

Blood alcohol level and early cognitive status after traumatic brain injury

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Statement of Purpose: This archival study sought to clarify the relationship between admission blood alcohol level (BAL) after traumatic brain injury (TBI) and subsequent neuropsychological functioning. It was hypothesized that BAL would be positively correlated with impairment on basic neuropsychological tests and that this relation would weaken as time since TBI increased.

Methods: Fifty-eight patients were tested within 60 days of their TBI. Correlational analyses were used to test the relation between neuropsychological performance and admission BAL.

Results: As expected, BAL was unrelated to demographic variables or lag time between TBI and time of testing. Bivariate correlations showed that higher BAL predicted poorer performance on a broad range of neuropsychological tests. Patients tested less than 30 days after their TBI showed the strongest effects.

Conclusions: Neuropsychological impairments detected 1-2 months after TBI may be affected by BAL at the time of hospital admission. The influence of BAL seems greatest during the first month post-injury, but may persist beyond 30 days in some areas of cognitive function. Blood alcohol at the time of injury may have a direct effect on cognitive functioning or may be a proxy for the effects of chronic alcohol use or abuse. Clinical implications are discussed.

Introduction

Alcohol and traumatic brain injury (TBI) are inextricably connected. People who sustain traumatic brain injury are more likely to have a history of significant alcohol related problems than the general population [1]. Alcohol use at the time of injury is thought to be involved in 47% of cases [2]. After brain injury, alcohol use typically subsides for a short period, but drinking rates subsequently increase from 1 month to 1 year after TBI [3]. Returning to drinking alcohol, at least within 1-2 years after brain injury, is thought to be a risk factor for recurrent traumatic brain injury [4, 5], development of post-traumatic seizures [6], cerebral atrophy [7], poorer cognitive recovery [8], poorer community integration [9], and deterioration of emotional and behavioural functioning during the post-acute recovery phase [10].

One of the most interesting putative relationships between alcohol and TBI is the effect that a significant blood alcohol level (BAL) at the time of injury may have

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on subsequent neuropsychological impairments. The two clinical neuropsychological studies currently available produce a confusing picture. Brooks and colleagues [11] obtained neuropsychological data on 134 people with severe brain injury within 7 years post-injury. Drinking at the time of injury was judged to be 'none', 'some' or 'drunk' by a relative of the injured person. The researchers found that estimates of drinking at the time of injury bore a clearer relationship to cognitive outcome than estimates of habitual drinking. Greater estimated drinking at the time of injury was associated with poorer performance on a number of measures, but most clearly on measures of memory, especially verbal learning. The authors cautioned that the effects of habitual drinking and age probably confounded the effect of drinking at the time of injury. Weaknesses of this study include having no objective measure of BAL at the time of injury and providing no information on the subjects' alcohol use during the years following TBI.

Kaplan and Corrigan [12] studied 88 persons with severe brain injury who had admission BALs available. Among patients with a BAL greater than zero ($n = 46$), BAL was positively correlated with time to rehabilitation admission. However, neuropsychological testing results obtained on average 67–82 days after the TBI, did not vary as a function of three levels of BAL.

Studies of the effects of alcohol intoxication on neurological outcomes also provide mixed results. Some studies have shown that alcohol intoxication at the time of brain injury is associated with lower levels of consciousness [13], longer length of coma [13], longer hospitalization, longer period of agitation [14] and lower cognitive level at discharge [14]. On the other hand, Ruff and colleagues [15] reported that intoxication at the time of injury was not a factor in predicting Glasgow Outcome Scale scores or mass lesions, whereas a history of chronic alcohol abuse history was associated with both. Kaplan and Corrigan [12] found no reliable relationship between BAL and length of post-traumatic amnesia. Other investigators have made the point that, for intoxicated patients, neurological assessments such as the Glasgow Coma Scale (GCS) are thought to be unreliable and to overestimate injury severity [16]. Alcohol intoxication seems to influence initial hospital treatment such as increasing the likelihood that patients will be intubated, require an intracranial pressure monitor or need ventilator support [2]. However, Gurney *et al.* admit that these findings are more likely to be due to the unwillingness of clinicians to rely solely on clinical neurological signs in intoxicated patients than to greater brain damage in intoxicated patients.

Taken together, the relevant literature could be interpreted to suggest that alcohol intoxication is most likely to affect early indicators of cognitive function, but that with time and physical recovery the neuropsychological influence of intoxication fades. To help clarify the relationship between blood alcohol and neuropsychological functioning the authors wanted to examine cognitive test data obtained soon after TBI, within 2 months of injury. The main hypothesis of this study was that higher blood alcohol levels at the time of injury would be associated with greater neuropsychological impairments soon after TBI. It was predicted that this relationship would be unaffected by potential confounding variables such as age, education level and brain injury severity. It was also predicted that the relationship between blood alcohol level and cognition would be weaker as time since injury increased.

Method

Subjects

This is an archival study based on 75 persons with TBI who were referred for brief neuropsychological assessment during their initial in-patient rehabilitation. A total of 68 patients had admission blood alcohol values available. Of those, 64 had completed the primary outcome measure, the Neurobehavioral Cognitive Status Examination (NCSE) [17]. Persons tested beyond this 60-day limit were excluded because there is evidence that many cognitive effects of alcoholism diminish after 2 months of abstinence [18]. Fifty-eight met the final inclusion criteria, having been tested within 60 days of brain injury onset. All procedures for this study were approved by a human subjects institutional review board.

Procedures

At Harborview Medical Center patients with TBI are tested on a routine basis during in-patient rehabilitation. All testing was performed in a standardized manner by trained psychometrists. Since testing was for clinical purposes, tests were administered at variable times post-injury, within the 60-day limit described above. The number of days between TBI onset and testing ranged from 4–56. Patients with very severe injuries (i.e. persons who remained in post-traumatic amnesia throughout their rehabilitation stay), non-English speakers and patients with severe comprehension deficits were not tested and are not represented in this study sample.

Measures

Neurobehavioral Cognitive Status Examination (NCSE). The NCSE is a brief screening instrument designed to measure ten areas of cognitive functioning that are summarized in a profile with age-corrected cutoffs. Areas measured include orientation, attention span, language comprehension, sentence repetition, object naming, constructional ability, verbal memory, mental arithmetic, similarities and judgment. Scores in each area fall into normal, mild, moderate or severe impairment. A validity study has shown that the NCSE is significantly more sensitive to neurological impairment than two widely used mental status examinations [19]. For most subscales, a more difficult screening item is administered first followed by several 'metric' items that are administered only if the subject fails the screening item. Previous researchers have recommended that investigators administer the metric components of the construction subscale regardless of whether the patient passed the screening component in order to reduce potential false-positive test results [20]. Therefore, the block design component of the subscale was always administered and subscale scores reflected performance on this task.

Trailmaking Tests, forms A and B (TMT-A; TMT-B). The TMT-A and TMT-B are widely used measures of psychomotor speed, concentration and cognitive flexibility.

Standard administration and scoring procedures were used [21]. The authors used the time to complete each test as dependent measures for this study.

Rey Auditory Verbal Learning Test (RAVLT). The RAVLT [21, 22] is a widely used, easily administered test which measures immediate memory span, verbal learning curve, recall post distraction and delayed recall. Subjects are read a list of 15 unrelated words five times. After each learning trial they are asked to recall as many words as they can. Subsequently they are asked to learn a second (distractor) list of words, after which they are asked to recall the first word list again (recall post-distraction). For the purposes of this study the authors used the total number of words recalled over the five learning trials and the number of words recalled post-distraction as the dependent measures.

Blood alcohol level (BAL). Blood samples were obtained by the emergency department medical personnel as part of the routine laboratory testing for all trauma patients. Standard laboratory procedures were used to determine serum alcohol concentration expressed here in terms of mg/dL. Levels ≥ 100 mg/dL indicate legal intoxication.

Traumatic brain injury severity was categorized into three ranges by the first author based on a combination of administration GCS and CT scan results [23]. In general, mild brain injury was characterized as having an initial GCS of 13–15 and negative head CT scan. Moderate injuries were characterized by GSC from 9–12 inclusive. Injuries were considered severe when the initial GCS was 8 or less.

Results

Participants ($n = 58$) were on average 34.9 years of ($sd = 13.8$; range 15–81). In total 69% were male and 83% were Caucasian, 10% African American, 3% Native American and 3% Hispanic. The average number of years of education was 12.5 ($sd = 2.3$; range 8–18). More than half of the sample (55%) had a positive blood alcohol screen on admission. Of those with a positive screen, 81% were legally intoxicated ($BAL \geq 100$ mg/dL). The average BAL for the entire sample was 97.5 ($sd = 104$; range 0–306) while the average BAL for the 32 participants with a positive screen was 176.8 ($sd = 73.6$; range: 14–306). Of the 58 patients, 9% had a mild brain injury, 48% had a moderate brain injury and 43% had a severe brain injury.

Before testing the main hypotheses, the authors attempted to identify potential confounding variables by computing correlations between the independent variable (BAL) and other factors including age, sex, years of education, brain injury severity and the number of days between TBI and the neuropsychological screening exam. There were no statistically significant correlations between BAL and potential confounding effects, though in the subsample of patients with a positive BAL, there was a non-significant trend for female gender to be positively correlated with higher BAL ($r = 0.29$; $p = 0.10$).

To test the main hypothesis, correlations were calculated between BAL and neuropsychological test scores. Correlations for the total sample as well as within the subgroup with positive BAL were examined to avoid the attenuation in correlations likely to occur due to the fact that 45% of the subjects had a BAL of zero. As table 1 shows, results were consistent with the hypothesis that those with higher

Table 1. Correlations between BAL and scores on neuropsychological tests

Neuropsychological test	Total sample	Tested ≤ 30 days from TBI	Tested 31–60 days after TBI	Positive BAL only
NCSE orientation	$r_{(58)} = -0.35^{**}$	$r_{(34)} = -0.43^*$	$r_{(24)} = -0.24$	$r_{(32)} = -0.35^*$
NCSE attention	$r_{(58)} = -0.11$	$r_{(34)} = -0.25$	$r_{(24)} = 0.17$	$r_{(32)} = -0.42^*$
NCSE comprehension	$r_{(58)} = 0.20$	$r_{(34)} = 0.19$	$r_{(24)} = 0.24$	$r_{(32)} = 0.21$
NCSE repetition	$r_{(58)} = -0.18$	$r_{(34)} = -0.27$	$r_{(24)} = 0.05$	$r_{(32)} = -0.26$
NCSE naming	$r_{(58)} = -0.38^{***}$	$r_{(34)} = -0.39^*$	$r_{(24)} = -0.37$	$r_{(32)} = -0.49^{***}$
NCSE visual-spatial	$r_{(58)} = -0.22$	$r_{(34)} = -0.19$	$r_{(24)} = -0.30$	$r_{(32)} = -0.28$
NCSE memory	$r_{(58)} = -0.36^{***}$	$r_{(34)} = -0.30$	$r_{(24)} = -0.46^*$	$r_{(32)} = -0.48^{***}$
NCSE calculation	$r_{(58)} = 0.03$	$r_{(34)} = -0.11$	$r_{(24)} = 0.24$	$r_{(32)} = -0.02$
NCSE similarities	$r_{(57)} = -0.27^*$	$r_{(34)} = -0.20$	$r_{(23)} = -0.41^*$	$r_{(31)} = -0.54^{***}$
NCSE judgment	$r_{(56)} = -0.19$	$r_{(33)} = -0.24$	$r_{(23)} = -0.10$	$r_{(30)} = -0.37^*$
RAVLT total	$r_{(55)} = -0.22$	$r_{(32)} = -0.42^*$	$r_{(23)} = 0.16$	$r_{(30)} = -0.47^{**}$
RAVLT recall	$r_{(48)} = -0.13$	$r_{(26)} = -0.26$	$r_{(23)} = 0.04$	$r_{(24)} = -0.22$
TMT-A time	$r_{(54)} = 0.28^*$	$r_{(32)} = 0.36^*$	$r_{(22)} = 0.06$	$r_{(28)} = 0.52^{***}$
TMT-B time	$r_{(36)} = 0.07$	$r_{(30)} = 0.02$	$r_{(22)} = 0.13$	$r_{(26)} = 0.19$

Note: numbers in parentheses are the sample sizes for each correlation.

* $p < 0.05$;

** $p < 0.01$;

*** $p < 0.005$.

BALs at the time of TBI would perform more poorly on a number of neuropsychological tasks. For the total sample, orientation, naming, verbal memory and similarities scores from the NCSE were all significantly and inversely correlated with BAL. Time to complete Trailmaking Test A was also positively correlated with BAL.

Restricting the analyses only to those with a positive BAL, a similar pattern of correlations was obtained, though the magnitude of the correlation coefficients were generally stronger (see table 1). In this subgroup analysis, attention span and judgment scores on the NCSE as well as the RAVLT total score were added to the list of variables significantly correlated with BAL.

Since no potential confounding factors were significantly correlated with BAL in the preliminary analyses, the authors did not control for these factors in the analyses reported above. However, on an exploratory basis they computed the same correlations in the subsample with positive BALs while controlling for the effects of age, sex, years of education, TBI severity and number of days between injury and testing. As expected, most of the significant correlational relationships were maintained (i.e. attention, verbal memory, similarities, Rey Total and Trailmaking A).

Next the authors examined the hypothesis that the relationship between BAL and neuropsychological performance would diminish with time. To do this participants tested ≤ 30 days post-injury were compared to those tested > 30 days post-injury. As table 1 illustrates, findings were consistent with predictions. The correlations between BAL and neuropsychological test scores was generally stronger and more reliable for those participants tested ≤ 30 days post-injury. After 30 days post-injury, only performance on the NCSE memory and similarities tasks were significantly correlated with BAL. On the other hand, the correlations between BAL and similarities and memory tended to be stronger in the group tested 31–60 days after TBI.

Discussion

These data support the hypothesis that alcohol intoxication influences neuropsychological test performance within the first 1–2 months after TBI. Simple bivariate correlations show that higher blood alcohol levels were predictive of poorer performance on a number of neuropsychological screening measures. Functions affected include orientation, concentration/mental speed, naming ability, verbal memory and verbal abstract reasoning. When the effects of potential confounding variables such as age, sex, years of education and brain injury severity were controlled, the relationship between BAL and test performance remained largely unchanged. When correlations were examined from data obtained 1 month versus 2 months post-injury, the relationship between BAL and neuropsychological screening measures tended to be diminished. Exceptions to this rule included the similarities and memory subtests of the NCSE which may somehow be more persistently influenced by alcohol.

There are at least two mechanisms by which blood alcohol level at the time of injury may influence neuropsychological performance after TBI. First, significant blood alcohol at the time of injury may influence the magnitude of traumatic brain injury via mechanisms such as haemodynamic and respiratory depression, altered homeostasis due to increased blood clotting time and blood-brain barrier impairment [24]. The data on this issue are complex, involve both animal and human studies, and seem to point to both neuroprotective and neurotoxic roles of alcohol in the context of acute brain injury [8, 24]. A review of these findings is beyond the scope of this paper, but it is the authors' judgment that the research in this area is not developed enough to suggest specific predictions about the impact of these factors on neuropsychological recovery in clinical populations.

The other mechanisms by which BAL may seem to influence cognitive outcomes is by acting as a proxy for chronic alcohol abuse or dependence. In this study data on recent alcohol consumption or alcohol problems are not available. However, the authors speculate that high blood alcohol levels at the time of injury are not usually one-time events, but rather an indication of high levels of alcohol use during the weeks and months prior to TBI. Indeed, a large case study at the Harborview Medical Center has found that 75% of trauma survivors who were injured while intoxicated also admitted a pattern of more chronic alcohol abuse [25].

If it is assumed for the moment that a positive BAL at the time of injury frequently reflects more chronic alcohol use, then the more well known neuropsychological effects of alcoholism can be invoked to explain differences in neuropsychological functioning after TBI. Studies of newly abstinent alcoholics show three general phases of neuropsychological recovery [18]. During the first 2 weeks of detoxification, widespread cognitive impairments are common including, distractibility, mild confusion, irritability, impaired attention and concentration, slower reaction times, poor verbal and nonverbal memory, impaired abstract reasoning, cognitive inflexibility and visual-spatial impairments. Such impairments are found among non-alcoholic drinkers as well as among people with alcoholism. Intermediate-term cognitive impairments fall within 2 weeks to 2 months after abstinence begins. This phase is characterized by impaired nonverbal abstract reasoning in the presence of intact verbal intelligence, as well as continued problems with complex attention, motor speed, verbal and nonverbal memory and more

challenging forms of verbal reasoning [18]. Residual impairments at this stage may be related to the severity of alcoholism. Finally, the long term phase of neuropsychological recovery lasts from 2 months to more than 2 years after drinking has stopped [26]. Age, drinking history and interim drinking seem to affect recovery in this phase [18, 26]. Residual neuropsychological impairments are those that are more classically associated with the organic effects of alcoholism and include impairments in the areas of nonverbal memory, nonverbal abstract reasoning, cognitive flexibility and visual-spatial abilities [18].

The limited literature on chronic alcohol problems and TBI generally supports the idea that alcoholics have poorer outcomes. Persons with more chronic alcohol problems have been found to be at greater risk of developing mass lesions as a consequence of TBI, were more likely to die and had poorer Glasgow Outcome Scale scores [15]. The authors suggested that these outcomes may be due to the brain atrophy, greater susceptibility to tearing in bringing vessels, platelet dysfunction and reduced levels of clotting factors associated with chronic alcoholism. In a large longitudinal study of persons with TBI with and without chronic alcohol problems, those with pre-existing chronic alcohol problems had poorer neuropsychological outcomes at 1 month and 1 year after TBI [27]. These investigators noted that difficulty of untangling the effects of confounding factors (e.g. low education) from the effects of chronic alcohol problems alone. In the present study, the authors note the apparent attenuation of BAL-related effects as time since injury increases. This pattern is consistent with the idea that the subjects are recovering from the intermediate neuropsychological effects of alcohol abuse as previously described as well as from TBI.

Several limitations of the study should be noted before concluding. Subjects were non-consecutive admissions to the authors' in-patient rehabilitation unit and, as an archival study, test data were obtained at varying times post-injury based on clinical need. Although patients with TBI are tested on a routine basis in this institution, it is likely that those with more mild injuries as well as those with very severe injuries are underrepresented. While these factor limit the generalizability of this study, previous studies relating BAL to neuropsychological outcomes have been restricted only to patients with severe TBI [11, 12]. In prior studies, the severity of TBI may have masked any alcohol-related impact on neuropsychological functioning. Future research should examine whether the relative impact of acute or chronic alcohol factors is greater among persons with milder injuries.

Unsuspectedly, there was no significant relationship between TBI severity and neuropsychological test performance. Clinical testing conditions may explain this lack of relationship, in that those with more severe injuries were likely tested later. These data show that time from injury to neuropsychological screening did increase as a function of TBI severity ($r = 0.29$; $p < 0.05$). This association probably attenuated the effects of brain injury severity on measured abilities. The study design would have been strengthened if the same subjects were tested twice. However, repeat testing as not part of the routine clinical examination procedures in this study.

Another limitation is that these data lacked information on recent alcohol consumption and lifetime alcohol problems to complement the data on admission BAL. Consequently the effects of acute intoxication cannot be separated from more chronic alcohol problems. Future studies should collect all three types of

data and examine the differential impact on each drinking factor on post-TBI recovery.

Finally, these results are based on brief neuropsychological screening tests rather than a comprehensive neuropsychological test battery. The screen and metric approach has been criticized as resulting in too many false negative results when screening for organic impairment among psychiatric patients. This issue was thought to be of particular concern for the construction scale [20]. Therefore, both the screening and metric component of the construction scale were administered as recommended [20]. The NCSE has demonstrated low specificity and low positive predictive value when used to screen for cognitive deficits in a psychiatric population where the base rate of organic impairment is low [28]. These indexes of clinical utility have not yet been examined among TBI survivors. On the other hand, in a mixed clinical population, most of the NCSE subtests have been found to have strong agreement with comparison WAIS-R subscales, the Boston Naming Test and the Hopkins Verbal Learning Test [29]. The NCSE has also been found to be sensitive to the cognitive effects of stroke and predictive rehabilitation outcomes such as length of stay, discharge Functional Independence Measure scores and outpatient therapy hours [30, 31]. Taken together, the NCSE seems to provide a reasonable estimate of cognitive impairments. Its limitations, primarily low sensitivity, would tend to reduce the likelihood of finding significant correlations with blood alcohol.

In conclusion, this study supports the hypothesis that higher admission blood alcohol levels after TBI predict lower scores on early measures of cognitive impairment. Based on the larger neuropsychological literature on the topic, as well as research on the neuropsychology of alcoholism, blood alcohol at the time of injury may have a direct effect on cognitive function or may be a proxy for the effects of more chronic alcohol use or abuse. The clinical implication is that early cognitive assessment after TBI may be influenced by alcohol related factors. Clinicians should routinely assess recent alcohol use and screen for a history of abuse or dependence. Additionally, clinicians should be cautious attributing early neuropsychological impairments only to TBI severity when there is evidence that acute or chronic alcohol factors are involved.

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