INTRODUCTION

Alcohol is clearly a major factor in serious and fatal motor vehicle crashes (1). The deterioration in performance as blood alcohol level increases has been demonstrated in laboratory studies (1) as well as epidemiological data showing the rapid increase in risk of crash as blood alcohol level increases (2-3). These relationships have provided the scientific basis for major anti-drunk driving legislation and public education programs. Indeed, the major argument against combining alcohol and driving has been this observed deterioration in performance. However, there is growing evidence that the alcohol and injury relationship is more than one of impaired judgment or performance.

LABORATORY STUDIES

Studies of alcohol and experimental hemorrhagic shock show that, compared to control animals, intoxicated animals tolerate acute blood loss less well (4-7). Myocardial functional capacity is diminished by intravenous administration of alcohol (8). Furthermore, studies involving a controlled nonpenetrating blow to the chest of dogs found that the impact alone created immediate transient major arrhythmias but only one fatality out of 9 subjects. In contrast, when the impact was combined with alcohol, 11 of 12 animals died within 90 minutes (9).

Several studies have examined injury to the CNS. A standardized cerebral contusion results in much more extensive lesions and edema in cats when alcohol is present (10). Several levels of impact applied to the exposed dura mater of either the spinal cord or the cerebral hemispheres in cats showed similar findings at higher levels of traumatic impact, with alcohol treated animals showing hemorrhage up to four times that of nontreated animals receiving the same impact. Twenty-seven animals receiving spinal cord impact were observed over several weeks. Of 13 receiving no alcohol, all were walking at the end of five days, although three walked poorly. In contrast, of 14 animals pretreated with alcohol, 12 remained totally paraplegic and only one walked well (11).

Four levels of spinal cord trauma in cats showed marked differences between alcohol and control animals at the two lower impact levels. After ten days all control animals walked or initiated hindpaw activity compared to none of the alcohol treated groups. At higher impact levels no animals showed evidence of
gait function. Similar results were obtained for the cortical evoked response and extent of tissue damage observed in histopathology studies (12). A study of ferrets showed consistent findings (13).

The extent of cortical damage resulting from a small stab wound in rats is much greater in alcohol treated animals. Furthermore, differences obtain even when alcohol is administered as much as 24 hours following injury (14-15).

CLINICAL STUDIES
Clinical studies have failed to confirm laboratory findings of a relationship between alcohol and extent of injury. Ward, et al. (16) examined 1198 patients admitted to a major trauma service. Blood alcohol levels and ISS, as well as other measures of injury severity and outcome, were obtained. The authors concluded that, if anything, alcohol had a protective effect and that patients with high blood alcohol levels experienced lower mortality rates.

In a similar study, Huth, et al. (17) examined automobile drivers admitted to a major trauma center. On the basis of severity of injury, hospital course, and long term outcome, the authors concluded that there were no effects of blood alcohol level. Thai, Bost, and Anderson (18) considered the effects of other drugs as well as alcohol in 615 trauma patients admitted to hospital. The presence of drugs other than alcohol was related to higher incidence of shock, severity of injury, and mortality, but no effects of alcohol were found.

MOTOR VEHICLE CRASH STUDIES
Two studies of motor vehicle crashes fail to confirm the clinical findings (19-20). Driver injury was examined in relation to alcohol use while taking into account other variables correlated with both alcohol and driver injury. These factors included vehicle speed, safety belt use, degree of vehicle deformation, driver age, vehicle weight, as well as others. When injury producing dimensions of a crash are considered, the drinking driver is found to be about twice as likely to be seriously injured or killed as the nondrinking driver. The effect of alcohol is greatest in the less damaging crashes.

DISCUSSION AND CONCLUSIONS
The findings from motor vehicle crashes are consistent with the controlled laboratory studies but not with the clinical studies. However, the clinical studies failed to consider the degree of impact experienced. If the alcohol-involved patient experienced a given level of injury as a result of a less severe crash, this information was lost.

Furthermore, alcohol may make a patient appear to be more injured than is actually the case. Symptomatology disappears with increasing sobriety, so that
the intoxicated patient appears to recover more quickly and completely (21). Moreover, the clinical studies have been confined to the most severe end of the continuum of injury. If the differential effects of alcohol are greatest at lower levels of impact, examining only more severe injury would reduce the likelihood of detecting alcohol effects.

The effects of alcohol may be greatest within a limited range of traumatic insult. An analogy might be the effectiveness of safety belts, where one would not anticipate major benefits in very low speed crashes, e.g., 5 mph, or very high speed, e.g., 90 mph. While the parameters of the potentiating effects of alcohol on injury remain to be determined, the evidence suggests that the greatest differential effects of alcohol may occur at relatively low levels of impact combined with high levels of alcohol.

It appears that alcohol increases injury not only by increasing the probability of an injury-producing event but also by rendering the body more vulnerable to injury from any given impact. Furthermore, studies of cerebral and spinal cord edema suggest that in the event of CNS damage, an intoxicated victim is more likely to become permanently impaired.

These findings indicate that the designated driver, a practice with a long history in the Scandinavian countries, is protective against the harmful effects of alcohol only in regard to impairment of judgment and performance. However, should a crash occur for whatever reason, the impaired passenger is at higher risk of injury than the sober passenger.

The findings also emphasize the need for routine measurement of blood alcohol level in patients presenting with serious injury in that the exacerbating effects of alcohol may complicate both treatment and eventual outcome.

Finally, these findings highlight the need for research aimed at reducing the secondary effects of alcohol on the trauma victim, that is, interventions that reduce the extent of hemorrhage and edema and hence long term impairment.

REFERENCES
5. Gettler, DT, Allbritten, FF Jr. (1963) Effect of alcohol intoxication on the respiratory exchange and mortality rate associated with acute...