

AN INVESTIGATION OF THE PSYCHOLOGICAL,
PHYSIOLOGICAL AND ENDOCRINOLOGICAL FACTORS
UNDERLYING TWO TYPES OF ALCOHOLISM.

by

Larry Loebenstein

Thesis submitted in partial fulfilment of the
requirements for the degree of Master of Science
in Clinical Psychology.

University of Cape Town

1978



The copyright of this thesis vests in the author. No quotation from it or information derived from it is to be published without full acknowledgement of the source. The thesis is to be used for private study or non-commercial research purposes only.

Published by the University of Cape Town (UCT) in terms of the non-exclusive license granted to UCT by the author.

ABSTRACT

Fifteen delta and eleven gamma alcoholics were given a moderate dose of alcohol. Changes in extroversion, neuroticism, anxiety, assertiveness, Basal Skin Conductance and urinary levels of epinephrine and norepinephrine were measured.

The changes that significantly differentiated the gamma group from the delta group were those measured by the psychological variables. In the sober state, measures of extroversion, anxiety and assertiveness, also differentiated the two groups. Although the physiological and biochemical changes were significant within each group, their differential responses were not significant. The results have therapeutic implications.

ACKNOWLEDGEMENTS

My appreciation is extended to my supervisor, Prof. P.D. Du Preez, for his incisive criticism at a critical time.

To Urbie Gomez, research biochemist at the Tygerberg Hospital, whose kindness, consideration and help was invaluable for the catecholamine assay.

To Debbie Bradshaw and Lester Gilbert for the statistical analysis.

To Toni MacMahon for her pressure typing.

To Harriet Storr for her affection and support.

To my favourite mentors, the patients of William Slater Hospital, who gave me their support for this project.

Finally, to the Medical Research Council for their financial assistance.

The opinions expressed in this work are those of the author and do not necessarily reflect those of the South African Medical Research Council.

CONTENTS

	<u>Pages</u>
<u>SUMMARY</u>	i - vii
<u>CHAPTER I</u>	I - 27
Introduction: Concepts of Alcoholism and their consequences for research.	
(I) The Disease Concept	
(2) The Problem-Drinking Approach	
(3) The Alcohol Dependence Syndrome	
<u>CHAPTER TWO</u>	28 - 45
The Aetiology of Alcohol.	
(I) Constitutional Factors	
(2) Cultural Factors	
(3) Pscho-analytic Theory	
(4) Personality Theories	
(5) Learning Theories	
<u>CHAPTER THREE</u>	46 - 51
The Heterogeneity of the Alcoholic Population.	
<u>CHAPTER FOUR</u>	52 - 67
The Psychopharmacology of Alcohol.	
(I) Physical Properties	
(2) Sites and modes of Action	
(3) Dependence Producing Effects	
(4) The Effects of Alcohol on Psychological Functioning	
(5) The Effects of Alcohol on Physiological Functioning	

- (6) The Effect of Alcohol on
Endocrine Functioning

CHAPTER FIVE

68 - 73

- (1) The Problem
- (2) Rationale
- (3) Hypotheses

CHAPTER SIX

74 - 97

- (1) Subjects
- (2) Procedure
- (3) Tests
- (4) Statistical Design

CHAPTER SEVEN

98 - 127

- (1) Results
- (2) Discussion and Conclusions
- (3) Implications for further
research

REFERENCES

I29 - I47

APPENDICES

I48 - I54

- (A) The Gambrill and Richie
Assertion Inventory
- (B) The Eysenck Personality
Inventory

SUMMARY

Research into the field of alcoholism has been characterized by the fruitless search for the "alcoholic personality". The recognition of the inherent heterogeneity of the alcoholic population has led to attempts to understand the alcoholic in his own right, rather than to attempt to understand him by differentiating him from non-drinkers.

The widespread acceptance of the disease concept of alcoholism has aided the understanding of physical addiction and has led to a deeper understanding and acceptance of the alcoholic by society. The disease concept has also generalized multidisciplinary intervention in the course of addictive alcoholism. The emphasis of the disease concept has therefore focussed on the causes of addiction. However, the concern of the psychotherapist is more properly addressed to the possible causes of readdiction. The goal of this research is to attempt to gain a better understanding of these latter causes.

Alcoholics often invoke a teleological explanation for their drinking, i.e. their interpretation of drinking is made in terms of the purpose that it serves. Mellor (1974) proposes that learning theory and alcohol-effects hypotheses provide an appropriate framework for the experimental investigation of

factors that could initiate an alcoholic's recourse to the use of alcohol.

Consistent with the view of heterogeneity, Jellinek (1960) identified two forms of alcoholism that present with contrasting symptoms:

- (1) Gamma Alcoholism: This is characterized by periodic bouts of uncontrolled drinking;
- (2) Delta Alcoholism: This form is characterized by continuous drinking that is reasonably controlled.

Several researchers have confirmed the utility of the above distinction. Vogel (1960) found that periodic drinkers tended to be extroverted, while continuous drinkers tended to be introverted. Walton (1968) confirmed this finding and established that gamma alcoholics were impulsive and aggressive. Gamma alcoholics were found to be concerned about the effects that their behaviour would have on others and tended towards being more hostile and intropunitive than delta alcoholics. Using psychiatric criteria, Stein, Niles and Ludwig (1968) found gamma alcoholics to be more disturbed than delta alcoholics. Tomsovic (1974) found that delta alcoholics tended to be bored, anxious and depressed and that they experienced difficulty in coping with their social roles. The gamma alcoholic appeared to drink for intermittent intensive psychic experiences in order to pre-empt his social role and obligations.

It can be concluded from the above studies that differences between gamma and delta alcoholics are not only restricted to their different patterns of alcoholic intake, but that they differ significantly with regard to other variables, viz. personality and psychiatric disturbances.

The extensive work on the effects of alcohol on the alcoholic reported by Mellor (1972) and van der Spuy (1972), failed to uphold the widely accepted view that the alcoholic benefits from his drinking. The majority of experimental studies that have concentrated on the alcoholic's drinking behaviour, have found that many individuals fail to follow group trends.

It seems reasonable to conclude that these studies would have yielded more positive and comprehensive results if they had recognized the non-homogeneity of their alcoholic sample.

Aim: The aim of this study was to investigate the effect of a moderate dose of alcohol on a sample of gamma and delta alcoholics. The rationale for the choice of variables to measure these effects was as follows:

(1) Important measures that were known to differentiate between gamma and delta alcoholics. The Eysenck Personality Inventory was chosen as extroversion was shown to be one of the factors that differ the one group

from the other and numerous studies involving neuroticism had been done on alcoholics.

(2) Measures that were relevant to the treatment of alcoholics viz. assertiveness and anxiety.

(3) Since the experiment is by nature a drug experiment, an attempt was made to measure the possible influence of physiological effects (Lader and Wing, 1966) by measuring levels of skin conductance (arousal) and catecholamine production (adrenaline and nonadrenaline).

Hypotheses

(1) Delta alcoholics will show a greater increase in extroversion under the influence of alcohol than gamma alcoholics.

(2) Delta alcoholics will show a greater decrease in neuroticism under the influence of alcohol than gamma alcoholics.

(3) Delta alcoholics will show a greater decrease in anxiety under the influence of alcohol than gamma alcoholics.

(4) Delta alcoholics will show a greater increase in assertive behaviour under the influence of alcohol than gamma alcoholics.

(5) Delta alcoholics will show a greater change in autonomic arousal under the influence of alcohol than gamma alcoholics.

(6) Delta alcoholics will show a lesser increase in epinephrine production under the influence of alcohol

than gamma alcoholics.

(7) Delta alcoholics will show a greater increase in norepinephrine production under the influence of alcohol than gamma alcoholics.

Method: 15 Delta and 11 Gamma alcoholics were given 3 tots of brandy such that they all achieved a blood alcohol content of approximately 0.065%. They were given the E.P.I., an anxiety scale, and an assertiveness scale before and after drinking. Their level of Basal Skin Conductance (B.S.C.) was monitored (through) at ten minute intervals from the start until the end of the experiment. Urine samples for catecholamine assays were taken before and after the experiment.

Results: (1) The change scores that differentiated the one group of alcoholics from the other were the psychological ones.

- (a) Extroversion: Delta greater increase than gamma
($P < 0,01$)
- (b) Neuroticism: Delta greater decrease than gamma
($P < 0,01$)
- (c) Anxiety: Delta greater decrease than gamma
($P < 0,01$)
- (d) Assertiveness: Delta greater increase than gamma
($P < 0,01$)

The difference in change as measured by (B.S.C.) and catecholamine levels were not significant.

Discussion: The results of the present research show that valid investigation of the effects of alcohol in alcoholics can only be achieved if the heterogeneity of the alcoholic sample is catered for. This study has also shown that the gamma-delta distinction is a valid one and that it is of heuristic value.

In addition, the research has shown that gamma and delta alcoholics differ in their response to a moderate dose of alcohol. Delta alcoholics become extroverted and assertive, and experience an accompanying reduction in anxiety and neuroticism. Gamma alcoholics are far less responsive to this dose and experience only a significant change in anxiety. The biological responses of both gamma and delta alcoholics showed almost identical responses to alcohol. Thus it can be concluded that similar future research consider psychological variables to be more useful than biological ones.

The results also confirm that the personalities of gamma and delta alcoholics differ substantially. Gamma alcoholics are more extroverted and assertive and less anxious than delta alcoholics.

The results of this study also have therapeutic implications. It is suggested that the delta alcoholic would benefit from therapy involving social skills and assertiveness training and therapies generally directed

towards the reduction of anxiety and the increasing of self confidence. The gamma alcoholic would appear to benefit more from a combination of intensive psychotherapy, aversive therapy and the prophylactic use of Antabuse.

CHAPTER I
INTRODUCTION

Anyone who has reflected on the many definitions of health and of mental health in particular, will, I think, conclude that there is no consensus, and he will see that when moral or social values are invoked, there are scarcely any limits to the behaviour which might be called morbid. Medical criteria are safer, that is, criteria essentially concerned with the integrity of physiological and psychological functions.

Sir Aubrey Lewis, 1963, p.1550

The current understanding of alcoholism has been confounded by the contradictory philosophical and scientific premises on which concepts of this disorder have been based. This almost total absence of concurrence is not only a consequence of logical and conceptual differences. It also exists as a result of the predilection of the various disciplines involved in the treatment of alcoholism for rejecting conceptions on the basis of their social consequences. The disease concept, for example, is accepted by some workers, since it destigmatizes alcoholic behaviour in an individual. It also allows the medical

profession to engage formally in the treatment of an implied pathophysiological disorder.

Equally, the disease concept might be rejected as it essentially proscribes intervention by non-medical disciplines or absolves the alcoholic from personal responsibility for his drinking behaviour.

Varying interpretations of terminology contribute to the problem of conceptual diversity -

No area of medicine is so bedevilled by semantic confusion as is the field of alcoholism.

Davies, 1969, p.1042

Many terms used to define alcoholism are at certain times employed exclusively and at other times interchangeably. Analogies putatively pertinent to the study of alcoholism, and originally adduced for purposes of argument, attain a functional independence. Clark (1975) indicates that semantic confusion leads from a description of what alcoholism is like, to what it actually is, in a more substantive form.

There can be little doubt, however, that the ongoing conflict of conceptual interest and definition is only a representation of the multiplicity of causative factors involved in human behaviour in general, and in alcoholism in particular.

CONCEPTS OF ALCOHOLISM AND THEIR CONSEQUENCES FOR RESEARCH

I. THE DISEASE CONCEPT

At present the disease concept of alcoholism is being used more frequently by more alcoholologists than any other concept. Not only is this approach accepted by the medical and allied professions, but it is held to be one of the basic tenets in the philosophy of a widely accepted fundamentalist therapeutic organization, such as Alcoholics Anonymous. Criticisms of this concept, on the basis of its operational or heuristic value, can be found in a plethora of articles in the literature. This is often to the exclusion of criticisms of other approaches.

The acceptance of the disease concept of alcoholism has, however, been questioned, as the following excerpt from The Lancet (1977) illustrates :

The view of the matter is therefore that the hunt for a definition of alcoholism should be abandoned as the pursuit of what was never more than an imagined animal: a medical diagnosis was being falsely imposed on behaviour and events of great variability.

Lancet I, 1977, pp.1087, 1088

Madden (1977), in a concise article, captures the essence of this definitional and diagnostic problem by indicating

that two approaches exist in the definition of alcoholism :-

- (i) A fundamental or essentialistic approach which views alcoholism as an endogenous condition. Alcoholism defined in such a manner is thus a phenomenon sui generis. The disease concept falls within the ambit of this approach.
- (ii) A scientifically conventional approach in which a term functionally or operationally defines and labels a designated group of phenomena.

It would appear to be fruitless to pursue the disease concept of alcoholism without having some understanding of the disease concept itself. A comprehensive review of the concept has often proved to be a chimerical goal and neither proponent nor opponent of the disease concept of alcoholism has embarked on this pursuit with much intellectual vigour. The reasons for this are partly historical, partly semantic, and a brief, and therefore conceptually limited review, will be attempted, with a special reference to alcoholism.

The concept of disease

The modern concept of illness, both physical and mental, appears to be based on the empirical and serendipitous discoveries of science and technology on the one hand, and the subsequent theoretical interpretation of these discoveries on the other.

Initially, the definition of illness was extremely limited

the emphasis being placed on endogenous physical changes resulting in a pathological condition. These changes were described in simplistic terms and perceived as anatomical lesions, caused by exogenous, autonomous organisms invading the body (Taylor, 1972). Progress in understanding diseases correlated closely with the strides made in the fields of the natural and biological sciences. The functional relationships between the anatomical systems were then seen to be important, especially with regard to diagnostic signs. This development is the basis for the first and probably the most objective description of the three elements that constitute a disease process, viz :

- (i) The identification of life processes which are changed and tend to result in various signs or symptoms which will frequently and consistently cluster together;
- (ii) The subjective description of symptoms by the person involved, which means in a somewhat classic sense, that the subject gives a verbal indication that some form of disease is present;
- (iii) The third element, and probably the most polemical, is that other individuals or society as a whole now deems that the above-mentioned factors, separately or in combination, constitute an impairment or derangement of normal functioning, and a disease or illness is ascribed to the individual.

The first two factors constitute the rationale for the clinical practice of eliciting signs and symptoms. This

fundamental paradigm, when applied to the understanding of the nature of alcoholism, is fraught with difficulties as Room (1972) has stated :

The Platonic assumption that empirical events are merely the projected shadows of underlying realities blurs the distinctions between testable mechanisms, plausible models and hypothetical analogies, since assertions about underlying realities are not easily falsifiable.

Room, 1972, p.1055

If we accept the above limitations placed on the first two stages of a nosological endeavour, we find that it is the third stage that has led to the most acrimonious debate. There is little doubt that of all the processes leading to a diagnosis, the labelling would tend to be the most capricious and hence the most subjective, and therefore the most liable to error and misinterpretation.

The tendentious arguments of the anti-psychiatry movement are directed towards the role of society and the "true" nature of mental illness in particular, i.e. that deviation or change in an individual's behaviour is labelled "pathological" or "diseased" as it violates the norms of society rather than being a process that is intrinsically disabling. The main proponents of anti-psychiatry (with Szasz (1961) in the vanguard) base their arguments on this belief. On close examination, however, one sees that their formulations are

based on the spurious assumption that society establishes the norm. Clearly this appears to be a misunderstanding of the logical corollaries stemming from the principle of eliciting signs and symptoms. The objective norms for diagnosis and the deviation therefrom, are established by the total functioning of the subject rather than society. Lewis (1955) stresses this point by stating that a disorder or disablement must be detectable at both a discrete and mental or physical level. Behavioural deviance or non-conformity per se could in fact be defined as health.

This situation is best illustrated with regard to the manifestations of typical alcoholic withdrawal symptoms induced by a deceleration of alcohol intake. Clearly the presence of severe tremor, auditory and visual hallucinations and seizures constitutes an involuntary impairment of the central nervous system. The deviance does not exist as a result of an alcohol intake over and above (or below) some established social norm, but is due to a particular individual's pattern of alcohol consumption.

It is evident therefore that the main features of disease are their characteristic signs and symptoms, however much importance is also placed on their syndromic and temporal manifestations. Of equal importance is the synergistic influence of a host (person), agent (ethanol) and an environment. If alcoholism is to be conceptualized as a disease, it should at least conform to the basic tenets expressed above.

Signs of Alcoholism

(A) There appears to be general agreement that loss of control (LoC) is a pathognomonic sign for the diagnosis of alcoholism (Keller, 1975). LoC is assumed to take place in addicted alcoholics and manifests itself in two forms :

- (a) An inability to consistently refrain from drinking (Delta alcoholism); and
- (b) An inability to consistently control or stop drinking once it has been initiated (Gamma alcoholism).

The genesis of LoC lies in Jellinek's (1960) classic text : "The Disease Concept of Alcoholism" and the above forms were the sine qua non for the diagnoses of the types of alcoholism, delta and gamma, respectively, which were regarded as disease forms.

It was assumed that LoC was a result of continued exposure to alcohol, and developed pari passu with :

- (i) increased tissue tolerance;
- (ii) adaptive cell metabolism;
- (iii) emergence of physical dependence in the form of withdrawal symptoms; and
- (iv) a strong psychological disposition to alcohol termed "craving".

Clarification of Jellinek's (1960) developmental view was made by Glatt (1975). The initial phase is seen to be that of "symptomatic drinking", not yet implying a disease, but certainly the phase where an increase in tolerance is developing. The second phase is assumed to be that of addictive drinking in which some irreversible, constitutionally based change has taken place and a disease process has been initiated. The third or chronic stage is characterized by secondary physical illness and continuous intoxication.

It is unfortunate, however, that Jellinek (1960) in his overzealous desire to conceptualize alcoholism as a disease, viewed LoC as a constant phenomenon once host and agent were in contact. His description is published in the well-known World Health Organization Report (1952) :

As soon as any small quantity of alcohol enters the organism a demand for more alcohol is set up which is felt as a physical demand by the drinker but could possibly be a conversion phenomenon. This demand lasts until the drinker is too intoxicated, or too sick, to ingest more alcohol.

World Health Organization
Second Report, 1952

The inherent variability of the LoC concept, initially denied by Jellinek (1952), has led to the serious

questioning of the value of the concept and the psychopharmacological and biological underpinnings on which the concept is based. It can be stated unequivocally that within any one alcoholic individual, there exists numerous responses to alcohol and not all excessive drinking behaviour need be characterized by loss of control. The disease concept of alcoholism is then rejected on the basis that it presents with inconsistent symptomatology.

Merry (1966) destroyed the inevitability of LoC after one drink in an experiment in which he gave disguised alcohol to alcoholics. Mello and Mendelson's study (1965) showed that alcohol consumption by alcoholics is largely dependent on the contingencies of reinforcement which accompany their drinking and not on ingestion per se. Furthermore, Davies (1962), and Bailey and Steward (1967) have shown that certain alcoholics can return to social drinking. Although LoC has been reported in institutionalized populations, Clark's (1976) four-year follow up study found less frequent and more inconsistent LoC in non-institutionalized individuals.

Critics of LoC tend to regard the phenomenon in an absolute way rather than in a relative one, a view very uncommon with regard to other psychopathologies. Semantic confusion appears to compound this error. LoC was thought by Jellinek (1960) and his followers to pertain only to gamma alcoholics whereas they exhibit only one form of LoC. Delta alcoholism, i.e., not being able

to abstain, is only another variant of the inability to exert a voluntary control over drinking behaviour.

Rohan (1978) believes that alcoholism is misperceived as having ontological existence but in fact is based on "mentalisms" such as LoC and craving. He regards these entities as explanatory fictions since they cannot be objectively defined or measured. It is beyond the scope of this study to take issue with this metaphysical viewpoint, which in effect invokes the mind-body dichotomy, but strong exception has been taken against adopting such an evaluistic framework.

Ludwig and Wikler (1974), protest that a subjective state (as opposed to an explanatory fiction) need not be reflected in behaviour in the first place. Behaviour can easily be controlled by cognitive processes such that it can even appear to be inconsistent with what a person might be experiencing, e.g. alcoholics commonly experience anger without expressing it. Rohan's (1978) preference for a strict operational approach to the study of alcoholism contributes little to the understanding of this complex problem. Less rigorously defined concepts, such as LoC, appear to be of far greater value in this area as they stem from the problem itself.

In agreement with this viewpoint, Glatt (1967) perceives LoC as a relative and progressive phenomenon, increasing in the latter stages of alcoholism with intra-

and inter-individualistic differences. This is dependent on the amount of alcohol taken and on an individual's psychological, physiological or social situation. This aspect can again be perceived as the juxtaposition of important elements in host, agent and environment and accompanying temporal factors. LoC is therefore a construct that is at least consistent with a disease concept of alcoholism.

- (B) Craving is also considered to be a pathognomonic sign in the diagnosis of alcoholism. It is considered to have both psychological and physiological manifestations, and is implicated in both the development and propagation of alcoholic behaviour. Craving shares with LoC a central position in the understanding of alcoholism as a disease, but the scientific value of this major tenet is questionable, both on the basis of the logical tautologies it causes, and the fact that no independent test exists to prove that it is at least partially a physiological phenomenon. The clinical value of craving is also questionable since it fails to distinguish between uncontrolled drinking in addicted individuals and those who simply at times exhibit undisciplined drinking as the basis of an assumed elective intoxication.

Both Isbell (1955) and Kissen (1974) defined craving in two forms :

- (a) "Symbolic" craving or a desire for alcohol not contingent with withdrawal symptoms. This form of

craving is assumed to occur during the developmental phase of addictive alcoholism but also accounts for relapse during the addictive and chronic phases. An intrinsic characteristic of symbolic craving is psychological dependence.

- (b) "Non-symbolic" craving or a need for alcohol is thought to occur after the onset of drinking, and alcohol is used to relieve withdrawal symptoms rather than to prevent them, i.e. withdrawal is initiated at the commencement of drinking rather than at its termination. This important yet often neglected point was confirmed by the experimental work of Gross and Lewis (1973), and Bergleiter (1973) who show that the function of the central nervous system of alcoholics is disrupted early in their drinking session. The strength of reinforcement and the propensity for generalization within this schedule, viz. alcohol leading to withdrawal leading to alcohol, is indeed great. In essence, "non-symbolic" craving is due to the reactivation of physical dependence.

It may be said that many workers assert that the link between the two forms of craving is at best tenuous, casting doubt on the validity of the construct as a whole. MacAndrew (1969), in a witty yet profound article, states that evidence for craving in the physiological sense, could be established on the basis that an individual becomes intoxicated even though there is a

strong desire to remain sober. It is argued that it is excessive drinking that establishes the presence of craving. This craving is then used as an explanation for drinking. The circularity of argument here is self-evident. This form of logical pursuit is seen to apply to constructs governing the disease concept of alcoholism in general.

Alcoholism and similar explanatory entities are reached through a process of circular reasoning. Not only is the presence of "alcoholism" deduced from the visible behaviour of harmful drinking, but the characteristics of alcoholism are constructed from this very same behaviour which is said to be persistent excessive and lacking in control.

Rohan, 1978, p.212

Certain animal studies by Deutsch and Coopmans (1973), Way, Loh and Shin (1968), and Myers and Veale (1968), have shown that excessive drinking can produce craving. They found that if rats become addicted to a variety of central nervous system depressants, they will subsequently show a preference for these substances on exposure to them even though they have remained abstinent for protracted periods of time. Other mechanisms for linking the two forms of craving and the reification of the construct were proposed by Wikler (1971). Concomitant with the development of physical dependence, the associated withdrawal states can

become classically conditioned to numerous stimuli that arise in temporal contiguity with them. Operant forces then contribute in the form of negative conditioning. The administration of the drug will continually and repeatedly bring about relief from the symptoms of withdrawal. This view is supported by clinical evidence that many addicts relapse when exposed to physical and emotional stimulus situations similar to those in which addiction took place.

Ludwig and Lyle (1964), and Wikler (1971) further maintain that somatic and cognitive states similar to those experienced during withdrawal, e.g. anxiety, agitation or depression, could equally bring about the same conditioned response, i.e. the repeated taking of the drug. Ludwig and Wikler (1974) feel that the subjective concomitants of drug-seeking behaviour may or may not be at a conscious level. The extent of cognitive specificity is related to the addict's capacity for symbolism. Craving, therefore, provides the addict with the necessary cognitive symbolism for certain goal-directed behaviour in the form of drug-taking, induced by exposure to a protracted and varied negative reinforcement schedule.

If one accepts the validity of the above-mentioned factors, permutational consequences follow. An essential factor would be the intensity of the drive or craving. Ludwig and Starke (1974) have shown alcoholics perceive

craving as being associated with various states of dysphoria, the intensity of the craving ranging from minor anxiety to strong somatic arousal and feelings of distress. In confirming the role of conditioning factors outlined above, it was found that strength of symbolic craving correlated highly with strength of previous withdrawal symptoms. This finding was supported by Barry (1974).

A further consequence of perceiving craving as a drive or motivational state is that, like all other drives, it is subject to at least some cognitive control. It might compete with other drives, or its strength could be of a minor magnitude such that it brings about a minimal response. Situational effects enhance the complexity of these factors. Schachter and Singer's (1972) classic work on obesity and the value of recognizing both external and internal cues for an excessive eating response is of particular importance here. This view is further complemented by Ormes' (1962) work on demand characteristics implicit in social situations that determine perceptions as well as responses. The inherent variability of the craving response appears to be that it is a necessary but not sufficient condition for drinking. This further negates Rohan's (1978) approach. Subjective states, such as craving and LoC, easily generate numerous testable hypotheses. These hypotheses do not conform to the scientific purity demanded by Rohan (1978) yet they appear of equal use in the understanding of alcoholism.

Craving is in essence a symptom of alcoholism insofar as its presence is based on the subjective reports of the individual. Thus at this point it probably represents the parting of the ways for the formal and praxiological scientist. It would seem inconceivable, at least to the practising clinician, to dispense with a concept that has perhaps little meaning to the formal scientist but is of great importance to the alcoholic. As Keller (1975) points out, the essential question for the alcoholologist is not the reason for addiction, but the reason for continued readdiction.

- (C) The appearance of withdrawal symptoms is accepted as a classic sign of having an addictive illness. This factor, as a contribution to an understanding of the disease concept, has been partially discussed previously in this chapter. Since tolerance to a drug is seen as a necessary condition for withdrawal symptoms to occur, this will be discussed more fully in the section dealing with the effects of alcohol.

The discussion of the disease model thus far has adhered to the classic view that a pathological condition is a reasonably predictable and ongoing process. Discussion has also centred on "hard" signs and symptoms and it would appear inappropriate to relinquish the problem here, without briefly mentioning that genetic factors, if implicated in alcoholism, would enhance the

disease view, as somatic factors would then be implicated as a causal nexus in the process. A more comprehensive discussion of this will be undertaken in the section on aetiological factors.

An inherent aspect of the disease concept is that it causes deleterious physical and mental changes in an individual. Van Dijk (1973) asserts that a disease process will be exemplified by at least the following characteristics :

(1) Decrease in competence, freedom and autonomy :

It can be argued that continuous and excessive alcohol consumption will acutely and chronically cause a decrease in competence. If physical dependence is manifest, an automatic diminution in autonomy will occur at least with regard to alcohol consumption. Keller (1976) regards disablement as a key factor in the diagnosis of disease and appears to concur with Van Dijk (1973) since loss of competence, freedom and autonomy implies disablement. Edwards and Gross (1976) also consider this to be an essential aspect in the diagnosis of alcoholism, but tend to circumscribe the issue to a narrowing of the drinking repertoire. The salience of drink-seeking behaviour now takes priority over other behaviour and this also implies a decrease in competence, freedom and autonomy.

(2) Disorganization and disintegration :

This takes the form of the various systemic illnesses that accompany excessive alcohol abuse. Korsakoff-Wernicke syndrome, cirrhosis and alcoholic pancreatitis are examples of mental and physical disablement.

(3) Environmental maladaptation :

Van Dijk (1973) considers maladaptation as a reciprocal dialectical relationship between man and his environment. This dialectic aspect comprises initiating active components as well as active and passive aspects. This is also manifest in the alcoholic's articulation of time. His behaviour patterns are orientated towards immediate and present goals, and little regard is paid to past experiences and future intentions. Maladaptation is therefore a logical consequence.

The discussion thus far has attempted to isolate and define the major tenets of this disease concept of alcoholism. The value of any concept that is used for investigative purposes should not be judged in isolation, but should be seen in relation to other conceptions. Neither should historical vicissitudes be assigned too great an importance. Clearly Jellinek's (1960) early conceptions lacked theoretical clarity owing to

his rather tautological assertions. This does not invalidate the disease concept but rather magnifies a less erudite view of the concept itself. What is being questioned is whether the disease concept is too simplistic or whether it serves the obvious virtues of parsimony.

Certainly alcoholism cannot be compared to a strict definition of disease as exemplified by those of the infectious kind. The involuntary aspect of the process of the disease and the manifestation of symptoms are central features absent in alcoholism. Paradoxically, it is the absence of the agent that produces some alcoholic symptomatology, e.g. withdrawal. Alcoholism does share many common properties with disease. For example, it presents with various disablements due to impaired physiological and psychological functioning. If left untreated and allowed to continue, it has a predictable course. Deficiencies in properties common to both certainly argue against unequivocal acceptance of the disease concept.

2. THE PROBLEM-DRINKING APPROACH

This approach views alcoholism from the perspective of the problems any drinker manifests as a result of his alcoholic intake. As Cahalan and Room (1974) indicate, the emphasis

of this approach is directed more towards social consequences, and the basic criteria for the forming of problem-drinking measures were a priori conceptualizations. This approach appears more consistent with Madden's (1977) alternative view of the disease concept in which the term "alcoholism", functionally or operationally, defines a designated group of phenomena.

A further impetus to this approach is the apparent ephemeral nature of the key symptoms illicited in diagnosing an addictive alcoholic illness :

In general, it may be fair to say that operationalized definitions of alcoholism of this addictive pattern closely resemble the operational definitions of non-addictive problem drinking. The problem, of course, is that the indicators that go to make up the operational definitions are quite removed from the essence of conceptual distinction; they require a long and tenuous chain of inferences from the concept to the operation and in the process of making these references, distinctions between competing conceptualizations of the phenomena are easily lost.

Clark, 1975, p.420

This approach therefore advocates the separation of problems and behaviour that constituted the interchangeable indicators of the underlying disease. Clark (1975) further articulates

this point of view by calling for a "double disaggregation" of the phenomena from an initial unitary phenomenon (disease):

- (1) To examine drinking problems separately in order to ascertain frequency of occurrence and whether the problems present as a cluster or in a disjunctive form.
- (2) To bring to bear the inherent advantage of longitudinal study that is implicit in this approach.

The emphasis is therefore not to question whether a person is an alcoholic or not, because once this distinction is made, the possibility of other causes, which could be revealed by separation of drinking and problems in longitudinal study, would be ignored. This is an important point. The emphasis of the approach is on the consequences that can arise as a result of alcohol abuse in a given combination of social structure and individual behaviour. Rudi and McGaughron (1961) emphasize that alcoholism is not a behavioural entity with a single aetiology and course, but is better viewed as a combination of disorders which share phenotypic properties of excessive alcohol ingestion. The view is taken that little progress can be made until finer distinctions are made among the alcoholic population.

The use of the essential-reactive distinction, initiated by Knight (1937), was consistent with the problem-orientated concept of alcoholism, and exists as an illustrative and historical approbation of the approach.

The essential alcoholic was seen to ingest alcohol in an indiscriminate manner, had an early onset of drinking in the absence of any precipitating event, and would use drugs to obtain high levels of the desired pharmacological action. Problems which accompany this drinking pattern are long-term economic and emotional dependence, hedonistic preoccupation, and the inability to form stable relationships. This results in serious marital problems, conflicts and immature feelings and emotions.

The reactive alcoholic's drinking was of later onset, and was usually related to an identifiable, precipitating event. Drinking was more intermittent and less erratic, and, because it was restricted, it was more responsible. Reactive alcoholics exhibited dependent and passive types of immature behaviour.

Although some advantages accrue from the problem approach and the "double disaggregation" process as illustrated above, it overlooks, at least to a degree, an individual's own contribution to his problem. Although the main proponents of this approach are sociologists, their investigative endeavours of analysing drinking habits, the consequences of drinking and the (social) circumstances in which these occur, place these investigators in and about the confines of Skinnerian psychology - a tight fit indeed. There appear to be other inherent difficulties in the approach. The degree to which any number of problems must cluster before they are utilized appears arbitrary. The view of problems

might differ from population to population as well as from one evaluator to another. Edwards and Gross (1976) see little value in using secondary damage with regard to job, person, property and family, as these parameters vary with environmental, social and cultural differences. As Edwards et al. (1976) indicate, it was found that social disabilities related to alcohol consumption did not easily lend themselves to testing and definition. A telling point that comes to mind is whether these persons would have suffered these particular problems without their abuse of alcohol.

Certainly the influence of alcohol on the central nervous system is by comparison a very stable variable. It would appear imprudent to ignore the perspicacity of Lewis (1963) who sees the integrity of psychological and physical functioning as the safest criteria with which to work. It is agreed that there appears to be a general problem system related to, or as a result of, alcohol consumption. A problem sub-system appears to be related to entities that could be classed as disabilities or diseases.

3. THE ALCOHOL DEPENDENCE SYNDROME

This appears to be a workable compromise between the disease concept and the problem approach. The problem-drinking approach to alcoholism is conceptually perhaps no more or less perjorative than the view of alcoholism as a disease. There is, however, a more conceptually advantageous position to adopt that caters for the semantic, social and scientific

difficulties that have arisen from both the disease and problem approaches. This is to view alcoholism as a dependence syndrome.

Edwards et al. (1976) explain this position in an extensive article. A syndrome is manifest when a number of phenomena tend to cluster with sufficient frequency as to constitute a recognizable occurrence. In itself the fact of dependence predicates the existence of a profound and central disability seen as a psychobiological reality and not as an arbitrary social label. This approach also recognizes that alcohol-related disabilities need not be related to the alcohol-dependence syndrome. Implicit in the approach at present is that no view of aetiology is adopted and therefore no assumptions are made as to the stage at which the syndrome should be termed a disease. In a more important and general sense, however, the syndrome is of multidimensional origin and exists in degrees. The core syndrome presents with identifiable subtypes.

The following criteria are used for the diagnosis of dependence :

(1) Altered behavioural state :

A central factor in diagnosis is that an alteration occurs in the individual's drinking behaviour. This is evidenced by diminished variability in drinking responses. Typically the person's alcohol intake becomes a logistic problem and scheduling of drinking

takes place in the face of negative cultural or individual consequences. There exists impaired control over drinking.

(2) Altered subjective state :

It is recognized that the use of subjective reports is often difficult as the basis for scientific data or proof, however, the argument to include what an individual states about his drinking behaviour in a diagnosis appears to be a useful one. Changes in an individual's subjective state appear to correlate with altered behavioural states, and reports of "craving" as previously discussed in its various forms and degrees are of importance.

(3) Altered psychobiological state :

The changes that are of importance in this regard are twofold :

- (i) the signs and symptoms of typical alcohol withdrawal states range from anxiety and sweating to hallucinations and grandmal seizures; and
- (ii) changes in tolerance occur.

It is known that on a neurophysiological level, an increase in tolerance is a necessary but not sufficient condition for withdrawal symptoms to occur. It is

usually reported that more alcohol is needed to bring about desired states. After years of heavy alcohol intake, a decrease in tolerance is also reported.

This study has adopted the above conception of alcoholism - and the criteria used to establish the alcohol-dependence syndrome were used for the purpose of diagnosis.

The alcohol dependence syndrome appears to be the most suitable concept of alcoholism to adopt for the purpose of research. Scientific methods require conceptual thinking that is both systematic and easily communicated. These are the central concerns of this approach. Classification must by necessity precede aetiology. The problem drinking approach is deficient with regard to systematic conceptualization. On the other hand, the disease approach, by definition, must seek a physiologically based aetiological process. However, the alcohol dependence syndrome is defined with sufficient precision to allow both the testing of empirical hypotheses and the integration and interpretation of data through the use of diverse theories. It is also independent of any social values. The author feels that this approach is at least consistent with the criteria expressed by Lewis (1963) for the purposes of investigating morbid behaviour.

CHAPTER TWO

THE AETIOLOGY OF ALCOHOLISM

Aetiology is defined as the science of the causes of disease. The term pertains to predisposing and direct causes and the mode of interaction of these causes leading, in this particular context, to a defined effect - alcoholism. Since, for the purpose of this study, alcoholism is treated as a generic term, the heterogeneity of this population, and evidence to support this view, will be discussed in Chapter Three.

This review will attempt to be representative rather than exhaustive as there is abundant work in the field making a total view prohibitive. If the alcohol-dependence syndrome is the subject to concern, then, according to Murphree (1976), there are instances where alcohol intake and diverse problems are correlated to such a degree that a transactional, if not causal, relationship appears incontrovertible. Quantification of the degree of causality remains a complex task, and the multiplicity of causal explanations appears to founder on the basis of either being simplistic or overinclusive.

(1) Constitutional Factors

Despite a good deal of research into the subject, no convincing evidence has been provided to make us believe that the future alcoholic is in some way marked out from his fellows by differences in anatomy, physiology or pathology or by abnormalities of metabolism

or of tissue chemistry.

Kessel and Walton, 1969, p.69

The above quotation, made almost a decade ago, is today as compelling as it was originally. Evidence that there is a significant association between alcoholism and other inherited characteristics of the above nature appears equivocal. Attempts to separate the genotypic and phenotypic properties of alcoholism have proved difficult. However, some progress has been made by using the method of temporal precedence and control in genetic studies. For the sake of clarity, supplementary evidence from animal studies will be offered in a later section.

Cruz-Coke et al. (1964, 1965, 1966) have shown that there is not only an association between colour blindness and cirrhosis of the liver but also between alcoholism and colour blindness. Since colour blindness is an X-linked recessive abnormality, it was deduced that alcoholism followed the same mechanism. These workers felt that the higher incidence of alcoholism in males comported well with this finding.

It appears that the results of replication studies were mixed. However, strong methodological objections were raised by Smith and Brinton (1971) who showed that colour vision improved in alcoholic subjects after clearing of the sensorium and nutritional treatment. Equally,

Nordmo's (1959) work on linking alcoholism to the A-blood group failed to be replicated and was methodologically unsound. Pelton et al. (1959), and Lester (1966) infer that innate factors are involved in a disturbance of carbohydrate metabolism in alcoholics. This appears to be partially related to anecdotal evidence that an inverse relationship exists between sucrose and alcohol consumption in alcoholics. Clearly, this could be so, and the experimental evidence could be open to a wide range of alternative interpretations (e.g. oral dependence), or could be attributed to acquired factors such as protracted and excessive alcohol abuse.

In contrast to the work done in an attempt to establish a relationship between alcoholism and genetically determined physiological factors, some investigators have researched the relationship between alcoholism and endogenous psychiatric conditions. Jellinek (1945) found that alcoholism in probands was largely dependent on psychopathic and psychic features in their parents. The rationale was that an individual was predisposed to alcoholism on the basis of a constitutionally based psychological and personality-related abnormality. Environmental factors of a sociocultural nature then precipitated the illness. The dubious contribution of Amark (1951) related epilepsy to epsilon alcoholism, a very rare subtype. A more useful contribution is that of Winokur, Rimmer and Reich (1971), and Winokur (1974)

who have shown that a significant correlation exists between alcoholic probands and a parent suffering from depressive spectrum disease. All these studies can at best suggest that some alcoholic illnesses might be secondary to an acquired psychiatric illness.

The use of animal analogues in the study of alcoholism is fraught with difficulties. Although many workers have shown inter- and intra-species variability in preference for alcohol (Arvola and Forsander, 1961; Rogers and McClean, 1962), none have succeeded in linking preference with quantities consumed. This implies that although the animals preferred drinking alcohol, they did not necessarily abuse it or even drink in excess. In many of these studies, the animals were forced to drink alcohol. It seems almost absurd that when certain animals are compelled to drink alcohol, and subsequently some drink more than others, a genetic predisposition towards alcohol abuse or addiction is assumed.

Following genotrophic theory, Williams (1959) showed that mice provided with a deficient diet will have a preference for alcohol. This was purported to support Pelton (1959) and Lester's (1966) work, but the argument on disturbances of carbohydrate metabolism in alcoholics appears spurious. If alcoholics (or animals) drink ethanol excessively because of nutritional and/or caloric deficiency, it should follow that they would have continued preference for high caloric diets established before the commencement of drinking. Lester and

Greenberg (1952) have shown that deprived rats, given a choice between water, alcohol and sucrose, show a preference for sucrose. Von Wartburg's (1970) work appears to have been much more rewarding. He has shown vast individual differences in the production of alcohol dehydrogenase, an enzyme involved in alcohol metabolism. He postulates that "alcoholic" animals therefore inherit an enzyme system different from non-drinking ones. There is some support for this work.

Although useful, there are at least two major pitfalls which exist in animal research. Lester (1966) shows that in the majority of studies using free and/or forced selection of alcohol, the rats appeared to control the intake of alcohol to levels below that of intoxication. Further, in an edited text (Waynen and Mendelson) (1977) which they devoted entirely to drinking behaviour in animals, it was found that numerous variables, viz. osmosensitivity, temperature of solids and liquids, temporal locus of eating and drinking, age and sex, and mode of food dispensation, appear to influence intake. Further research will be of little value if these variables are not controlled.

Although twin studies have formed the basis of most genetic studies, few have attempted to control for environmental factors. Only two studies will be quoted here, because, not only do they support the findings of the less-controlled studies, but are important contributions in their own right. Goodwin, Schulsinger,

Hermansen, Guze and Winokur (1973) in a study using 55 men, showed that probands removed from their parents, one of whom had a hospital diagnosis of alcoholism, had a significantly greater chance of developing alcoholism, than a control group without an alcoholic parent. With the exception of divorce, a possible consequence of alcoholism, the two groups did not differ with regard to other important variables such as other forms of psychopathology. This directly contradicts Jellinek's (1952) view that alcoholism is part of the fabric of an inherited disposition to personality disturbances. Goodwin et al.'s (1973) work clearly excludes the possibility that alcoholism is secondary to some other genetically determined psychiatric illness.

Schuckit, Goodwin and Winokur (1972) studied individuals who had either an alcoholic adopted parent or who were reared separately from their biological alcoholic parent with non-alcoholic surrogate parents. There was a greater significant incidence of alcoholism in the latter group and this finding enhances Goodwin's work (1970). Although this work is empirical in nature, it would seem prudent to accept that the mere fact of having had an alcoholic parent predisposes an individual to developing alcoholism. Circumspection needs to be exercised in evaluating the nature and mechanism of what is transferred.

(2) Cultural Factors

The work of Horton (1943) and of Bales (1946) contribute historically and comprehensively to an understanding of the influence of social, cultural and anthropological factors involved in the rate and development of alcoholism. Horton (1943) proposed that primitive man was in a state of constant anxiety owing to subsistence insecurity and problems derived from acculturation. If these circumstances occurred in societies where there was high acceptability of the utilitarian use of alcohol for the reduction of anxiety, they would lead to the continuous use of alcohol and a high probability of dependence.

Bales (1946) systematically expanded this view by the exposition of the following cultural formulations. Society will influence the rate of alcoholism by :

- (i) the attitude towards drinking that it engenders in its members;
- (ii) the conflicts that individuals will be exposed to by being members of that society; and
- (iii) the number of alternatives to satisfaction that it provides.

Bales hypothesized that a society can adopt four attitudes to drinking :-

- (a) prohibition;

- (b) ritual drinking, for religious reasons, a typical example being a Jewish ceremony;
- (c) convivial drinking, where drinking is a shared and pleasant experience; and
- (d) utilitarian drinking, where alcohol serves to alleviate suffering, increases pleasure or is used for medicinal purposes.

Bales (1941) illustrates his hypotheses by citing the low incidence of alcoholism among Jews where alcohol is used for ritual purposes and hedonistic use is subject to censure. Equally high rates among the Irish are attributed to a common utilitarian view.

Field (1962), in a follow-up study, has shown that excessive use of alcohol was related to the degree of strength in both the horizontal and vertical structure in a society. He opposed Horton's (1943) view and claimed that the reputed anxiety was actually induced by poorly organized cultures which did not supply their members with sustenance and which placed them in a position where acculturation could easily occur. O'Connor (1976) cited in Hare (1976) also opposes Bales's theory (1946) by showing that the Irish, both historically and currently, have an ambivalent attitude towards alcohol. She believes that this places the younger members of Irish society in a relatively difficult position with regard to drinking. Jewish attitudes by comparison are circumscribed and defined.

It is difficult to assess the contribution of cultural theories apart from their obvious sociometric value. They do not explain why the majority of members in a defined culture do not develop alcoholism, while the nature and exposition of these cultural theories dictate that they are essentially far too inclusive. No doubt such information aids the problem-drinking approach, but could easily obscure some real issues. Defined cultures might alternatively be seen as a collection of individuals sharing a relatively common genetic pool.

(3) Psycho-analytic theory

A common principle in psycho-analytic theory is that alcohol is used to free repressed material. McCord and McCord (1960) state the classic Freudian position that oral fixation, self-destruction and latent homosexuality are the three unconscious tendencies that motivate a person to drink. Suffice it to say that none of these factors have experimental verification but are clinically-derived hypotheses from intensive case studies. They therefore remain the subject of conjecture. Empirical testing by the above-mentioned workers, however, revealed little support for the hypotheses of oral fixation or latent homosexuality.

Menninger (1938) attempted to explain alcoholism on the basis of teleological factors, viz. a rather sedentary form of self-destruction to avert a greater

self-destruction as a result of anger towards one's parents. This also allows regression to overcome earlier oral frustration, and drinking represents symbolic revenge against parents.

Most analytical theories, therefore, appear to be based on a set of systematized beliefs. It seems fallacious to attempt to force all alcohol addicts into a common aetiological mould, a practice that appears to be at variance with clinical observation. The principle of over-determination typifies psycho-analytic theory which portends an excess of interpretative data with regard to alcoholism but which precludes useful discourse.

In fairness to psycho-analysis, it must be stated that its central concern remains the provision of frameworks for the understanding and possible treatment of individual cases. Although psychodynamic treatments have been widely used with dubious results, Glatt (1974) feels that they have singularly failed. Furthermore, the high incidence of alcoholism precludes use of analytical therapy from an economic point of view, and it provides no paradigm for prophylaxis.

(4) Personality theories

The rationale underlying these theories is that a certain constellation of personality characteristics will predispose an individual with certain of these traits in his make-up to alcoholism. Lisansky (1960) in her

extensive review, identified the following important characteristics that appear to be consistent with clinical observation :

- (a) An intense dependence-interdependence conflict;
- (b) An intensely strong need for dependence;
- (c) Poor defences against frustration and conflict;
- (d) Numerous unresolved love-hate ambivalences.

Support for this view has been given by Blane (1968). Pharmacologically, alcohol reduces conflict and it can therefore increase and decrease dependence; in the former it will produce counter-dependence with the illusion of freedom. Bacon (1974) has given further support for the dependency-conflict hypothesis in a cross-cultural study that took further cognizance of developmental variables. These formulations are reputed to account for the large discrepancy between male and female alcoholics; the latter have immunity since their dependency needs are met while males are expected to show competitiveness and independence.

Retrospective follow-back studies provide a useful means of identifying premorbid personality factors in alcoholics. McCord et al. (1960) in their classic Cambridge-Somerville studies show that in their formative years, alcoholics tend to be independent, aggressive, assertive and tend to exaggerate masculine behaviour.

The weakness of this study is that variability in age of onset of alcoholism was not controlled. Abelson (1973) has shown that the onset of alcoholism is earlier in gamma alcoholics than in delta alcoholics. Thus the study of McCord et al. (1960) could easily have identified predisposing characteristics of gamma alcoholism.

A similar study of this kind was undertaken by Jones (1968). The findings were, in substance, similar to those of McCord et al. (1960) and controlled for many variables. The difficulty here, however, was that the most important variable, alcoholism, was left undefined and the term "problem drinking" was used. The samples in neither study were representative since the majority of subjects were referred by social agencies, and probably represent bias toward alcoholics having anti-social problems rather than naturalistic or other relevant difficulties.

Certainly, the traits in the above-mentioned studies are those commonly observed clinically, and numerous modes of treatment are often empirically devised on this basis. The inherent advantages of this form of enquiry are numerous, the implication for prevention being of paramount importance. The inherent difficulty in this form of research is that of having to survey a large population owing to the low incidence of alcoholism. A further factor is that in the time taken for this population to develop alcoholism, other investigative pursuits might prove relatively more rewarding.

(5) Learning Theories

Consistent with learning principles, an increase in drinking behaviour is a result of its positive consequences. Since the effects of alcohol will be extensively discussed in Chapter Four, initial discussion will be restricted to the implications for research. Mellor (1974) asserts that any explanation involving alcohol effects, is essentially of a teleological nature. Teleology, in this sense, means that an effect precedes its cause. Drinking occurs for the purpose of overcoming a future event, e.g. drinking to avoid anxiety in a forthcoming social situation.

Here the epistemological factors are of extreme importance. Mellor (1974) asserts that the concept of purpose and the propositions pertaining to them, place them outside the ambit of objective empirical investigation. Essentially, they are generalizations that have been established by induction without hypothetico-deductive support as the effect precedes the cause. Teleological explanation, however, has considerable value as it has as its major goal the systematic organization of empirical laws. The Kantian viewpoint offers two reasons :

- (i) Questions asked about ends might obtain questions about means and therefore have heuristic value in identifying causal laws.

- (ii) Supplementary knowledge of available causal interpretations.

Learning theory hypotheses, therefore, appear to be the restatements of alcohol-effects hypotheses that are empirically testable. A typical example of this type of formulation is evident in the work of Ludwig and Wikler (1974) who have shown that craving and LoC can be explained by use of conditioning and other forms of learning theory.

Conger (1956) initiated research that espoused Hullian learning principles. This work was influential in launching further research that can be said to fall under the rubric of the Tension Reduction Theory (TRT). This theory assumes that tension is a generic term for an aversive state in an organism.

Empirical support for TRT can only be obtained by two forms of investigation :

- (1) The aim of this form would be to show that alcohol consumption modifies that type of behaviour which is thought to be indicative of an inner state of tension.
- (2) This second approach attempts to show that tension will increase alcohol consumption.

Conger's (1951) original study typified the first approach. Rats were conditioned to an approach-avoidance

conflict by the use of food and shock at the end of an alley. Since escape and avoidance are considered to be controlled by fear and anxiety, any changes in these particular behaviours, following the consumption of alcohol, would be seen to be indicative of support for TRT. Indeed, Conger (1951) found this to be the case.

The inferential leap to man then occurred, the claim being that alcohol reduces neurotic fears in man. Since tension reduction is seen to be contingent with alcohol intake, the tension reduction response becomes conditioned. However, the nature of the drinking produces negative physical, psychological and social consequences. In order to resolve this issue, Conger (1956) later made use of a temporal gradient of reinforcement to explain the limited influence of these negative consequences of alcohol abuse. This measure indicated that the negative consequences occurred long after the actual drinking and thus had little effect on weakening the drinking behaviour. Indeed, illness, guilt and social censure might even induce tension which would consequently reactivate the drinking response.

In regard to the second approach, Horton's (1943) sociological work could be considered as a natural test of TRT. The study by Masserman and Yun (1946) has shown that not only was alcohol made acceptable to rats in a conflict situation, but that the conflict increased their intake. However, results from human experimentation

(Mello, 1972) were at best equivocal and, as later evidence will show, it appears that "tension reduction" is largely dose dependent.

Cappell (1975), in a review of TRT, states that numerous alternative explanations can be made with regard to animal studies. These studies invariably take place in a restricted environment with highly circumscribed contingencies. No generalizations of these conditioned behaviours have been shown. Cappell's (1975) view appears limited. Alternate interpretations of the increase of alcohol intake could easily be invoked, viz. caloric value of alcohol or that alcohol was merely a substitution for a deprivation of liquid. Most human studies involved correlational significance. The link between independent and dependent variables could be incidental or related to other phenomena. More circumspection in research strategy is needed for TRT to be the basis for rewarding investigative endeavours in the future.

There have also been some approaches to explain alcoholism on the basis of State-dependent learning. Storm and Smart (1965) prefer a theory based on specific effects of alcohol, rather than one based on tension reduction. Described as "dissociation theory", it has been shown that as doses of alcohol increase, the transfer of experience to sober states is decreased. Further, behaviours learned during drinking generalize

along the various levels of intoxication. Goodwin (1974) found that if State-dependent learning does occur, it does so under extreme conditions of intoxication. A further important finding was that alcoholics showed no greater propensity for State-dependent learning than normals. Finally, one cannot understand why the "dissociation" is selective with the positive aspects of drinking having immunity and only the negative aspects being subject to alcoholic blackout. Furthermore, if dissociation does take place, it is difficult to understand how the State-dependent learning induces behaviours that are aimed at a return to a state of intoxication.

Bandura (1969) has attempted a comprehensive explanation of alcohol addiction on the basis of positive reinforcement. He accepts many of the TRT assumptions, at least those which account for alcohol's initial reinforcing ability, and therefore an individual's subsequent psychological addiction. There are added factors which reinforce continued consumption of alcohol with the development of physical addiction. Typical of these would be drinking in order to get the rewards of social interactions as a result of imbibing with companions. Alcohol consumption in this case would serve an instrumental rather than a reinforcing function.

Bandura (1969) states, however, that the above conditions are not sufficient to account for alcoholism. He feels that social learning is of great importance. The majority of persons who experience stress do not

become alcoholics. Individuals in a milieu that either circumscribes the use of alcohol, or encourages its use for stress or boredom, must necessarily be influenced in their use of the drug. This influence is achieved through vicarious learning or social modelling.

In summary, therefore, there appears to be substantial evidence to show that there is a multiplicity of causes for alcoholism. Some factors appear innate, some are related to the effects of the drug, while others appear to be acquired.

Logic would be foresworn if any one theory would attempt to accommodate all the above factors. From the perspective of research the complexity of the situation may appear parlous to the researcher. However, learning theory and its ability to incorporate the alcohol-effects hypotheses appears to be a useful paradigm for the purposes of research.

CHAPTER THREE

THE HETEROGENEITY OF THE ALCOHOLIC POPULATION

Jellinek (1960) proposed that there are at least two types of alcoholic drinking patterns that occur in individuals :

- (1) A gamma-type who suffers a loss of control of his drinking. This type is able to abstain from drinking for varying periods of time. However, once drinking has commenced, this person is unable to stop and this leads to a so-called "binge".

- (2) A delta-type who suffers an inability to abstain from drinking but who can control the intake of alcohol. The delta-type alcoholic is usually unable to abstain for longer than a period of twenty-four hours.

Jellinek (1960) and Pittman and Snyder (1962) invoked a social and cultural explanation to account for these different drinking patterns. Jellinek (1960) hypothesized that gamma alcoholics experienced a rapid onset of withdrawal and he felt that this led to continued (and hence "binge") drinking. The delta alcoholic was assumed to be less responsive to these withdrawal situations. Furthermore, Glatt (1974) felt that the two types might represent a difference between addiction and habituation. This latter phenomenon implies continuous use of a drug without an increase in the dose taken.

Several researchers have shown, however, that this distinction is related to differing personality factors. Vogel (1961) found that extroverts tended to drink in bouts and in company, while introverts tended to be continuous drinkers who were socially isolated. She did not directly relate gamma-delta type alcoholism to extroversion-introversion.

Walton (1968), however, was the first worker to show that there were numerous personality factors which played a part in determining the form of alcoholism. He showed that "loss-of-control" (gamma) alcoholics tended to be impulsive and aggressive as they feared the effects that their behaviours would have on others. He found that the direction of their excessive hostility tended to be intropunitive, as measured on the Foulds Hostility Scale. The delta alcoholics were significantly different from the gamma alcoholics on these measures. Although only seen as a trend, Walton (1968) confirmed Vogel's (1961) work which illustrated that gamma alcoholics tended to be more extroverted.

The findings of Stein, Niles and Ludwig (1968) were similar. The psychiatric histories of their patients lead them to conclude the following with respect to the loss-of-control alcoholic, who was significantly different to everyday drinkers on the following functions :

- (a) they tended to have a greater family history of psychiatric illness and alcoholism;
- (b) they showed a greater tendency to have been delinquent and also had a history of "dropping out";

- (c) they exhibited a greater degree of social disorganization that manifested itself in the form of police contact and constant geographic instability;
- (d) they had poor reactions to drinking; and
- (e) they suffered greater physiological damage which took the form of numerous and longer blackouts and a higher incidence of Delirium Tremens.

As Walton (1968) indicated in his study, care must be exercised in interpreting this data as it could be indicative of personality features that are a consequence of these two forms of alcoholism. Although it has not been commented on, there does appear to be some consistency in outcome in all these studies. A poor response to alcohol and longer and more numerous blackouts would be typical of the response that extroverted individuals would make to this CNS depressant. Eysenck's (1955) prediction is that extroverts with high cortical inhibition would respond poorly to alcohol. The low sedation threshold in extroverts is an obvious implicating factor. It is a pity that workers who differentiate alcoholics on the basis of their drinking patterns fail to integrate many similar findings through a common theoretical interpretation.

Abelsohn and Ben-Arie (1971) have shown that gamma alcoholics tend to have poor self-acceptance as compared to delta alcoholics. Abelsohn (1973) has shown that gamma alcoholics have a significantly earlier age of onset of alcoholism than delta alcoholics.

Tomsovic (1974) assumed that a particular psychiatric disorder or personality type was associated with gamma-type drinking. He could not confirm this using the Minnesota Multiphasic Personality Inventory. However, certain characteristics (some confirming previous work) significantly differentiated the two types. In summary he concludes :

Many binge drinkers seem to be seeking an intense psychic experience that pre-empts social obligations while the continuous drinker tries to achieve a steady tranquil state as he plays his social roles The continuous drinker, in contrast must learn to cope with his existential problem of boredom, depression and anxiety.

Tomsovic, 1974, p.563

When embarking on research, the assumption that the alcoholic population is a homogeneous one, is fraught with difficulties. An aggregate of scores obtained from an alcoholic sample will tend to average out and compress composite profiles. This could obfuscate real issues and significant differences. Van der Spuy (1972), using a defined homogeneous sample, found that, on close examination of his results, many individual subjects did not necessarily show group trends. Varying results from similar studies could be accounted for in terms of the differing composition of the alcoholic samples used.

This appears to be of special importance when alcoholics'

responses to alcohol are being used as the dependent variable.

Wallgren and Barry (1970) comment :

Most of the studies indicate beneficial effects shortly after drinking, especially if the quantity is small or moderate, whereas adverse effects on mood occur after prolonged consumption of large amounts. Conclusiveness of the findings is limited by the small number of subjects used in most of the experiments, because there is great variability among alcoholics in their account of how they experience the effects of alcohol. More comparisons are needed between gamma and delta alcoholics, or other categories of alcoholics.

Wallgren and Barry, 1970, p. 517

Naturally these factors have implications for treatment. If a heterogeneous population of alcoholics can be divided into types, then corresponding and appropriate treatments could be found for them. Initially, treatment modalities are usually empirically based, and the treatment of alcoholism appears to be no exception. Multidisciplinary treatment is probably a representation of the multiplicity of causes of this condition.

In general, Mellor (1974) makes a plea for a close inspection and utilization of multiple causes and their operation in alcoholism. Certainly one feels that theoretical

statements in the field thus far exhibit a characteristic lack of criteria for the independence and sufficiency of causes.

CHAPTER FOURTHE PSYCHOPHARMACOLOGY OF ALCOHOL(1) PHYSICAL PROPERTIES

Ethanol is a monohydric alcohol ($\text{CH}_3\text{CH}_2\text{OH}$, $M=46,07$) and is derived from the corresponding hydrocarbon ethylene (CH_3CH_3) by substitution of a hydrogen atom for a hydroxyl group. Ethanol occurs naturally as a result of the fermentation of a wide variety of substances and is easily derived synthetically by the above process.

Ethanol is totally miscible in all proportions in water, but itself behaves more like a solvent than a solute. It is absorbed by a process of simple diffusion as the intermolecular forces for this type of compound are very weak. This accounts for its low boiling point ($78,15^\circ\text{C}$). The diffusion process is aided by the small molecular diameter of alcohol, viz. $5,62 \text{ \AA}$. Kinetically, not only is the diffusion through the gastrointestinal wall rapid, but the molecule is easily absorbed through cell walls. (Wallgren and Borg, 1970, pp.17-38).

The ease of intestinal and cellular penetration and the rapidity of circulation of alcohol through the body, results in alcohol reaching the pharmacologically active sites in the cortex soon after ingestion. These contingencies, therefore, could be strongly reinforcing.

(2) SITES AND MODE OF ACTION

Ethanol acts as a depressant drug, whose action is poorly understood. Increasing amounts of the drug will eventually induce stupor and death when the lower levels of the brain, such as those controlling respiratory function, are depressed. Alcohol penetrates all cells in the body, but discussion will be restricted to its effect on the Central Nervous System (CNS).

It appears that the first area that is affected is the midbrain reticular activating system, (Ritchie, 1970). This effectively frees the lower areas from higher cortical control and results initially in less restrained behaviour. As blood alcohol concentration (BAC) increases, ethanol has a systematic and selective depressant effect on the various associative areas of the cortex. Mental processes affected by training and previous experience and an ability for fine discrimination are all impaired. Further ingestion of alcohol leads to poor perceptual abilities and motor incoordination. When a BAC of 400mg/100ml is reached, a state of surgical anaesthesia develops which can then be followed by coma and respiratory paralysis.

(3) DEPENDENCE PRODUCING EFFECTS

The essential pharmacological action of a drug that leads to its abuse is that of

enhancing pleasurable excitement, escape or relaxation - in a word 'reward', and of lessening painful boredom, anxiety or defensiveness - in a word 'punishment'.

Collier, 1971, p.45

A drug in itself will therefore have a potential abuse liability depending on its effect. Initial consumption, as an antecedent event in physical dependence, will thus be largely dependent on the psychopharmacology of the drug.

(a) Tolerance :

Although tolerance is essentially a physiological phenomenon, it would appear prudent to discuss it at this juncture as it seems to be a necessary condition for physical dependence.

Kalant, Le Blanc and Gibbons (1971) state that tolerance to a drug is such that an increase in quantity is required to produce the same specified degree of effect. Conversely, it may be stated that less effect may be produced by the same quantity of alcohol. There appear to be two hypothesized mechanisms that attempt to account for tolerance :

- (i) Dispositional tolerance : this refers to the mechanisms that are instrumental in transporting and altering alcohol, viz. absorption,

excretion, distribution and metabolism.

According to the above authors, there appears to be little support for the establishment of tolerance on the basis of transport variables. Although there is some support for increased metabolic rates in alcoholics, these results seem equivocal. This was confirmed by the extensive work of Mendelson (1968).

- (ii) Functional tolerance : this term is preferred to tissue tolerance as it implies that a difference exists between behavioural and physiological or biochemical tolerance. The divisive issue of mind and body will be avoided.

The studies of Mello (1972, 1973) confirm that a central adaptive process subserves behavioural tolerance. These studies, as well as those of Goldberg (1943), also serve to indicate that alcoholics can perform cognitive and behavioural tasks with significantly higher BAC levels than normals.

Functional tolerance also appears to be exhibited on a biochemical and physical level. Holmberg and Martens (1955) found that equivalent doses of alcohol caused greater electroencephalogram and electrocardiogram changes in

normals than in alcoholics. Mizoi et al. (1963) found similar results, using subjects ordered according to their drinking patterns, with regard to electromyographic responses.

No adequate theory or evidence currently exists to account for functional tolerance on a cellular level. It remains speculative whether the phenomenon is a result of an inherent predisposition, whether it is acquired, or if it is a combination of the two.

Most of the support is inferred, (Kalant et al. 1971). The occurrence of cross-tolerance that alcoholics have for other CNS depressant drugs is also inferred by exclusion of other hypothesized mechanisms. It appears, therefore, that after exposure to sufficient quantities of alcohol over an undefined period of time, tolerance results as a consequence of cellular adaption. Tolerance is therefore assumed to be indicative of a "pathological" change, which, in time, is the basis for physical dependence, the basic tenet of the disease concept.

(b) Physical dependence :

Although tolerance and physical dependence were once believed to be sequential and

inseparable aspects of the same underlying addictive process, our current understanding of this relationship suggests that, although physical dependence is invariably accompanied by the induction of tolerance, drug tolerance may occur independent of physical dependence.

Mello, 1976, p,182

This statement illuminates the fact that little is known about the development and persistence of alcohol dependence. Physical dependence is signalled by the precipitation of a typical alcoholic withdrawal syndrome due to a deceleration of alcohol intake. Amelioration of these symptoms is achieved by further ingestion of alcohol, indicating that normal neural activity cannot be achieved in the absence of alcohol, once introduction to alcohol takes place.

The hypothesized mechanisms for this phenomenon are beyond the scope of this study, but, far more important, is the role of physical dependence in the maintenance of drinking. More cogently, why is drinking resumed by most addicts in the absence of physical evidence of withdrawal?

(c) Psychological dependence :

It is an accepted pharmacological truism that only

CNS depressants cause physical dependence. It is generally accepted that a positive psychological disposition to a drug is a prerequisite for its continued use, which in turn pre-empts physical dependence. The quote by Collier (1971) is of special significance (see Section (3) of this chapter). There does, however, exist considerable controversy regarding the relationship between physical and psychological dependence.

It would seem advantageous, therefore, to define drug dependence in general. The 1964 World Health Organization Expert Committee defined drug dependence as follows :

A state arising from repeated administration of a drug on a periodic or continuous basis.

Psychic dependence is seen to be a state of mind in an individual which requires periodic or chronic administration of a drug for pleasure or the removal of discomfort.

Archibald (1970) has shown that withdrawal symptoms may be induced in persons who have a limited drinking history. Collier (1971) contends that psychological and physical dependence are the subjective and objective manifestations of the same phenomenon, i.e. physical dependence. This

latter view in particular appears simplistic. It is known that the majority of persons suffering from alcohol dependence, have a history of insidious onset. Tolerance is small, relative to other depressant drugs, as is the relative time taken for physical dependence to be manifest, viz. a relatively protracted period of time. It would seem more logical to assume that certain individuals initially show a preference for alcohol due to its effects on them. It is to these possible reinforcing effects that we shall now turn.

(4) THE EFFECTS OF ALCOHOL ON PSYCHOLOGICAL FUNCTIONING

According to Kassin (1974) there are four major psychopharmacologic effects of alcohol :

- (i) an euphoric effect;
- (ii) a disinhibiting effect;
- (iii) a relief from anxiety and depression; and
- (iv) a relief from physical dysphoria.

Kassin's view is simplistic and one feels that the following factors could be more relevant :

- (a) As stated previously, alcohol has a selective and successive effect on the CNS, hence most affective and behavioural changes are dose related.

- (b) Kissin's (1974) view takes no cognisance of tolerance.
- (c) Many of the above four defined effects are related and correlate highly on corresponding measurement scales, ostensibly held to be independent (Lubin and Zukerman, 1967).
- (d) No allowance for personality differences are made.
- (e) No allowance for the influence of an individual's affective state, contingent with alcohol use, is made.
- (f) The environmental determinants of emotional states are unaccounted for when alcohol is ingested.

A more useful approach appears to be that formed by Russell and Mehrabian (1975). In their view, emotions may be delimited along three independent, bipolar dimensions, viz. pleasure-displeasure, level of arousal, and dominance-submission. Level of arousal could range from sleep to excitement, while dominance-submission could range from submissiveness or lack of control to dominance and feelings of control over one's environment.

It we recognise the influence of dose and environment, a finer understanding of the psychological effects of alcohol will be achieved. This will enhance the understanding of the more important effects of alcohol on personality functioning.

Anecdotal and experimental evidence that alcohol, in small doses, has a mood elevating and anxiolytic effect pervades the literature, (Freed, 1970; Williams 1968). Depression (displeasure, low arousal, submissiveness) and anxiety (displeasure, high arousal, submissiveness) are therefore closely related. According to Russell and Mehrabian (1975), moderate doses of alcohol raise arousal and increase pleasure and dominance. Large doses decrease arousal and dominance and bring about slight increases in pleasure. This probably accounts for the high correlation found to exist between anxiety and depression in numerous studies, since they are seen to share common properties. (Lubin and Zuckerman, 1967).

Dominance is an important factor which plays a part in alcoholism. The resolve of many alcoholics (and normals) to drink to obtain "Dutch Courage" is well known. There has been previous comment on the influence of alcohol on avoidance behaviour. However, the approach of Russell and Mehrabian (1975) offers an avenue for the understanding of this particular form of behaviour within a certain dimension of personality, i.e. persons who feel passive, submissive, dependent, or impotent to act in their environment will free themselves from these feelings by the ingestion of moderate doses of alcohol. According to these authors, a moderate dose of alcohol would be accompanied by an increase in dominance behaviour.

Blake (1965) and Mortorano (1974) show that there is overwhelming clinical evidence to support the view that assertiveness (or lack of it) is an extremely important factor in the treatment of alcoholics. Lazarus (1968) espouses this viewpoint to the extent of claiming that appropriate treatment can only be achieved through assertiveness training. The above workers all conclude that, concomitant with an increase in assertiveness, an obvious indicator of dominance, is an increase in self-esteem which then leads to greater emotional freedom.

(5) THE EFFECT OF ALCOHOL ON PHYSIOLOGICAL FUNCTIONING

As has been previously mentioned, alcohol penetrates tissue easily, and causes numerous changes in systemic functioning. It would seem appropriate, therefore, to study physiological changes that affect personality and emotional functioning.

It has long been accepted that levels of autonomic arousal (Duffy, 1962; Hebb, 1955; Lindsey, 1951) are components of numerous and diverse emotional states. Duffy and Lacy (1946) showed that basal skin conductance (BSC) was an accurate indicator of sympathetic innervation. The higher the BSC, the greater the sympathetic innervation and the greater the degree of arousal in the organism. This approach is consistent with the analytical schema of Russell and Mehrabian (1975) who consider arousal a central feature of emotions.

In their classic monograph, Lader and Wing (1966), both discussed and illustrated the relationships between sedative drugs, emotions and various physiological measures. From this study one could extrapolate that there exists a relationship between the physiological effects of alcohol and emotional states. It is to these particular interactions that attention will be given.

The majority of studies conducted on the effects of alcohol on BSC have been restricted to normal subjects. Other studies that have made use of skin measures have employed the Galvanic Skin Response (GSR) as a dependent variable. This measure is assumed to represent autonomic reactivity. However, this form of data is not easily interpreted.

Work in this field has lead to much confusion. Greenberg and Carpenter (1957) and Carpenter (1957) showed that when normal subjects were given a dose of alcohol (0,5g/kg), there was an initial increase in BSC which was then followed by a decline. A factor which compounds confusion in this area is that the subjects responded differently to various types of alcoholic beverages, even though each individual's beverage contained the above-mentioned concentration of alcohol. The investigators also found that varying amounts of alcohol tended to change BSC levels. Coopersmith (1964) produced equally equivocal results. The most interesting

aspect of this study is that the great variability between the subjects' individual responses to alcohol were neglected.

Cutter, Green and Harford (1973) divided alcoholics into extroverts and introverts. They found that, consistent with Eysenck's (1967) view of arousal, there was a significantly higher incidence of risk taking (an index of arousal) in intoxicated introverted alcoholics than in intoxicated extroverted ones.

It seems, therefore, that the personality of the individual makes some contribution to the level of arousal induced by alcohol. A disregard of this phenomenon might reduce the validity of such research.

(6) THE EFFECT OF ALCOHOL ON ENDOCRINE FUNCTIONING

Alcohol has the ability to stimulate the sympathetico-adrenomedullary system (Perman, 1958). This is reflected in the increased levels of the catecholamines in urine. Results from acute studies in normals vary considerably. Abelin, Herren and Berli (1958) found that an acute ingestion of alcohol resulted in a twelve-fold increase in epinephrine and a four-fold increase in norepinephrine. In a similar study, Perman (1958) found a moderate increase in epinephrine but no increase in norepinephrine.

Ogata, Mendelson, Mello and Majchrowicz (1971) showed that there is abundant evidence that alcohol increases

catecholamine response. They further found that this response is dose related. This indicates that, unlike most physiological responses to alcohol, no tolerance exists as far as adrenal-medullary response is concerned. During withdrawal there is a further increase in catecholamine production, but this is probably related to other mechanisms.

An added feature of this study was the finding that many emotions compete during intoxication. For example, some of the subjects' emotions varied with respect to depression, aggression, anxiety and euphoria during the experiment. Confusion exists in the field as a result of these numerous diverse findings. Certainly what appears to compound the situation is that no work has resolved the question of whether sympathetic activation is related to a direct effect of alcohol on autonomic centres in the mid-brain, or whether it is an indirect consequence of behavioural changes due to alcohol ingestion. Wallgren and Barry (1970, p.175) comment:

Controlled studies capable of demonstrating the factors through which variability in the response arise, should yield information of great interest for our understanding the significance of the effects of ethanol.

(7) THE EFFECT OF ALCOHOL ON PERSONALITY FUNCTION

Why does a man drink heavily? He wouldn't

drink unless he got a change in personality that was satisfying to him ... Some of these people undergo surprising changes of personality. Meek men become belligerent, and so on.

Packard, 1962, pp.40-41

One of the most important observations made in the mammoth work of Mendelson and his co-workers (Mendelson, La Dou and Solomon, 1964; McGuire, Stein and Mendelson, 1966; Mello, 1972; Mello, 1976) is that extensive or chronic drinking appears to lead to a deterioration in mood-state in both normals and alcoholics. It seems reasonable to conclude, therefore, that any desirable change in an individual's behaviour can be expected to arise from the acute effects of alcohol.

Having reviewed work involving alcohol and mood-states, Van der Spuy (1972) concludes that the alcoholic appears to benefit less from alcohol than the non-alcoholic. When the alcoholic does benefit from drinking, the improvement is still less than that of non-alcoholics.

It seems incongruous, therefore, that alcoholics should drink at all. A possible explanation could be that there are certain behavioural changes induced by alcohol that are extremely rewarding to the alcoholic. The benefit that is derived could outweigh other

negative consequences that could even be concomitant with these positive effects. Partington (1970) reported that alcoholics saw themselves as dependent and submissive when sober, but dominant and independent while intoxicated. Keehn (1970) asked alcoholics to describe themselves on the Eysenck Personality Inventory, first when sober and then when they were in an intoxicated state. A significant difference in extroversion was found, i.e. the alcoholic felt more pleasant, aroused and dominant when intoxicated. Gross and Carpenter (1971) have shown that alcoholics exhibit higher neuroticism scores than normals, and that the alcoholics experienced this form of trait-anxiety as undesirable.

In summary, it appears that the acute effects of alcohol are desirable for alcoholics. The importance of this state to the alcoholic is at the expense of numerous negative psychological and physical consequences. The dimensions along which change is desirable appear to be related to passivity, submission and dependence, extroversion and neuroticism, anxiety and arousal. The aim of this study is to investigate which dimensions are important to two different types of alcoholics.

CHAPTER FIVE(1) THE PROBLEM

In the past, fruitful research into various aspects of alcoholism have been hampered, not only by the longevity of spurious notions concerning the phenomenon, but also by rather simplistic experimental approaches.

It seems almost incredible that the investigation of the "alcoholic personality" continued for so long, considering the dearth of information and understanding that it yielded. The plethora of traits, moods, feelings, dispositions and personality typologies that supposedly predisposed the alcoholic to his malady, eventually became infinite. Equally, the statistical axiom : Correlation does not imply Causation : went unheeded. This type of experimental work relied on the dubious value of inference, especially when aetiological factors were being investigated. As can be observed from the review of studies on aetiology and alcohol effects, many of the results seemed both simplistic and equivocal. Typically it assumed that alcoholics would drink because they were anxious, depressed or orally fixated. Other inferences were that alcohol reduces tension, avoidance behaviour and passivity or dependence. This criticism might appear excessive, but at best these studies would suffer from the deficiencies levelled at any form of ex post facto research.

Further difficulties accrued with the above approach. Even if we accept that cause-effect relationships have

been established, it is often difficult to interpret what constitutes the cause or the effect. As Walton (1968) has stated, many of the factors found in alcoholics could easily be results of their alcoholism rather than their causes.

These studies did make some positive contribution in the form of providing generally testable hypotheses for experimental research. However, their contribution was diminished by the rather pedestrian approach of their investigation of alcohol effects in general. Progress was further retarded by using normals as subjects for work on alcohol effects. The variables that these experimenters considered to be important, viz., cognitive, motor and perceptual functions, threw little light on the understanding of alcoholism.

Animal studies provided another avenue of experimental investigation for testing hypotheses concerning alcohol effects. The analysis of data from animal analogues can at best yield simple effects or relationships that are difficult to interpret. The climb up the phylogenetic scale would then prove to be an equally or more arduous task.

A further misapprehension in the investigation of alcohol effects was the acceptance of the notion of homogeneity. It was believed that there existed a uniform alcoholic population. This general acceptance lasted for decades. What appears to be especially

puzzling in this regard is that concurrent with this acceptance, the heterogeneity of other clinical entities, such as depression and schizophrenia, was easily recognized. A serious compounding factor was that at least one dimension along which this heterogeneity could be delineated, was a naturally occurring one, i.e. different drinking patterns. This phenomenon could at least avoid recourse to intuitive or theoretically based divisions.

It is only recently that a more determined effort (Mello, 1972 and Van der Spuy, 1972) attempted a more comprehensive approach to investigating the effects of alcohol on alcoholics. Their work has at least partially dispelled the unproven acceptance of the supposed benefits that alcoholics derive from their consumption of alcohol and the possible ameliorating effects that alcohol will have on various negative states such as tension and anxiety. Deficiencies in this work have already been discussed (p 42).

Mendelson et al. (1964) likewise attempted to analyse the effect of alcohol on physiological and biochemical functioning. Unfortunately, their work centred on chronic effects. A general criticism that can be levelled at all alcohol-effects experiments is their neglect of the heterogeneity of their samples.

(2) RATIONALE :

The main goal of this investigation was to assess the effect of an acute dose of alcohol on gamma and delta alcoholics.

These two types of naturally-occurring drinking patterns occupy opposite ends of a drinking continuum. It has also been shown that these two types of alcoholics have significantly different personality traits. Consistent with learning theory in general and the alcohol-effects hypotheses in particular, it seemed reasonable to assume that individuals will continue to use alcohol as a result of the positive effects it will have on them. It was therefore anticipated that these two groups of alcoholics would respond differently to alcohol as a result of their differing personalities and patterns of alcohol use.

The choice of dependent variables attempted to satisfy many factors :

- (a) To include measures that would reflect changes that alcoholics consider important when they drink;
- (b) To include measures that are considered important in the treatment of alcoholism;
- (c) To include physiological and biochemical variables;
- (d) To include measures based on a theoretical structure to avoid a purely empirically based investigation.

It thus seemed prudent to include measures from the EPI. Eysenckian theory would provide a theoretical structure for the investigation. The theory also provides drug postulates in which the role of CNS depressants, such as alcohol, have a central place.

Following the work of Vogel (1961), Walton (1968), Keehn (1970) and Tomsovic (1974), a composite picture emerges of the typical delta alcoholic : A quiet, introverted, rather anxious and neurotic individual who has difficulty coping with the normal vicissitudes of life. Lack of assertiveness is a typical problem. On the other hand, the gamma alcoholic is a person who has more inter-personal difficulties related to his impulses. He is unable to cope with intermittent problems. According to Ganong (1967, p.296) elevated levels of epinephrine are usually found in persons who deal with circumstances with which they are unfamiliar. Similarly raised norepinephrine responses occur in person dealing with difficult situations with which they are familiar. Since the latter difficulties appear consistent with those faced by delta alcoholics it seemed reasonable to assume they would exhibit raised norepinephrine levels. Equally circumstances associated with unfamiliar and intermittent difficulties could result in raised epinephrine levels in gamma alcoholics.

An investigation addressing itself to these specific problems has yet to be attempted.

(3) HYPOTHESES

- (1) Delta alcoholics will show a greater increase in extroversion under the influence of alcohol than gamma alcoholics.
- (2) Delta alcoholics will show a greater decrease in neuroticism under the influence of alcohol than gamma alcoholics.
- (3) Delta alcoholics will show a greater decrease in anxiety under the influence of alcohol than gamma alcoholics.
- (4) Delta alcoholics will show a greater increase in assertive behaviour under the influence of alcohol than gamma alcoholics.
- (5) Delta alcoholics will show a greater change in autonomic arousal under the influence of alcohol than gamma alcoholics.
- (6) Delta alcoholics will show a lesser increase in epinephrine production under the influence of alcohol than gamma alcoholics.
- (7) Delta alcoholics will show a greater increase in norepinephrine production under the influence of alcohol than gamma alcoholics.

CHAPTER SIX(1) SUBJECTS :

The subjects were 26 White male patients who were attending the William Slater Hospital, Cape Town. The patients were tested while seeking treatment at the hospital and the period of testing lasted from January 1977 to June 1978.

Ethical considerations partially governed the selection criteria. All the patients had had some alcohol in the previous 72 hours. These ethical considerations ensured that the experiment did not recommence drinking in any of the patients who had been abstinent for a lengthy period of time. Patients showing signs of acute intoxication or gross withdrawal symptoms were excluded. Most patients had been "nibbling", had ceased drinking in the previous 72 hours, or felt that their drinking might once again be getting out of control.

The diagnosis of alcoholism at the Hospital is consistent with the operational definition of alcoholism, advocated by Frightner, Robins, Guze, Woodruff, Winokur and Munoz (1972). A "definite" diagnosis of alcoholism is made when a patient manifests any symptoms in at least three of the four following groups :

- (A) (a) Any indication of alcoholic withdrawal, e.g. tremulousness;

- (b) A history of medical complication due to excessive drinking, e.g. polyneuropathy;
 - (c) Alcoholic blackouts;
 - (d) At least two alcohol benders which have lasted for 48 hours or more.
- (B)
- (a) The subject has not been able to stop drinking when he has wanted to;
 - (b) The subject has tried to discipline and control his drinking, e.g. only drinking with other people;
 - (c) Drinking before breakfast;
 - (d) Drinking non-beverage alcohol, e.g. after-shave lotion.
- (C)
- (a) Arrests for drinking;
 - (b) Drink-associated work trouble;
 - (c) Drink-associated fighting.
- (D)
- (a) The subject thinks that he drinks too much;
 - (b) The family objects to his drinking;
 - (c) Loss of friends because of drink;
 - (d) The subject feels guilty about his drinking.

In order to establish the pattern of alcoholism, the experimenter's classification was verified by two independent judges. The first was the Consultant Psychiatrist in charge of the Hospital, and the second was a psychiatric registrar, who knew the patients well. Agreement was one hundred percent. This was thought advisable as the pattern of alcoholism was the independent variable.

The criteria used for the classification of type was a modified version of that recommended by Walton (1968). The criteria conformed to those used to define the alcohol-dependent syndrome. Impaired Control rather than Loss of Control was used.

Gamma Alcoholism :

- (a) The problem experienced with drinking was a consistent inability to stop drinking once it starts;
- (b) Periodic intoxication;
- (c) Periods of abstinence.

Delta Alcoholism :

- (a) The problem was a consistent inability to abstain from drinking;
- (b) Drinking virtually every day;

(c) Relative absence of periodic intoxication.

The patients were all voluntary subjects. All delta alcoholics approached agreed to participate, while three gamma alcoholics refused. This could imply that the gamma alcoholic sample was not representative of the gamma population.

(2) PROCEDURE :

When suitable subjects were found, the experimenter introduced himself as a psychologist who was interested in investigating the effects of alcohol on people who had different types of drinking problems. The procedure of the experiment was then outlined. It was made explicit that the results would remain confidential, that the experiment was totally voluntary, and that medication to ensure easy cessation of drinking after the experiment would be prescribed.

Testing took place between 1400 and 1600 hours to control for the diurnal variation in basal levels of skin conductance and catecholamine production. A south-facing room was used in order to minimize temperature variation.

Although the above control measures were stringent, other variables were beyond the control of the experimenter. The majority of the patients were smokers. Smoking was not allowed for fifteen minutes before the experiment and for its duration. Some patients were on varying doses of medication, usually a benzodiazepine drug. These minor tranquillizers and smoking will affect anxiety, skin conductance and catecholamine production. However, alcohol administration would be expected to minimize these effects.

The patients voided their bladders before the

experiment. They were asked to sit in an easy chair and the electrodes for the skin conductance measures were then attached. In order to control for possible practice effects and the remote possibility of personality changes in the time between testing, half the gamma and half the delta subjects were assessed, first under the influence of alcohol, and then when they were sober. With the other half of the subjects, this procedure was reversed. The order of the tests was :

- (A) Sober :
 - (a) Eysenck Personality Inventory
 - (b) Assertion Inventory
 - (c) Anxiety Scale

- (B) Intoxicated:
 - (a) Assertion Inventory
 - (b) Eysenck Personality Inventory
 - (c) Anxiety Scale.

The initial reason for the above ordering of tests was that, if patients were going to be intoxicated, the most difficult test should be administered first, as the level of intoxication would then be at its lowest. This proviso proved unnecessary, as most patients had little difficulty in completing testing under both situations.

The administration of alcohol was achieved by a compromise between the methods of Fleetwood (1955) and

Anton (1965). A standard subject mass of 70kg was used. A 50ml standard tot of 80% proof South African brandy was given to the subjects. Each ml of brandy contained 0,33gms of alcohol. Three such tots were given :

- (i) At the beginning of the experiment. This had to be drunk within ten minutes;
- (ii) The second, poured after the completion of the first, also had to be drunk within ten minutes;
- (iii) The third, poured after completion of the second, also had to be drunk within ten minutes.

This volume of alcohol was adjusted according to the subject's mass, such that every subject received 0,70g alcohol/kg body mass within 30 minutes. This resulted in a maximum blood alcohol level of 0,065%.

Final measures and testing commenced 10 minutes after the last drink. The diuretic effect of alcohol ensured easy voiding of the bladder at the cessation of testing.

Provision was made for the testing of Afrikaans speaking subjects. However, all these patients (seven) elected to answer the tests in English. It did not appear that any language difficulties were experienced.

(3) TESTS :

(A) The Eysenck Personality Inventory (EPI)

The EPI was developed as the basic assessment tool for Eysenck's theory of personality. It purports to measure two dimensions of personality, viz. extroversion-introversion and neuroticism-stability.

Theoretical basis : Eysenck's (1955) theory of personality has undergone some changes since the enunciation of his original postulates in 1955. Not all of these revisions were satisfactory. Eysenck's (1955) theory has led to an enormous volume of experimental and clinical research. Only the features that have a bearing on testing will be discussed.

The theoretical construct of excitation/inhibition is proposed as a genotypic basis for personality. It is assumed that strong cortical excitation will lead to controlled introverted behaviour, while weak cortical excitation will lead to extroverted behaviour as the cortex exerts little control over lower level functioning. Individual differences, resulting from this assumed function of the CNS, were reflected in experimental work done on psychomotor performance, conditioning and perception. Phenotypic properties of personality are assumed to be the results of interaction of these genotypic properties and the environment, and

manifest themselves in the form of the primary traits of extroversion-introversion.

Typically, extroverts are sociable, group dependent, easy-going and enjoy change in their environment. Introverts are usually quiet, have few but close friends, are introspective, keep their feelings under control and generally rely on themselves for their well-ordered mode of life. There are 24 items in the EPI that reflect these prime traits.

The second source of individual variation is considered to be that governed by the dimension of neuroticism. Along with inhibition, this concept has had a rather chequered theoretical career, and it has been left largely to other workers (Claridge 1967) to expand and define this dimension. Originally, Eysenck (1955) simply regarded it as a form of drive, related to over-excitability of the sympathetic branch of the autonomic nervous system. In certain later revisions of his theory (Eysenck 1967), he conceded that neuroticism might be related to certain hypothalamic functions. In the latest revision of the manual of the EPI, Eysenck (1976) appears to rely very much on his original formulations. Neuroticism is assumed to reflect a form of trait-anxiety consistent with that measured by the Taylor Anxiety Scale. There are 24 items that measure the dimension of neuroticism in this scale.

Included in the Inventory is a nine item lie scale. No guidance for its use is given other than that a score exceeding five on the scale would cast doubt on the integrity of the responder. Bailey and Metcalf (1967) consider this score to be too low and advise six as a cut off point. In the present study no one subject scored more than five under either condition.

Reliability : Test-retest reliability and split-half reliability fall between 0,74 and 0,91 for the test. Such high reliability is extremely advantageous in acute studies, where scores are attributed to changes in the independent variable.

Validity : It is accepted that criterion-related validity is virtually impossible in the area of personality testing. Eysenck (1976) states that since the EPI is an improved version of the Maudsley Personality Inventory, the validity of the latter ensures the validity of the EPI. This seems acceptable but independent proof would be more satisfactory. Further validity was attempted using independent judges to rate individuals as extroverted or introverted, neurotic or stable. When members of this nominated group answered the EPI, there were clear differences between extroverted-introverted and neurotic-stable groups. A curious statement by Eysenck (1976) is that when there exists a lack of

agreement, it is the judges who are at fault! It is difficult to understand why he makes this assertion. The implication is that one rids oneself of the judges until they validate the test. This contribution to the validation of the test is unacceptable.

The EPI, however, provides the best index of extroversion. The measure of neuroticism is adequate and is made more acceptable for research due to its hypothesized orthogonal relationship with extroversion.

The acceptability of the EPI and Eysenckian theory is enhanced by virtue of its ability to make predictions regarding the influence of drugs on personality.

(B) The Anxiety Scale :

Zuckerman (1960) based his rationale for the development of his anxiety scale on the fact that previous scales, established on the basis of general levels of anxiety, showed large inter- and intro-individual variation. A further disadvantage of these scales (Taylor, 1953) was that they were personality scales of manifest anxiety or trait anxiety (Eysenck, 1964). Zuckerman's (1960) scale was thus devised to measure acute changes in anxiety, as they would occur in drug experiments.

An adjective check-list appeared ideal for this purpose. The final 21 items for the test were obtained through the careful use of criterion groups.

Reliability : This was established by the use of measures of internal consistency and test-retest reliability. Significant levels of $p < 0,001$ and $p < 0,05$ were found for the two forms of reliability respectively.

Validity : It was argued that if the scale could register significant changes in examination anxiety in students, it would be even more sensitive to changes in clinical anxiety. The scale was given to a group of students over a period of 21 days which included three exam days. Significant changes in anxiety were measured on these three days.

The scale consists of 21 items :

AFRAID	FRIGHTENED	TENSE
Calm	NERVOUS	Secure
DESPERATE	Joyful	TERRIFIED
Cheerful	PANICKY	Steady
FEARFUL	Loving	UPSET
Contented	SHAKY	Thoughtful
Happy	Pleasant	WORRYING

A score of 1 is given to a positive response to an anxiety adjective.

Instructions : "Here are a number of words that might describe how you feel. Please say 'yes' or 'no' if the words describe how you feel at the moment."

This test was administered before and after the drinking session.

(C) The Assertion Inventory :

Gambrill and Richey (1975) constructed a 40 item self-report assertion inventory. The purpose of this development was to facilitate the measuring of assertive behaviour in a wide range of situations which involved a variety of individuals.

Three types of information regarding assertiveness (or lack of it) can be derived from the questionnaire :

- (a) Degree of discomfort in relation to specific situations;
- (b) The judged probability of engaging in assertive behaviours; and
- (c) Identification of situations in which a person would like to be more assertive.

For the purpose of this study only the judged probability of engaging in assertive behaviours was used, as the subject of interest was the degree to which alcohol affected assertive behaviours.

Items for the scale were chosen following a perusal of the literature and clinical reports which concerned frequently occurring assertion difficulties. The 40 items fell into the following categories :

- (i) turning down requests;

- (ii) expressing personal limitations;
- (iii) initiating social contacts;
- (iv) expressing positive feelings;
- (v) handling criticism;
- (vi) differing with others;
- (vii) assertions in service situations; and
- (viii) giving negative feed-back.

Scoring on the probability of engaging in this behaviour was done on a 1-5-point scale which is as follows :

- 1 = always do it;
- 2 = usually do it;
- 3 = do it about half the time;
- 4 = rarely do it;
- 5 = never do it.

The sum of these scores is the total assertion score. Low scores indicate high probability of assertive behaviour.

Reliability : Test-retest reliability, estimated by Pearson's correlation co-efficient, was 0,87 for discomfort and 0,81 for response probability. This indicated a high degree of reliability.

Validity : Hersen, Eisler and Miller (1973) have shown that deficits in assertive behaviour are common to a wide range of clinical conditions. Scores between a clinical and normal group were significantly different on response probability when this scale was used ($p < 0,002$). Although not stated, this gives the scale concurrent validity. A significant difference ($p < 0,002$) in response probability scores was also found when the scale was administered before and after assertiveness training to persons requesting this form of help.

Instructions : A slightly modified version of the written instructions was made : "Most people experience difficulty in handling interpersonal situations requiring them to assert themselves in some way, for example, turning down a request, asking a favour, giving someone a compliment, expressing disapproval or approval and so on. I am going to name some situations and I want you to tell me the probability or likelihood of your displaying this sort of behaviour as you feel at the moment. Tell me if you would always do it, usually do it, do it about half of the time, rarely do it, or never do it. For example : Initiate conversation with a stranger; would you :

- (i) always do it;
- (ii) usually do it;

- (iii) do it about half the time;
- (iv) rarely do it; or
- (v) never do it;

as you are feeling now."

Most subjects, under both conditions of testing, had little difficulty in answering the questions. Occasionally statements needed repeating or interpretation. Two slight alterations were made; item 33 was changed from "Quit a job", to "Leave a job". Item 34 was changed from "Resist pressure to 'turn on'", to "Resist pressure to take drugs".

(D) Measures of Skin Conductance:

Skin conductance measures were recorded on a two channel Beckman Dynograph, using the new Type 9842 Galvanic Skin Response Coupler.

The circuit used was that described for the Time-Mark System (Beckman, 1972). The TM switch was put ON, and a 1 sec time constant was chosen at 10 ua constant current excitation.

Calibration: The instrument was calibrated according to instructions for the Type 9842 Coupler given by Beckman (Bulletin Nr 613744, 1972). This involves using standard resistances and plotting their equivalent Time-Mark period. For the sake of convenience this latter measure was converted to Time-Mark frequency. A graph of Time-Mark frequency versus Basal Resistance was then plotted and the relationship was linear (See Fig. I)

Although not advocated by the manufacturers, an obvious check is to hold the two electrodes together as this would give zero resistance. The Time-Mark frequency should thus cut the Y-axis. This was confirmed and indicates a highly accurate and sensitive machine.

The electrodes used were Beckman Biopotential Skin Electrodes with an area of 0,6 sq.cm. A bipolar placement on the medial phalanx of the 1st and 2nd finger of the left

hand was used. A double-sided adhesive disc was employed to attach the electrodes and this, together with Beckman's electrolyte paste ensured proper contact between electrode and the surface of the skin.

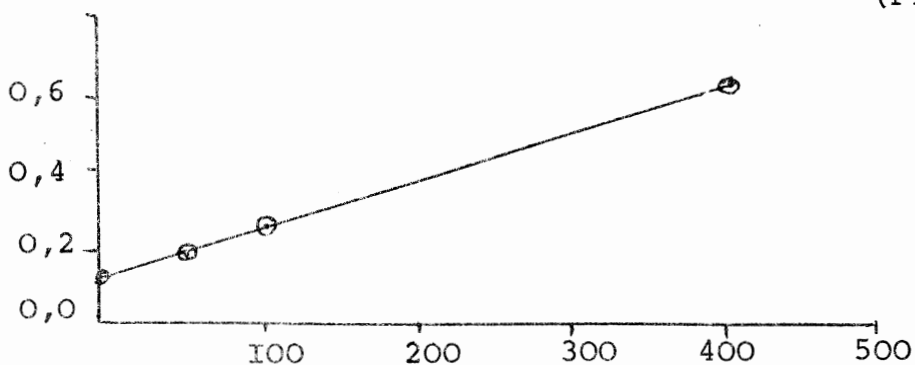
Results were converted to standard units of measurement, i.e. $\mu\text{mho}'\text{s}/\text{cm}^2$. Since a bipolar placement was used, the calculation was as follows :

$$\begin{aligned} \text{Total Basal Resistance} &= xk\Omega \\ &= 1000x\Omega \\ \text{Therefore Basal Resistance} &= \frac{1000x}{2} \\ \text{Therefore Basal Conductance} &= \frac{2}{1000x} \mu\text{mho}'\text{s} \\ &= \frac{2}{1000x} \times 10^6 \mu\text{mho}'\text{s} \\ &= \frac{2}{x} \times 10^3 \mu\text{mho}'\text{s} \\ &= \frac{2}{0,6x} \times 10^3 \mu\text{mho}'\text{s}/\text{cm}^2 \end{aligned}$$

Measurements were taken before drinking and ten minutes after each drink.

Time mark Frequency (Hz) vs Basal Resistance (Komms).

(Fig. I.)



(E) Estimation of Catecholamines : Epinephrine (E) and Norepinephrine (NE)

A modification of the method of Bohuon (1974) was used.

Collection : The urine samples were collected before and after the experiment. They were acidified at pH 2 with concentrated Perchloric acid and frozen at -20°C until the time of assay.

Extraction : To a 25ml aliquot of urine was added 860ul of concentrated Perchloric acid and this was agitated for five minutes. This was centrifuged at 30,000xg, using a Sorvall RC2-B refrigerated centrifuge, for 10 minutes. The supernatant liquid was transferred to a beaker and diluted to 65ml with 0,4M Perchloric acid. To this was added 700mg of Alumina (Sigma Chem. Co. Neutral Grade) that had been purified according to the method of Anton and Sayre (1962), 400mg of the salt of disodium ethylenediamine tetra-acetic acid (EDTA) and 20mg Sodium Pyrosulphite.

The pH of the solution was adjusted to 8,5 by the addition of 5M NaOH and carefully agitated for five minutes. This solution was then transferred to a glass centrifuge tube and centrifuged at 2000 RPM for ten minutes, using a bench-type centrifuge. The supernatant was drawn off without disturbing

the Alumina and was subsequently washed four times with distilled water. The catecholamines were eluted from the Alumina with 5ml 0,1M HClO₄.

Solutions : (S)

- (a) 0,1M Perchloric acid adjusted to pH 2,1 with 5M NaOH;
- (b) 0,5M Potassium Phosphate buffer at pH 7;
- (c) Buffer solution pH 2 : 25ml of 0,2M KCl adjusted to pH 2 with 0,2M HCl and diluted to 100ml with distilled water;
- (d) Solution of 0,00075M Potassium Ferricyanide;
- (e) Solution of 0,1M Ascorbic acid;
- (f) Alkaline ascorbate solution : 18ml 5M NaOH; 0,4ml ethylene diamine; 2ml S (5);
- (g) Sodium Hydroxide-ethylene diamine mixture; 18ml 5M NaOH; 0,4ml ethylene diamine;
- (h) Standard solution of norepinephrine : 39,8mg norepinephrine (bitartrate) dissolved in 100ml of 0,1 Perchloric acid. This was kept at 4°C as stock. Solution obtained by dilution 1:1000 with S.(a).
- (i) As for S.(h) except mass of epinephrine used was 36,4mg.

(500ul of S(h) and S(i) contain 100ng of amine).

Estimation : The principle of estimation was the oxidation of the Perchloric acid eluent at pH 7 and pH 2 by Potassium Ferricyanide, and a blank was run for every determination. Four tubes were thus required for every determination, which was conducted in series.

To each tube was added successively :

1ml Perchloric acid eluate;

500ul S(b) or S(c)

50ul S(d)

The oxidation was allowed to proceed for exactly 180s. and was stopped by the addition of 1ml of S(f).

Into the blank tubes was introduced 1ml of S(g) and 15 minutes later 100ul of S(e).

The Spectrofluometric analysis was done by an Aminco-Bowman Spectrofluorimeter. Wavelengths of 395nm (excitation) and 505nm (emission) were set. Standardization was obtained for a series by replacing the Perchloric-eluates with 500ul of S(h) and S(i). The meter reading was set at 100 with the fluorescence given by the standard NE oxidized at pH 7.

Creatinine was estimated using the modified Jaffee reaction, automated by Chasson, Grady and Stanley (1961) for the Technicon Auto-analyzer II.

Calculations :

a = net fluorescence of the standard (100ng)
of NE at pH 7

b = net fluorescence of the standard (100ng)
of NE at pH 2

c = net fluorescence of the standard (100ng)
of E at pH 7

d = net fluorescence of the standard (100ng)
of E at pH 2

X = net fluorescence of determination at pH 7

Y = net fluorescence of determination at pH 2.

$$\text{NE} = 100 \times \frac{X \times d}{c} - Y$$

$$\frac{a \times d}{c} - b$$

$$= P \text{ ng NE} \times 5$$

$$= 5 P \text{ ng NE/25ml urine}$$

$$\text{E} = \frac{100Y - \frac{P \times b}{100}}{d}$$

$$= Q \text{ ng E} \times 5$$

$$= 5Q \text{ ngE/25ml urine.}$$

Creatinine values were converted to g/25ml urine.

Therefore NE = 5P ng NE/g Creatinine

E = 5Q ng E/g Creatinine.

Only II delta and 9 gamma subjects were used. One sample was lost when a defective tube broke in the centrifuge. One subject failed to produce 25ml of urine, while another spoiled a sample. Three other sets of samples were lost due to an undetected power failure in a freezer.

(4) STATISTICAL DESIGN

Since two different groups were measured on the same variable, before and after identical treatment, a TWO-WAY ANALYSIS OF VARIANCE with repeated measures was used.

CHAPTER SEVEN

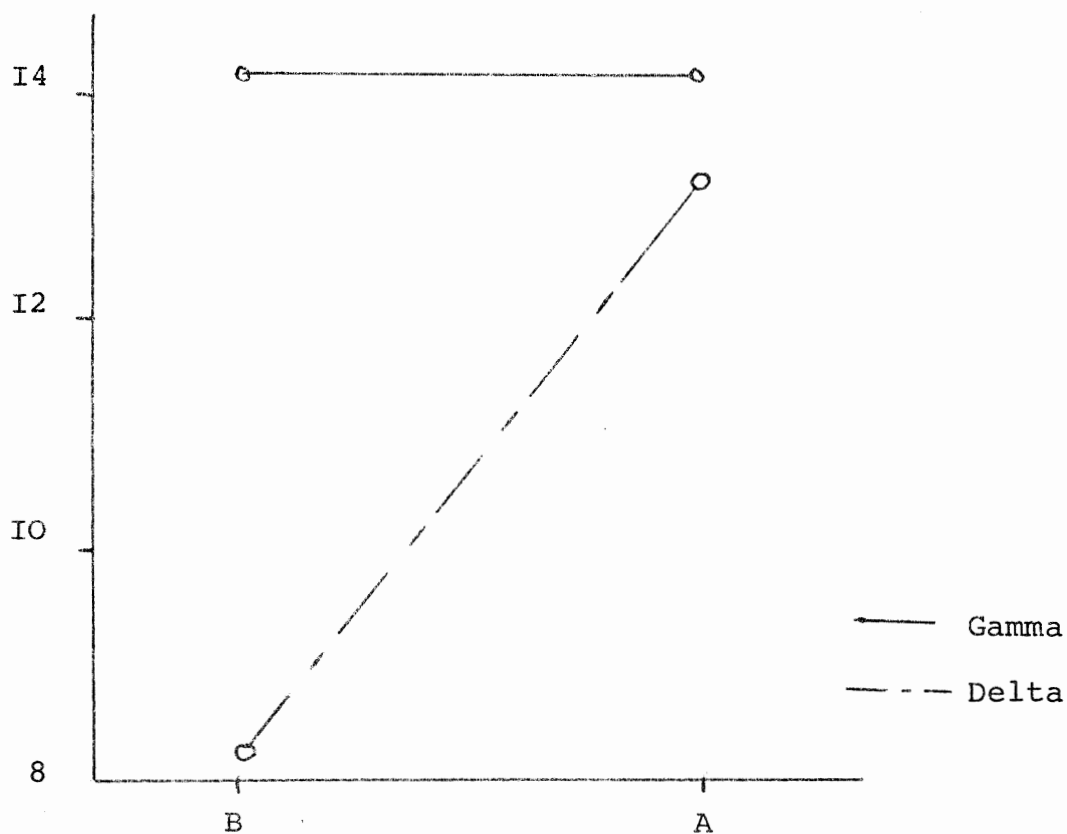
(1) RESULTS :

(A) EXTROVERSION :

Table of Mean Values. (I : I)

	Before	After	Overall
Delta	8,2	13,7	11,0
Gamma	15,3	15,4	15,3
Overall	11,2	14,4	12,8

Graph of Cell Means: Extroversion versus Before/After. (Fig. 2)



ANOVA Table. (I : 2)

SOURCE	SS	df	MS	F-ratio
<u>Between Subjects</u>				
Groups: Delta/Gamma	240,3	I	240,3	15,1**
Subjects	381,7	24	15,9	
<u>Within Subjects</u>				
Before/After	100,4	I	100,4	24,5**
Interaction	94,0	I	94,0	22,94**
Residual	98,3	24	4,1	

**p 0,01

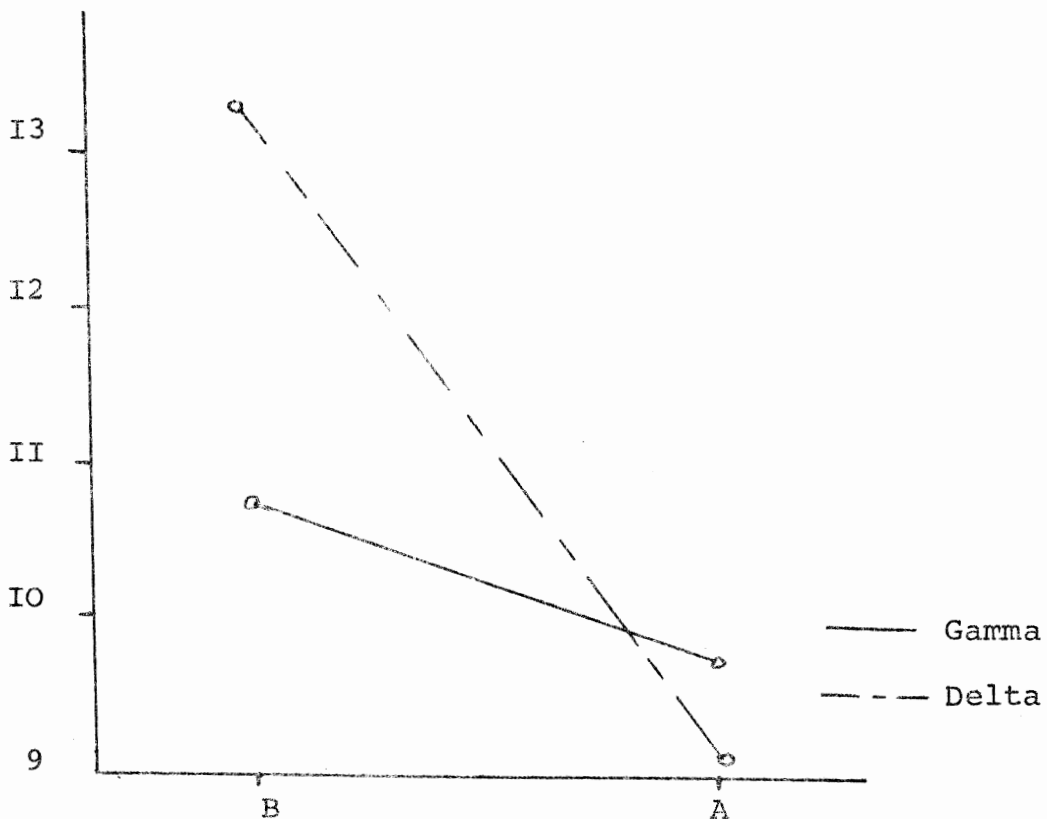
Table of Studentized Range Statistics. (I : 3)

	t'	df ₁	df ₂
<u>Before vs After</u>			
Delta	-13,94**	2	24
Gamma	-0,23	2	24
<u>Delta vs Gamma</u>			
Before	-11,4**	2	48
After	-2,6	2	48

Gamma alcoholics are significantly more extroverted than delta alcoholics before drinking. However, this difference is minimal after drinking. Delta alcoholics are significantly more extroverted after drinking, whereas gamma alcoholics remain virtually the same.

B) NEUROTICISM:Table of Mean Values. (2 : 1)

	Before	After	Overall
Delta	13,2	9,1	11,1
Gamma	10,7	9,7	10,2
Overall	12,2	9,3	10,8

Graph of Cell Means: Neuroticism versus Before/After. (Fig. 3)

ANOVA Table. (2 : 2)

SOURCE	SS	df	MS	F-ratio
<u>Between Subjects</u>				
Groups: Delta/Gamma	10,4	1	10,4	0,2
Subjects	1081,8	24	45,5	
<u>Within Subjects</u>				
Before/After	83,6	1	83,6	22,5**
Interaction	31,1	1	31,2	8,4**
Residual	88,8	24	3,7	

**p 0,01

Table of Studentized Range Statistics. (2 : 3)

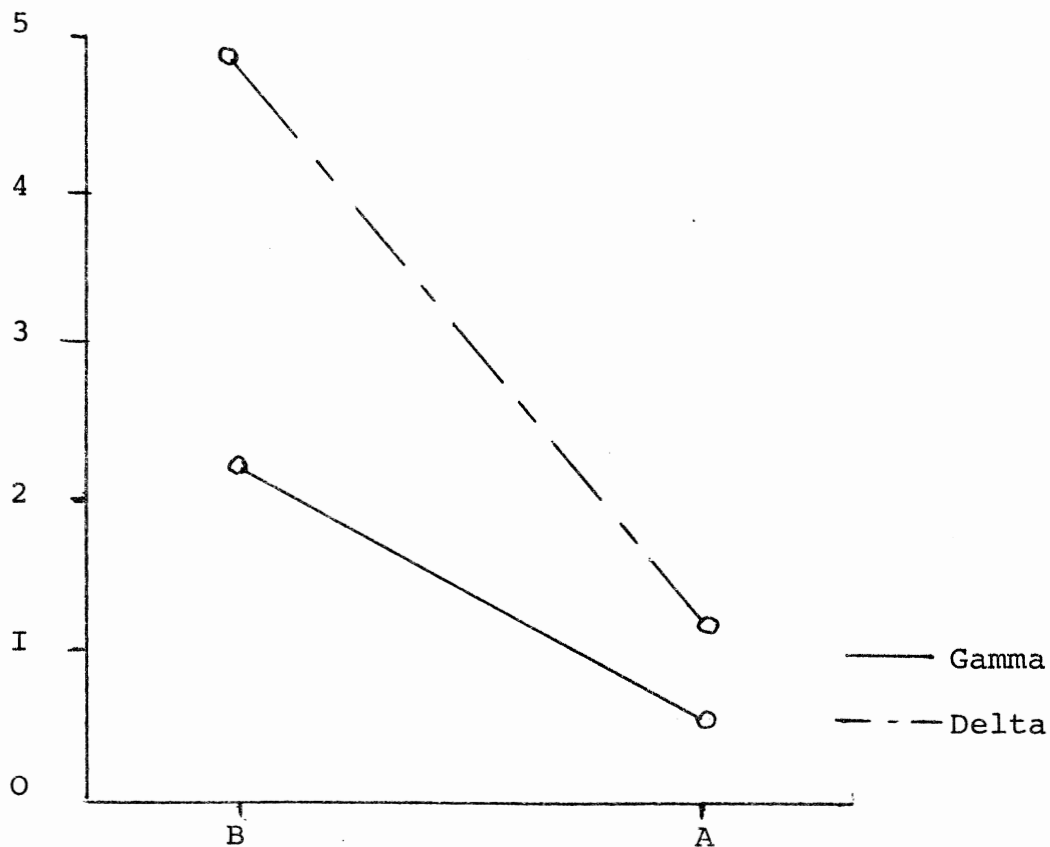
	t'	df ₁	df ₂
<u>Before vs After</u>			
Delta	10,95**	2	24
Gamma	2,65	2	24
<u>Delta vs Gamma</u>			
Before	2,62	2	48
After	-0,70	2	48

**p 0,01

There is no significant difference between gamma and delta alcoholics on measures of neuroticism before or after drinking. There is, however, a significant reduction in delta alcoholics' neuroticism after drinking. Although there is a reduction in gamma alcoholics' neuroticism after drinking, this change is not significant.

(C) ANXIETY:Table of Mean Values. (3 : 1)

	Before	After	Overall
Delta	4,8	1,3	3,1
Gamma	2,2	0,5	1,3
Overall	3,7	1,0	2,3

Graph of Cell Means: Anxiety versus Before/After. (Fig. 4)

ANOVA Table. (3 : 2)

SOURCE	SS	df	MS	F-ratio
<u>Between Subjects</u>				
Groups: Delta/Gamma	38,8	1	38,8	4,9*
Subjects	191,1	24	8,0	
<u>Within Subjects</u>				
Before/After	85,6	1	85,6	52,7**
Interaction	9,6	1	9,6	5,9*
Residual	39,0	24	1,6	

*p 0,05

**p 0,01

Table of Studentized Range Statistics. (3 : 3)

	t'	df ₁	df ₂
<u>Before vs After</u>			
Delta	13,87**	2	24
Gamma	6,91**	2	24
<u>Delta vs Gamma</u>			
Before	6,06**	2	48
After	2,05	2	48

**p 0,01

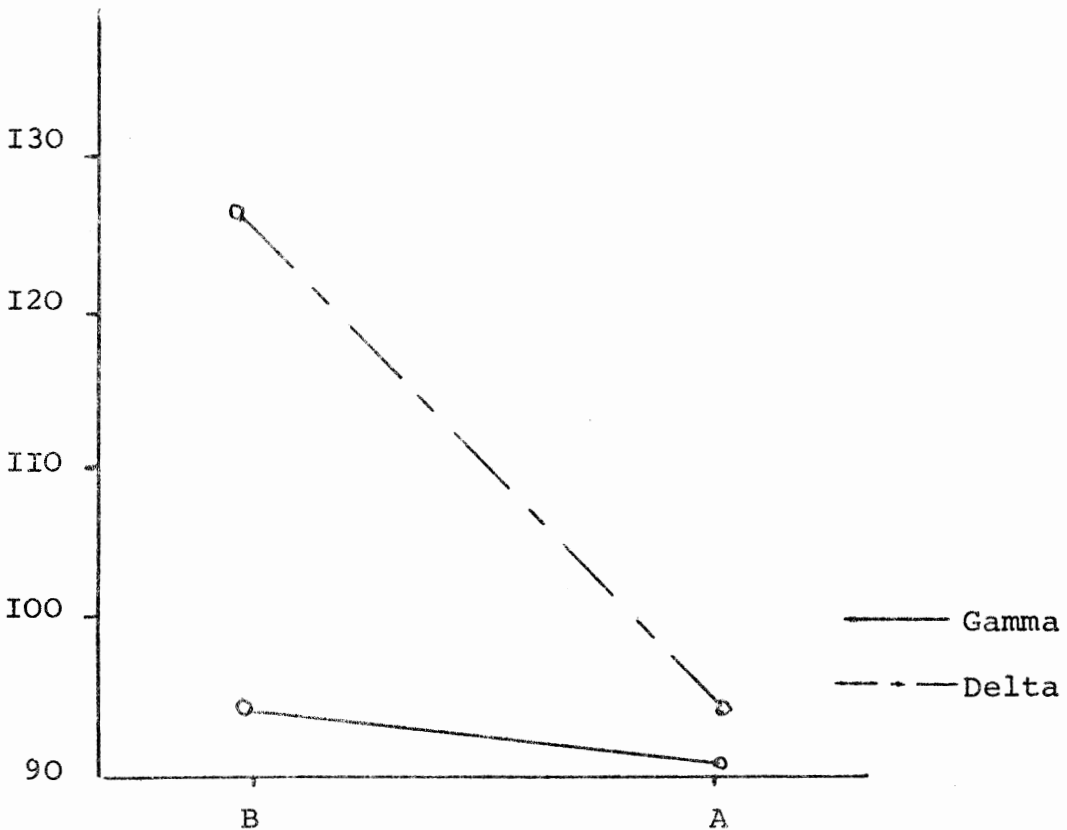
There is a significant reduction in anxiety in both gamma and delta alcoholics after drinking. Before drinking, however, the delta alcoholics were significantly more anxious than the gamma alcoholics. Therefore alcohol has a greater anxiolytic effect on delta alcoholics.

(D) ASSERTIVENESS:

Table of Mean Values. (4 : 1)

	Before	After	Overall
Delta	126,6	94,2	110,4
Gamma	94,0	90,9	92,5
Overall	112,8	92,8	102,8

Graph of Cell Means: Assertiveness versus Before/After. (Fig. 5)



ANOVA Table. (4 : 2)

SOURCE	SS	df	MS	F-ratio
<u>Between Subjects</u>				
Groups: Delta/Gamma	4087,4	1	4087,5	9,32**
Subjects	10520,7	24	438,4	
<u>Within Subjects</u>				
Before/After	3996,8	1	3996,8	47,20**
Interaction	2725,7	1	2725,7	32,20
Residual	2023,3	24	84,7	

**p 0,01

Table of Studentized Range Statistics (4 : 3)

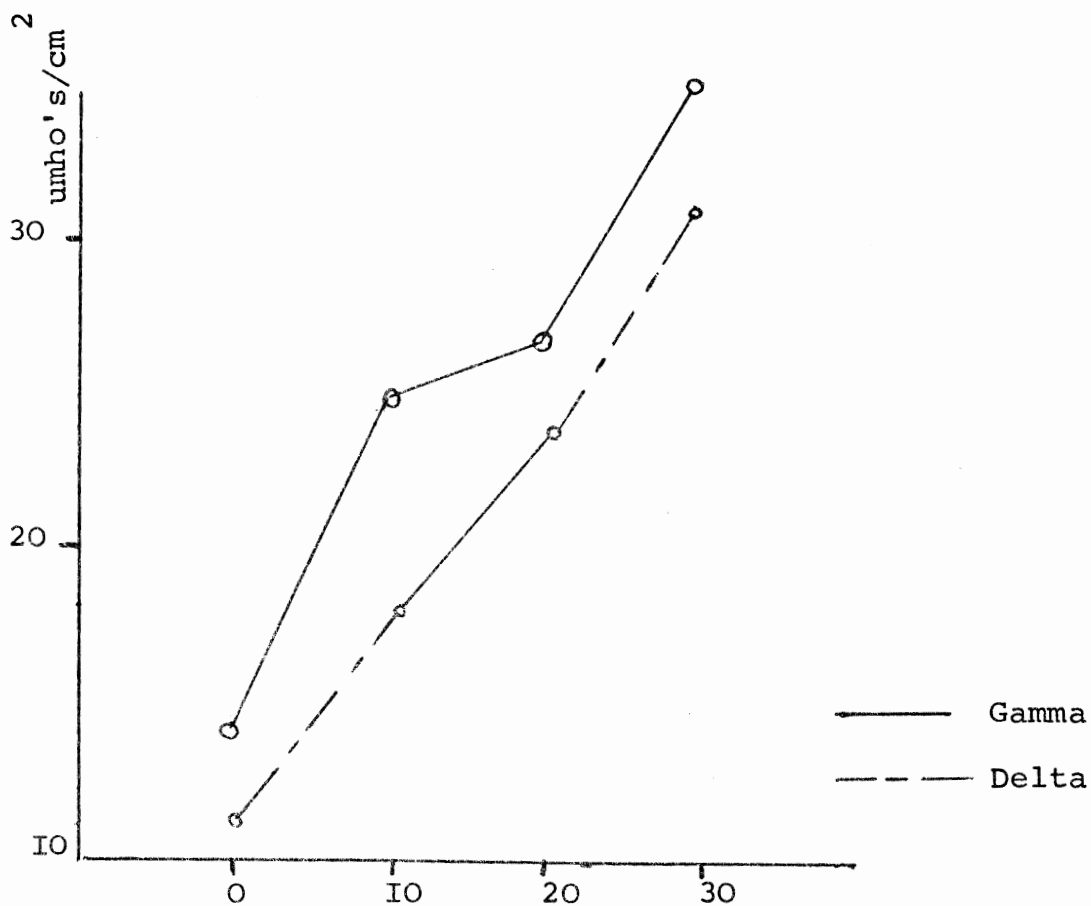
	t'	df ₁	df ₂
<u>Before vs After</u>			
Delta	18,06**	2	24
Gamma	1,82	2	24
<u>Delta vs Gamma</u>			
Before	10,22**	2	48
After	0,97	2	48

**p 0,01

There is a significant increase in assertiveness exhibited by delta alcoholics after drinking, while change in assertiveness in gamma alcoholics is minimal after the drinking episode. Before commencement of drinking, the gamma alcoholics were significantly more assertive than delta alcoholics.

(E) SKIN CONDUCTANCE:Table of Mean Values. (5.: I)

	0	1	2	3	Overall
Delta	10,1	18,5	24,3	30,6	20,9
Gamma	13,4	25,1	28,7	35,3	25,6
Overall	11,5	21,3	26,1	32,6	22,9

Graph of Cell Means: Skin Conductance versus Minutes. (Fig. 6)

ANOVA Table. (5 : 2)

SOURCE	SS	df	MS	F-ratio
<u>Between Subjects</u>				
Groups: Delta/Gamma	576,8	1	576,8	0,59
Subjects	233003,7	24	971,0	
<u>Within Subjects</u>				
Before/After	6085,6	3	2028,5	34,3*
Interaction	36,0	3	12,0	0,2
Residual	4247,5	72	59,0	

*p 0,01

Pairwise Comparison: Table of Studentized Range Statistics(5 : 3)

<u>Pairs</u>	<u>t'</u>	<u>df₁</u>	<u>df₂</u>
0-1	-6,51**	4	72
0-2	-9,73**	4	72
0-3	-14,04**	4	72
I-2	-3,2	4	72
I-3	-7,5**	4	72
2-3	-4,3*	4	72

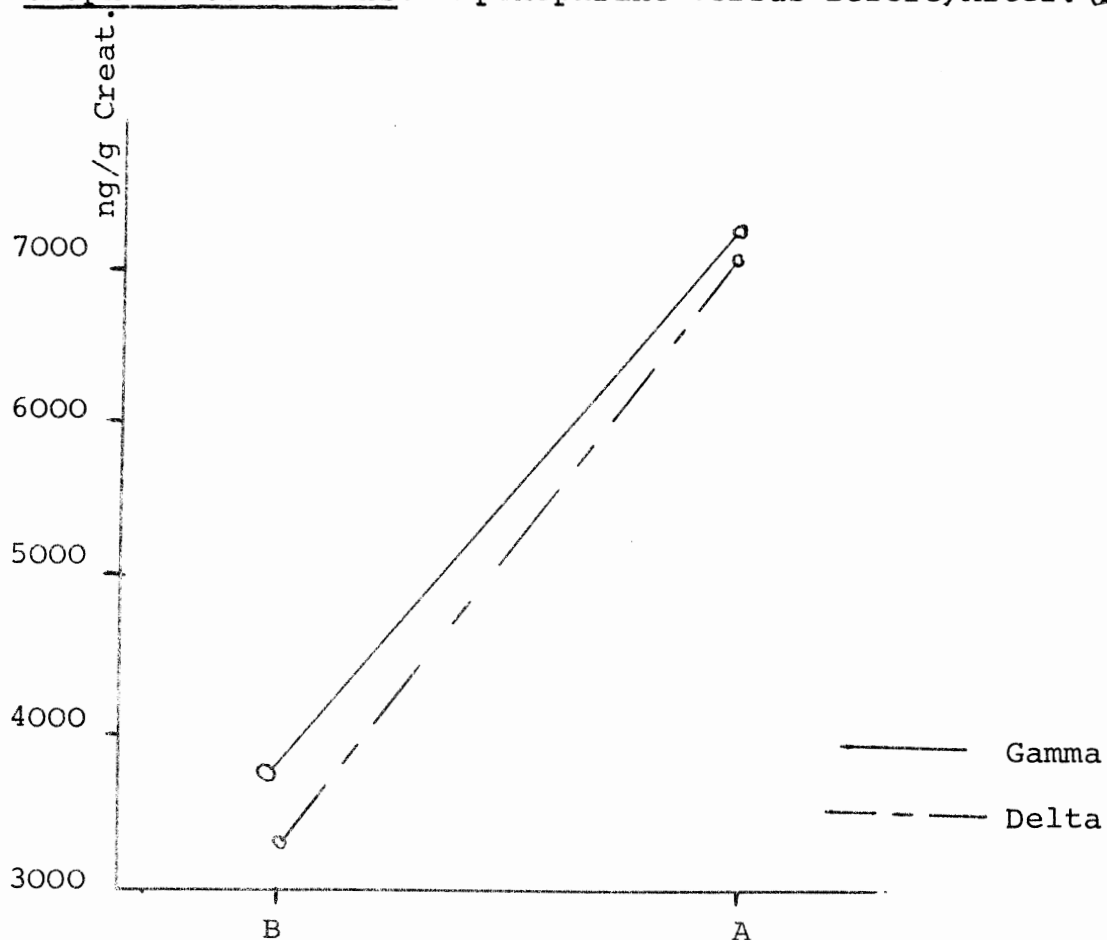
**p 0,01

*p 0,05

Although the gamma alcoholics' skin conductance was higher than that of the delta alcoholics', this difference was not significant at any stage of the experiment. However, there were significant increases in skin conductance at every stage of the experiment (with pairwise comparison) except between 20 and 30 minutes.

(F) EPINEPHRINE:Table of Mean Values. (6 : I)

	Before	After	Overall
Delta	3398,5	7042,2	5220,3
Gamma	3860,7	7222,6	5541,7
Overall	3606,5	7123,4	5364,9

Graph of Cell Means: Epinephrine versus Before/After. (Fig. 7)

ANOVA Table. (6 : 2)

SOURCE	SS	df	MS	F-ratio
<u>Between Subjects</u>				
Groups: Delta/Gamma	1022676	1	1022676	,15156
Subjects	121459573	18	6747754	
<u>Within Subjects</u>				
Before/After	121469662	1	121469662	
Interaction	196586,0	1	196596,0	19,0**
Residual	114974808	18	6387489	,03

**p 0,01

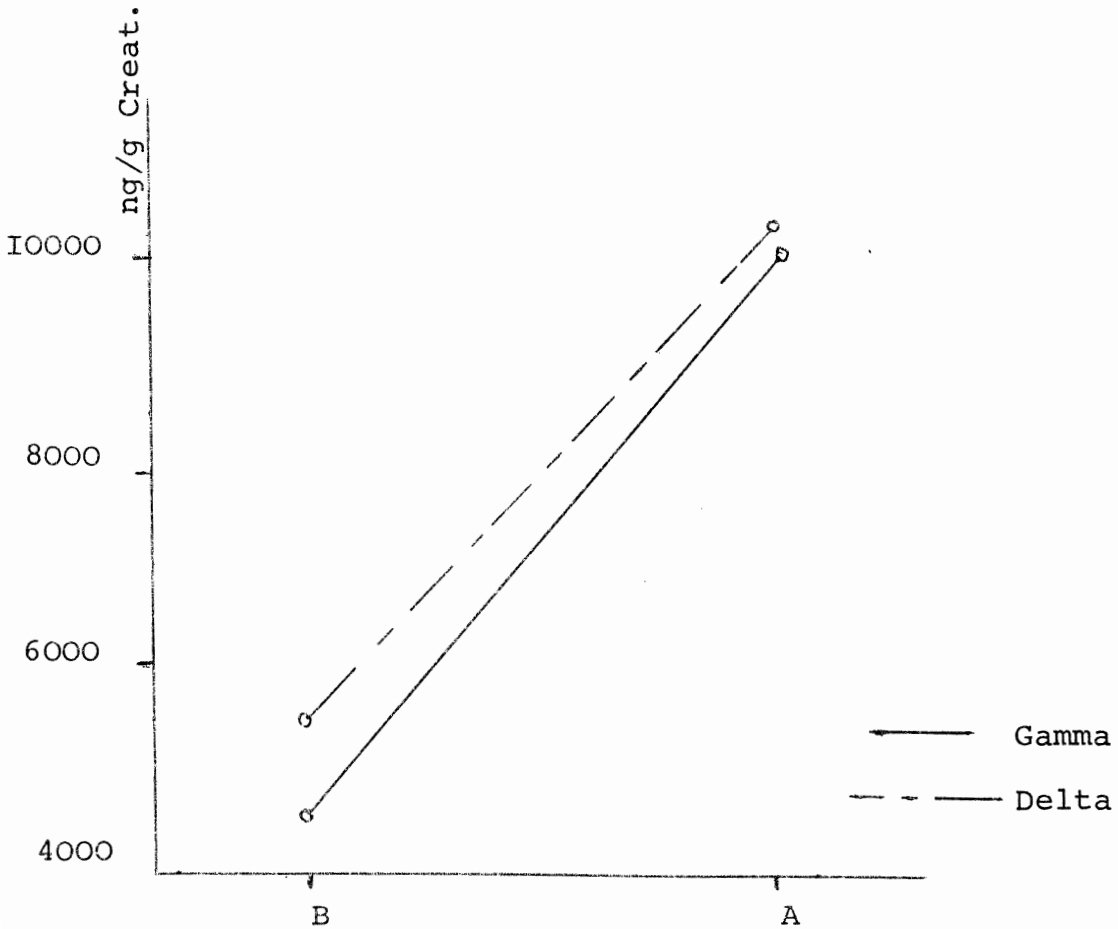
Both groups' urinary epinephrine content increased after drinking. However, no group increased more significantly than the other.

F) NOREPINEPHRINE:

Table of Mean Values. (7 : I)

	Before	After	Overall
Delta	6021,7	11151,6	8586,7
Gamma	4832,3	10097,1	7464,7
Overall	5486,5	10677,1	8081,8

Graph of Cell Means: Norepinephrine versus Before/After. (Fig. 8)



ANOVA Table. (7 : 2)

SOURCE	SS	df	MS	F-ratio
<u>Between Subjects</u>				
Groups: Delta/Gamma	I2462044	I	I2462044	,64
Subjects	350757480	I8	I9486526	
<u>Within Subjects</u>				
Before/After	267422520	I	267422520	4I,04**
Interaction	450I9	I	450I9	,0067
Residual	II7288353	I8	65I60I9	

**p 0,0I

There was a significant increase in norepinephrine response in both groups. However, no group differed significantly from the other.

(2) DISCUSSION AND CONCLUSIONS :

(A) EXTROVERSION :

As has been demonstrated, (Table I : 3), delta alcoholics become significantly more extroverted ($p < 0,01$) under the influence of alcohol. This result is consistent with predictions made by Eysenckian Theory. The gamma alcoholics, whose extroversion scores were greater before the intake of alcohol, remain virtually the same after drinking.

At this point it seems appropriate to discuss the Law of Initial Values. Initially proposed by Wilder (1950) and adopted by Lacey and Lacey (1962) and Eysenck (1967), it has been found to be applicable to a variety of physiological and psychological functions in general and to autonomic reactivity and extroversion in particular. The Law of Initial Values states that increments of response level are greater for functions that initially operate at a low level than they are for functions that operate at a high level. Further, there is a tendency to a "paradoxical reaction" when initial level of functioning is extreme in the direction of a stimulus. In the present study, alcohol is considered as the stimulus and degree of extroversion is considered as the level of functioning. The results appear consistent with the Law of Initial Values. The "paradoxical reaction" was noted in some of the gamma

patients who become more introverted under the influence of alcohol.

This study gives experimental verification to Keehn's (1970) ex post facto investigation of the effects of alcohol on extroversion. The alcohol level produced in the experiment would approximate that usually taken by delta alcoholics, as a BAC of 0,065% is below that usually associated with intoxication (about six drinks). It seems reasonable to conclude, therefore, that the increase in the delta alcoholics' extroversion is not only significant, but it is desired by them. The delta alcoholic will tend to become more sociable, care-free and less introspective once the desired level has been reached.

The gamma alcoholics' change in extroversion is of interest. After approximately six drinks there is a strong probability that most gamma alcoholics would consistently continue to drink. The Law of Initial Values, along with the curvilinear relationship between alcohol, dose and extroversion, would then predict that gamma alcoholics would become initially slightly more extroverted and subsequently introverted. Consistent with the findings of Tomsovic (1974), this phenomenon would effectively change the extroverted gamma alcoholic from an easy-going, impulsive and gregarious person to an introverted and withdrawn one, who would then relinquish

his usual social role.

The advantage of the present study's experimental design and statistical analysis is evident in their ability to compare significant differences between the groups before and after drinking. The Before Comparison of Means (Table I : 3) confirms both Vogel (1961) and Walton's (1968) findings that gamma alcoholics are more extroverted ($p < 0,01$) than delta alcoholics. There is no significant difference after drinking. This study's hypothesis regarding extroversion is accepted.

(B) NEUROTICISM :

The results (Table 2 : 3) concerning neuroticism confirm the hypothesis ($p < 0,01$) that delta alcoholics will show a significant decrease in neuroticism after drinking. The gamma alcoholics' scores decrease under the same conditions, but this finding is not statistically significant.

It is difficult to interpret these results due to the lack of information on neuroticism in Eysenck's formulations. This situation is compounded by the fact that there exists a tenuous link between "arousal" and neuroticism. If it is assumed that there is a relationship between neuroticism and autonomic or hypothalamic functions (Eysenck, 1967), then an interpretation could be made by using the

Law of Initial Values. The present study's results are consistent with the changes predicted by this law. Since delta alcoholics have higher neuroticism scores when sober, they will have a greater response to alcohol with regard to neuroticism than gamma alcoholics. It remains highly speculative what changes would have occurred in the neuroticism scores of the gamma alcoholic group if they had continued to drink - a phenomenon that would be consistent with their drinking pattern.

Keehn's (1970) study, which likewise made use of the EPI, showed that alcoholics considered that their neuroticism was not reduced by drinking alcohol. Keehn's work is partially supported in this study. However, Keehn did not divide his sample into gamma and delta alcoholics. If there were a greater number of gamma than delta alcoholics in his sample, this imbalance could account for his finding.

Jensen (1958) found that hospitalized introverts were significantly more neurotic than hospitalized extroverts. The findings in this study follow the above trends. It should be remembered, however, that the sample in the present study was divided according to drinking patterns and not on the basis of extroversion-introversion.

(C) ANXIETY :

As Table 3 : 3 indicates, there is a significant change in anxiety ($p < 0,01$) in both gamma and delta alcoholics after drinking. Since the delta alcoholics were significantly more anxious ($p < 0,01$) than the gamma alcoholics, it can be concluded that alcohol had a greater anxiolytic effect on the delta alcoholics. Hypothesis 3 is therefore supported.

The significant difference in anxiety between the groups before drinking can possibly be ascribed to the fact that this investigation took place in the afternoon. The delta alcoholics would associate this time of day with their need to imbibe more alcohol.

(D) ASSERTIVENESS :

As it is clearly shown by Table 4 : 3, delta alcoholics' assertiveness increases significantly more ($p < 0,01$) than gamma alcoholics following the ingestion of alcohol. Thus hypothesis 4 is accepted. Delta alcoholics will produce more assertive behaviour in social situations under the influence of alcohol than when they are sober. Under the same conditions, gamma alcoholics show a marginal increase in assertiveness. It was interesting to note that some gamma alcoholics became less assertive under the influence of alcohol and thus it seems acceptable to

hypothesize that this psychological function might follow the Law of Initial Values. Equally, assertiveness might be related to some physiological variable such as arousal or anxiety. Table 4 : 3 also indicates that gamma alcoholics are more assertive than delta alcoholics when they are both sober ($p < 0,01$). This seems to support Tomsovic's (1974) view that delta alcoholics have difficulty with those normal everyday situations, which require their being assertive and confident in their social roles. The problems of gamma alcoholics appear to be more individualized and specific.

(E) SKIN CONDUCTANCE :

As illustrated by Table 5 : 2, hypothesis 5 is not supported by the data. For the entire duration of the experiment, the level of skin conductance of the gamma alcoholics was higher than that of the delta alcoholics. This difference is not statistically significant. There is likewise no significant increase in level of autonomic arousal due to the effects of alcohol when the two groups of alcoholics are compared. The results are totally consistent with the Law of Initial Values and the findings of McDonnell and Carpenter (1959), who found that low doses of alcohol increased levels of skin conductance.

The findings of the present study are, however, not insignificant. The autonomic arousal of gamma

and delta alcoholics did not differ under the experimental conditions. Delta alcoholics could be expected to maintain elevated levels of arousal due to their continuous pattern of drinking. Gamma alcoholics would continue drinking and, since there exists a curvilinear relationship between BAC levels and arousal during a "binge", their level of arousal would drop. The type of drinking that gamma alcoholics engage in would depress their level of autonomic arousal. Whether this lower level is desired by this type of alcoholic remains speculative. The rate of absorption and the metabolism of alcohol are of importance. Jones (1974) has shown that extroverts tend to respond more to the depressant effects of alcohol when they are experiencing a decline in BAC. Since the two above factors have complex relationships, the results of the present study are restrictive and should be treated with caution.

(F) CATECHOLAMINES :

It can be seen from Table 6 : 2 and Table 7 : 2 that the data does not support hypotheses 6 and 7.

However, support is given to the usual finding which is that there exists a significant increase ($p < 0,01$) in epinephrine and norepinephrine after the ingestion of alcohol. Catecholamine levels and production appear to be identical. It appears that the large

discrepancy in recorded results regarding catecholamine production (Wallegren and Barry, 1970, pp.174-176) is related to methodological deficiencies or other variables. The latter appears to be linked to a subject's emotional disposition and his perception of the experimental situation. According to Mason (1972, p.35), most studies done on psychiatric patients indicate that some endocrine abnormality occurs during the course of their illness. This could present the researcher with considerable difficulties when his work involves the comparison between normals and psychiatric patients or between one class of psychiatric patient and another.

Psycho-endocrine responses show a curvilinear relationship with chronic alcohol ingestion (Mello, 1972), but an additive effect (Ogata et al. 1971) during withdrawal periods. Controlling these variables appears important when engaging in psycho-endocrine research.

The overall findings of the present research indicate that psychological responses to alcohol are the variables that optimally differentiate the gamma group of alcoholics from the delta group. Following an acute ingestion of alcohol, delta alcoholics tend to become more extroverted and assertive with a concomitant reduction in anxiety and neuroticism. Gamma alcoholics become less neurotic and anxious but this change is of a lesser magnitude than that of the

delta group. In a sober state, delta alcoholics tend to be significantly more introverted, anxious and less assertive than gamma alcoholics. The personality of the intoxicated delta alcoholic approximates the personality of the sober gamma alcoholic.

Physiologically and biochemically, both groups experience an increase in arousal with a concomitant increase in catecholamine production. One of the more important findings of the present study is the rapid autonomic and catecholamine response to alcohol. Skin conductance increases were measured within 10 minutes and an approximately three-fold increase in catecholamine production was measured within one hour. If these effects are desirable, strong positive reinforcement of drinking behaviour will occur.

More specifically, the experimental findings of this study show that the understanding of alcoholic drinking behaviour will be diminished if researchers rely on a simplistic, unidimensional analysis of behaviour. Recourse to molar constructs such as "tension" or "dependence" will prove unproductive. Alcohol studies are essentially drug experiments and should be treated as such. The present work has shown conclusively that two groups of alcoholics respond differently to alcohol on many measures but

especially psychological ones. It is imperative that researchers differentiate their population in at least one dimension. Significant results from one group might be negated by insignificant ones from another. This study strongly suggests that the gamma-delta distinction is an important one.

A further important finding is that there are considerable benefits to be derived from analysing alcohol effects on various response levels. Although the results of the measures of skin conductance and catecholamine production were not significant, this information is still important. Lader and Wing (1966), in their exemplary psychophysiological drug study, have shown that it is important to rank variables in order of their importance. This ranking would usefully exclude unimportant measures that might have been included for the sake of completeness on theoretical or other grounds. Highly predictive response measures could be obscured by useless ones if the process of ordering is not used. If similar research is undertaken in the future, it would seem to be more beneficial to address oneself to problems involving psychological change rather than physiological ones.

If we retain the perspective that this research was essentially a drug experiment, generalization of results would be more appropriate if we view the independent variable, i.e. gamma and delta alcoholics,

in the following terms : gamma alcoholism is characterized by the intermittent self-administration of large doses of alcohol, while delta alcoholism is essentially the result of the continuous self-administration of moderate doses of alcohol. Although one could ask why the gamma alcoholic drinks so much, one could equally wonder why the delta alcoholic by comparison drinks so moderately.

The schema of Russell and Mehrabian (1975) provides an explanatory framework. Moderate doses of alcohol increase pleasure, arousal and dominance. This is reflected behaviourally by increases in affiliation (Mehrabian and Ksionsky, 1974) and, according to Docter and Bernal (1970), a tendency to be more demanding and assertive. There will also be a tendency (Cutter et al., 1973) towards risk taking. This could explain why there is a significant change in extroversion, assertiveness and a decrease in neuroticism in delta alcoholics after the consumption of alcohol. Increases in skin conductance and catecholamine production provide the level of arousal for these psychological changes to occur.

It is difficult to interpret the gamma alcoholics' responses to the alcohol dose given in the experimental situation. Jellinek (1960) proposed that gamma

alcoholics have a greater tolerance for alcohol and would thus need a greater dose to induce changes that are equivalent to those induced in the delta alcoholics. Gamma alcoholics might also need a "trigger" situation to induce their drinking. This situation was absent in the present experimental situation and might account for the refusals by the three gamma alcoholics to take part in the experiment. It is for this reason that the majority of results in this study that pertain to gamma alcoholics have been speculative and inferred. A more general observation would be that alcohol consumption decreases general social functioning in the gamma alcoholic but increases it in the delta alcoholic.

This study has highlighted an anomaly. Why do persons in general report a reduction in anxiety after the ingestion of alcohol when drinking is accompanied by an increase in arousal and sympatho-adrenalmedullary response? Even if we accept McDonnell and Carpenter's (1954) explanation that anxiety and arousal have an inverted U relationship, a person would have to be at the apex of the curve to benefit from the consumption of alcohol. The schema of Russell and Mehrabian (1975) provides little help. Although a moderate dose of alcohol would increase pleasure and dominance, it would also increase the person's

high level of arousal. A large dose of alcohol would reduce the level of arousal but increase submissiveness and would induce a minimal increase in pleasure.

The only explanation that the author can offer to resolve this problem is that the initial central effects of alcohol, especially those which influence the associative areas of the cortex, are instrumental in reducing these autonomic effects.

Indications for therapy.

A comprehensive review of the results of this study indicates that the delta alcoholic would benefit from therapy involving assertiveness training, social skills development, benzodiazepine therapy and forms of systematic desensitization and group psychotherapy. These forms of treatment would provide the delta alcoholic with the skills and self confidence which he previously acquired by the continuous ingestion of alcohol. The treatment suggested would diminish his dependent and passive behaviour patterns and would enable him to cope with the vicissitudes of everyday life.

The problems of the gamma alcoholic are not as evident. He has most of the skills that treatment would provide for the delta alcoholic.

Thus therapeutic strategies for the gamma alcoholic are far less definitive. Consistent with other studies reviewed (Walton, 1968 and Tomsovic, 1974), the gamma alcoholic was found to be more complex and disturbed. He is more likely to respond to intensive psychotherapy, behaviour modification and antabuse therapy. The last two forms of therapeutic intervention would be especially beneficial if gamma alcoholism is mediated more by physical than psychological addiction.

CONCLUSIONS.

It can be concluded that:

1 (a) Delta alcoholics exhibit a greater increase in extroversion and assertiveness following the ingestion of a moderate dose of alcohol than gamma alcoholics.

(b) Delta alcoholics exhibit a greater decrease in neuroticism and anxiety following the ingestion of a moderate dose of alcohol than gamma alcoholics.

2 (a) In the sober state delta alcoholics are significantly more anxious but significantly less extroverted and assertive than gamma alcoholics.

3 (a) There is no significant difference between gamma and delta alcoholics catecholamine or Basal Skin Conductance response following the ingestion

of a moderate dose of alcohol.

(b) When sober there is no difference between gamma and delta alcoholics in their level of urinary catecholamine content, Basal Skin Conductance or level of neuroticism.

(3) IMPLICATIONS FOR FURTHER RESEARCH.

This study highlights the need to devise a suitable experiment that will provide more information about the gamma alcoholic. In the present study the amount of alcohol ingested was held constant, at the expense of obtaining suitable information about the gamma alcoholic. A suggestion would be to allow alcohol to be consumed on a self-selection basis. Different settings might change responses and different conditions, i.e., administration of alcohol in groups or in natural settings, could prove a useful substitute for the restrictive (hospital) environment.

A further variable that is important is the control of chronicity. This is of special importance as chronicity and tolerance are intimately related. Earlier in this study, it was suggested that high tolerance could account for the minimal changes that alcohol produced in gamma alcoholics after the ingestion of alcohol. Equally, significant changes could be attributed to chronic alcoholism which is associated with a low tolerance for alcohol.

However useful the delineation of gamma and delta group has proved, it must be stressed, particularly with regard to therapy, that the patient's right to individuality remains a prime concern. It is hoped that this research has contributed something of value to the understanding and treatment of alcoholism.

REFERENCES

- ABELIN, I.
HERREN, C.
BERLE, W. On the effect of alcohol on the production of adrenalin and nor-adrenalin in man. Helvetia Medica Acta, 1958, 25, 591-600.
- ABELSOHN, D.S. The age variable in alcoholism
Unpublished Masters Thesis, University of Cape Town, 1973.
- ABELSOHN, D.S.
BEN-ARIE, O. Self-acceptance and patterns of alcohol drinking. Unpublished Honours Thesis, University of Cape Town, 1971.
- AMARK, C. A study in alcoholism: Clinical, social, psychiatric and genetic investigations. Acta Psychiatrica and Neurologica Scandanavica, 1951, Suppl. 70.
- ANTON, A.H. Ethanol and urinary catecholamines in man. Clinical Pharmacology and Therapeutics, 1965, 6, 462-467.
- ARVOLA, A.
FORSANDER, O.A. Comparison between water and alcohol consumption in six animal species in free choice experiments. Nature, 1961, 191, 819-820.
- ARCHIBALD, P. 3rd, Leonard Ball Oration, Melbourne. Reprinted in 29th International Congress on Alcoholism and Drug Dependence, ed. L.G. Kiloh, Butterworth, Australia, 1971.

- BACON, M.K. The Dependency-Conflict Hypothesis and the frequency of drunkenness; Further evidence from a cross-cultural study. Quarterly journal of Studies on Alcohol, 1974, 35, 863-876.
- BAILEY, J.E.
METCALF, M. The MPI and the EPI. A comparative study on depressive patients. British Journal of Social and Clinical Psychology, 1969, 8(1), 50-54.
- BAILEY, M.B.
STEWART, J. Normal drinking by persons reporting previous problem drinking. Quarterly Journal of Studies on Alcohol, 1967, 28, 303-315.
- BALES, R.F. Cultural differences in rates of alcoholism. Journal of Studies on Alcohol, 1946, 6, 480-499.
- BANDURA, A. Principles of Behaviour Modification. New York: Holt, Rineman & Winston, 1969.
- BARRY, H. Psychological factors in alcoholism. In B. Kissen & H. Bergleiter (Eds.), The Biology of Alcoholism (Vol. 3). New York: Plenum, 1974.
- BECKMAN Beckman Service Bulletin (Offner division). Special Instructions for Type 9842 Coupler, 1972.
- BERGLEITER, H.
GROSS, M.M.
PARJEZZ, B. Recovery functions and clinical symptomatology in acute alcoholization and recovery. In M.M. Gross (Ed.), Experimental Studies of Alcoholic Intoxication and Withdrawal. New York: Plenum, 1973.

- BLAKE, B.G. The application of behaviour therapy to the treatment of alcoholism. Behaviour Research and Therapy, 1965, 3, 75-85.
- BLANE, H.T. The Personality of the Alcoholic: Guises of Dependency. New York: Harper & Row, 1968.
- BOHUON, C. The biological active amines. In H.C. Curtis & M. Roth (Eds.), Clinical Biochemistry. Principles and Methods (Vol.2) Berlin: Walter de Gruyter, 1974.
- CAHALAN, D.
ROOM, R. Problem drinking among American men. New Brunswick: Rutgers Centre of Alcohol Studies, 1974.
- CAPPELL, H. An evaluation of tension models of alcohol consumption. In R.J. Gibbons, Y. Israll, H. Kalant, R.E. Popham, W. Schmidt, & R.G. Smart (Eds.), Research advances in alcohol and drug problems. (Vol.2). New York: John Wiley, 1975.
- CARPENTER, J.A. Effects of alcoholic beverages on skin conductance. An exploratory study. Quarterly journal of Studies on Alcohol, 1957, 18, 1-18.
- CHASSON, A.L.
GRADY, H.T.
STANLEY, H.T. The automated Jaffee Reaction. American Journal of Clinical Pathology, 1961, 35, 83-88.

- CLARIDGE, G.S. Personality and Arousal. London: Pergamon Press, 1967.
- CLARK, W.B. Conception of alcoholism:Consequences for research. Addictive Diseases: An International Journal, 1975, I(4), 395-430.
- CLARK, W.B. Loss of control, heavy drinking and drinking problems in a longetudinal study. Journal of Studies on Alcohol, 1976, 37, 9.
- COLLIER,H.D.S. Biochemical, pharmacological aspects of dependence and reports on Marihuana research. H.M. van Praag, R. de Erven & R. Bolm (Eds.), 1971.
- CONGER, J.J. The effects of alcohol on conflict behaviour in the albino rat. Quarterly journal of Studies on Alcohol, 1951, 12, 1-29.
- CONGER, J.J. Reinforcement theory and the dynamics of alcoholism. Quarterly journal of Studies on Alcohol, 1956, 17, 296-305.
- COOPERSMITH, S. The effects of alcohol on reactions to affective stimuli. Quarterly journal of Studies on Alcohol, 1964,25, 459-475.
- CRUZ-COKE, R. Colour-blindness and cirrhosis of the liver. Lancet II,1964, 1064.

- EDWARDS, G. Alcohol related problems in the dis-
GROSS, M.M. ability perspective. Journal of Studies
KELLER, M. on Alcohol, 1976, 37(9), I360-I380.
MOSER, J.
- EYSENCK, H.J. A dynamic theory of anxiety and hysteria.
Journal of Mental Science, 1955, 101,
28-51.
- EYSENCK, H.J. The biological basis of personality.
Massachusetts: Springfield Thomas, 1967.
- EYSENCK, H.J. Manual of the Eysenck Personality In-
EYSENCK, S.B.G. ventory. Kent: Hodder & Stoughton,
1976.
- FIELD, P.G. A cross cultural study of drunkenness.
In D.J. Pittman & C.R. Snyder (eds.),
Society; Culture and Drinking Patterns.
New York: John Wiley, 1962.
- FLEETWOOD, M.F. Biochemical experimental investigations
of emotions and chronic alcoholism.
In O. Diethelam (Ed.), Etiology of Chronic
Alcoholism. 1955.
- FREED, E.X. Alcoholism and manic-depressive disorder:
Some perspectives. Quarterly journal
of Studies on Alcohol, 1970, 31, 62-89.
- GAMBRILL, E.D. An Assertion Inventory for use in
RICHEY, C.A. assessment and research. Behaviour
Therapy, 1975, 6, 551-561.

- GANONG, W.F. Review of Medical Physiology (3rd Ed.).
Los Altos, California: Lange Medical
Publications, 1967.
- GLATT, M.M. The question of moderate drinking despite
"Loss of Control". British Journal of
Addiction, 1967, 62, 267-274.
- GLATT, M.M. A guide to addiction and its treatment.
Lancaster: Medical and Technical Publica-
tions Co. Ltd., 1974.
- GLATT, M.M. The alcoholisms 4: The birth of the
disease concept. Nursing Times, 1975,
71, 822-823.
- GOLDBERG, L. Quantitative studies on alcohol tolerance
in man. Acta Physiologica Scandanavica,
1943, 5(Suppl. 16).
- GOODWIN, D.N.
SCHULSINGER, F.
HERMANSEN, L.
GUZE, S.B.
WINOKUR, G. Alcohol problems in adoptees raised
apart from alcohol biological parents.
Archives of General Psychiatry, 1973,
28, 238-243.
- GOODWIN, D.W. Alcoholic blackout and state-dependent
learning. Federal proceedings, 1974,
33, 1833-1835.
- GREENBERG, L.A.
CARPENTER, J.A. The effect of alcoholic beverages on skin
conductance and emotional tension. I.
Wine, whisky and Alcohol. Quarterly
Journal of Studies on Alcohol, 1957, 18
190-204.
- GROSS, W.F.
CARPENTER, L.L. Alcoholic Personality: Reality or fiction?
Psychological Reports, 1971, 28, 375-378.

- GROSS, M.M.
LEWIS, E. Prevalence of withdrawal manifestations during experimental alcoholization and withdrawal. In M.M. Gross (Ed.), Experimental Studies of Alcohol Intoxication and withdrawal. New York: Plenum, 1973.
- HARE, B.D. Alcohol Dependence. London: Butterworth, 1976.
- HEBB, D.O. Drives and the CNS (Conceptual nervous system). Psychological Review, 1955, 62, 243-254.
- HERSEN, M.
EISLER, R.M.
MILLER, R. Development of assertive responses: clinical measurement and research considerations. Behaviour Research and Therapy, 1973, II, 505-522.
- HOLMBERG, G.
MARTENS, S. Electroencephalographic changes in man correlated with blood alcohol concentration and some other conditions following standardized ingestion of alcohol. Quarterly journal of Studies on Alcohol, 1955, I6, 411-424.
- HORTON, D.L. The function of alcohol in primitive societies: A cross cultural study. Quarterly journal of studies on alcohol, 1943, 4, 199.
- ISBELL, H. Craving for alcohol. Quarterly journal for Studies on Alcohol, 1955, I6, 38-42.
- JELLINEK, E.M. Heredity of the alcoholic. Quarterly journal of Studies on Alcohol, 1945, 6, 105-114.

- JELLINEK, E.M. The Disease Concept of Alcoholism.
New Haven: Hillhouse Press, 1960.
- JENSEN, A.R. The Maudsley Personality Inventory.
Acta Psychologica Scandanavica, 1958,
14, 312-325.
- JONES, M.C. Personality correlates and antecedents
of drinking patterns in adult males.
Journal of Consulting Psychology, 1968,
32, 2-12.
- KALANT, H.
LEBLANC, A.E.
GIBBONS, R.J. Tolerance to, and dependence on,
Ethanol. In Y. Isael & J. Mardones
(Eds.), Biological Basis of Alcoholism.
Toronto: John Wiley, 1971.
- KEEHN, J.D. Neuroticism and extraversion: Chronic
alcoholics reports on effects of
drinking. Psychological Reports, 1970,
27, 767-770.
- KELLER, M. Alcoholism: The image and the reality.
Alcoholism, Zogreb, 1975, II (NoI),
18-27.
- KELLER, M. The nature of addiction: Some second
thoughts. Alcoholism, Zogreb, 1975,
II (NoI), 28-32.
- KELLER, M. The disease concept of alcoholism re-
visited. Journal of Studies on Alcohol,
1976, 37 (No II).
- KESSEL, N.
WALTON, H. Alcoholism. England: Penguin, 1967
(revised).

- KISSIN, B. The pharmacodynamics and natural history of alcoholism (pI-36). In B. Kissin & H. Bergleiter (Eds.), Biology of Alcoholism (Vol. 3). New York: Plenum 1974.
- KNIGHT, R.P. The dynamics and treatment of chronic alcohol addiction. Bulletin of the Menninger Clinic, 1937, I, 233-250.
- LACEY, J.I.
LACEY, B.C. The law of initial value in the longitudinal study of autonomic constitution: Reproducibility of autonomic responses and response patterns over a four year interval. Annals of New York academy of Science, 1962, 98, 1257-1290.
- LADER, M.H.
WING, L. Physiological measures, sedative drugs and morbid anxiety. Maudsley Institute of Psychiatry Monograph, 1966.
- LAZARUS, A.A. Behaviour therapy and beyond. New York: McGraw-Hill, 1968.
- LESTER, D. Self selection of alcohol by animals, human variation and the etiology of alcoholism: A critical review. Quarterly journal of studies on Alcohol, 1966, 27, 395-438.
- LESTER, D.
GREENBERG, L. Nutrition and the etiology of alcoholism. The effect of sucrose, saccharin and fat on the self-selection of ethyl alcohol by rats. Quarterly journal of studies on Alcohol, 1952, 13, 553.

- LEWIS, A.J. Health as a social concept. British Journal of Sociology, 1955, 4, 109-124.
- LEWIS, A.J. Medicine and the affections of the mind. British Medical Journal, 1963, 2, 1549-1557.
- LINDSEY, D.B. Emotion. In S.S. Stevens (Ed.), Handbook of Experimental Psychology. New York: Wiley, 1951.
- LISANSKY, E.S. The aetiology of addiction. The role of psychological predisposition. Quarterly Journal of Studies on Alcohol, 1960, 21, 314-340.
- LUBIN, B. Affective and perceptual-cognitive patterns in sensitivity training groups. Psychological Reports, 1967, 2, 365-376.
- LUDWIG, A.M. The experimental production of narcotic drug effects and withdrawal symptoms through hypnosis. International Journal of Clinical and Experimental Hypnosis, 1964, 12, 1-17.
- LUDWIG, A.M. Aspects of craving. Quarterly journal of Studies on Alcohol, 1974, 35, 899-905.
- STARKE, L.H.
- LUDWIG, A.M. "Craving" and relapse to drink. Quarterly journal of Studies on Alcohol, 1974, 35, 108-130.
- WIKLER, A

- MacANDREW, C. On the notion that certain persons who are given to frequent drunkenness suffer from a disease called alcoholism. In S.C. Plog & R.B. Edgerton (Eds.), Changing Perspectives in Mental Illness. New York: Holt, Rinehart & Winston, 1969.
- MADDEN, J.S. On defining alcoholism. British Journal of Addiction, 1977, 71, 145-148.
- MASON, J.W. Organization of Psychoendocrine Mechanisms; A Review and Reconsideration of Research. In N.S. Greenfield & R.A. Sternback (Eds.), Handbook of Psychophysiology. New York: Holt, 1972.
- MASSERMAN, J.H. An analysis of the influence of alcohol on experimental neurosis in cats. Psychosomatic Medicine, 1946, 8, 36-52.
- YUM, K.S.
- McCORD, W. Origins of Alcoholism. Stanford, California: Stanford University Press, 1960.
- McCORD, J.
- GUDEMAN, J.
- McDONNELL, G.J. A study of the relation between anxiety and skin conductance and the effect of alcohol on the conductance of subjects in a group. Quarterly journal of Studies on Alcohol, 1954, 20, 33-38.
- CARPENTER, J.A.
- McGUIRE, M.T. Comparative psychosocial studies of alcoholic and non-alcoholic subjects undergoing experimentally induced ethanol intoxication. Psychosomatic Medicine, 1966, 28, 13-26.
- STEIN, S.
- MENDELSOM, J.H.

- MELLO, N.K. Operant analysis of drinking
 MENDELSON, J.H. patterns of chronic alcoholics.
Nature, London, 1965, 206, 43-46.
- MELLO, N.K. Behavioural studies in alcoholism.
 In B. Kissen & H. Bergleiter (Eds.)
The Biology of Alcoholism (Vol.II)
Physiology and Behaviour. New York:
 Plenum, 1972.
- MELLO, N.K. Short-term memory function in
 alcohol addicts during intoxication.
 In M.M. Gross (Ed.), Alcohol
Intoxication and Withdrawal:
Experimental Studies. New York:
 Plenum, 1973.
- MELLO, N.K. Some Issues in research on the
 biology of alcoholism. In W.J.
 Filstead, J.J. Rossi & M. Keller,
 (eds.), Alcohol and Alcohol Problems.
 Cambridge: Ballinger, 1976.
- MELLOR, C.S. Aetiology in Alcoholism. In
 N. Kessel, A Hawker & H. Chalke
 (Eds.), First International Medical
Conference on Alcoholism. London:
 B. Edsel & Co, 1974.
- MENDELSON, J.H. Experimentally induced chronic
 LA DOU, J intoxication and withdrawal in
 SOLOMON, P alcoholics. Part Three Psychiatric
 Findings. Quarterly journal of
Studies on Alcohol, 1964, Suppl.2,
 40-52.

- MENDELSON, J.H. Ethanol - I - C^{I4} metabolism in alcoholics and non-alcoholics. Science, 1968, 159, 319-320.
- MENNINGER, K. Man against himself. New York: Brace & Co., 1938.
- MERRY, J. 'The loss of control myth'. Lancet II 1966, 167-168.
- MIZOI, Y. Electromyographic studies for the diagnosis of mild drunkenness.
KIMURA, A. Japanese Journal of Legal Medicine,
OLGA, N. 1963, 17, 1-8. (Abstract, Quarterly
ISBIDO, T. journal of studies on Alcohol, 1964, 25, 759).
- MOTORANO, R.D. Mood and social perception in Four Alcoholics Effects of drinking and assertion training. Quarterly journal of Studies on Alcohol, 1974, 35(2), 445-458.
- MURPHREE, H.B. Some possible origins of alcoholism. In W.J. Filstead, J.J. Rossi & M. Keller (eds.), Alcohol and Alcohol Problems. Massachusetts: Ballinger, 1976.
- MYERS, R.D. Alcohol preference in the rat:
VEALE, W.L. Reduction following depletion of brain serotonin. Science, 1968, 160, 1469-1471.
- NORDMO, S.M. Blood groups in schizophrenia, alcoholism and mental deficiency. American Journal of Psychiatry, 1959, 116, 460.

- OGATA, M. Adrenal function and alcoholism. II
MENDELSON, J.H. Catecholamines. Psychosomatic
Medicine, 1971, 33, 159.
MELLO, N.K.
MAJCHROWICZ, E.
- ORME, M.T. On the social psychology of the
psychological experiment; with
particular reference to demand
characteristics. American
Psychologist, 1962, 17, 776-783.
- PACKARD, V. The Hidden Persuaders. London:
Pelican, 1961.
- PARTINGTON, J.T. Dr Jekyll and Mr High; multidimensional
scaling of alcoholic self-evaluations.
Journal of Abnormal Psychology, 1970,
75, 131-138.
- PELTON, R.B. Metabolic characteristics of
WILLIAMS, R.J. Alcoholics: I Response to glucose
ROGERS, L.L. stress. Quarterly journal of
Studies on Alcohol, 1959, 20,
28-32.
- PERMAN, E.S. The effect of ethyl alcohol on the
secretion from the adrenal medulla
in man. Acta Physiologica Scan-
danavica, 1958, 44, 241-247.
- PITTMAN, D.J. Society, Culture and Drinking
Patterns. New York: John Wiley,
SNYDER, C.R. 1962.
- RITCHIE, J.M. The Aliphatic Alcohols. In L.
Goodman & A. Gilman (Eds.), The
Pharmacological Basis of Thera-
peutics (ed. 4). New York:
Macmillan, 1970.

- RODGERS, D.A. Mouse strain differences in various
McCLEAN, G.E. concentrations of alcohol.
Quarterly journal of Studies on
Alcohol, 1962, 23, 26-33.
- ROHAN, W.P. Comment on the "NCA" criteria
for the diagnosis of alcoholism:
An empirical evaluation study.
Journal of Studies on Alcohol,
1978, 39, 211-218.
- ROOM, R The Alcoholists Addiction.
Quarterly journal of Studies on
Alcohol, 1972, 33, 1049-1059.
- RUDIE, R. Differences in development ex-
McGAUGHRON, L. periences, defensiveness, and
personality organization between
two classes of problem drinkers.
Journal of Abnormal and Social
Psychology, 1961, 62, 659-665.
- RUSSELL, J.A. The mediating role of emotions in
MEHRABIAN, A. alcohol use. Journal of Studies
on Alcohol, 1975, 36, 1508-1538.
- SCHACTER, S. Cognitive, Social and Physiological
SINGER, J.E. determinants of emotional states.
Psychological Review, 1972, 69,
379-399.
- SCHUKIT, M.A. A study of alcoholism in half
GOODWIN, D.W. sibling. American Journal of
Psychiatry, 1972, 128, 122-126.
- SMIT, J.W. Colour-vision defects in alcoholism.
BRINTON, G.A. Quarterly journal of Studies on
Alcohol, 1971, 32, 41-44.

- STEIN, L.F. The Loss of Control Phenomenon
NILES, D. in Alcoholics. Quarterly journal
LUDWIG, A.M. of studies on Alcohol, 1968, 29,
598-602.
- STORM, T. Dissociation: A possible explanation
SMART, R.G. of some features of alcoholism,
and its implications for its
treatment. Quarterly journal of
Studies on Alcohol, 1965, 26(I),
III-III5.
- SZASZ, T.T.S. The Myth of Mental Illness. New
York: Hoeber-Harper, 1961.
- TAYLOR, J.A. A personality scale of manifest
anxiety. Journal of Abnormal and
Social Psychology, 1953, 48,
285-290.
- TAYLOR, F.K. A logical analysis of the Medical-
Psychological Concept of Disease.
Psychological Medicine, 1972,
2(I), II.
- TOMSOVIC, M. "Binge" and Continuous drinkers:
Characteristic and Treatment
follow up. Quarterly journal of
Studies on Alcohol, 1974, 35,
558-564.
- VAN DER SPUY, H. The influence of alcohol on the mood
I.J. of the alcoholic. British Journal
of Addiction, 1972, 67, 255-265.

- VAN DIJK, W.C. Problems concerning the application of the medical model in alcoholism. In N.Kessel, A. Hawker, H. Chalke (Eds.), First International Medical Conference on Alcoholism. London: B Edsell & Co., 1974.
- VOGEL, M.D. The relationship of personality factors to drinking patterns of alcoholics. Quarterly journal of Studies on Alcohol, 1961, 22, 394-400.
- VON WARTBURG, J. Alcohol dehydrogenase distribution in tissues of different species. In R.E. Popham (Ed.), Alcohol and Alcoholism (Chp. 3). Toronto: University of Toronto Press, 1970.
- WALLGREN, H. The Action of Alcohol. Amsterdam: Elsevier Publishing Co., 1970.
- BARRY, H.
- WALTON, H.J. Personality as a determinant of the form of alcoholism. British Journal of Psychiatry, 1968, 114, 761-766.
- WAY, E.L. Morphine tolerance, physical dependence, and synthesis of brain 5 hydroxytryptamine. Science, 1968, 162, 1290-1292.
- LOH, H.
- SHIN, F.H.
- WEYNEN, A.M.W. Drinking Behaviour. New York: Plenum, 1977.
- MENDELSON, J.
- WIKLER, A. Some implications of conditioning theory for problems of drug abuse. Behavioural Science, 1971, 16, 92-97.

- WILDER, J. The law of initial values. Psychosomatic Medicine, 1950, 12, 392.
- WILLIAMS, A.F. Social drinking, anxiety and depression. Journal of Personality and Social Psychology, 1968, 3, 689-693.
- WINOKUR, G. Alcoholism IV. Is there more than
RIMMER, J. one type of alcoholism? British
REICH, T. Journal of Psychiatry, 1971, 118
525-531.
- WINOKUR, G. The division of depressive illness
into depression spectrum disease and
pure depressive disease. International
Pharmoco-psychiatry, 1974, 9, 5-13.
- WORLD HEALTH ORGANIZATION Expert Committee on Mental Health:
Alcoholism subcommittee. (Second report)
Geneva: WHO, 1952.
- WORLD HEALTH ORGANIZATION Expert Committee. Technical Report
Services, 1964, 9, 273.
- World Health Organization and a new
perspective on alcoholism. Lancet,
1977, 1, 1087-1088.
- ZUCKERMAN, M. The development of an Adjective Check
List for the measurement of Anxiety.
Journal of Consulting Psychology,
1960, 24(5), 457-462.

APPENDIX A

THE ASSERTION INVENTORY.

Many people experience difficulty in handling interpersonal situations requiring them to assert themselves in some way, for example, turning down a request, asking a favour, giving someone a compliment, expressing disapproval or approval, etc. Please indicate your degree of discomfort or anxiety in the space provided before each situation listed below. Utilize the following scale to indicate degree of discomfort:

- 1 = None
 2 = a little
 3 = a fair amount
 4 = much
 5 = very much

Then, go over the list a second time and indicate after each item the probability or likelihood of your displaying the behaviour if actually presented with the situation. For example, if you rarely apologize when you are at fault, you would mark a "4" after that item, Utilize the following scale to indicate response probability:

- 1 = always do it
 2 = usually do it
 3 = do it about half the time
 4 = rarely do it
 5 = never do it

Situation	Response Probability	
	INTOX	SOBER
1. Turn down a request to borrow your car	3	4
2. Compliment a friend	2	2
3. Ask a favour of someone	3	3
4. Resist sales pressure	1	2
5. Apologize when you are at fault	1	2
6. Turn down a request for a meeting or date	2	3
7. Admit fear and request consideration	2	2
8. Tell a person you are intimately involved with when he/she says or does something that bothers you	2	2
9. Ask for a raise	2	4
10. Admit ignorance in some area	1	2
11. Turn down a request to borrow money	2	3
12. Ask personal questions	3	4
13. Turn off a talkative friend	3	3
14. Ask for constructive criticism	2	2
15. Initiate a conversation with a stranger	2	3
16. Compliment a person you are romantically involved with or interested in	2	3
17. Request a meeting or a date with a person	1	3
18. Your initial request for a meeting is turned down and you ask the person again at a later time	1	3
19. Admit confusion about a point under discussion and ask for clarification	1	1

Situation.	Response probability
20. Apply for a job	<u>2 3</u>
21. Ask whether you have offended someone	<u>1 2</u>
22. Tell someone that you like them	<u>2 2</u>
23. Request expected service when such is not forthcoming, e.g. in a restaurant	<u>2 2</u>
24. Discuss openly with the person his/her criticism of your behaviour	<u>2 3</u>
25. Return defective items, e.g. store or restaurant	<u>2 2</u>
26. Express an opinion that differs from that of the person you are talking to	<u>2 2</u>
27. Resist sexual overtures when you are not interested	<u>2 2</u>
28. Tell the person when you feel he/she has done something that is unfair to you	<u>2 2</u>
29. Accept a date	<u>1 2</u>
30. Tell someone good news about yourself	<u>1 2</u>
31. Resist pressure to drink	<u>1 1</u>
32. Resist a significant person's unfair demand	<u>2 2</u>
33. Leave a job	<u>1 4</u>
34. Resist pressure to take drugs	<u>1 2</u>
35. Discuss openly with the person his/her criticism of your work	<u>1 2</u>
36. Request the return of borrowed items	<u>2 2</u>
37. Receive compliments	<u>3 3</u>
38. Continue to converse with someone who disagrees with you	<u>2 2</u>
39. Tell a friend or someone with whom you work when he/she says or does something that bothers you	<u>2 2</u>
40. Ask a person who is annoying you in a public situation to stop	<u>3 3</u>

APPENDIX B

EYSENCK PERSONALITY INVENTORY

by H. J. Eysenck and Sybil B. G. Eysenck

PERSONALITY QUESTIONNAIRE**FORM A**

NAME..... AGE.....

OCCUPATION..... SEX.....

N=

E=

L=

Instructions

Here are some questions regarding the way you behave, feel and act. After each question is a space for answering "YES" or "NO".

Try to decide whether "YES" or "NO" represents your usual way of acting or feeling. Then put a cross in the circle under the column headed "YES" or "NO". Work quickly, and don't spend too much time over any question; we want your first reaction, not a long-drawn out thought process. The whole questionnaire shouldn't take more than a few minutes. Be sure not to omit any questions.

Now turn the page over and go ahead. Work quickly, and remember to answer every question. There are no right or wrong answers, and this isn't a test of intelligence or ability, but simply a measure of the way you behave.



UNIVERSITY OF LONDON PRESS LTD



FORM A

YES NO

- 1. Do you often long for excitement? YES NO
- 2. Do you often need understanding friends to cheer you up? YES NO
- 3. Are you usually carefree? YES NO
- 4. Do you find it very hard to take no for an answer? YES NO
- 5. Do you stop and think things over before doing anything? YES NO
- 6. If you say you will do something do you always keep your promise, no matter how inconvenient it might be to do so? YES NO
- 7. Does your mood often go up and down? YES NO
- 8. Do you generally do and say things quickly without stopping to think? YES NO
- 9. Do you ever feel "just miserable" for no good reason? YES NO
- 10. Would you do almost anything for a dare? YES NO
- 11. Do you suddenly feel shy when you want to talk to an attractive stranger? YES NO
- 12. Once in a while do you lose your temper and get angry? YES NO
- 13. Do you often do things on the spur of the moment? YES NO
- 14. Do you often worry about things you should not have done or said? YES NO
- 15. Generally, do you prefer reading to meeting people? YES NO
- 16. Are your feelings rather easily hurt? YES NO
- 17. Do you like going out a lot? YES NO
- 18. Do you occasionally have thoughts and ideas that you would not like other people to know about? YES NO
- 19. Are you sometimes bubbling over with energy and sometimes very sluggish? YES NO
- 20. Do you prefer to have few but special friends? YES NO
- 21. Do you daydream a lot? YES NO
- 22. When people shout at you, do you shout back? YES NO
- 23. Are you often troubled about feelings of guilt? YES NO
- 24. Are all your habits good and desirable ones? YES NO
- 25. Can you usually let yourself go and enjoy yourself a lot at a lively party? YES NO
- 26. Would you call yourself tense or "highly-strung"? YES NO
- 27. Do other people think of you as being very lively? YES NO

- | | YES | NO |
|--|-----------------------|-----------------------|
| 28. After you have done something important, do you often come away feeling you could have done better? | <input type="radio"/> | <input type="radio"/> |
| 29. Are you mostly quiet when you are with other people? | <input type="radio"/> | <input type="radio"/> |
| 30. Do you sometimes gossip? | <input type="radio"/> | <input type="radio"/> |
| 31. Do ideas run through your head so that you cannot sleep? | <input type="radio"/> | <input type="radio"/> |
| 32. If there is something you want to know about, would you rather look it up in a book than talk to someone about it? | <input type="radio"/> | <input type="radio"/> |
| 33. Do you get palpitations or thumping in your heart? | <input type="radio"/> | <input type="radio"/> |
| 34. Do you like the kind of work that you need to pay close attention to? | <input type="radio"/> | <input type="radio"/> |
| 35. Do you get attacks of shaking or trembling? | <input type="radio"/> | <input type="radio"/> |
| 36. Would you always declare everything at the customs, even if you knew that you could never be found out? | <input type="radio"/> | <input type="radio"/> |
| 37. Do you hate being with a crowd who play jokes on one another? | <input type="radio"/> | <input type="radio"/> |
| 38. Are you an irritable person? | <input type="radio"/> | <input type="radio"/> |
| 39. Do you like doing things in which you have to act quickly? | <input type="radio"/> | <input type="radio"/> |
| 40. Do you worry about awful things that might happen? | <input type="radio"/> | <input type="radio"/> |
| 41. Are you slow and unhurried in the way you move? | <input type="radio"/> | <input type="radio"/> |
| 42. Have you ever been late for an appointment or work? | <input type="radio"/> | <input type="radio"/> |
| 43. Do you have many nightmares? | <input type="radio"/> | <input type="radio"/> |
| 44. Do you like talking to people so much that you never miss a chance of talking to a stranger? | <input type="radio"/> | <input type="radio"/> |
| 45. Are you troubled by aches and pains? | <input type="radio"/> | <input type="radio"/> |
| 46. Would you be very unhappy if you could not see lots of people most of the time? | <input type="radio"/> | <input type="radio"/> |
| 47. Would you call yourself a nervous person? | <input type="radio"/> | <input type="radio"/> |
| 48. Of all the people you know, are there some whom you definitely do not like? | <input type="radio"/> | <input type="radio"/> |
| 49. Would you say that you were fairly self-confident? | <input type="radio"/> | <input type="radio"/> |
| 50. Are you easily hurt when people find fault with you or your work? | <input type="radio"/> | <input type="radio"/> |
| 51. Do you find it hard to really enjoy yourself at a lively party? | <input type="radio"/> | <input type="radio"/> |
| 52. Are you troubled with feelings of inferiority? | <input type="radio"/> | <input type="radio"/> |
| 53. Can you easily get some life into a rather dull party? | <input type="radio"/> | <input type="radio"/> |
| 54. Do you sometimes talk about things you know nothing about? | <input type="radio"/> | <input type="radio"/> |
| 55. Do you worry about your health? | <input type="radio"/> | <input type="radio"/> |
| 56. Do you like playing pranks on others? | <input type="radio"/> | <input type="radio"/> |
| 57. Do you suffer from sleeplessness? | <input type="radio"/> | <input type="radio"/> |

PLEASE CHECK TO SEE THAT YOU HAVE ANSWERED ALL THE QUESTIONS