EFFECT OF ACUTE ALCOHOL INTOXICATION ON INJURY TOLERANCE AND OUTCOME

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In 1984, NASS data indicates that alcohol was involved in at least 43% of fatalities and 29% of non-fatal serious injuries resulting from U.S. motor vehicle accidents. An earlier clinical study suggests over 50% of non-fatal injuries involve alcohol. Alcohol intoxication has long been known to increase the probability of a crash by affecting driver judgment and performance. More recent traffic accident data indicate that acute intoxication may also result in greater likelihood of serious injury for otherwise comparable accident severity. Clinical data further indicates that acute intoxication of victims of car crashes confounds the attending physician’s diagnosis of injury severity and complicates acute medical management.

This paper analyses these previous studies and presents data from our laboratories on the interaction of alcohol with cardiac or spinal cord contusion. Our data show that acute intoxication significantly increases the risk of immediate or delayed fatality after cardiac injury, and that post-contusion spinal cord pathology and functional recovery are worsened by acute intoxication. These findings together with data from previous studies in other experimental models of cardiac and central nervous system injury suggest that there is increased severity of at least some types of injury resulting from motor vehicle accidents through direct physiologic effects of acute intoxication.

MYOCARDIAL CONTUSION

Myocardial contusion is estimated to occur in about 18% of serious trauma victims, most commonly resulting from blunt chest impact, and frequently remains undiagnosed, even when cardiac performance is altered. Previous studies found that acute intoxication prior to trauma increased the mortality rate for constant severity closed chest injury. With our animal model we used a controlled, direct cardiac contusion technique with more reproducible outcome at a severity normally resulting in only transient arrhythmia and recovery of normal cardiac performance by one hour post-trauma. We found that rapid intravenous administration of alcohol (blood alcohol level=65mg/dl) prior to injury resulted in 66% mortality during the first hour after injury. Further studies used oral administration of alcohol, and defined a dose-response relation (Fig. 1) between level of pre-injury intoxication and probability of fatality. Fatality resulted from disruption of cardiac
excitation-contraction coupling, causing deterioration of blood pressure and loss of effective cardiac output within 20 minutes.

With less severe contusion, acute intoxication increased cardiac irritability for several hours after injury, resulting in more frequent and longer duration runs of pre-ventricular contractions compared to non-intoxicated control response. Cardiac irritability may have serious consequences in the multiply injured victim or in patients with premorbid heart disease. The cellular and subcellular mechanisms for both decoupling and myocardial irritability after cardiac contusion injury remain uncertain, though we hypothesize that transmembrane calcium flux and distribution and alterations in myocardial energy metabolism are involved.

Figure 1: Percent mortality for standardized cardiac injury.

Figure 2: Local iron content as indicator of hemorrhage.

CENTRAL NERVOUS SYSTEM

Mechanical injury to the brain or spinal cord initiates a progression of pathological changes in neural and vascular elements responsible for much of the permanent loss of function. We used a reproducible animal model of moderate severity spinal cord injury to determine the effects of acute intoxication (100mg/dl) on injury physiology and outcome, and found that functional recovery during the three hour monitoring period was blocked in the alcohol pre-treated animals, while sober controls showed the expected partial recovery. Further, the extent of hemorrhage within the cord, as measured by tissue iron content reflecting extravasation of red blood cells, was significantly increased in the alcohol-intoxicated group (Fig. 2), which may contribute to greater anatomic damage.

We have also conducted in vitro studies of spinal cord conduction following
mechanical compression. Conduction in the isolated spinal cord recovers to levels comparable to control recordings following 50% compression, probably because local hemorrhage and ischemia are prevented in the isolated system. However, when ethanol is added to the bathing medium for a final concentration of 100mg/dl (to simulate a blood alcohol concentration of 100mg/dl) we found that axonal conduction did not recover to control levels after 50% compression. Addition of ethanol to the bath without compression had minimal effects on conduction, and membranes could be made resistant to the effects of acute exposure through repeated intoxication in vivo for two weeks prior to the experiment. These results suggest that even low levels of alcohol which do not normally affect conduction have membrane effects which impair recovery of conduction after mechanical injury to nerve axons.

Other investigators have also shown that intoxication coupled with standardized experimental spinal cord injury worsens functional outcome. Injury severity normally resulting in only moderate spasticity caused permanent paralysis when delivered in conjunction with elevated blood alcohol levels. The extent of spinal cord necrosis was increased for moderate severities of trauma, indicating a reduction in threshold for spinal cord injury with serious functional consequences. Our data suggest that the mechanisms for alcohol interaction with CNS injury response leading to increased pathology and impaired recovery involve effects on both vascular and neural elements. We hypothesize that alcohol reduces membrane stability directly, thus increasing local hemorrhage, impairing restoration of electrolyte gradients necessary for recovery of axonal conduction and rendering membranes more accessible to proteolytic or peroxidative degradation.

PROJECTED EFFECTS OF ALCOHOL INTOXICATION ON TRAUMATIC INJURY

Experimental and field accident studies support the hypothesis that alcohol increases injury severity and worsens outcome for myocardial and central nervous system injury. Since the physiologic response to injury is a key contributor to functional outcome in myocardial and central nervous system injury, understanding the contribution of acute intoxication to injury physiology may offer an opportunity to significantly reduce mortality and morbidity associated with such injuries. More sensitive and quantitative evaluation procedures are needed for clinical studies than are provided by the current anatomic and fatality measures.
The contribution of acute alcohol intoxication to injury mortality and morbidity can be estimated by combining risk of injury data from the University of North Carolina with alcohol involvement data from the Fatal Accident Reporting System (FARS) and National Accident Sampling System (NASS).\textsuperscript{1,4} Our calculations indicate that even without allowance for the expected reduction in accident rates, fatalities could be reduced by 32\% and serious injury by 12\% if alcohol intoxication while driving automobiles were eliminated. This represents a substantial possibility for reduction in occupant injury and fatality resulting from traffic accidents.

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