"AROUSAL" AND ALCOHOLISM: PSYCHOPHYSIOLOGICAL RESPONSES TO ALCOHOL

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It is assumed that any comprehensive theory of alcoholism presupposes an understanding of the biological mechanisms of action subserving or associated with this disorder. It is further assumed that the determinants of excessive drinking behavior pertain not only to environmental events and contingencies but to interoceptive bodily cues induced by alcohol itself (1). Unfortunately, though alcohol represents one of the oldest and most widely used drugs known to man, there is a surprisingly scant and often contradictory literature relevant to its physiological and neurophysiological effects in alcoholics. Almost all prior studies (a) pertain to animals rather than humans, (b) pertain to normal, healthy male volunteers rather than habituated or detoxified alcoholics, thereby ignoring critical factors of metabolic and tissue tolerance (2), (c) employ extremely small, non-representative samples (d) do not take into account the "Mellanby effect" (3) (i.e., differential effects of alcohol pertaining to the ascending and descending limb of the blood alcohol curve) or time-dose-response characteristics and (e) do not control for the extremely important influences of mental set and physical setting.

If the actions of alcohol are to be investigated, then it should prove helpful to organize and interpret observations within some reasonable, theoretical framework. Since the psycho-neurophysiological concepts of "arousal" and "activation" have been adopted to interpret many kinds of psychopathology (4), they may likewise prove useful as explanations of the biological basis for certain kinds of alcoholic behavior. In this regard, perhaps the most intriguing but yet unreplicated study is that of Kissin, et al. (5), which assayed six physiological systems in alcoholics. According to these investigators, alcoholics showed evidence of an
impaired sympathetic nervous system, overactive parasympathetic nervous system, underactive 17-hydroxycorticoid and 17-ketosteroid production and overactive muscle tension functioning. With the exception of 17-ketosteroid production, the actions of alcohol appeared to "normalize" the functioning of these systems. While Coopersmith and Woodrow (6) concluded that alcoholics and non-alcoholics did not differ in arousal level, as measured by basal skin conductance, Garfield and McBrearty (7) demonstrated that the ingestion of small to moderate amounts of alcohol raised basal conductance but decreased over-all reactivity in alcoholics. Other scattered reports indicate that low doses of alcohol administered to alcoholics heightened "vigilance" behavior (8) and increased critical flicker fusion discrimination (9), a measure presumed to be associated with "arousal" (10,11). Aside from the acute effects of alcohol, chronic administration characteristically seems associated with greater "emotionality" and anxiety (12,13). With findings such as these, the simplistic classification of alcohol as a depressant drug, especially in alcoholics, must be open to serious question.

The present study derives from a prior one (14), investigating the effects of the "first drink" of alcohol on craving, alcohol acquisition behavior and selected physiological responses, in which certain "discrepant" findings were obtained. In brief, despite the differential effects produced by high and low doses of alcohol on tremor and CNV amplitudes, both doses were associated with increases in heart and respiratory rate, as well as percent time alpha activity. Although increased alpha activity represents a constant reproducible effect of alcohol (8,15), reports of the acute effects of alcohol on heart and respiratory functions in alcoholics are mostly anecdotal and somewhat contradictory (16-18). More important, there have been no systematic attempts made to reconcile the apparent "dissociation" between these autonomic nervous system (ANS) and central nervous system (CNS) responses. Increases in heart and respiratory rate are consistent with the concept of "sympathetic dominance" or ANS "arousal" (21) while increases in alpha activity have become traditionally associated with the concept of CNS "inhibitory" functioning (22,23).

Since our own theory of relapse to drink (14) presupposes the presence of a "conditioned withdrawal syndrome" or comparable state of "arousal," it seemed necessary to clarify the meaning of this apparent dissociation of ANS and CNS indices. In essence, we wished to determine whether it was possible for alcoholics to respond in a "normal" or "aroused" manner during an "inhibitory" or "depressed" electrocortical state or to respond in a "normal" or "retarded" manner in the presence of peripheral signs of ANS.