil & Oded, 2003). It seems that the cued recall procedure has facilitated memory retrieval, and as a result the learning rate has improved. In conclusion, learning rate following TBI, when measured by free recall, is frequently impaired. Inefficient organization and learning strategy is probably the major reason for this deficit.

Semantic Organization. Findings are inconsistent regarding the question whether patients with TBI demonstrated an ability to utilize semantic knowledge to assist their memory and learning. On the one hand, some studies reported reduced ability to benefit from semantic knowledge. For example, these patients demonstrated reduced ability to spontaneously utilize semantic knowledge during word-list memory tests and when requested to answer questions regarding general knowledge (Perri, Carlesimo, Loasses, & Caltagirone, 2000). Goldstein, Levin, Boake, and Lohrey (1990) have shown that semantic (categorical) encoding of words yields better recognition and cued recall than physical (letters) or acoustic (rhyme) encoding in survivors of TBI, but to a lesser degree than in controls. Stallings, Boake, and Sherer (1995) showed on the CVLT that patients with TBI exhibited reduced semantic clustering, indicating a qualitative deficit underlying the quantitative deficits in learning and memory. Vakil et al. (1992) using the Logical Memory subtest of the WMS, reported that patients following TBI did not show differential delayed recall according to the relative importance of the information in the story (i.e., better retention of the more important information units) (but see Haut et al., 1990a, 1991a). It is interesting to note that these patients did not differ from controls when judging the relative importance of the units of information of the story. This dissociation led the researchers to conclude that the difficulty of patients with TBI is not with semantic knowledge per se, but rather with utilization of this knowledge in the memory process. Forgetting rate between 30 seconds to 60 minutes for patients with TBI was equivalent to that of controls, when free recall of unrelated words was used. However, when free recall of related words was tested, controls' retention of the material was significantly better than with unrelated words. In contrast, patients with TBI did not take advantage of the semantic organization of the material and showed the same rate of forgetting as with the unrelated words (Carlesimo, Sabbadini, Loasses, & Caltagirone, 1997).

Other studies, however, were able to show that under certain circumstances, patients after TBI do take advantage of their semantic knowledge. For instance, just like controls, patients' performance improved when a list of related words was presented in a clustered order (Levin & Goldstein, 1986). However, closer analysis of the results indicated that the patients tended to use clustering and subjective organization spontaneously to improve their recall to a lesser degree than the controls. Consistent with this finding, Goldstein et al. (1990) reported that patients benefited from semantic encoding, but to a lesser degree than controls. Goldstein, Levin, and Boake (1989) found that patients demonstrated normal release from proactive interference upon shifting semantic category, thus reflecting sensitivity to semantic categorization.

Several researchers have offered to reconcile the seemingly contradictory findings with regard to the utilization of semantic knowledge by patients with TBI in the process of learning and memory. It is suggested that these patients have difficulties in applying an active or effortful learning strategy, so that when a task is demanding in terms of the processing required, performance by patients with TBI becomes defective. However, when a passive strategy or automatic processes are sufficient, then their performance is within the normal range (Levin, 1989a; Levin & Goldstein, 1986; Perri et al., 2000; Vakil et al., 1992).

Forgetting Rate. Delayed Recall Index generated from the WMS-R was significantly impaired (Index score = 74.3) in a sample of patients suffering from very severe TBI even ten years post-injury (Zec et al., 2001). Wilson (1992) reported that 42% of 26 patients with TBI tested five to ten years post-injury scored below 50 on the Delayed Recall Index of the WMS-R. Reid and Kelly (1993) found that patients with TBI were impaired on all five indices of the WMS-R, but impairment was most pronounced in the delayed memory index. A disproportionate forgetting rate of a word list studied repeatedly 20 minutes earlier was reported for patients with TBI when using the CVLT (Crosson, Novack, Trenerry, & Craig, 1988; Haut & Shutty, 1992; Vanderploeg et al., 2001), and similarly when using the Rey AVLT (Blachstein et al., 1993; Geffen et al., 1994; Zec et al., 2001). Haut et al. (1990a, 1991a) and Vakil et al. (1992) reported that patients with TBI showed an accelerated forgetting rate of a short story as compared to controls (i.e., Logical Memory subtest of the WMS). Unlike controls, patients' retention of information over time was not related to its relative importance to the story (Vakil et al., 1992). Carlesimo et al. (1997) compared the forgetting rate (between 30 seconds and 60 minutes) of patients with TBI and controls using four memory procedures: free recall of unrelated stimuli, free recall of related stimuli, cued recall, and recognition. The most pronounced difference between the groups in this study was found with free recall of related words. By contrast with the controls, patients did not take advantage of the semantic organization of the material and showed the same rate of forgetting as with the unrelated words. When external cues are provided (i.e., cued recall and recognition), the groups' retention over time did not differ significantly.

When the groups' initial acquisition (i.e., baseline) differs, the comparison of their forgetting rate should be interpreted cautiously. In such a case, a different rate of forgetting may not necessarily reflect a consolidation deficit, but rather a deficit in encoding evident in the impaired acquisition. In an attempt to address this difficulty, the groups' initial acquisition was equated either by longer exposure to stimuli or by adding learning trials. DeLuca et al. (2000) demonstrated that when patients were equated with controls on the initial acquisition, their forgetting rate did not differ either on delayed recall or on recognition. These findings suggest that the impaired forgetting rate reported in other studies may

reflect poor acquisition rather than poor retention of information. A different approach was taken by Hart (1994) in order to address the problem of differentiating between a consolidation and encoding deficit when groups differ on baseline performance. If the patient group shows a faster forgetting rate for all stimuli when testing different stimuli, Hart suggested that this could be interpreted as reflecting a consolidation problem. However, if patients show an accelerated forgetting rate on only some of the stimuli, it would be interpreted as reflecting an encoding deficit of the particular stimuli. Accordingly, Hart tested the rate of forgetting of words, pictures, and designs with patients suffering from moderate to severe TBI. Results showed that patients had a faster forgetting rate for pictures, but not for words or designs. According to the rationale presented above, consistent with DeLuca et al. (2000), Hart interpreted the results as indicating an encoding rather than a consolidation deficit in patients after TBI. Unlike controls, patients failed to apply a strategy of dual coding to the pictures presented. Such a strategy was less applicable to words and designs, and thus patients did not differ from controls.

Vakil and Oded (2003) compared the forgetting rate of controls and patients with TBI on three memory tests: cued recall, priming, and saving. Saving is defined as the advantage of relearning of a list of word pairs over the original learning of the same list. It was found that the overall number of pairs of words recalled immediately and retained over time was impaired in patients with TBI as compared with controls. However, patients with TBI demonstrated significant saving in relearning old, as compared to new, pairs of words even after two weeks' delay. The authors attribute these results to the fact that in the saving procedure, just as in other implicit tasks, participants are not requested explicitly to recall previously learned material at the relearning stage, thereby enabling normal performance by the patient group.

Thus, although most studies using free recall demonstrated accelerated forgetting rate for patients with TBI, it does not necessarily indicate a retention deficit, if baseline performance of the patient group was not equated to that of the control group. For further discussion of the question whether the memory impairment following TBI reflects an encoding, consolidation or retrieval deficit, see Vanderploeg et al. (2001).

Sensitivity to Interference (Proactive and Retroactive). Proactive interference refers to the interference of earlier information with the acquisition or retention of additional new information (Underwood, 1957). Retroactive interference, however, occurs when earlier learning interferes with later learning. The CVLT and the Rey AVLT enable measurement of proactive interference (i.e., trial 1 of list A vs. list B) and retroactive interference (i.e., comparison of the trials of list A before and after the interference of list B). Shum et al. (2000) found that patients were more vulnerable than controls to retroactive interference when verbal memory was tested with the Rey AVLT, but not when visual memory was tested with the Shum Visual Learning Test. Crosson et al. (1988) found that, unlike controls, patients following TBI were not sensitive to proactive interference when measured with the CVLT.

A sensitive paradigm for assessment of proactive interference is the presentation of new words from the same category, which results in decreased recall capacity (i.e., buildup of proactive interference). Upon shifting the category of words in the next trial, recall capacity increases (i.e., release from proactive interference) (Wickens et al., 1963). Using this procedure, Goldstein et al. (1989) showed that patients with TBI are sensitive to category shift, thus indicating preserved conceptual encoding in these patients. As can be seen, very few studies tested the effect of TBI on proactive and retroactive interference. The paradigms used differ considerably, so it is questionable whether they tap the same

cognitive processes. More systematic research is required in order to be more conclusive about the nature of the effect of TBI on proactive and retroactive interference.

Retrograde Amnesia - Autobiographical Memory. Retrograde amnesia is defined as the loss of memory of events experienced prior to the time of onset of the brain damage. The precise measurement of retrograde amnesia is problematic, primarily because it is difficult to accurately quantify the information acquired prior to the injury by post-onset testing (for further discussion of these difficulties, see Richardson, 2000). Carlesimo et al. (1998) reported a high prevalence of retrograde amnesia among patients following TBI. The impairment involves both autobiographical memory and memory for public events extending back to early acquired knowledge. In two case reports (Markowitsch et al., 1993; Mattioli, Grassi, Perani, & Cappa, 1996), although severe retrograde amnesia was observed following TBI, only mild anterograde amnesia was found. In the former case report, MRI showed damage to both temporal poles and the lateral portion of the right prefrontal cortex. In the latter case using PET scan, a significant bilateral reduction of metabolism in the hippocampus and anterior cingulate cortex was observed. Thus, despite the dearth of information concerning retrograde amnesia in patients with TBI, the findings seem to indicate consistently that such impairment is frequently observed in this patient group.

Prospective Memory. Groot, Wilson, Evans, and Watson (2002) describe prospective memory as follows: "Prospective memory involves remembering to perform previously planned actions at the right time or within the right time interval or after a certain event takes place while being involved in other activity" (p. 645). Groot et al. assessed the prospective memory of 36 patients with brain injury (of whom only 22 were following TBI) and 28 control participants. Participants' prospective memory was evaluated using the Cambridge Behavior Prospective Memory Test, which includes 4 time-based and 4 event-based tasks. The results showed that patient group performance on the prospective memory tasks was significantly poorer than that of the control group. Interestingly, it was also found that test scores of prospective memory were significantly associated with retrospective memory and executive function test scores. Kinsella et al. (1996) reported that patients who sustained severe TBI had poorer prospective memory compared to controls. It is noteworthy that prospective memory, more than standard memory tests, was associated with memory functioning as evaluated by the patients. Consistent with the above findings, following severe TBI patients were impaired on time-, event-, and activity based prospective memory tests (Shum, Valentine, & Cutmore, 1999). Hence, difficulty with prospective memory seems to be one of the common consequences of TBI.

Implicit Memory

Implicit tests of memory (Schacter, 1987) are frequently divided into two major subtypes: priming or item-specific, and skill or procedural learning (Moscovitch, Goshen-Gottstein, & Vierzen, 1994; Squire & Zola-Morgan, 1991). Moscovitch et al. define these two subtypes as follows: "Memory for the item typically is inferred from changes in the efficiency or accuracy with which the item is processed when it is repeated… Procedural tests, on the other hand, are not concerned with acquisition of a particular item, but rather with learning a general cognitive or sensorimotor skill… Here, too, memory is inferred from changes in performance with practice" (p. 621). In the next two sections, studies investigating priming effect and skill-learning abilities in patients with TBI are reviewed.

Priming. Priming has been divided into perceptual (e.g., word fragment completion task) and conceptual priming (e.g., category production task) (Blaxton, 1992). This dissociation is supported by studies manipulating level of processing. While deep encoding leads to stronger conceptual priming as compared to shallow encoding, perceptual priming is not affected by this manipulation (Srinivas & Roediger, 1990). The divided attention manipulation provides further support for this dissociation. Conceptual priming effect was reduced or eliminated under divided attention condition as compared to full attention condition, but perceptual priming was the same under both attentional conditions (Mulligan, 1998). Shum, Sweeper, and Murray (1996) tested patients with two explicit memory tasks and two implicit memory tasks under shallow and deep encoding. Regardless of level of encoding, when compared to controls patients were found to be impaired on the explicit memory tasks. However, implicit memory was impaired only under deep encoding. Vakil and Sigal (1997) tested the same group of patients with TBI on perceptual priming (i.e., partial-word identification) and conceptual priming (i.e., category production) tasks in addition to a declarative memory task (i.e., free recall). The groups did not differ significantly from each other on the perceptual priming task, but patients performed more poorly than controls on the conceptual priming task and on the declarative task. Other studies with patients following TBI partially support this dissociation between perceptual and conceptual priming. These patients were tested in several studies with the word stem completion task, a perceptual priming task similar to the word fragment completion task. Vakil, Biederman, Liran, Groswasser, and Aberbuch (1994) reported that patients with TBI demonstrated priming ability similar to that of normal controls when tested with a word stem completion task. Consistent with these two studies, Watt, Shores, and Kinoshita (1999) reported preserved priming in patients with TBI when measured with the word stem completion task under full attention condition. However, unlike controls, patients under divided attention condition showed a decreased priming effect. The authors interpreted their results to indicate that, even though the word stem completion task is an implicit memory task, it requires attentional resources that are impaired following TBI. Similarly, Schmitter-Edgecombe (1996) reported intact performance of patients with TBI on a perceptual priming task (i.e., tachistoscopic identification). Divided attention affected the patients' performance disproportionately due to factors other than memory. Contrary to the above reports, Markowitsch and Haerting (1996) found that perceptual priming, tested with a picture fragment completion task, was impaired in patients afflicted with TBI as compared to controls. Perri et al. (2000) reported that these patients showed normal lexical facilitation on a semantic priming task. This result does not necessarily contradict the report on impaired conceptual priming by Vakil and Sigal (1997), as the authors concluded that semantic priming is intact in patients with TBI because it requires automatic access to semantic memory. It is reasonable to claim that category production is a task more demanding than semantic priming on lexical decision, and therefore leads to impaired performance by these patients. Vakil and Oded (2003) found that implicit memory (i.e., word stem completion) is preserved in patients following TBI only when based on reactivation of preexisting knowledge, but not when dependent on forming new associations.

Skill Learning. It is well documented that TBI has minimal effect on well-practiced skills acquired prior to the injury (Schmitter-Edgecombe & Nissley, 2000; Vakil et al., 1994). The question is whether these patients are capable of acquiring new skills following TBI.

The serial reaction time (SRT) task (Nissen & Bullemer, 1987) is a widely used skill-learning task that could generate indirect and direct measures of sequence learning. In this

task participants are presented with asterisks appearing in a repeated spatial sequence. They are asked to respond as fast as possible on a key whose location corresponds to the position of the appearance of the asterisk. The increase in reaction time from a repeated sequence block to a random sequence block is used as an indirect measure of sequence learning. A direct measure of sequence learning is the accuracy with which participants predict the spatial sequence of asterisks. Mutter, Howard, and Howard (1994) reported that patients suffering from severe, but not mild, TBI were impaired on the indirect measure of sequence learning, but demonstrated normal performance on the direct measure of sequence learning. McDowall and Martin (1996) tested patients with TBI with the SRT task and reached contradictory results: Patients with severe TBI performed normally on the implicit sequence learning measure of the SRT task (explicit memory for sequence was not tested). In an attempt to address some of the methodological difficulties with the previous two studies, Vakil, Kraus, Bor, and Groswasser (2002) tested patients who sustained TBI with explicit and implicit measures of sequence learning using the SRT task. The findings suggest that the patient group shows a unique pattern of results—impairment on both the explicit and implicit measures of sequence learning. This pattern of results does not correspond to either that of patients with amnesia or to that of patients with damage to the basal ganglia (e.g., Parkinson's disease). Although patients suffering from amnesia showed impaired sequence learning when measured explicitly, their implicit sequence learning is preserved (Nissen & Bullemer, 1987). However, patients with Parkinson's disease show the opposite pattern (Ferraro, Balota, & Connor, 1993). The Tower of Hanoi Puzzle (TOHP) is also one of the tasks used frequently to measure skill learning (Cohen, Eichenbaum, Deacedo, & Corkin, 1985). Using this task Vakil, Gordon, Birnstok, Aberbuch, and Groswasser (2001) showed that the control group performed the task faster than the group of patients with TBI, and the learning rate of the control group, as measured by number of moves, was steeper than that of the patient group.

In a series of studies using search-detection tasks, Schmitter-Edgecombe and colleagues have demonstrated that patients with TBI were able to develop automatic/skillful processes, although at a slower rate (Schmitter-Edgecombe & Beglinger, 2001; Schmitter-Edgecombe & Rogers, 1997). In a study using a semantic-category visual search task, patients with TBI were able to develop skilled performance not significantly different from that of normal controls. Likewise, these patients showed a normal learning rate on a perceptually based implicit task (Nissley & Schmitter-Edgecombe, 2002).

These contradictory findings regarding the effect of TBI on skill learning might possibly be reconciled by distinguishing between tasks known to be sensitive to the functioning of the frontal lobes (i.e., TOHP and SRT), and perceptual tasks (i.e., search-detection task) that are not mediated by the frontal lobes. Due to the vulnerability of the frontal lobes in TBI, the former tasks are impaired, but not the latter. This hypothesis needs to be tested empirically.

Context and Source Memory

Following the review of explicit and implicit tests of memory, studies investigating context and source memory in patients with TBI are surveyed at this stage, since contextual memory can be tested explicitly and implicitly. The distinction between target and contextual information is determined by differential attention allocated to various components of the environment. This is consistent with Mayes, MacDonald, Donlan, Pears, and Meudel's (1992) definition of context as "information that falls on the periphery of attention" (p. 268). A similar distinction was introduced by Schacter, Harbluk, and McLachlan (1984)

between item (or fact) and source memory. Source memory refers to the background information of an item or event, such as its temporal order, spatial location, or modality of presentation. Memory for contextual information could be assessed explicitly and implicitly. For example, asking participants about the modality, font, or voice in which a word was originally presented would be considered as explicit measures of context, which is viewed as the equivalent of source memory. The facilitation due to correspondence of context (e.g., modality or font) of the words in learning and test would be considered as an implicit measure of context or context effect (see Vakil, Openheim, Falck, Aberbuch, & Groswasser, 1997).

Several studies reported that memory for contextual information in patients with TBI was impaired when measured directly (i.e., source memory). When asked to judge the spatial location of words presented at the study phase, the performance of these patients was impaired as compared to that of controls following intentional and incidental learning (Vakil & Tweedy, 1994). Similarly, patients with TBI were impaired in judging the frequency of occurrence of words from the study word list (Levin, Goldstein, High, & Williams, 1988; Tweedy & Vakil, 1988). Dywan, Segalowitz, Henderson, and Jacoby (1993) reported that source memory was impaired in these patients when a fame judgment task was used. The results with regard to temporal order judgment are not as consistent. Some studies reported that patients following TBI were impaired in temporal order judgment of a word list (Vakil, Blachstein, & Hoofien, 1991; Vakil & Tweedy, 1994) or actions (Cooke & Kausler, 1995) whether learned intentionally or incidentally. However, other studies reported that patients with TBI were not significantly different from controls in temporal order judgment. Vakil, Sherf, Hoffman, and Stern (1998) reported that following eight repetitions of a word list in the same order, no group difference was found in temporal order judgment of these words. The authors attributed these results to the numerous repetitions of the list that may have enabled the patient group to compensate for the difficulty detected in a previous study with only five repetitions of the list (Vakil et al., 1991). Inconsistent with Cooke and Kausler, who found impaired temporal order for activities, Schmitter-Edgecombe and Wright (2003) reported intact temporal order for performed activities in patients with TBI. The authors point to three differences when comparing their own study to that of Cooke and Kausler: Participants in their study spent more time completing each task, the context in which each task was encoded was more distinct, and finally, participants completed less activities (8 as compared to 12 and 24). Each one or a combination of these differences made the temporal order judgment easier and as such, less sensitive to TBI.

In a series of studies, Vakil and colleagues have tested context memory in patients with TBI using explicit and implicit measures of memory following the same learning episode. In accordance with the previously reported studies, patients affected by TBI were quite consistently shown to be impaired relative to controls on all the explicit memory tests of target information (e.g., word recall and recognition) and of context information (e.g., modality and temporal order judgment). However, when contextual information was tested implicitly, the patient and control groups did not differ significantly, that is, the groups showed the same magnitude of context effect (cf. Vakil et al., 1991; re: temporal order judgment; cf. Vakil et al., 1994, re: frequency judgment; cf. Vakil, Golan, Grunbaum, Groswasser, & Aberbuch, 1996, re: perceptual context; cf. Vakil et al., 1997, re: modality of presentation).

Intrusion errors and false alarms reflect difficulty in attributing a particular word to its original list. As such, they could be viewed as source memory errors in recall and recognition, respectively. Hannay et al. (1979) and Crosson et al. (1988) reported a high rate of

false alarms in a recognition task. Findings with regard to intrusion errors are inconsistent. A number of studies have reported a high intrusion rate of extra list words in patients with TBI (Crosson et al., 1988; Levin & Goldstein, 1986; Levin et al., 1979; Novack et al., 1995). However, Brooks (1975) noticed that these patients tend to have fewer intrusion errors. Brooks interpreted these results as an indication of poor long-term memory caused by deficient encoding. Using signal detection analysis, Brooks (1974b) found that as compared to controls, patients following TBI showed lower memory capacity and more conservative response bias (i.e., unwillingness to guess). Further systematic studies are needed in order to reconcile conflicting findings on the effect of TBI on response bias as expressed by false alarm rate and intrusion errors.

Speed of Processing and Attention

Following diffuse brain injury, it would not be unexpected to find that in addition to memory, several cognitive domains (e.g., attention and speed of processing) are affected. Some researchers have suggested that these cognitive deficits are the source of the memory impairment observed in patients who have sustained TBI.

Speed of processing was shown to be reduced in patients following TBI in a large range of cognitive tasks. Fisher, Ledbetter, Cohen, Marmor, and Tulsky (2000) and Axelrod, Fichtenberg, Liethen, Czarnota, and Stucky (2001) found that the speed of processing derived from the WAIS-III is very sensitive to TBI. Martin, Donders, and Thompson (2000) found that the only measure that was sensitive to the severity of injury was the Processing Speed score from the WAIS III, 100.90 and 88.10, for patients with mild and moderate to severe TBI, respectively. Madigan, DeLuca, Diamond, Tramontano, and Averill (2000) showed that speed of processing for patients with TBI was slower than that of healthy controls, even when accuracy of performance was equated across the groups. Based on meta-analysis, Ferraro (1996) concluded that patients with TBI are significantly slower than controls on cognitive tasks involving simple- and choice-reaction time. Semantic organization was not impaired in patients following TBI, but their access to semantic information was slower compared to that of controls (Haut, Petros, Frank, & Haut, 1991b). While attempting to attribute the memory deficit in these patients to speed of processing, some studies have demonstrated that when controlling for speed of information processing, the disadvantage of the group with TBI compared to the control group was eliminated (Timmerman & Brouwer, 1999; Veltman, Brouwer, van Zomeren, & van Wolffelaar, 1996). However, Gronwall and Wrightson (1981) showed independence between speed of processing and verbal memory in patients with TBI. Azouvi et al. (1996) have shown that even when speed of processing is controlled statistically, supervisory strategies were impaired. Thus, the reports are quite conclusive about the effect of TBI on speed of processing. However, more research is needed in order to achieve a better understanding of the nature of the relations between speed of processing and memory difficulties in patients with TBI.

Deficits in *attention* and concentration are also reported as common symptoms following TBI (Binder, 1986; McKinlay, Brooks, Bond, Martinage, & Marshall, 1981; Oddy et al., 1985; Stuss et al., 1983; van Zomeren & van den Burg, 1985). It is well established that attention is composed of a variety of cognitive processes subserved by different neural systems (for review, see Posner & Petersen, 1990). In most studies involving patients who sustained TBI, they were found to be impaired on a range of attentional processes: *Selective attention* (Cremona-Meteyard, Clark, Wright, & Geffen, 1992; Schmitter-Edgecombe & Kibby, 1998; van Zomeren, 1981); *Divided attention* (Leclercq

et al., 2000, Park, Moscovitch, & Robertson, 1999); and *Sustained attention* (Loken, Thornton, Otto, & Long, 1995, but see Brouwer & van Wolffelaar, 1985). The hypothesis that memory impairment in patients with TBI stems from an attentional deficit was investigated in several studies. To test this hypothesis, Schmitter-Edgecombe (1996) and Watt et al. (1999) compared memory performance encoded under focused versus divided attention. Both studies found that although explicit memory was impaired in patients following TBI, divided attention did not affect patients' memory disproportionately as compared to that of controls. These results are inconsistent with the hypothesis that memory difficulties observed following TBI are generated by an attentional deficit. Mangels, Craik, Levine, Schwartz, and Stuss (2000) did find a disproportionate effect of divided attention on memory in patients with mild head injury. The more severely injured patients were divided into two subgroups based on their strategy at learning. Under divided attention, the patient subgroup that preferred to focus on the encoding task showed intact memory performance. However, the subgroup that preferred to focus on the distracting task (i.e., digit-monitoring) did show impaired memory under focused attention and disproportionate memory impairment under divided attention. The authors' conclusion is that the memory deficits observed in patients following TBI are secondary to deficits in attentional resources. These findings draw attention to the heterogeneity of patients affected by TBI, and to the fact that careful classification of patients can lead to interesting results. In conclusion, although patients who sustained TBI are consistently reported to have a variety of attention deficits as well as a variety of memory deficits, more systematic research is required with these patients in order to gain a better understanding of the interrelations between these two cognitive domains.

Discussion

This review demonstrates the extensive research conducted to investigate the effect of moderate to severe TBI on a wide range of memory processes. In an attempt to characterize the profile of memory deficit following TBI, the findings were classified into theoretically driven memory categories. Following this review, in the next sections I will try to address several questions with theoretical as well as clinical implications.

Could Memory Impairment Following TBI Be Characterized As Amnesia?

This survey demonstrates that TBI affects a large range of memory aspects. The question of resemblance between the memory impairment profile following TBI and that of patients suffering from global amnesia has theoretical and diagnostic implications. Parkin (1997) (pp. 87–88) lists five major features that are characteristic of the amnesic syndrome. In the next section memory impairment following TBI is evaluated in light of Parkin's criteria for amnesia.

1. *"No evidence of impaired short-term storage as measured by tasks such as digit span on WMS-R…"* Most studies reviewed in the Working Memory section reported impaired working memory as measured by digit span (Haut et al., 1990b) and by other measures (Christodoulou et al., 2001). Thus, this criterion is not fulfilled.
2. *"Semantic memory and other intellectual functions, as measured by tests such as WAIS-R, generally intact…"* The comparison between intelligence and memory is very important, because it determines whether or not memory is selectively impaired and whether it is not secondary to a more general cognitive deterioration (Milner, 1975;

Weiskrantz, 1985). Intelligence Quotient (IQ) is generated from the different versions of the Wechsler Adult Intelligence Scale (WAIS: Wechsler, 1955; WAIS-R: Wechsler, 1981; WAIS-III: Wechsler, 1997a). Parallel to the IQ is the Memory Quotient (MQ) generated from the different versions of the Wechsler Memory Scale (WMS: Wechsler, 1945; WMS-R: Wechsler, 1987). In the latest version, WMS-III (Wechsler, 1997b), summary scores were changed to Immediate Memory and General Memory (composed of delayed memory test scores). According to some researchers the discrepancy between IQ and MQ is the most critical criterion for amnesia. As such, a diagnosis of amnesia would be applied if the difference between intelligence (i.e., IQ) and memory (i.e., MQ) measures is 12 points (Milner, 1975) or 15 points (Weiskrantz, 1985). Levin et al. (1988), used a criteria of 15 points disparity between Verbal IQ and Verbal memory, and between Performance IQ and Visual memory. For a discussion of the difficulties in comparing the IQ and MQ scores derived from the different editions of the WAIS and WMS, see Prigatano (1978) and Richardson (2000) (pp.105–109).

Compared to estimated premorbid intelligence, TBI was shown to affect intelligence by approximately 3–4 points on the WAIS-R (Johnstone, Hexum, & Ashkanazi, 1995). However, Freeman, Godfrey, Harris, and Partridge (2001) reported that a large proportion of their TBI sample was detected as intellectually impaired. Tremont, Hoffman, Scott, Adams, and Nadolne (1997) used the Oklahoma Premorbid Intelligence Estimation (OPIE) in order to estimate the premorbid IQ of patients who sustained TBI. This test is based on demographic information (e.g., age & occupation) in addition to the Vocabulary and Picture Completion subtests from the WAIS-R. In general the patients with moderate to severe TBI showed a greater discrepancy between the estimated IQ and index scores generated from the WAIS-R and the WMS-R. For the severe to moderate group the estimated premorbid IQ score derived from the OPIE was 104.09 compared to IQ score of 92.98 and MQ score of 90.93. Thus, the estimated reduction in intelligence score is 11.11 points, and the memory reduction compared to estimated intelligence is slightly higher, 13.16 points. There is a negligible difference (2.05) between the current IQ and MQ scores. Interestingly the largest discrepancy, 19.07 points, was found between the estimated premorbid intelligence score and the delayed recall index (85.02) from the WMS-R. In a sample of patients with moderate to severe TBI studied by Levin et al. (1988), about one third had an IQ score below 85, which is one standard deviation below normal. Among the two thirds whose IQ score was in the normal range of intelligence (85 and above), about one fourth demonstrated impaired memory. This subgroup of patients with moderate to severe TBI satisfies the criteria of amnesia as defined above, as a discrepancy between IQ and MQ scores. In a series of longitudinal studies Mandleberg and colleagues (Mandleberg, 1975, 1976) observed significant improvement of intelligence scores over time, as measured by the WAIS, in patients who sustained TBI. This finding emphasizes the importance of the time after injury when testing was performed. Martin et al. (2000) compared the performance of patients with mild TBI to moderate and to severe TBI, on IQ scores generated from the WAIS III and the General Ability Measure for Adults (GAMA). Neither the GAMA IQ nor the WAIS III FSIQ were sensitive to severity of TBI. Patients with mild injury have an IQ score of 101.28 and 99.69 on the GAMA and WAIS III, respectively. Patients with moderate to severe injury have 99.94 and 93.10 on the GAMA and WAIS III, respectively.

Table 1 provides a selective list of studies that compared patients' intelligence and memory quotients. As can be seen in Table 1, the IQ-MQ gap in Corkin et al.'s (1985) sample is the largest (22 points). As noted by the authors, this sample is not

representative of the TBI population due to sampling limitations and the small number of patients (i.e., 5). The second largest IQ-MQ difference, of 14.6 points, was reported by Zec et al. (2001). Their sample represents patients with *very* severe TBI, with an average of 65.8 (*SD* = 61.8) days in coma. With the above two exceptions, findings reported in Table 1 demonstrate the IQ range in patients with TBI to be between 82.24 and 89.05 (weighted mean 88.28, about one standard deviation below normal). The memory scores range between 75.8 and 86.0 (weighted mean 85.95, about one standard deviation below normal), slightly lower than the intelligence scores (weighted mean of IQ-MQ 2.36). Thus, on the average as a group, patients suffering from TBI do not fulfill one of the fundamental criteria for pure amnesia, as intelligence is not preserved. In addition, the criterion of a gap of at least 12 points (Milner, 1975) or 15 points (Weiskrantz, 1985) between IQ and MQ is not fulfilled. This conclusion implies that unlike in amnesia, memory impairment observed in patients with TBI could be viewed, at least to some degree, as a consequence of a more general cognitive deficit. Hence, the second criterion for amnesia is not satisfied.

3. *"A severe and permanent anterograde amnesia is present…on tests of recall…"* Most studies found anterograde amnesia in patients following TBI (reviewed in the Immediate Memory, Learning Rate and Forgetting Rate sections) as measured, for instance, by a free recall test. However, the impairment in most cases is not as severe as in amnesia. This is reflected in a smaller gap between IQ and MQ as compared to the gap observed in patients with amnesia. However, the smaller gap is also attributed to the reduced IQ score as reported above. Hence, this criterion is partially fulfilled.
4. *"Retrograde amnesia will inevitably be present, but its extent can be extremely variable."* As reviewed in the Retrograde Amnesia section, this criterion is completely satisfied. For example, Carlesimo et al. (1998) reported a high prevalence of retrograde amnesia among patients with TBI.
5. *"Procedural memory, as measured by skill learning, perceptual learning and priming will also be relatively intact."* A different pattern of findings emerges from the review of the literature of implicit memory (i.e., skill learning and priming) when tested in patients with amnesia and in patients with TBI. Patients with amnesia have been shown quite consistently to have preserved skill-learning (procedural) ability for a large range of tasks (cf. Cohen & Squire, 1980; *re:* mirror reading; Cohen et al., 1985, re: TOHP; Nissen & Bullemer, 1987; re: SRT). By contrast, as reported in the Skill Learning section, patients with TBI were impaired on several skill-learning tasks (e.g., SRT task, Vakil et al., 2002 and TOHP task, Vakil et al., 2001). These patients were able to develop automatic/skillful processes on a search-detection task, although at a *slower* rate (Schmitter-Edgecombe & Beglinger, 2001; Schmitter-Edgecombe & Rogers, 1997). As proposed in the skill learning section, a possible way to reconcile these contradictory findings is to distinguish between tasks involving frontal lobe functioning (i.e., TOHP and SRT) and perceptual tasks not involving frontal lobe functioning (i.e., search-detection task). The former tasks are impaired while the latter tasks are preserved following TBI.

   Patients with amnesia were shown to have preserved priming effect, whether perceptual (e.g., partial-word identification task) or conceptual (e.g., category-production task) (Cermak, Verfaellie, & Chase, 1995). Patients with TBI showed preserved perceptual priming, and unlike patients with amnesia, were impaired compared to controls on the conceptual priming task (Vakil & Sigal, 1997). In conclusion, Parkin's fifth criterion is not fulfilled since unlike patients with amnesia, implicit memory is not consistently preserved in patients with TBI.

Table 1
Intelligence (IQ) compared to memory (MQ) in patients who sustained moderate to severe TBI

	n	Intelligence test	IQ	Memory test	MQ	IQ-MQ
Black (1973)	50	WAIS	84.50	WMS	79.1	5.40
Corkin et al. (1985)*	5	WAIS & WAIS-R	98.00	WMS	75.8	22.20
Solomon et al. (1986)	126	WAIS	93.85	WMS	90.71	3.14
Corrigan and Hinkeldey (1987)	38	WAIS	89.05	WMS	79.92	9.13
Corrigan and Hinkeldey (1987)	98	WAIS-R	82.24	WMS	85.31	–3.07
Tremont et al. (1997)	41	WAIS-R	92.98	WMS-R	90.93	2.05
Zec et al. (2001)*	24–29	WAIS-R	91.50	WMS-R	76.9	14.60
Fisher et al. (2000)	22	WAIS-III	86.50	WMS-III		
				IM	78.90**	7.60
				GM	81.90	4.60
Axelrod et al. (2001)	38	WAIS-III	85.60	WMS-III		
				IM	83.50**	2.10
				GM	86.00	–0.40
Weighted Mean	**413**		**88.28**		**85.95**	**2.33**

WAIS-R = Wechsler Adults Intelligence Scale–Revised; WMS-R = Wechsler Memory Scale–Revised; IM = Immediate Memory; GM = General Memory.
*These studies are not included in the calculation of the weighted mean (see text).
**The mean of IM and GM scores was used for the calculation of the weighted mean.

In sum, the profile of memory impairment following TBI is inconsistent with most of the features characteristic of the amnesic syndrome as presented by Parkin (1997). However, it should be noted that as demonstrated by Levin et al. (1988), it is possible to detect a subgroup of patients that do satisfy the criteria of amnesia. The differences between patients with TBI and amnesia may reflect their divergent underlying neuropathology. While medial-temporal lobe and diencephalic damage is the primary cause of global amnesia (Squire & Zola-Morgan, 1991), the frontal lobes (in addition to other structures) are most frequently involved following TBI (Adams, 1975; Crosson et al., 1993). Thus, the question addressed in the next section is:

Is There a Similarity Between Characteristics of Memory Deficit Following Frontal Lobe Damage and TBI?

Frontal lobe injury frequently occurs as a consequence of TBI (Adams, 1975; Crosson et al., 1993). Researchers have recently emphasized the cardinal role of lesions to the frontal lobes as an explanation for the behavioral sequelae typically observed following TBI (Bigler, 1990; for review, see Stuss & Gow, 1992). Although the frontal lobes are not a unitary system, it is widely accepted that one of their fundamental roles is to mediate the executive functions (Shallice & Burgess, 1991; Stuss & Benson, 1984). These functions are characterized by purposeful and self-directive behavior (Lezak, 1982) and include planning, decision-making, judgment, and self-perception and self-monitoring (Tranel, Anderson, & Benton, 1994). Consistent with this, patients sustaining frontal lobe damage present difficulties in memory tasks that require effortful organization of information in the learning and retrieval phases (Shimamura, 1995). According to Moscovitch (1994), "the frontal lobes are 'working-with-memory' structures that operate on the input to the hippocampal component and the output from it" (p. 279). As such, the frontal lobes contribute to the implementation of strategic approach and conceptual elaboration of information at the encoding as well as at the retrieval stages of the memory process.

Verbal Fluency and the Wisconsin Card Sorting Test, which are sensitive to frontal lobe damage, were found to be impaired in patients with TBI. However, these patients were not impaired on the Tower of London task, which is also sensitive to frontal lobe damage (Cockburn, 1995, but see McDowell et al., 1997). Anderson, Bigler, and Blatter (1995) also reported that patients following TBI were impaired compared to controls on the Wisconsin Card Sorting Test and on the Halstead Category Test. These tests, however, which are considered to be "frontal lobe" tests, were not sufficiently sensitive to distinguish between subgroups of patients, with or without evidence of frontal lobe damage. An interesting observation was made by Spikman, Deelman, and van Zomeren (2000): Structured executive tasks are less sensitive to TBI than are daily life unstructured executive tasks (e.g., Executive Route Finding task), because they provide external cueing to cope with the problem. The Tinkertoy test is an unstructured executive test designed by Lezak (1995). It was found to be sensitive to frontal lobe damage in patients who sustained TBI (Martzke, Swan, & Varney, 1991). Furthermore, this test was able to discriminate between employed and unemployed patients with TBI (Bayless, Varney, & Roberts, 1989). Bublak, Schubert, von Cramon, and von Cramon (2000) demonstrated that patients following TBI were impaired compared to controls on a task requiring preplanning of a sequence of actions under different working memory demands. The authors suggest that this deficit may contribute to the rigid and inflexible behavior observed in patients with TBI.

Researchers attempting to characterize the most common vulnerable memory processes following TBI frequently end up describing it in a way that very much resembles the expected memory deficits following frontal lobe damage. The conclusion of quite a few researchers is that patients afflicted by TBI have difficulties in applying active or effortful strategy, either at the encoding phase or at the retrieval phase of learning. However, when a passive strategy or automatic processes are sufficient, their performance is within the normal range (Levin, 1989a; Levin & Goldstein, 1986; Perri et al., 2000; Vakil et al., 1992). This conclusion is also consistent with findings on performance of patients with TBI on implicit memory tasks. As suggested in the Skill Learning section, contradictory findings with regard to the effect of TBI on skill learning may possibly be reconciled by distinguishing between tasks known to be sensitive to the functioning of the frontal lobes, and perceptual tasks that are not mediated by the frontal lobes. As a consequence of the vulnerability of the frontal lobes in patients suffering from TBI, the former tasks are impaired (SRT task, Vakil et al., 2002 and TOHP task, Vakil et al., 2001), but not the latter (search-detection task, Schmitter-Edgecombe & Beglinger, 2001; Schmitter-Edgecombe & Rogers, 1997). As in the case of priming tasks, patients who sustained TBI did not differ significantly from controls on the perceptual priming task, but performed more poorly than controls on the conceptual priming tasks (Vakil & Sigal, 1997).

The similarity between the characteristics of memory deficit in patients suffering frontal lobe injury and TBI goes beyond the fundamental difficulty with memory tasks that require strategic encoding or retrieval. These findings have very important implications for rehabilitation. As pointed out by Spikman et al. (2000), structured tasks are more resistant to the effects of injury because they provide external cueing to cope with the problem. Accordingly, patients are expected to reach higher performance levels under a structured environment when external cues are available.

In order to reach a more definitive conclusion about the hypothesis that memory function following moderate and severe TBI resembles frontal lobe dysfunction, it must be tested directly. A possible method to test this hypothesis is by using patients with focal frontal lesion as controls and comparing them to patients with focal frontal lesions from TBI and to patients with TBI without focal frontal findings.

Do Studies on Memory of Patients With TBI Contribute to the Understanding of Normal and Disturbed Memory Processes?

There is no doubt that much has been learned about normal memory functioning from studies with either individual (e.g., H.M.) or groups of patients with impairment of different memory systems (e.g., amnesia and Parkinson's disease). Breakdown of memory processes could help to reveal underlying independent sub-processes that seem inseparable in normal memory functioning. Deficient versus preserved types of memory in H.M. led to the important distinction between declarative and procedural memory, respectively (Cohen, 1984). In addition, such findings indicate which brain structures are involved in these processes (i.e., hippocampus in the case of H.M.). Another example is the double dissociation between declarative and skill-learning tasks reported in patients with amnesia and in patients with Parkinson's disease (Knowlton, Mangels, & Squire, 1996). The findings of this study indicated that while declarative memory is mediated by limbic and diencephalon structures (i.e., amnesia), skill learning is mediated by the basal ganglia (i.e., Parkinson's disease).

Among the findings reported in this review, there are numerous studies demonstrating that some patients with TBI are impaired on one memory type and preserved on a different

memory type. Such findings are an indication for dissociation between these two memory types. Examples of such dissociations are: between declarative, priming and saving tasks (Vakil & Oded, 2003); preserved perceptual and impaired conceptual priming (Vakil & Sigal, 1997); preserved implicit and impaired explicit measures of contextual information (Vakil et al., 1996, 1997); and preserved automatic and impaired effortful processes (Schmitter-Edgecombe & Beglinger, 2001; Schmitter-Edgecombe & Rogers, 1997).

Other findings with this patient group showed the contribution of different cognitive processes to learning and memory. Several studies have demonstrated the effect of inefficient organization and learning strategy on learning and memory (Blachstein et al., 1993; Levin et al., 1979; Paniak et al., 1989). Similarly, difficulties in the utilization of semantic knowledge were associated with deficient memory in this patient group (Levin & Goldstein, 1986; Perri et al., 2000; Vakil et al., 1992). These findings demonstrated how impaired cognitive processes, such as categorization and use of strategy, could affect learning and memory. The examples listed above show that several dissociations between different memory types are supported by findings in patients with TBI.

Thus, just as other patient populations with deficient memory (e.g., amnesia and Parkinson's disease) have contributed to finer delineation of memory processes, such research with patients who sustained TBI has also enriched our knowledge of normal memory and its disorders. However, unlike patients with localized damage, in the case of patients with TBI the association between a deficient memory process and a specific brain structure should be made more cautiously.

Factors Contributing to Heterogeneity Among Patients With TBI

In the literature on the effect of TBI on memory (and other cognitive domains), several variables probably contribute to the heterogeneity among patients with TBI that leads to the inconsistent findings reported in some cases. The major factors described below contribute potentially to the variability observed in the performance of patients with TBI. Accordingly, some minimal standards of methodology are suggested that would hopefully lead to more consistent findings with this patient group. As described below, information about some of the variables should be reported in every study of patients with TBI, while information about other variables should be used as exclusion criteria, unless they are the issues addressed in the study. As can be seen in Table 2, even though the selection criterion for the studies reviewed was all studies that tested memory functioning after moderate to severe TBI, there is a large variability among studies in several categories. First, some studies did not provide basic information about the patient group (e.g., years of education, source of patients, or some of the severity measures). Also clearly demonstrated in Table 2 is the wide range between studies and within studies, of sample size, time after injury, source of patients, and age of patients. As will be discussed below, there is no doubt that this variability contributed to the inconsistent findings in the literature on the effect of TBI on memory.

Information Should be Reported about the Following Variables:

1. The nature of the TBI. As reported above, TBI frequently leads to widespread, diffuse axonal injury (Ommaya & Gennarelli, 1974). Abnormalities on MRI have been found frequently in the temporal and frontal lobes (Adams, 1975; Crosson et al., 1993), in addition to ventricular enlargement (Anderson & Bigler, 1995; Levin et al., 1981). In an attempt to reduce the variability of patients in terms of their injury, Anderson et al.

Table 2

Demographic description of the sample group of patients with TBI who participated in the studies reviewed, and the major findings in the memory category tested

Study	Characterization of group[a]	Sample size (M/F)	Age (Y) (Mean or Median)	Education (Y) (Mean or Median)	Coma duration (H/D/Y) (Mean or Median)	GCS (Mean)	PTA duration (Min/D/W)	TAO (W/M/Y) (Mean or Median)	Source/s of patients	Memory category tested	Major findings
Azouvi et al. (1996)	1: Severe	18 (14/4)	16–42 (25.1)	8–15 (11.5)	1–75 D (18.6)	4–8 (6.1)	-	4–50 W	-	Working memory	Impaired
	2: Severe	11	16–34 (23.6)	8–15 (11.2)	1–75 D (20.1)	4–8 (6.3)		4–25 W		Working memory	Impaired
Baddeley et al. (1987)	Severe	20 (15/5)	3–18 (25.4)	-	-	-	3 D–6 M	7 M–10 Y	-	Immediate memory - verbal	Impaired
Bennett-Levy (1984)	Severe	39 (32/7)	<35	-	-	-	1 W–3 W	39.1–39.5 M	Infirmary	Immediate memory- verbal	Mixed
Blachstein et al. (1993)	Moderate - severe	30	19–57 (30.27)	8–16	1–150 D	-	-	1–15 Y	Rehab. Institute	Forgetting rate	Impaired
										Learning rate	Impaired
Brooker & George (1984)	Severe[c]	14	(49.33)	(12.03)	-	-	0	>6 M	Medical center	Immediate memory - visual	Impaired
Brooks (1975)	Mild - severe	30 (28/2)	15–60 (36.5)	-	-	-	1–100 D (28.8)	0.5–60 M	Referred for evaluation following HI	Working memory	Preserved
Brooks (1976)	-	82 (73/9)	16–60 (31.7)	(15.6)	-	-	>2 D	0–24 M	Hospital	Working memory	Impaired
Carlesimo et al. (1998)	Severe	20 (16/4)	18–36	6–14	5–60 D	<8	-	3–46 M	Rehab. program	Retrograde amnesia	Impaired
Carlesimo et al. (1997)	Severe	20 (14/6)	14–43	5–15	3–90 D	<8	-	2–64 M	Rehab. program	Semantic organization	Impaired
										Forgetting rate	Impaired
Christodoulou et al. (2001)	Moderate - severe	9 (5/4)	(32.67)	(13.89)	-	(5.71)	-	(51.33) M		Working memory	Impaired
Constantinidou & Neils (1995)	Moderate - severe	24 (21/3)	16–51 (29.8)	7–18 (12.3)	(20.918) D	<12	-	(16.41) M	Rehab. program	Learning rate	Impaired
Constantinidou et al. (1996)	Moderate - severe	31 (21/10)	16–50 (25.52)	7.5–18 (12.52)	(7) D	<12	-	(6) M	Rehab. program	Learning rate	Impaired
Cooke & Kausler (1995)	Mild - severe	30	18–55 (29.2)	-	>6 H	-	-	9–242 M	Rehab. program	Context - direct	Impaired

Continued

Table 2
Continued

Study	Characterization of group[a]	Sample size (M/F)	Age (Y) (Mean or Median)	Education (Y) (Mean or Median)	Coma duration (H/D/Y) (Mean or Median)	GCS (Mean)	PTA duration (Min/D/W)	TAO (W/M/Y) (Mean or Median)	Source/s of patients	Memory category tested	Major findings
Crosson et al. (1988)	-	33 (33/0)	(34.21)	(12.67)	0–60 D[b]	-	5–120 D[b]	-	Medical center	Forgetting rate Proactive interference	Impaired Preserved
Curtiss et al. (2001)	-	151	32.3	<12–>16	-	-	1 Min–30 D		Medical center	Working memory	Preserved
	-	150	30.6	<12–>16	-	-	1 Min–30 D	-	Medical center	Immediate memory - verbal	Impaired
DeLuca et al. (2000)	Moderate - severe	28 (21/7)	19–55 (34.65)	13.5	<1–76 D	3–10	-	13–74 M	1: Support group 2: Rehab. hospital	Learning rate Forgetting rate	Impaired Mixed
Demery et al. (2002)	1: Mild (50%) 2: Moderate (30%) 3: Severe (20%)	160 (97/63)	(34)	(13.56)	<.5 H	9–12	<24 H	(12.4) M	Health science center	Learning rate	Mixed
Deshpande et al. (1996)	Mild - severe	88	17–67 (33.6)	6–18 (11.8)	-	3–18	-	-	Hospital	Learning rate	Mixed
Dywan et al. (1993)	Moderate - severe	13 (11/2)	18–42 (27.1)	10–14 (12)	0–60 D	-	0–120 D	1–15 Y	-	Context direct	Impaired
Gardner & Vrbancic (1998)	Moderate - severe	93	17–75	-	-	-	-	-	Hospital	Learning rate	Impaired
Geffen et al. (1994)	Moderate - severe	18 (18/0)	15–28 (21.3)	9–16 (10.6)	-	-	3–90 D	1–48 M	-	Learning rate	Impaired
										Forgetting rate	Impaired
Goldstein et al. (1989)	Severe	14 (10/4)	19–49 (27.4)	10–16 (12.9)	1–91 D	4–7	-	14–150 M	Rehab. center	Semantic organization	Preserved
Goldstein et al. (1990)	Severe	16	20–49 (26.9)	12.8	-	<8	-	14–150 M	Rehab. hospital	Semantic organization	Impaired
Groot et al. (2002)	-	36 (26/10)	19–60 (35.61)	9–17 (12.42)	-	-	-	0–60 M	1: Day center 2: Rehab. center	Prospective memory	Impaired
Hannay et al. (1979)[e]	1: I	47 (39/8)	<50	-	I: 0	-	-	-	-	Immediate memory - visual	Impaired
	2: II				II: 0–1 D (0.9)	(8)					

Continued

Table 2
Continued

Study	Characterization of group[a]	Sample size (M/F)	Age (Y) (Mean or Median)	Education (Y) (Mean or Median)	Coma duration (H/D/Y) (Mean or Median)	GCS (Mean)	PTA duration (Min/D/W)	TAO (W/M/Y) (Mean or Median)	Source/s of patients	Memory category tested	Major findings
	3: III				III: 1.3–23 D (14)	(5)					
Hart (1994)	Moderate - severe	6 (4/2)	22–46 (31.8)	11–16 (12.5)	.042–14 D	6–13	-	8–53 M	-	Forgetting rate	Mixed
Haut & Shutty (1992)	-	70 (54/16)	(29)	(12)	-	-	-	(7.7) M	Neuropsych. evaluation	Learning rate	Mixed
										Forgetting Rate	Impaired
Haut et al. (1990a)	Moderate - severe	20 (15/5)	(24.45)	(11.6)	-	-	-	(3.65) Y	-	Immediate memory -verbal	Impaired
										Forgetting rate	Impaired
Haut et al. (1991a)	1: Moderate-	16	25.3	12.9	-	(11.1)	-	56.1 D	Rehab. facility	Immediate memory - verbal	Impaired
	2: Severe	16	25.8	12.9	-	(5.2)	-	56.8 D	Rehab. facility	Forgetting rate	Impaired
Haut et al. (1990b)	Severe	12	20–57 (30.1)	12–18 (13.2)	>5 D (18.67)	-	-	>1 Y (48.17) M	Rehab. center	Working memory	Impaired
Kersel et al. (2001)	Severe	65 (49/16)	16–57 (28)	-	-	-	-	153–497 D	Hospital	Immediate memory - verbal	Impaired
Kinsella et al. (1996)	Severe	24 (18/6)	18–63 (32.5)	-	-	-	(7.79) W	-	Rehab. center	Prospective mem-ory	Impaired
Levin & Goldstein (1986)	Severe	12 (7/5)	21–37 (27)	9–20 (12)	2–105 D	-	-	23–112 M	Rehab. facility	Semantic organization	Mixed
Levin et al. (1976)[e]	1: I	9 (8/1)	18–48	6–13.5	0	-	-	1–15 D	Hospital	Working memory	Impaired
	2: II	9 (9/0)	17–31	8–13	0–3 H	-	-	1–465 D	Hospital	Working memory	Impaired
	3: III	6 (6/0)	22–45	3–14	10–21 D	-	-	25–419 D	Hospital	Working memory	Impaired
Levin et al. (1979)	Severe	27	16–45 (21)	8–16 (12)	0–28 D	<8	-	153–3452 D	Neurosurgery service	Learning rate	Impaired
Levin et al. (1988)	1: Severe[c]	15 (7/8)	21–37 (26)	9–20 (12)	2–150 D	-	-	7–112 M	Rehab. facility	Context - direct	Impaired
	2: Severe	16 (13/3)	18–32 (23.5)	12–14 (12)	4–365 D	-	-	13–102 M	Rehab. facility	Context - direct	Impaired

Continued

Table 2
Continued

Study	Characterization of group[a]	Sample size (M/F)	Age (Y) (Mean or Median)	Education (Y) (Mean or Median)	Coma duration (H/D/Y) (Mean or Median)	GCS (Mean)	PTA duration (Min/D/W)	TAO (W/M/Y) (Mean or Median)	Source/s of patients	Memory category tested	Major findings
Markowitsh & Haerting (1996)	-	15 (11/4)	(43)	>8	-	-	-	-	-	Perceptual Priming	Impaired
McDowell et al. (1997)	Severe	25 (21/4)	15–55 (32)	9–18	>6 H	<8		6 W–120 M	Rehab. hospital	Working memory	Impaired
Milders (1998)	Severe	12 (11/1)	20–45 (31.4)	4–7 (4.8)	-	-	12–150 D (39.2)	0.7–5 Y (2.2) Y	1: Rehab. center 2: Hospital	Immediate memory - visual	Impaired
Millis et al. (1994)	Moderate -severe	65 (47/18)	17–75 (29.9)	7–16 (12)	-	3–12	-	-	Rehab. hospital	Learning rate	Mixed
Mutter et al. (1994)	1: Mild	12 (9/3)	(29.25)	(13.92)	<20 min	13–15		1–17 D (8.92) D	Hospital	Skill learning	Mixed
	1: Moderate[c]	11	(36.82)	(13.36)	-	>13	-	133–712 D (314.45) D	Hospital	Skill learning	Mixed
	2: Severe[c]	12	(31.42)	(13.33)	-	<13	-	112–1049 D (463.75) D	Hospital	Skill learning	Mixed
Nissley & Schmitter-Edgecombe (2002)	Severe	19 (16/3)	(35.20)	(14.32)	>24 H	<8	>7 D	>12 Y	1: Rehab. program 2: Support groups	Skill learning	Preserved
Paniak et al. (1989)	Severe	21 (12/9)	14–53,[d] 1: 25.6 2: 26.7	12	2–60 D	-	-	1: 33–183 D 2: 367–876 D	Rehab. hospital	Learning rate	Impaired
Perri et al. (2000)	Severe	15 (11/ 4)	(22.3)	(11)	2–57 D	-	-	1–38 M	Hospital	Conceptual priming	Preserved
										Semantic organization	Impaired
Reid & Kelly (1993)	-	20 (17/3)	16–74 (31.9)	>8 (11.4)	-	3–15	3–150 D	18–191 D	Rehab. unit	Immediate memory - visual	Impaired
										Forgetting rate	Impaired
Schmitter-Edgecombe (1996)	Severe	27 (14/13)	(32.62)	(13.96)	>1 D	-	>7 D	>1 Y	1: Laboratory 2: Rehab. program 3: Support group	Perceptual priming	Preserved

Continued

Table 2
Continued

Study	Characterization of group[a]	Sample size (M/F)	Age (Y) (Mean or Median)	Education (Y) (Mean or Median)	Coma duration (H/D/Y) (Mean or Median)	GCS (Mean)	PTA duration (Min/D/W)	TAO (W/M/Y) (Mean or Median)	Source/s of patients	Memory category tested	Major findings
Schmitter-Edgecombe & Rogers (1997)	Severe	10 (9/1)	20–40 (28.40)	12–16, 13.9	>2 D	-	>7 D	>1 Y	1: Laboratory 2: Support group	Skill learning	Preserved
Schmitter-Edgecombe & Beglinger (2001)	Severe	18 (15/ 3)	(32.53)	(13.61)	>2 D	<8 (n=14)	>14 D	1–27 Y	1: Rehab. program 2: Chapter of the National HI foundation 3: Support group	Skill learning	Preserved
Schmitter-Edgecombe & Wright (2003)	Severe	30 (24/6)	(34.02)	(14.03)	(3.72 W)	-	(12.03 W)	1–29 Y (11.24) Y	1: Rehab. program 2: National HI foundation 3: Support group	Context - direct	Preserved
Shum et al. (1999)	Severe	12	16–37 (23.50)	(11.42)	1–12 (4.71) W	<8		>1 Y	Rehab. center	Prospective memory	Impaired
Shum et al. (1996)	Severe	16 (14/2)	16–51	9–13 (11.06)	-	<8	2 W–3 M (6) W	>1 Y	Rehab. center	Perceptual priming	Mixed
										Conceptual priming	Mixed
Shum et al. (2000)	1: Severe	14 (10/ 4)	17–52 (28.214)	7–12 (10.714)	-	3–8	31–225 D	2 M–9M 1 W	Hospital	Immediate memory -visual	Impaired
										Learning rate	Impaired
	2: Severe	14 (11/ 3)	17–47 (24.071)	10–14 (11.286)	-	3–7	20–135 D	1 Y 2 W–6 Y 3 M	Hospital	Retroactive interference	Mixed
Skelton et al. (2000)	Moderate- severe	12 (9/3)	19–49 (35.67)	10–16 (13.1)	-	-	-	-	Hospital	Immediate memory- visual	Mixed
Stablum et al. (1994)	Severe	14 (11/3)	19–57 (32)	5–13 (9.5)	-	3–8	-	6–127 M	Hospital	Working memory	Impaired
Stallings et al. (1995)	-	40 (30/10)	(29. 3)	(12.8)	-	3–12	-	1–167 M	Hospital	Semantic organization	Impaired

Continued

Table 2
Continued

Study	Characterization of group[a]	Sample size (M/F)	Age (Y) (Mean or Median)	Education (Y) (Mean or Median)	Coma duration (H/D/Y) (Mean or Median)	GCS (Mean)	PTA duration (Min/D/W)	TAO (W/M/Y) (Mean or Median)	Source/s of patients	Memory category tested	Major findings
Tweedy & Vakil (1988)	-	45 (33/12)	18–49 (28.67)	(14)	-	-	-	>1 Y	Medical center	Context - direct	Impaired
Vakil et al. (1992)	-	40	18–48 (30)	8–15 (11)	-	-	-	-	Rehab. center	Immediate memory -verbal	Impaired
										Semantic organization	Impaired
										Forgetting rate	Impaired
Vakil et al. (1991)	-	35	19–45 (29)	8–15 (11)	-	-	-	-	Rehab. center	Context - direct	Impaired
										Context - indirect	Preserved
Vakil et al. (2002)	Severe	20 (15/5)	18–55 (32.85)	8–20 (13.30)	2–60 D	3–13	-	5–47 M	Hospital	Skill learning	Impaired
Vakil et al. (1996)	Moderate- severe	15 (12/3)	16–44 (29.47)	10–19(12.40)	0.2–45 D	4–9	-	11–42 W	Rehab. hospital	Context -direct	Impaired
										Context - indirect	Preserved
Vakil et al. (2001)	Moderate- severe	26 (17/9)	17–57 (27.32)	10–15 (12.25)	2–120 D	3–11	-	3–72 M	Rehab. hospital	Skill learning	Impaired
Vakil & Oded (2003)	Moderate- severe	20 (19/1)	20–42 (28.35)	10–15 (12.75)	1–14 D	4–12	-	26–152 M	Neuropsych. treatment and rehab. unit	Forgetting rate	Mixed
										Perceptual priming	Mixed
										Learning rate	Impaired
Vakil & Blachstein (1997)	Moderate- severe	20 (19/1)	17–50 (26.30)	10–19 (11.85)	4–60 D	3–10	-	6–67 W	Rehab. hospital	Context – direct	Impaired
										Context - indirect	Preserved
Vakil & Sigal (1997)	Moderate- severe	24 (19/5)	18–46 (28.3)	10–17 (12.3)	1–99 D	-	-	12–137 M	Rehab. hospital	Perceptual priming	Preserved
										Conceptual priming	Impaired
Vakil & Tweedy (1994)	1: Moderate	20 (14/6)	18–49 (28.7)	-	>1 H	-	-	>1 Y	Rehab. institute	Context - direct	Impaired

Continued

Table 2
Continued

Study	Characterization of group[a]	Sample size (M/F)	Age (Y) (Mean or Median)	Education (Y) (Mean or Median)	Coma duration (H/D/Y) (Mean or Median)	GCS (Mean)	PTA duration (Min/D/W)	TAO (W/M/Y) (Mean or Median)	Source/s of patients	Memory category tested	Major findings
	2: Moderate	25 (19/6)	19–49 (28.64)	-	>1 H	-	-	>1 Y	Rehab. institute	Context - direct	Impaired
Vanderploeg et al. (2001)	Moderate- severe	55 (45/10)	37–44 (40.6)	<12–>16	1 M–30 D	-	1H–30 D	<3 M–>1 Y	Defense and Veterans' HI Program	Forgetting rate	Impaired
										Learning rate	Preserved
Watt et al. (1999)	1: Severe	12 (9/3)	18–35 (28.66)	11.91	>2 D	-	>7 D	>3 Y	Organizations that provide respite care	Perceptual priming	Mixed
Zec et al. (2001)	Severe	32 (24/ 8)	(34.4)	(12.7)	>14 D[b]	-	-	>2 Y	1: Community 2: Care facility	Learning rate	Impaired
										Immediate memory - verbal	Impaired
										Immediate memory - visual	Impaired
										Forgetting rate	Impaired

(M/F) = male/female; Min = minute; H = hour; D = day; W = week; M = month; Y = year; HI = head injury; GCS = Glasgow Coma Scale; PTA = posttraumatic amnesia; TAO = time after onset; Mixed = the major findings varied with the subgroups or different conditions. See text for more information.

[a] According to at least one of the defining criteria.

[b] A minimal amount of people exceeded the upper limit.

[c] As defined by the author despite not being in accordance with the classical definition.

[d] Patients were tested within 6 M of regaining consciousness and then again after at least 1 Y.

[e] Hannay & Levine classify their subjects in 3 severity groups. I = consciousness on admission and throughout and absence of neurological deficits. II= comatose for <1 D although they may have been stuporous for a longer interval and may have developed neurological deficits. III = comatose >1 D and had neurological deficits.

[*] TBI is used in this review because it is a broader term than the alternatives (e.g., closed-head injury - CHI).

(1995) distinguished between subgroups of patients with TBI, with and without evidence of frontal lobe damage. Wilson, Hadley, Wiedmann, and Teasdale (1995) distinguished between two subgroups of patients with TBI, the first with diffuse injury (primarily to the corpus callosum and the brainstem) and the second group with focal injury (primarily to the frontotemporal areas). Thus, it is imperative that neuroimaging data (e.g., MRI) be reported for each patient participating in the study. Newer imaging techniques such as DTI that document diffuse axonal injury and volumetric measures may provide additional information about the integrity of brain structures and may prove useful in creating subgroups for investigation.

2. *How is TBI acquired?* Although in most studies with patients who sustained TBI injury is a result of motor vehicle accident, some studies have included in their sample patients with penetration injury, falls, or assault (e.g., Boake, Freelands, Ringholz, & Nance, 1995; Constantinidou & Neils, 1995; Constantinidou et al., 1996; Kesler, Adams, Blasey, & Bigler, 2003). This is an empirical question: Do the different causes of TBI yield a similar pattern of injury, and as such a similar pattern of deficit? In an attempt to reduce variability we recommend using a homogeneous group, or alternatively, if the sample size is sufficiently large, comparing the performance of the subgroups of patients.
3. *Severity of injury (measured by length of coma, GCS, PTA duration, time after onset, and extent of lesion):* Severity of injury is usually reported, and in most studies the distinction between mild and moderate to severe injury (defined by length of coma, GCS score, and PTA) is maintained. Nevertheless, several researchers reported significant relations between various severity measures and memory. Stuss et al. (2000), combining severity of injury measures as well as demographic data, identified four patient subgroups that predicted outcome (measured as continuous memory of words after 24 hours) more accurately than GCS alone. Quite a few studies demonstrated the relation between length of PTA and severity of memory impairment (Bennett-Levy, 1984; Brooks, 1974a). Time after onset should also be taken into consideration. Sbordone, Liter, and Pettler-Jennings (1995) reported that even 10 years post-injury, patients' performance continued to improve. Similarly, improvement of intelligence scores over time was reported by Mandleberg (1975, 1976). Novack et al. (1995) tested patients with TBI in two sessions, the first within five months post-onset of PTA, and the second session within six months of the first session. Results indicated improvement from one session to the other on several memory measures in the test, although normal performance was not attained even in the second session. Hannay et al. (1979) found that recognition of line drawings was associated with length of coma. Levin's (1989b) conclusion, based on a review of the literature, is that the relationship between measures of severity of injury and memory performance of patients with TBI is moderate. Nevertheless, it seems that the classification of severity of TBI into mild, moderate and severe injury, based on a combination of length of coma, GCS score, and PTA (Williamson et al., 1996), is shown to be informative by several studies (e.g., Mangels et al., 2000).
4. *Age at onset of injury:* In most studies testing the effect of TBI on memory, patients are in their twenties, reflecting the age range (15–24) in which TBI occurs most frequently (Levin et al., 1982) (see Table 2). However, several studies that tested the effect of TBI on older individuals found that, as compared to younger patients, aging aggravates the outcome of the older patient group in a variety of domains, including cognition (Goldstein & Levin, 1995b; Katz & Alexander, 1994; Reeder, Rosenthal, Lichtenberg, & Wood, 1996) (for review on the outcome following TBI in the elderly see Rapoport & Feinstein, 2000). Thus, in studies with a wide age range of patients, reports on the

effect of TBI might be confounded with the effect of age. It is therefore recommended that patients' age range not exceed 15 years and that elderly participants not be included, or if included then as a separate group.

5. *Premorbid intelligence:* Premorbid intelligence is usually unavailable, but can be estimated either by number of years of education or by tests such as the National Adult Reading Test (NART) (see Lezak, 1995). Hannay et al. (1979) did not find education and age to be related to recognition of line drawings in patients with TBI. Nevertheless, estimates of premorbid intelligence might be associated with more demanding memory tasks than recognition, which require strategic encoding or retrieval. Freeman et al. (2001) found the discrepancy between the estimated premorbid intelligence score, using the OPIE, and the delayed recall index from the WMS-R as the measure most sensitive to TBI.
6. *Participation in rehabilitation program:* In many studies the patients are recruited from a rehabilitation program because they are readily available (see Table 2). On the one hand it is possible that these patients are referred to a rehabilitation program because of the severity of their cognitive impairments compared to those not referred to such a program. On the other hand in some rehabilitation programs patients are selected on the basis of learning potential (see Levin & Goldstein, 1986). Furthermore, in most programs patients receive cognitive remediation training. It is expected that such remediation treatment would affect performance at study (e.g., by utilization of mnemonic strategies), yet such information is not always controlled or even provided. Moreover, it is important to compare patients' performance pre- and post-training.

Variables That Should be Used as Exclusion Criteria (Unless They Are the Focus of the Study):

1. *Emotional/psychiatric state (e.g., presence of depression)*: Numerous studies have reported the emotional consequences of TBI. Furthermore, cognitive performance, including memory, was found to be affected by emotional state. For example, Gass and Apple (1997) reported that cognitive complaints were associated with emotional distress on the MMPI. Similarly, memory self-reports were associated with the severity of depression (Alfano, Neilson, & Fink, 1993; Boake et al. 1995). Therefore, it is recommended that at the very least, screening for depression should be conducted using one of the depression scales, in order to exclude from the sample patients who score in the pathological range.
2. *Pre-existing developmental/cognitive disorders:* Patients with a history of developmental disorders such as learning disabilities or attention deficit disorder (ADD) should not be included in a standard sample unless the developmental disorder is one of the issues addressed in the study.
3. *Pre-existing neurological disorders:* Many neurological disorders (e.g., Parkinson's) are known to affect cognition, including memory (Ferraro et al., 1993). Therefore, in a case where such a patient has sustained TBI, it would be almost impossible to sort out clearly the effect of the injury from the effect of the pre-existing condition. It would thus be preferable that such patients not be included in the study.
4. *Alcohol or drug abuse:* It is well documented that substance abuse causes neuropathological changes in the brain, such as reduced cerebral gray matter in alcoholics (Jernigan et al. 1991) or cerebral atrophy in cocaine abusers (Pascual-Leone, Dhuna, & Anderson (1991). Several studies reported that patients with TBI who tested positive compared to patients who tested negative on alcohol or drug abuse at the time of injury, were impaired on a range of neuropsychological tests, including the WAIS-R and the WMS-R (Kelly, Johnson, Knoller, & Winslow, 1997; Wilde et al., 2004).

5. *Patients in a litigation process:* many individuals who have sustained TBI are involved at one time or another in a litigation process that might affect their motivation to cooperate, intentionally (i.e., malingering) or even unintentionally. It is not surprising that these patients have no interest in making an effort or in demonstrating their full abilities. Hence, such a patient should not be included in a research study, first of all because of the question of validity of performance, and secondly to avoid placing such a patient in a conflicting situation. For example, the study by Ropacki and Elias (2003) tested the cognitive reserve theory in patients with closed head injury. In this study one of the exclusion criteria was that "At the time of evaluation, no patients were involved in litigation related to their head injury or pursuing either a worker's compensation or social security claim…" (pp. 646–647).

The importance of many of the variables discussed in the last two sections, those that should be reported and those that should be used as exclusion criteria, could be viewed from the perspective of brain capacity, also called cognitive reserve, and the threshold theory (Satz, 1993). With this theory, Satz attempted to explain confusing findings of patients with similar characteristics of brain injury and yet with different neurocognitive outcomes. It is claimed by this theory that on the one hand, cognitive or brain reserves can protect a patient with brain damage from the expression of pathological symptoms, or delay the onset of cerebral disease and mental decline as a function of age. On the other hand, reduced cognitive or brain reserves (e.g., earlier brain injury) would lead to greater vulnerability in the case of a new injury or disease (e.g., Alzheimer). Among the measures used for cognitive reserve are: general intelligence, educational level, and occupational attainment. Measures commonly used for brain reserve are: total intracranial volume (TICV) and ventricle-to brain ratio. Ropacki and Elias (2003) tested patients with a range of severity of mild to severe TBI. Based on premorbid history, that is presence or absence of mental or neurological illness, alcoholism, or drug abuse, patients were divided into positive or negative premorbid history, respectively. The results indicate that the group with the positive premorbid history (i.e., with reduced cognitive reserve) was more impaired cognitively compared to the group with the negative premorbid history. Cognitive impairment was measured by pre-post PIQ difference and VIQ/PIQ discrepancy. Kesler et al. (2003) tested the cognitive reserve hypothesis on 25 patients with TBI ranging in severity from mild to severe (i.e., GCS ranged = 3–14). Premorbid intelligence was estimated on the basis of the American College Testing Program (ACT) scores. Post injury intelligence was measured using the WAIS-R. TICV was used as an estimate of premorbid brain volume. In agreement with the cognitive reserve hypothesis, premorbid brain volume and education level reduced the negative effect of TBI on cognitive performance.

Thus far, I have addressed preexisting factors, and attempts to deal a priori with these factors in order to reduce the heterogeneity of patients' performance. Several studies have taken a different approach to cope with the heterogeneity of patients with TBI, by dividing their patient group into subgroups a posteriori, based on the characteristics of their performance. An example of such an approach is the study by Curtiss et al. (2001) which, by using cluster analysis procedure, identified different subgroups of patients characterized by particular deficits: consolidation, retention, or retrieval processes. As reported above, Mangels et al. (2000) divided the patients with moderate to severe TBI into two subgroups based on their strategy at learning. One group's memory was intact, while the other group's memory was impaired and disproportionately affected by divided attention. Other examples are the studies by Millis and Ricker (1994) and Deshpande et al. (1996), which used the CVLT to detect five subgroups of patients with TBI (only four were interpretable):

active, passive, disorganized, and deficient. Another example is the study by Demery et al. (2002), which identified two subgroups, one with normal and the other with moderate-to-severe impairment.

These findings should be taken as a cautionary sign against attempts to characterize these patients as a homogeneous group. One of the future challenges in this field is to identify subgroups of patients with TBI by adopting either an a priori or an a posteriori approach, or a combination of both. An a priori approach would require reliable characterization based first of all on the unique nature of their injury, either in terms of the structural damage to specific brain regions (e.g., based on MRI), the severity of injury, or in terms of the dysfunction of specific brain areas (e.g., based on fMRI or PET). The a posteriori approach is based on patients' memory profile (possibly using additional cognitive measures). An example of such an approach is the one used by Glisky and colleagues with elderly individuals tested for 'item' and 'source' memory (Glisky, Polster, & Routhieaux, 1995; Glisky, Rubin, & Davidson, 2001). In these studies elderly participants were divided preexperimentally into different groups based on their performance on tests sensitive to frontal lobes functioning (e.g., Wisconsin Card Sorting Test) and medial temporal lobes (e.g., Logical memory I, subtest of the WMS-R). The classification of the test was done on the basis of factor analysis. When applied to patients who have sustained TBI, such a procedure would enable the classification of patients with predominantly frontal lobe impairment versus patients with predominantly medial temporal lobe impairment. These subgroups of patients are expected to differ on a variety of memory tests. Consistent with the findings by Glisky and colleagues, item and source memory are predicted to be dependent on the functioning of the medial temporal lobes and the frontal lobes, respectively. Such a research strategy has the potential of explaining much of the variability in findings reported in the literature on the effect of TBI on memory. Furthermore, classification into subgroups could lead to a more precise prognosis and to more efficient rehabilitation.

Methodological Issues.

1. Patients selection: The importance of reporting whether participants attended a rehabilitation program was discussed earlier. This point touches upon a broader methodological issue, namely selection bias. The danger is that studies will be conducted on more accessible samples of patients, which do not necessarily represent the larger population of patients who sustained TBI. Selecting patients on the basis of inclusion and exclusion criteria from consecutive referrals could reduce some selection bias (see Boake et al., 1995).
2. *Matched control group:* Very few studies have used another patient group as a control group, such as orthopedic patients involved in a traumatic accident rather than healthy control individuals (Bennett-Levy, 1984; Brooks, 1974a; Richardson & Snape, 1984; Wilson et al., 1995; Zec et al., 2001). Unlike normal healthy control participants, such a control group is a better match for patients who have sustained TBI because these patients experienced a similar trauma (with the exception of TBI) with all the emotional sequelae (e.g., depression, posttraumatic stress disorder) and other experiences such as hospitalization. Lannoo et al. (2001) have found that their control group, trauma patients with injuries to parts of the body other than the head, was also impaired on several neuropsychological tests including memory. As described above, these factors are known to have an effect on memory and other cognitive tasks. Thus, comparison to normal healthy control individuals might have exaggerated the effect of TBI per se on memory.

3. *Sample size, variability, and statistical power:* It is obviously more difficult to recruit patients than healthy individuals to participate in a study. Therefore, many of the studies are conducted on a small number of patients (see Table 2). In addition the variability in the patients' performance leads to large variance scores. These two factors reduce the power of statistical analyses. Thus, lack of significant effects should be interpreted cautiously. Hence, statistical power should also be reported.

In conclusion, TBI causes memory deficits expressed in a large range of memory tasks. Memory impairment is probably the most debilitating cognitive consequence of TBI, but it is only one aspect among other impaired cognitive domains such as attention, speed of processing, and executive functions. This conclusion has important implications for approaches to compensation for memory problems after TBI, in terms of the need to adjust the remediation program to the specific memory deficit characteristics (for review, see, Sohlberg & Mateer, 2001; Wilson, 1992, 2004). The profile of the memory deficit in patients with TBI resembles that of patients with frontal injury rather than that of patients with amnesia. This is not surprising, given the nature of TBI, which frequently affects the frontal lobes. Nevertheless, the conclusion regarding similarity between patients with TBI and those with frontal lobe injury should be drawn cautiously. As could be expected by the nature of the injury, and as demonstrated by different researchers (Curtiss et al., 2001; Demery et al., 2002; Deshpande et al., 1996; Millis & Ricker, 1994), patients with TBI are not a homogeneous group. As demonstrated by Levin et al. (1988) it is possible to find a subgroup of patients who do satisfy the criteria of amnesia. Therefore, patients who have sustained TBI should be divided into different subgroups, either a priori according to the range of factors listed above (e.g., nature and severity of injury), or a posteriori based on their specific memory deficit characteristics. The challenge would then be to detect the relations between these two types of factors.

References

Adams, J. H. (1975). The neuropathology of head injury. In P. J, Binken, & G. W, Bruyn (Eds.), *Handbook of clinical neurology* (pp. 35–65). New York: Elsevier.

Alfano, D. P., Neilson, P. M., & Fink, M. P. (1993). Long-term psychosocial adjustment following head or spinal cord injury. *Neuropsychiatry, Neuropsychology, and Behavioral Neurology*, *6*, 117–125.

Anderson, C. V., & Bigler, E. D. (1995). Ventricular dilation, cortical atrophy, and neuropsychological outcome following traumatic brain injury. *Journal of Neuropsychiatry and Clinical Neuroscience*, *7*, 42–48.

Anderson, C. V., Bigler, E. D., & Blatter, D. D. (1995). Frontal lobe lesions, diffuse damage, and neuropsychological functioning in traumatic brain-injured patients. *Journal of Clinical and Experimental Neuropsychology*, *17*, 900–908.

Arcia, E., & Gualtieri, C. T. (1993). Association between patient report of symptoms after mild head injury and neurobehavioural performance. *Brain Injury*, *7*, 481–489.

Axelrod, B. N., Fichtenberg, N. L., Liethen, P. C., Czarnota, M. A., & Stucky, K. (2001). Performance characteristics of postacute traumatic brain injury patients on the WAIS-III and WMS-III. *Clinical Neuropsychology*, *15*, 516–520.

Azouvi, P., Jokic, C., Van der Linden, M., Marlier, N., & Russel, B. (1996). Working memory and supervisory control after severe closed-head injury: A study of dual task performance and random generation. *Journal of Clinical and Experimental Neuropsychology*, *18*, 317–337.

Baddeley, A. (2000). The episodic buffer: A new component of working memory. *Trends in Cognitive Science*, *4*, 417–423.

Baddeley, A., Harris, J., Sunderland, A., Watts, K. P., & Wilson, B. A. (1987). Closed head injury and memory. In H. S, Levin, J, Grafman, & H. M, Eisenberg (Eds.), *Neurobehavioural recovery from head injury* (pp. 295–319). Oxford: Oxford University Press.

Baddeley, A. D., & Hitch, G. J. (1974). Working memory. In G. H, Bower (Ed.), *The psychology of learning and motivation: Advances in research and theory*. Vol.8 (pp. 47–89) New York: Acedemic Press..

Bayless, J.D., Varney, N. R., & Roberts, R. J. (1989). Tinker toy test performance and vocational outcome in patients with closed-head injuries. *Journal of Clinical and Experimental Neuropsychology*, *11*, 913–917.

Bennett-Levy, J. M. (1984). Long-term effects of severe closed head injury on memory: Evidence from a consecutive series of young adults. *Acta Neurologica Scandinavica*, *70*, 285–298.

Bigler, E. D. (1990). Neuropathology of traumatic brain injury. In E. D, Bigler (Ed.), *Traumatic brain injury* (pp. 13–49). Austin, TX: PRO-ED.

Bigler, E. D., Johnson, S. C., Anderson, C. V., & Blatter, D. D. (1996). Traumatic brain injury and memory: The role of hippocampal atrophy. *Neuropsychology*, *10*, 333–342.

Binder, L. M. (1986). Persisting symptoms after mild head injury: A review of post concussive syndrome. *Journal of Clinical and Experimental Neuropsychology*, *8*, 323–346.

Blachstein, H., Vakil, E., & Hoofien, D. (1993). Impaired learning in patients with closed-head injuries: An analysis of components of the acquisition process. *Neuropsychology*, *7*, 530–535.

Black, R. W. (1973). Cognitive and memory performance in subjects with brain damage secondary to penetrating missile wounds and closed head injury. *Journal of Clinical Psychology*, *29*, 441–442.

Blaxton, T. A. (1992). Dissociations among memory measures in memory-impaired subjects: Evidence for a processing account of memory. *Memory & Cognition*, *20*, 549–562.

Boake, C., Freelands, J. C., Ringholz, G. M., & Nance, M. L. (1995). Awareness of memory loss after severe closed-head injury. *Brain Injury*, *9*, 273–283.

Brooker, A. E., & George, J. C. (1984). Visual recognition memory of severely head-injured patients. *Perceptual and Motor Skills*, *59*, 249–250.

Brooks, D. N. (1974a). Recognition memory and head injury. *Journal of Neurology, Neurosurgery and Psychiatry*, *37*, 794–801.

Brooks, D. N. (1974b). Recognition memory after head injury: A signal detection analysis. *Cortex*, *10*, 224–230.

Brooks, D. N. (1975). Long- and short-term memory in head injured patients. *Cortex*, *11*, 329–340.

Brooks, D. N. (1976). Wechsler Memory Scale performance and its relationship to brain damage after severe closed head injury. *Journal of Neurology, Neurosurgery and Psychiatry*, *39*, 593–601.

Brooks, N., McKinlay, W., Symington, C., Beattie, A., & Campsie, L. (1987). Return to work within the first seven years of severe head injury. *Brain Injury*, *1*, 5–19.

Brouwer, W. H., & van Wolffelaar, P. C. (1985). Sustained attention and sustained effort after closed head injury: Detection and 0.10 Hz heart rate variability in a low event rate vigilance task. *Cortex*, *21*, 111–119.

Bublak, P., Schubert, T., von Cramon, M. G., & von Cramon, D. Y. (2000). Differential demands on working memory for guiding a simple action sequence: Evidence from closed-head-injured subjects. *Journal of Clinical and Experimental Neuropsychology*, *22*, 176–189.

Buschke, H., & Fuld, P. A. (1974). Evaluation of storage, retention, and retrieval in disordered memory and learning. *Neurology*, *24*, 1019–1025.

Carlesimo, G. A., Sabbadini, M., Bombardi, P., Di Porto, E., Loasses, A., & Caltagirone, C. (1998). Retrograde memory deficits in severe closed head injury patients. *Cortex*, *34*, 1–23.

Carlesimo, G. A., Sabbadini, M., Loasses, A., & Caltagirone, C. (1997). Forgetting from long-term memory in severe closed-head injury patients: Effect of retrieval conditions and semantic organization. *Cortex*, *33*, 131–142.

Cermak, L. S., Verfaellie, M., & Chase, K. A. (1995). Implicit and explicit memory in amnesia: An analysis of data-driven and conceptually-driven processes. *Neuropsychology*, *9*, 281–290.

Christodoulou, C., DeLuca, J., Ricker, J. H., Madigan, N. K., Bly, B. M., Lange, G., Kalnin, A. J., Liu, W. C., Steffener, J., Diamond, B. J., & Ni, A. C. (2001). Functional magnetic resonance imaging of working memory impairment after traumatic brain injury. *Journal of Neurology, Neurosurgery and Psychiatry*, *71*, 161–168.

Cockburn, J. (1995). Performance on the Tower of London test after severe head injury. *Journal of the International Neuropsychological Society*, *1*, 537–544.

Cohen, N. J. (1984). Preserved learning capacity in amnesia: Evidence for multiple memory systems. In L.R, Squire, & N, Butters (Eds.), *Neuropsychology of memory* (pp. 83–103). New York: The Guilford Press.

Cohen N. J. Eichenbaum H. Deacedo B. S. Corkin S. (1985). Different memory systems underlying acquisition of procedural and declarative knowledge.D. S., OltonGamzu, E. S. Corkin, (Eds) *Memory dysfunctions: An integration of animal and human research from preclinical and clinical perspectives* (pp. 54–71). New York: New York Academy of Sciences.

Cohen, N. J., & Squire, L. R. (1980). Preserved learning and retention of pattern-analyzing skill in amnesia: Dissociation of knowing how and knowing that. *Science*, *210*, 207–210.

Constantinidou, F., & Neils, J. (1995). Stimulus modality and verbal learning after moderate to severe closed head injury. *Journal of Head Trauma Rehabilitation*, *10*, 90–100.

Constantinidou, F., Neils, J., Bouman, D., & Lee, L. (1996). Pictorial superiority during verbal learning tasks in moderate to severe closed head injury: Additional evidence. *Journal of General Psychology*, *123*, 173–184.

Cooke, D. L, & Kausler, D. H. (1995). Content memory and temporal memory for actions in survivors of traumatic brain injury. *Journal of Clinical and Experimental Neuropsychology*, *17*, 90–99.

Corkin, S, Cohen, N. J, Sullivan, E. V, Clegg, R. A, Rosen, T. J, & Ackerman, R. H. (1985). Analyses of global memory impairment of different etiologies. *Annals of the New York Academy of Sciences*, *444*, 10–40.

Corrigan, J. D., & Hinkeldey, N. S. (1987). Comparison of intelligence and memory in patients with diffuse and focal injury. *Psychological Reports*, *60*, 899–906.

Cremona-Meteyard, S. L., Clark, C. R., Wright, M. J., & Geffen, G. M. (1992). Covert orientation of visual attention after closed head injury. *Neuropsychologia*, *30*, 123–132.

Crosson, B., Novack, T. A., Trenerry, M. R., & Craig, P. L. (1988). California Verbal Learning Test (CVLT) performance in severely head-injured and neurologically normal adult males. *Journal of Clinical and Experimental Neuropsychology*, *10*, 754–768.

Crosson, B., Sartor, K. J., Jenny, A. B., Nabors, N. A., & Moberg, P. J. (1993). Increased intrusions during verbal recall in traumatic and nontraumatic lesions of the temporal lobe. *Neuropsychology*, *7*, 193–208.

Curtiss, G., Vanderploeg, R. D., Spencer, J., & Salazar, A. M. (2001). Patterns of verbal learning and memory in traumatic brain injury. *Journal of the International Neuropsychological Society*, *7*, 574–585.

Delis, D. C., Kramer, J. H., Kaplan, E., & Ober, B. A. (1987). *California Verbal Learning Test: Research edition*. San Antonio, TX: The Psychological Corporation.

DeLuca, J., Schultheis, M. T., Madigan, N. K., Christodoulou, C., & Averill, A. (2000). Acquisition versus retrieval deficits in traumatic brain injury: Implications for memory rehabilitation. *Archives of Physical and Medical Rehabilitation*, *81*, 1327–1333.

Demery, J. A., Pedraza, O., & Hanlon, R. E. (2002). Differential profiles of verbal learning in traumatic brain injury. *Journal of Clinical and Experimental Neuropsychology*, *24*, 818–827.

Deshpande, S. A., Millis, S. R., Reeder, K. P., Fuerst, D., & Ricker, J. H. (1996). Verbal learning subtypes in traumatic brain injury: A replication. *Journal of Clinical and Experimental Neuropsychology*, *18*, 836–842.

Dywan, J, Segalowitz, S. J., Henderson, D., & Jacoby, L. (1993). Memory for source after traumatic brain injury. *Brain and Cognition*, *21*, 20–43.

Ferraro, F. R. (1996). Cognitive slowing in closed-head injury. *Brain and Cognition*, *32*, 429–440.

Ferraro, F. R., Balota, D. A., & Connor, L. T. (1993). Implicit memory and the formation of new associations in nondemented Parkinson's disease individuals and individuals with senile dementia of the Alzheimer type: A serial reaction time (SRT) investigation. *Brain and Cognition*, *21*, 163–180.

Fisher, D. C., Ledbetter, M. F., Cohen, N. J., Marmor, D., & Tulsky, D. S. (2000). WAIS-III and WMS-III profiles of mildly to severely brain-injured patients. *Applied Neuropsychology*, *7*, 126–132.

Fontaine, A., Azouvi, P., Remy, P., Bussel, B., & Samson, Y. (1999). Functional anatomy of neuropsychological deficits after severe traumatic brain injury. *Neurology*, *53*, 1963–1968.

Frankowski, R. H., Annegers, J. F., & Whitman, S. (1985). Epidemiological and descriptive studies. I: The descriptive epidemiology of head trauma in the United States. In D. P, Becker, & J. T, Povlishock (Eds.), *Central nervous system trauma status report* (pp. 33–43). Bethesda, MD: National Institute of Neurological Disorders and Strike.

Freeman, J., Godfrey, H. P. D., Harris, J. K. J., & Partridge, F. M. (2001). Utility of demographic equation in detecting impaired NART performance after TBI. *British Journal of Clinical Psychology*, *40*, 221–224.

Gardner, S. D., & Vrbancic, M. I. (1998). Which California Verbal Learning Test factors discriminate moderate and severe head injury from normals? *Brain and Cognition*, *37*, 10–13.

Gass, C. S., & Apple, C. (1997). Cognitive complaints in closed-head injury: Relationship to memory test performance and emotional disturbance. *Journal of Clinical and Experimental Neuropsychology*, *19*, 290–299.

Geffen, G. M., Butterworth, P., Forrester, G. M., & Geffen, L. B. (1994). Auditory verbal learning test components as measures of the severity of closed-head injury. *Brain Injury*, *8*, 405–411.

Glanzer, M., & Cunitz, A. R. (1966). Two storage mechanisms in free recall. *Journal of Verbal Learning and Verbal Behavior*, *5*, 351–360.

Glisky, E. L., Polster, M. R., & Routhieaux, B. C. (1995). Double dissociation between item and source memory. *Neuropsychology*, *9*, 229–235.

Glisky, E. L., Rubin, S. R., & Davidson, P.S. R. (2001). Source memory in older adults: An encoding or retrieval problem? *Journal of Experimental Psychology: Learning, Memory, and Cognition*, *27*, 1131–1146.

Goldstein, F. C., & Levin, H. S. (1995a). Post-traumatic and anterograde amnesia following closed head injury. In A. D, Baddeley, B. A, Wilson, & F. N, Watts (Eds.), *Handbook of memory disorders* (pp. 187–209). New York: John Wiley & Sons.

Goldstein, F. C., & Levin, H. S. (1995b). Neurobehavioral outcome of traumatic brain injury in older adults: Initial findings. *Journal of Head Trauma Rehabilitation*, *10*, 57–73.

Goldstein, F. C., Levin, H. S., & Boake, C. (1989). Conceptual encoding following severe closed head injury. *Cortex*, *25*, 541–554.

Goldstein, F. C., Levin, H. S., Boake, C., & Lohrey, J. H. (1990). Facilitation of memory performance through induced semantic processing in survivors of severe closed-head injury. *Journal of Clinical and Experimental Neuropsychology*, *12*, 286–300.

Gronwall, D., & Wrightson, P. (1981). Memory and information processing capacity after closed head injury. *Journal of Neurology, Neurosurgery and Psychiatry*, *44*, 889–895.

Groot, Y. C. T., Wilson, B. A., Evans, J., & Watson, P. (2002). Prospective memory functioning in people with and without brain injury. *Journal of the International Neuropsychological Society*, *8*, 645–654.

Hannay, H. J., Levin, H. S., & Grossman, R. G. (1979). Impaired recognition memory after head injury. *Cortex*, *15*, 269–283.

Hart, R. P. (1994). Forgetting in traumatic brain-injured patients with persistent memory impairment. *Neuropsychology*, *8*, 325–332.

Haut, M. W., Petros, T. V., & Frank, R. G. (1990a). The recall of prose as a function of importance following closed head injury. *Brain Injury*, *4*, 281–288.

Haut, M. W., Petros, T. V., & Frank, R. G. (1991a). Semantic sensitivity in the acute phase of recovery from moderate and severe closed head injury. *Neuropsychology*, *5*, 81–88.

Haut, M. W., Petros, T. V., Frank, R. G., & Haut, J. S. (1991b). Speed of processing within semantic memory following severe closed head injury. *Brain and Cognition*, *17*, 31–41.

Haut, M. W., Petros, T. V., Frank, R. G., & Lamberty, G. (1990b). Short-term memory processes following closed head injury. *Archives of Clinical Neuropsychology, 5*, 299–309.

Haut, M. W., & Shutty, M. S. (1992). Patterns of verbal learning after closed head injury. *Neuropsychology, 6*, 51–58.

Henry-Feugeas, M. C., Azouvi, P., Fontaine, A., Denys, P., Bussel, B., & Maaz, F. et al. (2000). MRI analysis of brain atrophy after severe closed-head injury: Relation to clinical status. *Brain Injury, 14*, 597–604.

Huisman, T. A. G. M., Schwamm, L. H., Schaefer, P. W., Koroshetz, W. Z., & Shetty-Alva, N. et al. (2004). Diffusion tensor imaging as potential biomarker of white matter injury in diffuse axonal injury. *American Journal of Neuroradiology, 25*, 370–376.

Isaacs, E. B., & Vargh-Khdem, F. (1989). Differential course of development of spatial and verbal memory span: A normative study. *British Journal of Developmental Psychology, 7*, 377–380.

Jernigan, T. L., Butters, N., DiTraglia, G., Schafer, K., Smith, T., Grant, I., Schuckit, M., & Cermak, L. (1991). Reduced cerebral gray matter observed in alcoholics using magnetic resonance imaging. *Alcoholism: Clinical and Experimental Research, 15*, 418–427.

Johnstone, B., Hexum, C. L., & Ashkanazi, G. (1995). Extent of cognitive decline in traumatic brain injury based on estimates of premorbid intelligence. *Brain Injury, 9*, 377–384.

Katz, D. I., & Alexander, M. P. (1994). Traumatic brain injury: Predicting course of recovery and outcome for patients admitted to rehabilitation. *Archives of Neurology, 51*, 661–670.

Kelly, M. P., Johnson, C. T., Knoller, N., & Winslow, M. M. (1997). Substance abuse, traumatic brain injury and neuropsychological outcome. *Brain Injury, 11*, 391–402.

Kersel, D. A., Marsh, N. V., Havill, J. H., & Sleigh, J. W. (2001). Neuropsychological functioning during the year following severe traumatic brain injury. *Brain Injury, 15*, 283–296.

Kesler, S. R., Adams, H. F., Blasey, C. M., & Bigler, E. D. (2003). Premorbid intellectual functioning, education, and brain size in traumatic brain injury: an investigation of the cognitive reserve hypothesis. *Applied Neuropsychology, 10*, 153–162.

Kinsella, G., Murtagh, D., Landry, A., Homfray, K., Hammond, M., O'Beirne, L., Dwyer, L., Lamont, M., & Ponsford, J. (1996). Everyday memory following traumatic brain injury. *Brain Injury, 10*, 499–507.

Knowlton, B. J., Mangels, J. A., & Squire, L. R. (1996). A neostriatal habit learning system in humans. *Science, 273*, 1399–1402.

Kotapka, M. J., Graham, D. I., Adams, J. H., & Gennarelli, T. A. (1992). Hippocampal damage in fatal non-missile human head injury. *Acta Neuropathologica, 83*, 530–534.

Kraus, A. (1993). Epidemiology of head injury. In P. R, Cooper (Ed.), *Head injury* (3rd ed., pp.1–25). Baltimore:Williams & Wilkins.

Lannoo, E., Colardyns, F., Jannes, C., & De Soete, G. (2001). Course of neuropsychological recovery from moderate-to-severe head injury: A 2-year follow-up. *Brain Injury, 15*, 1–13.

Leclercq, M., Couillet, J., Azouvi, P., Marlier, N., Martin, Y., Strypstein, E., & Rousseaux, M. (2000). Dual task performance after severe diffuse traumatic brain injury or vascular prefrontal damage. *Journal of Clinical and Experimental Neuropsychology, 22*, 339–350.

Levin, H. S. (1989a). Memory deficit after closed head injury. *Journal of Clinical and Experimental Neuropsychology, 12*, 129–153.

Levin, H. S. (1989b). Memory deficit after closed head injury. In F, Boller, & J, Grafman (Eds.), *Handbook of neuropsychology*(Vol. 3 pp.183–207). Amsterdam: Elsevier Science.

Levin, H. S., Benton, A. L., & Grossman, R. G. (1982). *Neurobehavioral consequences of closed head injury*. (p. 55). New York: Oxford University Press.

Levin, H. S., & Goldstein, F. C. (1986). Organization of verbal memory after severe closed-head injury. *Journal of Clinical and Experimental Neuropsychology, 8*, 643–656.

Levin, H. S., Goldstein, F. C., High, W. M., & Eisenberg, H. M. (1988). Disproportionately severe memory deficit in relation to normal intellectual functioning after closed head injury. *Journal of Neurology, Neurosurgery and Psychiatry, 51*, 1294–1301.

Levin, H. S., Goldstein, F. C., High, W. M., & Williams, D. (1988). Automatic and effortful processing after severe closed head injury. *Brain and Cognition, 7*, 283–297.

Levin, H. S., Grossman, R. G., & Kelly, P. J. (1976). Short-term recognition memory in relation to severity of head injury. *Cortex*, *12*, 175–182.

Levin, H. S., Grossman, R. G., Rose, J. E., & Teasdale, G. (1979). Long-term neuropsychological outcome of closed head injury. *Journal of Neurosurgery*, *50*, 412–422.

Levin, H. S., Meyers, C. A., Grossman, R. G., & Sarwar, M. (1981). Ventricular enlargement after closed head injury. *Archives of Neurology*, *38*, 623–629.

Levine, B., Cabeza, R., McIntosh, A. R., Black, S. E., Grady, C. L., & Stuss, D. T. (2002). Functional reorganization of memory after traumatic brain injury: A study with H(2)(15)0 positron emission tomography. *Journal of Neurology, Neurosurgery and Psychiatry*, *73*, 173–181.

Lezak, M. D. (1979). Recovery of memory and learning functions following traumatic brain injury. *Cortex*, *15*, 63–72.

Lezak, M. D. (1982). The problem of assessing executive functions. *International Journal of Psychology*, *17*, 281–297.

Lezak, M. D. (1995). *Neuropsychological assessment* (3rd ed.). New York: Oxford University Press.

Loken, W. J., Thornton, A. E., Otto, R. L., & Long, C. J. (1995). Sustained attention after severe closed head injury. *Neuropsychology*, *9*, 592–598.

Madigan, N. K., DeLuca, J., Diamond, B. J., Tramontano, G., & Averill, A. (2000). Speed of information processing in traumatic brain injury: Modality-specific factors. *Journal of Head Trauma Rehabilitation*, *15*, 943–956.

Mandleberg, I. A. (1975). Cognitive recovery after severe head injury. 2. Wechsler Adult Intelligence Scale during post-traumatic amnesia. *Journal of Neurology, Neurosurgery, and Psychiatry*, *38*, 1227–1232.

Mandleberg, I. A. (1976). Cognitive recovery after severe head injury. 3. WAIS verbal and performance IQs as a function of post-traumatic amnesia duration and time from injury. *Journal of Neurology, Neurosurgery, and Psychiatry*, *39*, 1001–1007.

Mangels, J., Craik, F., Levine, B., Schwartz, M., & Stuss, D. (2000). Chronic deficits in item and context memory following traumatic brain injury: A function of attention and injury severity. *Brain and Cognition*, *44*, 107–112.

Mapou, R. L. (1992). Neuropathology and neuropsychology of behavioral disturbances following traumatic brain injury. In C. J, Long, & L. K, Ross (Eds.), *Handbook of head trauma: Acute care to recovery. Critical issues in neuropsychology* (pp. 75–89). New York: Plenum Press.

Markowitsch, H. J., Calabrese, P., Liess, J., Haupts, M., Durwen, H. F., & Gehlen, W. (1993). Retrograde amnesia after traumatic injury of the fronto-temporal cortex. *Journal of Neurology, Neurosurgery and Psychiatry*, *56*, 988–992.

Markowitsch, H. J., & Haerting, C. (1996). Interdependence of priming performance and brain damage. *International Journal of Neuroscience*, *85*, 291–300.

Martin, T. A., Donders, J., & Thompson, E. (2000). Potential of and problems with new measures of psychometric intelligence after traumatic brain injury. *Rehabilitation psychology*, *45*, 402–408.

Martzke, J. S., Swan, C. S., & Varney, N. R. (1991). Posttraumatic anosmia and orbital frontal damage: neuropsychological and neuropsychiatric correlates. *Neuropsychology*, *5*, 213–225.

Mattioli, F., Grassi, F., Perani, D., & Cappa, S. F. (1996). Persistent post-traumatic retrograde amnesia: A neuropsychological and (18F) FDG PET study. *Cortex*, *32*, 121–129.

Mayes, A. R., MacDonald, C., Donlan, L., Pears, J., & Meudel, P. R. (1992). Amnesics have a disproportionately severe memory deficit for interactive context. *The Quarterly Journal of Experimental Psychology*, *45A*, 265–297.

McDowall, J., & Martin, S. (1996). Implicit learning in closed-head-injured subjects: Evidence from an event sequence learning task. *New Zealand Journal of Psychology*, *25*, 2–6.

McDowell, S., Whyte, J., & D'Esposito, M. (1997). Working memory impairments in traumatic brain injury: Evidence from a dual-task paradigm. *Neuropsychologia*, *35*, 1341–1353.

McKinlay, W. W., Brooks, D. N., Bond, M. R., Martinage, D. P., & Marshall, M. M. (1981). The short-term outcome of severe blunt head injury as reported by relatives of the injured persons. *Journal of Neurology, Neurosurgery, and Psychiatry*, *44*, 527–533.

Milders, M. (1998). Learning people's names following severe closed-head injury. *Journal of Clinical and Experimental Neuropsychology*, *20*, 237–244.

Millis, S. R., & Ricker, J. H. (1994). Verbal learning patterns in moderate and severe traumatic brain injury. *Journal of Clinical and Experimental Neuropsychology*, *16*, 498–507.

Milner, B. (1975). Hemisphere specialization: Scope and limits. In F. O, Schmitt, & F. G, Warden (Eds.), *The neurosciences: Third study program*. Cambridge: MIT Press.

Moscovitch, M. (1994). Memory and working with memory: Evaluation of a component process model and comparisons with other models. In D. L, Schacter, & E, Tulving (Eds.), *Memory systems 1994*. Cambridge: MIT Press.

Moscovitch, M., Goshen-Gottstein, Y., & Vierzen, E. (1994). Memory without conscious recollection: A tutorial review from a neuropsychological perspective. In C, Umila, & M, Moscovitch (Eds.), *Attention and performance*. (Vol. 15 pp. 619–660) Cambridge, MA: MIT Press.

Mulligan, N. W. (1998). The role of attention during encoding in implicit and explicit memory. *Journal of Experimental Psychology: Learning, Memory and Cognition*, *24*, 27–47.

Mutter, S. A., Howard, J. H., & Howard, D. V. (1994). Serial pattern learning after head injury. *Journal of Clinical and Experimental Neuropsychology*, *16*, 271–288.

National Center for Injury Prevention and Control. (1999). *Traumatic brain injury in the United States: A report to the Congress*. Atlanta: GA, Centers for Disease Control and Prevention.

Neil, J., Miller, J., Mukherjee, P., & Huppi, P. S. (2002). Diffusion tensor imaging of normal and injured developing human brain — a technical review. *NMR in Biomedicine*, *15*, 543–552.

Nissen, M. J., & Bullemer, P. (1987). Attentional requirements of learning: Evidence from performance measures. *Cognitive Psychology*, *19*, 1–32.

Nissley, H. M., & Schmitter-Edgecombe, M. (2002). Perceptually based implicit learning in severe closed-head injury patients. *Neuropsychology*, *16*, 111–122.

Novack, T. A., Kofoed, B. A., & Crosson, B. (1995). Sequential performance on the California Verbal Learning Test following traumatic brain injury. *Clinical Neuropsychologist*, *9*, 38–43.

Numan, B., Sweet, J. J., & Ranganath, C. (2000). Use of the California Verbal Learning Test to detect proactive interference in the traumatically brain injured. *Journal of Clinical Psychology*, *56*, 553–562.

Oddy, M., Coughlan, T., Tyerman, A., & Jenkins, D. (1985). Social adjustment after closed head injury: A further follow-up seven years after injury. *Journal of Neurology, Neurosurgery, and Psychiatry*, *48*, 564–568.

Ommaya, A. K., & Gennarelli, T. A. (1974). Cerebral concussion and traumatic unconsciousness. *Brain*, *97*, 633–654.

Paniak, C. E., Shore, D. L., & Rourke, B. P. (1989). Recovery of memory after severe closed-head injury: Dissociations in recovery of memory parameters and predictors of outcome. *Journal of Clinical and Experimental Neuropsychology*, *11*, 631–644.

Park, N. W., Moscovitch, M., & Robertson, I. H. (1999). Divided attention impairments after traumatic brain injury. *Neuropsychologia*, *37*, 1119–1133.

Parkin, A. J. (1997). Normal age-related memory loss and its relation to frontal lobe dysfunction. In P, Rabbitt (Ed.), *Methodology of frontal and executive function* (pp. 177–190). Hove, East Sussex, England: Psychology Press.

Pascual-Leone, A., Dhuna, A., & Anderson, D. C. (1991). Cerebral atrophy in habitual cocaine abuse: A preliminary CT study. *Neurology*, *41*, 34–38.

Perri, R., Carlesimo, G. A., Loasses, A., & Caltagirone, C. (2000). Deficient intentional access to semantic knowledge in patients with severe closed-head injury. *Cortex*, *36*, 213–225.

Posner, M. I., & Petersen, S. E. (1990). The attention system of the human brain. *Annual Review of Neuroscience*, *13*, 25–42.

Prigatano, G. P. (1978). Wechsler Memory Scale: A selective review of the literature. *Journal of Clinical Psychology*, *34*, 816–832.

Rapoport, M. J., & Feinstein, A. (2000). Outcome following traumatic brain injury in the elderly: A critical review. *Brain Injury*, *14*, 749–761.

Reeder, K. P., Rosenthal, M., Lichtenberg, P., & Wood, D. (1996). Impact of age on functional outcome following traumatic brain injury. *Journal of Head Trauma Rehabilitation*, *11*, 22–31.

Reid, D. B., & Kelly, M. P. (1993). Wechsler Memory Scale—Revised in closed head injury. *Journal of Clinical Psychology*, *49*, 245–254.

Rey, A. (1964). *L'Examen clinique en psychologie*. Paris: Press Universitaires de France.

Richardson, J. T. E. (2000). *Clinical and neuropsychological aspects of closed head injury* (2nd ed.). Philadelphia, PA: Psychology Press.

Richardson, J. T., & Snape, W. (1984). The effects of closed head injury upon human memory: An experimental analysis. *Cognitive Neuropsychology*, *1*, 217–231.

Ricker, J. H., Mueller, R. A., Zafonte, R. D., Black, K. M., Millis, S. R., & Chugani, H. (2001). Verbal recall and recognition following traumatic brain injury: A [O-15]-water positron emission tomography study. *Journal of Clinical and Experimental Neuropsychology*, *23*, 196–206.

Ropacki, M. T., & Elias, J. W. (2003). Preliminary examination of cognitive reserve theory in closed head injury. *Archives of Clinical Neuropsychology*, *18*, 643–654.

Rugg-Gunn, F. J., Symms, M. R., Barker, G. J., Greenwood, R., & Duncan, J. S. (2001). Diffusion imaging shows abnormalities after blunt head trauma when conventional magnetic resonance imaging is normal. *Journal of Neurology, Neurosurgery, and Psychiatry*, *70*, 530–533.

Russell, W. R., & Nathan, P. W. (1946). Traumatic amnesia. *Brain*, *69*, 183–187.

Satz, P. (1993). Brain reserve capacity on symptom onset after brain injury: A formulation and review of evidence for threshold theory. *Neuropsychology*, *7*, 273–295.

Sbordone, R. J., Liter, J. C., & Pettler-Jennings, P. (1995). Recovery of function following severe traumatic brain injury: A retrospective 10-year follow-up. *Brain Injury*, *9*, 285–299.

Schacter, D. L. (1987). Implicit memory: History and current status. *Journal of Experimental Psychology: Learning, Memory and Cognition*, *13*, 501–518.

Schacter, D. L., & Crovitz, H. F. (1977). Memory function after closed head injury: A review of the quantitative research. *Cortex*, *13*, 150–176.

Schacter, D. L., Harbluk, J. L., & McLachlan, D. R. (1984). Retrieval without recollection: An experimental analysis of source amnesia. *Journal of Verbal Learning and Verbal Behavior*, *23*, 593–611.

Schmitter-Edgecombe, M. (1996). Effects of divided attention on implicit and explicit memory performance following severe closed head injury. *Neuropsychology*, *10*, 155–167.

Schmitter-Edgecombe, M., & Beglinger, L. (2001). Acquisition of skilled visual search performance following severe closed-head injury. *Journal of the International Neuropsychological Society*, *7*, 615–630.

Schmitter-Edgecombe, M., & Kibby, M. K. (1998). Visual selective attention after severe closed head injury. *Journal of the International Neuropsychological Society*, *4*, 144–159.

Schmitter-Edgecombe, M., & Nissley, H. M. (2000). Effects of divided attention on automatic and controlled components of memory after severe closed-head injury. *Neuropsychology*, *14*, 559–569.

Schmitter-Edgecombe, M., & Rogers, W. A. (1997). Automatic process development following severe closed head injury. *Neuropsychology*, *11*, 296–308.

Schmitter-Edgecombe, M., & Wright, M. J. (2003). Content memory and temporal order memory for performed activities after severe closed-head injury. *Journal of Clinical and Experimental Neuropsychology*, *25*, 933–948.

Shallice, T., & Burgess, P. W. (1991). Deficits in strategy application following frontal lobe damage in man. *Brain*, *114*, 727–741.

Shimamura, A. P. (1995). Memory and the prefrontal cortex. In J, Grafman, K. J, Holyoak, & F, Boller (Eds.), *Structure and functions of the human prefrontal cortex.* (Vol. 769 pp. 151–159). New York: Annals of the New York Academy of Sciences.

Shum, D. H. K., Harris, D., & O'Gorman, J. G. (2000). Effects of severe traumatic brain injury on visual memory. *Journal of Clinical and Experimental Neuropsychology*, *22*, 25–39.

Shum, D., Sweeper, S., & Murray, R. (1996). Performance on verbal implicit and explicit memory tasks following traumatic brain injury. *Journal of Head Trauma Rehabilitation*, *11*, 43–53.

Shum, D., Valentine, M., & Cutmore, T. (1999). Performance of individuals with severe long-term traumatic brain injury on time-, event-, and activity-based prospective memory tasks. *Journal of Clinical and Experimental Neuropsychology*, *21*, 49–58.

Skelton, R. W., Bukach, C. M., Laurance, H. E., Thomas, K. G. F., & Jacobs, W. J. (2000). Humans with traumatic brain injuries show place-learning deficits in computer-generated virtual space. *Journal of Clinical and Experimental Neuropsychology*, *22*, 157–175.

Sohlberg, M. M., & Mateer, C. A. (2001). *Cognitive rehabilitation: An integrative neuropsychological approach*. New York: NY, Guilford Press.

Solomon, G. S., Greene, R. L., Farr, S. P., & Kelly, M. P. (1986). Relationships among Wechsler Intelligence and Memory Scale Quotients in adult closed head injured patients. *Journal of Clinical Psychology*, *42*, 318–323.

Spikman, J. M., Deelman, B. G., & van Zomeren, A. H. (2000). Executive functioning, attention, and frontal lesions in patients with chronic CHI. *Journal of Clinical and Experimental Neuropsychology*, *22*, 325–338.

Squire, L. R., & Zola-Morgan, M. (1991). The medial temporal lobe system. *Science*, *253*, 1380–1386.

Srinivas, K., & Roediger, H. L. (1990). Classifying implicit memory tests: Category association and anagram solution. *Journal of Memory and Language*, *29*, 389–412.

Stablum, F., Leonardi, G., Mazzoldi, M., Umilta, C., & Morra, S. (1994). Attention and control deficits following closed head injury. *Cortex*, *30*, 603–618.

Stallings, G. A., Boake, C., & Sherer, M. (1995). Comparison of the California Verbal Learning Test and the Rey Auditory Verbal Learning Test in head-injured patients. *Journal of Clinical and Experimental Neuropsychology*, *17*, 706–712.

Stuss, D. T., & Benson, D. F. (1984). Neuropsychological studies of the frontal lobes. *Psychological Bulletin*, *95*, 3–28.

Stuss, D. T., Benson, D. F., Kaplan, E. F., Weir, W. S., Naeser, M. A.Lieberman, I. et al. (1983). The involvement of orbitofrontal cerebrum in cognitive tasks. *Neuropsychologia*, *21*, 235–248.

Stuss, D. T., Binns, M. A., Carruth, F. G., Levine, B., Brandys, C. F., Moulton, R. J., Snow, W. G., & Schwartz, M. L. (2000). Prediction of recovery of continuous memory after traumatic brain injury. *Neurology*, *54*, 1337–1344.

Stuss, D. T., & Gow, C. A. (1992). "Frontal dysfunction" after traumatic brain injury. *Neuropsychiatry, Neuropsychology, and Behavioral Neurology*, *5*, 272–282.

Teasdale, G., & Jennett, B. (1974). Assessment of coma and impaired consciousness: A practical scale. *Lancet*, *2*, 81–84.

Timmerman, M. E., & Brouwer, W. H. (1999). Slow information processing after very severe closed head injury: Impaired access to declarative knowledge and intact application and acquisition of procedural knowledge. *Neuropsychologia*, *37*, 467–478.

Tranel, D., Anderson, S. W., & Benton, A. (1994). Development of the concept of "executive function" and its relationship to the frontal lobes. In F, Boller, & H, Spinnler (Eds.), *Handbook of neuropsychology*.(Vol. 9 pp. 125–148). Amsterdam: Elsevier.

Tremont, G., Hoffman, R. G., Scott, J. G, Adams, R. L., & Nadolne, M. J. (1997). Clinical utility of Wechsler Memory Scale-Revised and predicted IQ discrepancies in closed head injury. *Archives of Clinical Neuropsychology*, *12*, 757–762.

Tweedy, J. R., & Vakil, E. (1988). Evaluating evidence for automaticity in frequency of occurrence judgments: A bias for bias? *Journal of Clinical and Experimental Neuropsychology*, *10*, 664–674.

Underwood, B. J. (1957). Interference and forgetting. *Psychological Review*, *64*, 49–60.

Vakil, E., Arbell, N., Gozlan, M., Hoofien, D., & Blachstein, H. (1992). Relative importance of informational units and their role in long-term recall by closed-head-injured patients and control groups. *Journal of Consulting and Clinical Psychology*, *60*, 802–803.

Vakil, E., Biederman, Y., Liran, G., Groswasser, Z., & Aberbuch, S. (1994). Head injured patients and control group: Implicit vs. explicit measures of frequency judgment. *Journal of Clinical and Experimental Neuropsychology*, *16*, 539–546.

Vakil, E., & Blachstein, H. (1997). Rey AVLT: Developmental norms for adults and the sensitivity of different measures to age. *The Clinical Neuropsychologist*, *11*, 356–369.

Vakil, E., Blachstein, H., & Hoofien, D. (1991). Automatic temporal order judgment: The effect of intentionality of retrieval on closed-head-injured patients. *Journal of Clinical and Experimental Neuropsychology*, *13*, 291–298.

Vakil, E., Golan, H., Grunbaum, E., Groswasser, Z., & Aberbuch, S. (1996). Direct and indirect measures of contextual information in brain-injured patients. *Neuropsychiatry, Neuropsychology, and Behavioral Neurology*, *9*, 176–181.

Vakil, E., Gordon, Y., Birnstok, S., Aberbuch, S., & Groswasser, Z. (2001). Declarative and non-declarative sequence learning tasks: Closed-head injured patients versus control participants. *Journal of Clinical and Experimental Neuropsychology*, *23*, 207–214.

Vakil, E., Kraus, A., Bor, B., & Groswasser, Z. (2002). Impaired skill learning in patients with severe closed-head injuries as demonstrated by the serial reaction time (SRT) task. *Brain and Cognition*, *50*, 30, 4–315.

Vakil, E., & Oded, Y. (2003). Comparison between three memory tests: Cued recall, priming and saving closed-head injured patients and controls. *Journal of Clinical and Experimental Psychology*, *25*, 27, 4–282.

Vakil, E., Openheim, M., Falck, D., Aberbuch, S., & Groswasser, Z. (1997). The indirect influence of modality on the direct memory for words and their modality: Closed-head injured patients versus control participants. *Neuropsychology*, *11*, 545–551.

Vakil, E., Sherf, R., Hoffman, M., & Stern, M. (1998). Direct and indirect memory measures of temporal order and spatial location: Control versus closed-head injured subjects. *Neuropsychiatry, Neuropsychology, and Behavioral Neurology*, *11*, 212–217.

Vakil, E., & Sigal, J. (1997). The effect of level of processing on conceptual and perceptual priming: Control versus closed-head-injured patients. *Journal of the International Neuropsychological Society*, *3*, 327–336.

Vakil, E., & Tweedy, J. R. (1994). Memory for temporal order and spatial position information: Test of the automatic-effortful distinction. *Neuropsychiatry, Neuropsychology, and Behavioral Neurology*, *7*, 281–288.

Vanderploeg, R. D., Crowell, T. A., & Curtiss, G. (2001). Verbal learning and memory deficits in traumatic brain injury: Encoding, consolidation, and retrieval. *Journal of Clinical and Experimental Neuropsychology*, *23*, 185–195.

van Zomeren, A. H. (1981). *Reaction time and attention after closed head injury*. Lisse, Switzerland: Swets & Zeitlinger.

van Zomeren, A. H., & van den Burg, W. (1985). Residual complaints of patients two years after severe head injury. *Journal of Neurology, Neurosurgery, and Psychiatry*, *48*, 21–28.

Veltman, J. V., Brouwer, W. H., van Zomeren, A. H., & van Wolffelaar, P. C. (1996). Central executive aspects of attention in subacute severe and very severe closed head injury patients: Planning, inhibition, flexibility, and divided attention. *Neuropsychology*, *10*, 357–367.

Watt, S., Shores, E. A., & Kinoshita, S. (1999). Effects of reducing attentional resources on implicit and explicit memory after severe traumatic brain injury. *Neuropsychology*, *13*, 338–349.

Wechsler, D. (1945). A standardized memory scale for clinical use. *Journal of Psychology*, *19*, 87–95.

Wechsler, D. (1955). *Wechsler Adult Intelligence Scale*. New York: Psychological Corporation.

Wechsler, D. A. (1981). *Wechsler Adult Intelligence Scale—Revised*. New York: Psychological Corporation.

Wechsler, D. (1987). *Manual for the Wechsler Memory Scale—Revised*. San Antonio, TX: Psychological Corporation.

Wechsler, D. (1997a). *Wechsler Adult Intelligence Scale* (3rd ed.). San Antonio, TX: Psychological Corporation.

Wechsler, D. (1997b). *Wechsler Memory Scale* (3rd ed.). San Antonio, TX: Psychological Corporation.

Weiskrantz, L. (1985). Issues and theories in the study of amnesic syndrome. In N. M, Weinberger, K. F, Berham, & G, Lynch (Eds.), *Memory systems of the brain*. New York: Guilford Press.

Wickens, D. D., Born, D. G., & Allen, C. K. (1963). Proactive inhibition and item similarity in short-term memory. *Journal of Verbal Learning and Verbal Behavior, 10*, 382–385.

Wilde, E. A., Bigler, E. D., Gandhi, P. V., Lowry, C. M., Blatter, D. D., Brooks, J., & Ryser, D. K. (2004). Alcohol abuse and traumatic brain injury: Quantitative magnetic resonance imaging and neuropsychological outcome. *Journal of Neurotrauma, 21*, 137–147.

Williamson, D. J. G., Scott, J. G., & Adams, R. L. (1996). Traumatic brain injury. In R.L., Adams, & O. A., Parsons, & et al. (Eds.), *Neuropsychology for clinical practice: Etiology, assessment, and treatment of common neurological disorders* (pp. 9–64). Washington, DC, US: American Psychological Association.

Wilson, B. (1992). Recovery and compensatory strategies in head injured memory impaired people several years after insult. *Journal of Neurology, Neurosurgery, and Psychiatry, 55*, 177–180.

Wilson, B. A. (2004). Management and remediation of memory problems in brain-injured adults. In A. D, Baddeley, M. D, Kopelman, & B. A, Wilson (Eds.), *The essential handbook of memory disorders for clinicians* (pp. 199–226). New York: John Wiley & Sons.

Wilson, J.-T. L., Hadley, D. M., Wiedmann, K. D., & Teasdale, G. M. (1995). Neuropsychological consequences of two patterns of brain damage shown by MRI in survivors of severe head injury. *Journal of Neurology, Neurosurgery and Psychiatry, 59*, 328–331.

Zec, R. F., Zellers, D., Belman, J., Miller, J., Matthews, J., Ferneau-Belman, D., & Robbs, R. (2001). Long-term consequences of severe closed head injury on episodic memory. *Journal of Clinical and Experimental Neuropsychology, 23*, 671–691.