ORIGINAL ARTICLE

Association Between Alcohol and Mortality in Patients With Severe Traumatic Head Injury

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Hypothesis: Admission blood alcohol concentration (BAC) is associated with in-hospital death in patients with severe brain injury from blunt head trauma.

Design: Retrospective cohort study.

Setting: Academic level I trauma center in Toronto, Ontario.

Patients: Using trauma registry data, between January 1, 1988, and December 31, 2003, we identified 1158 consecutive patients with severe brain injury from blunt head trauma.

Intervention: There was no active intervention. The primary exposure of interest was the BAC at admission, stratified into the following 3 levels: 0, no BAC; 0 to less than 230 mg/dL, low to moderate BAC; and 230 mg/dL or greater, high BAC.

Main Outcome Measure: In-hospital death.

Results: In patients with severe brain injury, low to moderate BAC was associated with lower mortality than was no BAC (27.9% vs 36.3%; $P=.008$). High BAC was associated with higher mortality than was no BAC (44.7% vs 36.3%), although this was not statistically significant ($P=.10$). These associations were all statistically significant after adjusting for demographic data and injury factors using logistic regression analysis. The odds ratio for death was 0.76 (95% confidence interval, 0.52-0.98) for low to moderate BAC compared with no BAC. The odds ratio for death was 1.73 (95% confidence interval, 1.05-2.84) for high BAC compared with no BAC.

Conclusions: Low to moderate BAC may be beneficial in patients with severe brain injury from blunt head trauma. In contrast, high BAC seems to have a deleterious effect on in-hospital death in these patients, which may be related to its detrimental hemodynamic and physiologic effects. Alcohol-based fluids may have a role in the management of patients with severe brain injury after they have been well resuscitated.

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TRAUMATIC BRAIN INJURY (TBI) from blunt head trauma is a leading cause of death and disability in young adults.[1] The pathophysiology of TBI is such that not all neurologic damage occurs immediately but evolves with time.[2] This secondary brain injury results from ongoing ischemia and contributes to the overall mortality of TBI. Reducing secondary brain injury is the basis of the medical management of TBI.[2]

Alcohol is a major risk factor for injury; 30% to 50% of all patients hospitalized with trauma are intoxicated at the time of injury.[3] Even so, the effect of alcohol on TBI outcomes is still controversial. Many animal studies have reported negative effects of alcohol on TBI outcomes.[4-8] Other studies, however, have reported no effect on TBI outcomes[9] or have even suggested that alcohol may be neuroprotective.[10-17]

Similarly, clinical studies have reported conflicting results. Some of the controversy may be the result of heterogeneous study objectives. For example,

See Invited Critique at end of article

some investigators were challenging the anecdotal belief that alcohol relaxes victims, thereby resulting in less severe injuries. Therefore, they compared Injury Severity Scores (ISS) and mortality between drivers with blood alcohol concentration (BAC) greater than 0 and those with BAC of 0. Alcohol use was associated with more severe injuries and, therefore, higher mortality.[18-20] Waller et al[21] controlled for in-

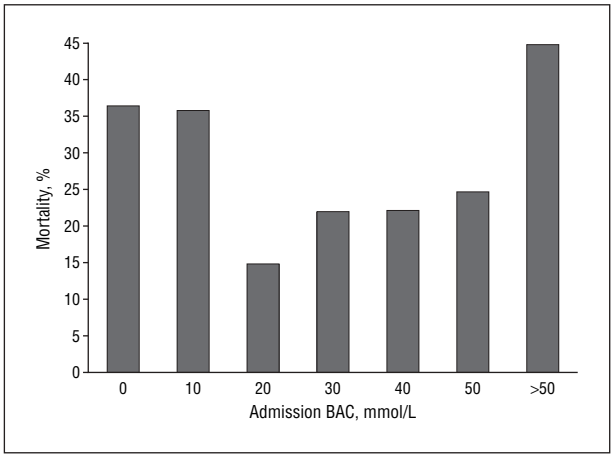

Figure. Case fatality vs increasing admission blood alcohol concentration (BAC) in patients with severe traumatic brain injury.

jury severity by using vehicle deformation as a surrogate and found that alcohol was still associated with an increased likelihood of death.

Other investigators focused on the public health implications of alcohol intoxication and looked for differences in outcome between legally intoxicated vs nonintoxicated patients (BAC <100 mg/dL or ≥100 mg/dL). In these studies, intoxication was associated with either worse outcome[22] or no difference in outcome.[23-27]

Only 2 studies have reported that alcohol was associated with improved trauma outcomes. Both sets of investigators found that an admission BAC greater than 0 was associated with reduced trauma-related mortality compared with BAC of 0, given injuries of equal severity.[28,29] Ward et al[28] concluded that alcohol may have some pharmacologic effect in the postinjury period that reduced mortality. The objective of our study was to determine whether admission BAC is associated with in-hospital death in patients with severe TBI after blunt head trauma, given injuries of equal severity.

METHODS

The trauma registry at Sunnybrook and Women's College Health Sciences Centre, an urban level I trauma center in Toronto, Ontario, was used to identify all trauma patients evaluated between January 1, 1988, and December 31, 2003 (16 years). Adult patients (age, 15-90 years) with a blunt mechanism of injury and who arrived directly from the scene of injury were selected for further analysis. Patients who were referred from other hospitals or who had sustained any penetrating injury or burns were excluded.

Patient demographic data, injury mechanism, ISS, abbreviated ISS (AIS), length of hospital stay (days), intensive care unit stay (days), total units of blood transfused, and in-hospital outcome (death or survival) were determined from our trauma registry. The ISS and AIS were calculated by trauma registry staff after discharge or death in each patient.

PATIENTS WITH SEVERE TRAUMATIC BRAIN INJURY: STUDY GROUP

Our study group of patients with severe TBI was selected by identifying all patients with an AIS score of 4 or 5 for the head and neck region.

PATIENTS WITH SEVERE TORSO INJURY: TRACER CONDITION

We identified severe torso injury, with no or mild head injury, as a tracer condition. This group included all patients with AIS-chest or AIS-abdomen scores of 4 or 5 who also had AIS–head and neck score of 3 or less.

INDEPENDENT VARIABLE: ADMISSION BAC

Our trauma registry includes the BAC in each patient at arrival at our trauma center. A histogram was constructed for mortality vs increasing BAC to look for a possible dose effect in patients with severe TBI (**Figure**). Patients were observed to have lower mortality when BAC was less than 230 mg/dL and higher mortality when BAC was 230 mg/dL or greater. Survival, therefore, was analyzed at 3 BAC levels: 0 mg/dL, no BAC; less than 230 mg/dL, low to moderate BAC; and 230 mg/dL or greater, high BAC.

OUTCOME MEASURE

The primary outcome was defined as in-hospital death in the study group of patients with severe TBI. We also analyzed mortality in patients with the tracer condition of severe torso injury to determine whether there was a lack of difference. No difference would be expected, given the hypothesized neuroprotective effects of alcohol.

STATISTICAL ANALYSIS

We compared in-hospital mortality in patients with severe TBI whose blood tests at admission demonstrated no, low to moderate, and high BAC, using the χ^2 test and, where appropriate, the Fisher exact test. In addition, we constructed a multivariate model by subjecting baseline demographic and injury factors to stepwise logistic regression analysis and, thereby, obtained an adjusted comparison of risk of death in patients whose blood tests at admission demonstrated no, low, and high BAC. We checked for main effects for all covariates and then analyzed for interactions. The same analysis was performed in patients with the tracer condition of severe torso injury, with mild or no head injury, to determine whether there was a lack of difference.

All *P* values are 2-tailed and adjusted for multiple comparisons using the Bonferroni method, where applicable. Means and odds ratios were reported with 95% confidence intervals, and all data were analyzed using SAS software (version 8.02; SAS Institute Inc, Cary, NC).

RESULTS

During the 16-year study period, 12 105 patients were evaluated at our trauma service. Of these, 4099 patients were adults (age, 15-90 years) with blunt trauma who arrived directly from the scene of injury. The BAC from the initial trauma room blood tests was reported as unknown in 424 patients (10.3%), and they were excluded from subsequent analysis. Thus, 3675 patients met the inclusion criteria and had a known BAC.

Patients with unknown BAC were older than those with known BAC (mean age, 44.4 vs 41.1 years) and had a shorter length of hospital stay (15.8 vs 19.6 days). Also, those with unknown BAC had higher mortality than those with known BAC (31.4% vs 14.2%). A higher percentage of deaths occurred in the trauma room in patients

Table 1. Baseline Characteristics in Patients With Severe Traumatic Brain Injury by Admission Alcohol Status*

Characteristic	Blood Alcohol Concentration, mg/dL		
	0 (n = 740)	<230 (n = 315)	≥230 (n = 103)
Sex, %			
Men	68.4	76.2	89.3
Women	31.6	23.8	10.7
Age, y	44.3 (42.8-45.9)	38.3 (36.1-40.4)	42.1 (39.3-44.9)
Injury Severity Score	37.0 (36.1-37.9)	37.3 (35.8-38.7)	34.9 (32.7-37.0)
PRBC, No. of units transfused	4.7 (4.0-5.4)	5.1 (4.1-6.1)	2.9 (1.7-4.1)
Length of stay, d			
Hospital	30.4 (26.3-34.5)	32.7 (27.0-38.3)	26.6 (16.9-36.3)
Intensive care unit	4.9 (4.4-5.5)	5.2 (4.4-6.0)	4.6 (3.1-6.1)

Abbreviation: PRBC, packed red blood cells.
*Data are given as mean (95% confidence interval) unless otherwise indicated.

Table 2. Baseline Characteristics of Patients With Severe Torso Injury by Admission Alcohol Status*

Characteristic	Blood Alcohol Concentration, mg/dL		
	0 (n = 343)	<230 (n = 162)	≥230 (n = 23)
Sex, %			
Men	62.4	74.7	78.3
Women	37.6	25.3	21.7
Age, y	44.7 (42.7-46.7)	41.2 (38.3-44.1)	36.9 (32.6-41.2)
Injury Severity Score	32.8 (31.8-33.8)	31.8 (30.4-33.2)	31.9 (28.2-35.5)
PRBC, No. of units transfused	9.4 (7.7-11.1)	6.8 (4.9-8.6)	9.2 (4.7-13.7)
Length of stay, d			
Hospital	24.1 (21.0-27.1)	20.8 (17.4-24.2)	17.3 (11.6-23.0)
Intensive care unit	6.1 (4.8-7.4)	4.2 (2.6-5.8)	4.0 (1.2-6.9)

Abbreviation: PRBC, packed red blood cells.
*Data are given as mean (95% confidence interval) unless otherwise indicated.

with unknown BAC compared with those with known BAC (48.5% vs 12.6%).

Of the 3675 patients who met inclusion criteria and had a known BAC, 1158 patients were identified as having severe TBI and 528 patients were classified as having severe torso injuries with only mild or no head injuries. Baseline characteristics of the study group are given in **Table 1**. Admission blood tests in the 1158 patients demonstrated that 740 (63.9%) had no BAC, 315 (27.2%) had low to moderate BAC, and 103 (8.9%) had high BAC. Patients with BAC greater than 0 were more likely male and younger compared with patients with BAC of 0. Male patients were more likely to have a higher BAC compared with female patients. No obvious differences were apparent in ISS, blood transfusion requirements, or length of hospital stay.

Similarly, in the group with severe torso injury, patients with a BAC greater than 0 were more likely to be male and younger compared with patients with a BAC of 0. Otherwise, ISS, transfusion requirements, and length of hospital stay were similar for patients with a BAC greater than 0 and a BAC of 0. Baseline characteristics in patients with the tracer condition are given in **Table 2**.

In patients with severe TBI, the overall risk of dying in the hospital was significantly lower in patients with a low to moderate BAC compared with no BAC (27.9% vs 36.3%; $P=.008$). Patients with a high BAC had higher mortality compared with patients with no BAC; this finding bordered on being statistically significant (44.7% vs 36.3%; $P=.10$). There was also a trend suggesting that patients with a high BAC died earlier during their hospital stay than did patients with no BAC (**Table 3**).

To gauge the robustness of this finding, we analyzed mortality in patients with the tracer condition. As expected, there were no significant differences in mortality between patients with a low BAC compared with no BAC (15.4% vs 14.9%; $P=.90$) and between patients with a high BAC compared with no BAC (13.0% vs 14.9%; $P=1.0$). There still was a trend, however, suggesting that patients with a high BAC died earlier in their hospital stay compared with patients with no BAC (**Table 4**).

Table 3. Time to Death in Patients With Severe Traumatic Brain Injury

Variable	Blood Alcohol Concentration, mg/dL		
	0	<230	≥230
No. of patients	740	315	103
No. of deaths	269	88	46
Time to death, d*	9.8 (3.4 – 16.1)	7.3 (2.9 – 11.7)	4.8 (2.3-8.3)

*Data are given as mean (95% confidence interval).

Table 4. Time to Death in Patients With Severe Torso Injury

Variable	Blood Alcohol Concentration, mg/dL		
	0	<50	≥50
No. of patients	343	162	23
No. of deaths	51	25	3
Time to death, d*	8.3 (1.8-14.9)	6.5 (1.1 – 18.7)	1.0

*Data are given as mean (95% confidence interval).

We used logistic regression to create a multivariate model to control for underlying confounders. Variables were screened at $P=.25$ and were included in the final model if $P<.05$. Sex; age; ISS; units of transfused red blood cells; AIS-chest, AIS-abdomen, and AIS–head and neck; year of admission; and mechanism of injury (occupants of motorized vehicle vs other) were all used in the model.

Except for sex, year of admission, and AIS-chest and AIS-abdomen, all of these variables had main effects that were found to be significant in patients with severe TBI (**Table 5**). Admission BAC was also found to be an independent predictor of death. A low to moderate BAC was associated with better survival than no BAC (odds ratio, 0.76; 95% confidence interval, 0.52-0.98), and a high BAC was associated with worse survival compared with no BAC (odds ratio, 1.73; 95% confidence interval,

Table 5. Independent Predictors of Death*

Variable	Study Group	Control Group
Low BAC vs no BAC	0.76 (0.52-0.98)	1.6 (0.82-3.03)
High BAC vs no BAC	1.73 (1.05-2.84)	2.02 (0.48-8.51)
Age	1.02 (1.01-1.03)	1.06 (1.05-1.08)
Injury Severity Score	1.03 (1.00-1.05)	1.06 (1.01-1.11)
PRBC, No. of units transfused	1.04 (1.02-1.06)	1.04 (1.02-1.06)
Mechanism of injury, motor vehicle crash vs other	0.54 (0.40-0.74)	0.43 (0.24-0.77)
AIS-chest	0.98 (0.87-1.11)	1.12 (0.86-1.47)
AIS-abdomen	1.06 (0.92-1.2)	1.33 (1.03-1.70)
AIS-head/neck	10.8 (6.5-17.9)	0.91 (0.70-1.19)
Sex, M vs F	0.85 (0.62-1.18)	0.75 (0.52-1.08)

Abbreviations: AIS, Abbreviated Injury Scale Score; BAC, blood alcohol concentration; PRBC, packed red blood cells.
*Data are given as odds ratio (95% confidence interval).

1.05-2.84; Table 5). No interactions were found to have significant effects and were excluded from the final model. The final model was found to have good discriminatory power (receiver operating characteristic curve, 0.82) and adequate calibration ($P=.15$, Hosmer-Lemeshow goodness-of-fit test).

We similarly constructed a multivariate model for our tracer condition using the same baseline characteristics as for our study group. Age, ISS, AIS-abdomen, units of blood transfused, and mechanism of injury were also found to have main effects that were significant predictors of death. Sex, date of admission, AIS-chest, and AIS–head and neck were not significant predictors. Also, as expected, low and high admission BAC were not significant predictors for death for our tracer condition (odds ratio, 1.6; 95% confidence interval, 0.82-3.03, and odds ratio, 2.02; 95% confidence interval, 0.48-8.51, respectively; Table 5). No interactions were found to be significant. The final model had good discriminatory power (receiver operating characteristic curve, 0.86) and adequate calibration ($P=.60$, Hosmer-Lemeshow goodness-of-fit test).

COMMENT

Alcohol use has been well established as the most important personal risk factor for fatal injuries, contributing to approximately one third of all deaths from injury.[30] Intoxication increases the risk of all types of injury, including motor vehicle collisions, falls, assaults, and self-inflicted injuries, by impairing motor skills, reaction time, and judgment.[31,32] Furthermore, acute alcohol use has been shown to be associated with more severe injuries in drivers,[18-20] in part because intoxicated victims were not using safety devices such as seat belts or helmets at the time of injury.[33,34]

Although there is no disagreement about the negative effects of alcohol on the risk of injury and the likelihood of sustaining more severe injuries, the effects of alcohol on traumatic brain injury outcomes in the postinjury period is still controversial. Some investigators believe that alcohol is deleterious in patients with TBI. Studies have shown that a high BAC can cause hypotension and apnea in animal models of TBI and unresuscitated hemorrhagic shock.[4-6] Also, in canine models of TBI and shock, investigators have found that alcohol can increase susceptibility to hemorrhagic shock by eliminating the host homeostatic compensatory mechanisms of preserving cerebral oxygenation and perfusion.[35,36] This failure of homeostasis may account for the observation by Luna et al[37] that intoxicated motorcyclists with critical head injury had twice the mortality of nonintoxicated motorcyclists, even after controlling for helmet use and ISS.

In contrast, Fabian and Proctor[9] found that the physiologic consequences of alcohol intoxication do not affect TBI outcomes in a swine model if adequate resuscitation takes place. Similarly, Huth et al[24] evaluated injured automobile drivers who were aggressively resuscitated in a trauma center and found that alcohol did not affect hospital course or outcome.

Basic science investigators are also beginning to elucidate potential beneficial pharmacologic effects of alcohol if administered after injury has occurred. Specifically, experimental evidence shows that alcohol in low to moderate doses (<240 mg/dL) can reduce secondary brain injury.[10-13] These neuroprotective effects seem to be mediated by alcohol's inhibition of *N*-methyl-D-aspartate receptor-mediated excitotoxicity. Mitigation of excitotoxicity can reduce secondary brain injury by attenuating the tendency of injured neurons to release neurotransmitters that cause further injury and death.[14] This effect is analogous to an experimental finding in stroke research in which alcohol and caffeine were shown to reduce the volume of cerebral infarction.[15-17] These neuroprotective effects were absent, however, at high doses of alcohol.

We studied adult patients with trauma who had severe TBI from blunt head injury. We found that a low to moderate BAC at admission was associated with improved mortality when compared with no BAC. Patients with TBI with a high BAC had higher mortality compared with patients with no BAC. Furthermore, this effect was specific to head injury. Alcohol was not significantly associated with mortality in patients with our tracer condition of severe torso injury with no or mild head injuries.

Our findings are consistent with both studies that suggest potential neuroprotective effects of alcohol in low to moderate concentrations and studies that suggest that alcohol detrimentally affects the host homeostatic compensatory response to shock. Our study was performed within a large, well-organized, urban trauma system. Therefore, the negative effects of alcohol may be overshadowed by its neuroprotective effects, especially in the context of short time to arrival at the hospital and aggressive resuscitation by emergency medical service staff before arrival at the hospital. At low and moderate doses (<240 mg/dL), alcohol was shown in animal studies to be neuroprotective[10-13]; in our study, BAC less than 230 mg/dL was likewise associated with a survival benefit, compared to BAC of 0.

Increased mortality was observed in patients with severe TBI with a high BAC. It is presumed that, at such high doses, alcohol reduced the compensatory response to shock despite resuscitation. At high doses (>240 mg/dL), alcohol was shown in animal studies to be deleterious to neurologic outcomes.[10-13] In our study, a BAC of 230 mg/dL or greater was shown to be associated with

higher mortality than a BAC of 0. Also, there was a trend in both our study group and in our tracer group to suggest that patients with a high BAC were more likely to die earlier after trauma compared with patients with no BAC. This finding would be consistent with the experimental finding that high doses of alcohol can reduce the host ability to compensate for hemorrhagic shock.[35,36]

There are major sociologic implications from implying that intoxicated patients with severe TBI have better outcomes than nonintoxicated patients. We stress that our study only examined the role of alcohol on outcome in the postinjury phase because we examined only in-hospital deaths. Fifty percent of all trauma-related deaths occur in the prehospital setting.[38,39] Alcohol-related deaths tend to be overrepresented in this subgroup of patients who die in the prehospital setting[40] because alcohol use increases the likelihood of severe injury[18,19] and impedes the body's ability to compensate for shock.[35] Overall, people are still at much greater risk of dying if they drive while intoxicated. What our study implies is that there may be a role for an alcohol-based resuscitation fluid in improving outcomes in adequately resuscitated patients with severe head injury.

LIMITATIONS

Our study is not a randomized controlled study, and, therefore, the results may be exaggerated by confounders and biases. Approximately 10% of patients who met inclusion criteria had an unknown BAC, and these patients were systematically different from those with a known BAC. These patients were older and were much more likely to die in the trauma room than were those with a known BAC. Like patients who died in the prehospital setting, patients with an unknown BAC were probably more likely to have a BAC greater than 0.[40] These patients were more likely to die in the trauma room because they were more severely injured and possibly because alcohol had detrimental effects on their homeostatic compensatory mechanisms.

For patients with a known BAC, we attempted to adjust for known predictors of trauma-related death, such as age,[41] sex,[42] and ISS,[43] in our multivariate model. We also adjusted for mechanism of injury in our final model. Alcohol may affect mortality in occupants of motorized vehicles in many ways apart from any pharmacologic effect in the postinjury period. Intoxicated drivers tend to drive faster and more recklessly[31] than do nonintoxicated drivers, and do not use safety devices such as seat belts as often.[33]

We also adjusted for the year of admission. Our study included patients admitted during 16 years. Trauma care has improved during this time. Improvements in care may have preferentially benefited nondrinking drivers because increased public health efforts have resulted in fewer patients who were intoxicated while driving in more recent years than at the beginning of our study.[44]

USE OF AIS TO DEFINE SEVERE TBI

We used AIS–head and neck score greater than 3 as our operational definition of severe TBI to avoid the selection bias of using a physiologic score (ie, Glasgow Coma Scale), which misclassifies intoxicated patients with mild TBI as having severe TBI.[45] Using AIS–head and neck, however, has important limitations. One major problem is that AIS–head and neck is also dependent, though to a much smaller extent than the Glasgow Coma Scale, on the duration of unconsciousness[46] and, therefore, does not completely eliminate the bias of using a physiologic score.

We attempted to estimate the effect of this bias on our study group by examining length of hospital stay and duration of intensive care unit stay across the BAC groups and found that they were almost identical. If alcohol intoxication resulted in a significant bias, one would assume that the length of hospital stay and intensive care unit stay would be significantly shorter in the groups with BAC greater than 0.

Another problem with using AIS–head and neck is that it is not specific for brain injury.[46] Cervical spine fractures and skull or facial fractures would also be coded within the framework of AIS–head and neck. This results in a nonspecific measure of severe TBI because patients with mild head injury with associated injuries would be misclassified as having severe TBI. However, skull or facial fractures and cervical spine fractures are often associated with severe TBI[47,48] and are likely to be equally distributed across BAC levels.

Using AIS–head and neck will likely result in overestimation of the number of patients with severe TBI. In our study, almost one third of the patients had severe TBI, using AIS–head and neck score greater than 3 as our operational definition. This is substantially higher than Centers for Disease Control and Prevention estimates for the United States.[49] However, this discrepancy is only partially due to our use of AIS–head and neck. Another reason is that at our institution isolated mild to moderate head injuries are referred through the emergency department directly to the neurosurgical service and are not included in our trauma registry. Our trauma registry, therefore, preferentially includes patients with severe TBI.

USE OF UNITS OF PACKED RED BLOOD CELLS AS A SURROGATE FOR HYPOTENSION

Another limitation of our study was the use of units of transfused red blood cells as a surrogate for hypotension as a covariate in our final model. Even one episode of hypotension has been shown to dramatically increase mortality in patients with severe brain injury.[50,51] There are numerous situations, however, where blood transfusion requirements poorly correlate with hypotension. False-positive findings occur when patients receive transfusions based solely on hemoglobin-based transfusion triggers. In the past, a common transfusion trigger was a hemoglobin level less than 100 g/dL, irrespective of hemodynamic status.[52] Trauma patients treated using these protocols would receive blood transfusions despite having a normal hemodynamic status. False-negative findings occur in patients who have episodes of hypotension but no requirement for blood transfusion. False-negative findings would occur, therefore, if patients had class II shock, as defined by the American College of Surgeons in the Advanced Trauma Life Support Course for Physicians[53]; the hypotension would re-

spond to crystalloid resuscitation alone and they would have no requirement for blood transfusion.

The major effect of using transfusion requirements as a surrogate for hypotension comes from the false-negative findings. Alcohol is known to have hemodynamic effects, and can cause hypotension.[45] Alcohol-induced hypotension is treatable with fluid resuscitation only and would not require blood transfusion. As a result, transfusion requirements would underestimate the degree of hypotension in patients with BAC greater than 0.

False-positive findings would more likely affect estimates of hypotension across time, not across BAC groups. Trauma patients early in the study would more likely receive blood despite having a normal hemodynamic status, because of more liberal transfusion practices, compared with patients later in the study. There should be no significant differences in transfusion practices across BAC groups.

STARTING TIME BIAS

Starting time bias[54] is associated with using admission BAC as our independent variable. Alcohol is metabolized quickly. Therefore, patients with a BAC greater than 0 at the time of injury may be misclassified as having a BAC of 0 at admission because the alcohol in their blood may be eliminated via metabolism. We minimized this bias by only including patients who arrived directly from the scene of injury. On average, patients arrived at our institution within 1 hour after injury (data not shown).

COMORBIDITIES

We did not adjust for comorbidities in our multivariate models because these are not reliably coded in our trauma registry. Persons who are alcohol dependent tend to have underlying chronic diseases,[55] and the injured patient with a BAC greater than 0 is more likely to have chronic alcoholism than is the nondrinker.[56] Milzman et al[57] demonstrated that preexisting disease is a strong predictor of trauma-related mortality. They reported 25% mortality in patients with 3 preexisting diseases compared with 3.2% in healthy persons.

This bias likely results in an underestimate of the beneficial effects of a low to moderate BAC compared with no BAC because those patients with BAC greater than 0 were more likely to have comorbidities. However, this bias likely results in an overestimate of the deleterious effects of a high BAC compared with no BAC. The observed increase in mortality may be accounted for by the comorbidities in the high BAC group because these patients are more likely to have chronic alcoholism.

TIME OF INJURY

Time of injury also biases our results. Alcohol-associated trauma tends to occur during the night and on weekends.[58] Investigators have observed an association between after-hours and weekend hospitalization, and increased mortality.[59] It is presumed that this effect is secondary to the presence of fewer staff, the absence of more experienced staff, and larger relative workloads during these times.

CONCLUSIONS

We conducted a retrospective cohort study in which we examined the effect of alcohol on mortality in patients with severe TBI. Compared with no BAC, at admission, a low BAC was associated with lower mortality and a high BAC was associated with higher mortality. Alcohol may have neuroprotective effects at low and moderate doses; however, these effects are likely overshadowed at higher doses by its hemodynamic and physiologic effects. There may be a role for alcohol-based resuscitation fluids in well-resuscitated patients with severe traumatic head injury. Prospective studies are needed to confirm this result.

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