Alcohol and head injury

The pathophysiologic changes associated with acute and chronic alcohol exposure in the setting of traumatic brain injury are complex. Experimental data indicate that ethanol intoxication can exacerbate brain injury through several mechanisms including hemodynamic and respiratory depression, blood-brain barrier disruption, and derangements in hemostasis. Alcohol, however, is also a potent inhibitor of N-methyl-D-aspartate (NMDA) receptor-mediated excitotoxicity, and thus is neuroprotective. In contrast to the effects of acute intoxication, chronic alcohol exposure appears to result in upregulation of NMDA receptor activity and downregulation of gamma-aminobutyric acid (GABA) receptor function. This imbalance, it is hypothesized, can result in a surge of excitotoxicity following alcohol withdrawal. Trauma-related excitotoxic cell damage may be significantly potentiated by this alcohol-induced receptor imbalance that is unmasked as withdrawal occurs. Clinical and epidemiologic investigations of alcohol and outcome after a head injury have not consistently demonstrated a measurable effect from either acute or chronic alcohol use. Multiple factors including the timing of intoxication in relation to the time of injury, the degree and chronicity of intoxication, as well as the influence of other secondary injury processes appear to determine the net effect of alcohol in a given individual. Further clinical and experimental investigations aimed at defining the impact of alcohol use on outcome after head injury are warranted [1)].

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Kelly DF. Alcohol and head injury: an issue revisited. J Neurotrauma. 1995 Oct;12(5):883-90. doi: 10.1089/neu.1995.12.883. PMID: 8594215.