THE EFFECTS OF ALCOHOL ON COGNITIVE, PSYCHOMOTOR, AND AFFECTIVE FUNCTIONING

Report and Recommendations Prepared by an Expert Working Group for the Royal Commisision Into Aboriginal Deaths in Custody

Janet Greeley & William Gladstone (Eds)

NDARC Monograph No. 8

Monograph No. 8

The Effects of Alcohol on Cognitive, Psychomotor, and Affective Functioning

Edited by

Janet Greeley
and
William Gladstone

National Drug and Alcohol Research Centre
University of New South Wales
PO Box 1
Kensington NSW 2033
Australia

ISBN 0 947229 12 4

Monograph No. 8

The Effects of Alcohol on Cognitive, Psychomotor, and Affective Functioning

*Report and Recommendations Prepared by an Expert Working Group for the Royal Commission into Aboriginal Deaths in Custody

Edited by

Janet Greeley
and
William Gladstone

Editorial assistant: Wendy Swift

National Drug and Alcohol Research Centre
Monograph No. 8

ISBN 0 947229 12 4

Funded by the National Campaign Against Drug Abuse

TABLE OF CONTENTS

Acknowledgements

The editors and contributors wish to thank Mrs Eva Congreve, Librarian at the National Drug and Alcohol Research Centre, for her tremendous assistance in locating and despatching reference materials. The editors also thank the numerous other staff members of the NDARC for their assistance at various stages in the preparation of this document. A special thank you to Mr Michael Tilbury, School of Law, University of New South Wales for providing the list of references on expert testimony (Appendix F).

Contributors

F. HARDING BURNS, MBBS, FRACP, FRCP

Dr Burns is currently Visiting Physician in Drug and Alcohol Services at Royal Prince Alfred Hospital, Concord Hospital, and Northside Clinic. He is a member of the WHO Collaborative Project on the Identification and Treatment of Persons with Harmful Alcohol Consumption. In 1980 Dr Burns became President of the Alcohol and Drug Foundation, Australia.

GREGORY BRADFORD CHESHER, MSc, PhD

Dr Chesher was reader in the Department of Pharmacology, University of Sydney until the end of 1986, when he took voluntarily early retirement. He is presently an Honorary Associate in that department and an Honorary Visiting Fellow of the National Drug and Alcohol Research Centre, University of New South Wales. Dr Chesher's research interests in the field of psychopharmacology include the effects of drugs on human cognitive and performance skills, and pharmacological and social perspectives and drug dependence.

JOHN ELLARD, MBBS, Dip Psych Med, FRACP, FRANZCP, FRCP, OA

Dr John Ellard is Chairman of the psychiatric practice at Northside Clinic, Sydney and Editor of Modern Medicine of Australia. He has served on Committees of Inquiry into psychosurgery and the needs of the mentally ill, and has published extensively on topics in psychiatry. Dr Ellard is currently Chairman, Practice Standards Committee, Royal Australian and New Zealand College of Psychiatrists; Chairman, Medical Committee; and a Member of the Advisory Board, Institute of Criminology.

PATRICIA FAGAN, MBBS, DCH, DRCOG

Dr Fagan has worked for seven years as a General Practitioner at a Community Controlled Aboriginal Health Service in Sydney. In her position at the Aboriginal Medical Service, she has been involved in educating, both formally and informally, medical students and other health professionals in different aspects of Aboriginal health. Dr Fagan is currently enroled in a Masters of Public Health degree at the University of Sydney and for the past two years has served on the AIDS Education Subcommittee.

WILLIAM GLADSTONE, BSc(Hons), PhD

Dr Gladstone is Senior Tutor in Biological Sciences at the University of Sydney. He has worked as researcher and project officer for studies into the employment of nurses in New South Wales; education about drugs and alcohol for Australian medical students; and revision of the methadone guidelines.

JANET GREELEY, BSc(Hons), MA, PhD

Dr Greeley is Lecturer at the National Drug and Alcohol Research Centre, University of New South Wales. A psychopharmacologist by training, her principal research interests centre on the role of learning in the development of drug tolerance and dependence. She studied, for 6 years, at the Addiction Research Foundation of Ontario, Canada. Dr Greeley has wide professional interests in the drug and alcohol field and has published and edited a number of scientific papers and monographs.

ALLAN HODDA, BSc, MSc

Mr Hodda joined the New South Wales Department of Health in 1969 and has worked in the Division of Analytical Laboratories until the present time. Most of his analytical experience is in forensic toxicology and he is currently the Assistant Government Analyst in charge of the Forensic Science Branch incorporating forensic toxicology. He has had several secondments outside the Laboratories including two years as co-ordinator of Scientific Services for the Health Department. He is currently actively involved in developing drug analysis facilities for the detection of drugs in intoxicated drivers in New South Wales.

ERNEST HUNTER, MBBS, MPM, MA, FRANZCP, Dip Amer Board Psych & Neurol.

Dr Hunter is an Australian medical graduate trained in adult, child, and cross-cultural psychiatry in the United States. He has been a research fellow with the New South Wales Institute of Psychiatry, and is currently continuing his research among Aboriginal populations in the Kimberley region of Western Australia.

MARTIN JACKSON, BSc (Hons), MSc

Mr Jackson is Clinical Neuropsychologist at the Regional Brain Damage Unit, Royal Prince Alfred Hospital, Sydney. His work involves the assessment of alcohol-related brain damage, counselling of clients and their families, making recommendations for rehabilitation programmes, and assisting in such programmes. He has written on the topics of chronic alcohol-related brain damage, social drinking and drug-related brain damage.

JEAN A. JANS

Ms Jans has worked in the field of drugs and alcohol for more than 10 years, with Aboriginal and non-Aboriginal people. As Principal Health Worker, she was involved in Aboriginal health in South Australia. She is currently Programs Officer in the Alcohol and Drug Dependence Service, Queensland.

DAVID McDONALD, BA, Dip Soc Wk, MA

Mr McDonald has a professional background in social welfare and community health, having experience in fieldwork, research, teaching and administration in these areas in Australia, Papua New Guinea and Canada. In recent years he has served as Deputy Director of the Northern Territory Drug and Alcohol Bureau and, in the 1986-1988 period, as Director of the National Drug Abuse Information Centre. He is currently seconded from the Commonwealth Department of Community Services and Health to the Royal Commission Into Aboriginal Deaths in Custody where he is Deputy Head of Research.

JOHN SAUNDERS, MBBS, MD, MRCP

Dr Saunders is Head of Drug and Alcohol Services and the New South Wales State-wide Training Unit at Royal Prince Alfred Hospital, Sydney. He has specialised in gastroenterology and alcohol and drug problems at King's College Hospital and the Addiction Research Unit, London. He is a member of the Drug Offensive Council of New South Wales, a member of the National Committee for Alcohol and Drug Education in Medical Schools and is Editor of the Australian Drug and Alcohol Review. His research interests include the metabolism of alcohol, alcohol and drug interactions and susceptibility to alcohol dependence and its physical consequences. He has co-authored with Dr J Christopher Clarke "Alcoholism and Problem Drinking: Theories and Treatment" and has published around 100 papers and reviews.

SISTER PAT SWAN, RCN, RPN, RGN

Sr Swan has had many years of nursing and counselling experience working in different areas of mental health as well as direct nursing care, both in institutional and community health centre settings, including acute and chronic care, patient assessment, program development, rehabilitation and administration. She is actively involved in Aboriginal health issues and programs on a community, state, national and international level. She is a former executive member of the National Aboriginal and Islander Health Organisation. Sr Swan is currently a member of the NSW Aboriginal Health Resource Committee and is employed by AMS Redfern in health promotion, counselling and program development. She is also on a panel to assist in the development of a National Strategy for HIV/AIDS.

WENDY SWIFT, BA(Hons)

Ms Swift is a Research Assistant at the National Drug and Alcohol Research Centre.

Abbreviations

BAC - blood alcohol concentration

g% - grams per cent

mg - milligram(s)

g - gram(s)

ml - millilitre(s)

l - litre(s)

Monographs

No. 1. Greeley, J. & Gladstone, W. (Eds.) (1987). *Methadone Programs in Australia:Policy and Practice: Proceedings From the National MethadoneWorkshop, 30th-31st March, 1987.* National Drug & Alcohol Research Centre.

No. 2. Prescott, J. (1987). *Issues and Priorities in Methadone Research.* National Drug & Alcohol Research Centre.

No. 3. Greeley, J. & Heather, N. (Eds.) (1987). *Towards a National Drug & Alcohol Research Network: Proceedings from the First Annual NDARC Research Symposium, November, 1987.* National Drug & Alcohol Research Centre.

No. 4. Swift, W. & Greeley, J.(Eds.) (1988). *The Future of the Addiction Model: Proceedings From a Seminar, Prince of Wales Hospital, August, 1988.* National Drug & Alcohol Research Centre.

No. 5 . Prescott, J. & McCall, G. (Eds.) (1988) *Kava: Use and Abuse in Australia and the South Pacific: Proceedings from the Symposium on Kava, University of New South Wales, 11th November, 1988.* National Drug & Alcohol Research Centre.

No. 6. Greeley, J. & Heather, N. (Eds.) (1988) *Perspectives on Drug and Alcohol Dependence: Proceedings from a Symposium held during the XXIV International Congress of Psychology, Sydney, August 30, 1988.* National Drug & Alcohol Research Centre.

No. 7. Grenyer, B. F. & Solowij, N. (Eds.) (1989) *Cognitive-Behavioural Approaches to the Treatment of Drug and Alcohol Problems: Proceedings from the Second National Drug & Alcohol Research Centre Annual Symposium, Sydney, December 16, 1988.* National Drug & Alcohol Research Centre.

No. 8. Greeley, J. & Gladstone, W. (Eds.) (1989) *The effects of alcohol on cognitive, psychomotor, and affective functioning. Report and recommendations prepared by an Expert Working Group for the Royal Commission into Aboriginal Deaths in Custody.* National Drug & Alcohol Research Centre.

Technical Reports

No. 1. Keats, J. A., Webster, R. A. & O'Brien,S. T. (1989). *Social Processes in Alcohol Dependence: An Analysis of the Australian Inventory of Alcohol Usage.* National Drug & Alcohol Research Centre.

No. 2. Westbrook, R. F. & Greeley, J. D. (1989). *Some Effects of the Opioid Antagonist, Naloxone,Upon the Rat's Reactions to a Heat Stresor.* National Drug & Alcohol Research Centre.

No. 3. Chesher, G., Lemon, J., Gomel, M. & Murphy, G. (1989). *The Effects of Methadone as Used in a Methadone Maintenance Program, on Driving-Related Skills* . National Drug & Alcohol Research Centre.

No. 4. Pedic, F. (1990). *A Review of the National Drug Poisonings Case Reporting System.* National Drug & Alcohol Research Centre.

No. 5. Pedic, F. & Flaherty, B. (1990). *Early Warning Indicators of Changes in Drug Use: A New South Wales Perspective.* National Drug & Alcohol Research Centre.

No. 6. Heather, N. (1990). *"Assisted Spontaneous Remission" from Alcohol Problems: Effects of a Controlled Drinking Self-help Manual With and Without Added Telephone Contact.* National Drug & Alcohol Research Centre.

The Effect of Alcohol on Cognitive, Psychomotor, and Affective Functioning

Chapter 1. Introduction

Janet Greeley

The purpose of this report is to review and evaluate research on the assessment of a person's ability to carry out certain behaviours while under the influence of the drug, alcohol. Intoxication by alcohol is often found to be a relevant factor in forensic investigations. It is well known that alcohol affects behaviour and that when an individual is alleged to have acted under its influence, this factor should be considered.

Alcohol can affect both mental and physical functions necessary for processing and acting on information. In everyday language, cognitive, psychomotor, and affective functioning might be described as the thoughts, actions, and mood-states which are component parts of virtually all human behaviour. This report is a review of research findings and clinical reports on the effects of alcohol on behaviour. The precision of the behavioural measurements used to assess capacity to function while under the influence of alcohol varies considerably - from well-controlled laboratory tests to reports of brief clinical assessments of intoxication. Laboratory studies attempt to measure precisely, particular components of cognitive, psychomotor and affective functioning; clinical assessments, on the other hand, tend to employ gross rating scales that provide commentary on the general demeanor of the person and his/her capacity to perform basic functions such as speaking and walking properly. The type of measure most appropriate to use in estimating capabilities to perform certain acts while under the influence of alcohol will depend largely on the cognitive and psychomotor requirements of the behaviour in question.

There are many factors which can influence the effect that a given dose of alcohol will have on mental and physical functioning. Of particular interest, is the difference between experienced and inexperienced drinkers in their reactions to alcohol. Prior drinking history can affect both sensitivity to the effects of an acute dose of alcohol as well as cognitive, psychomotor, and affective functioning in the absence of alcohol.

Eight areas were identified as having specific relevance to the evaluation of behaviour while under the influence of alcohol. The subject areas and the rationales for each are given below.

An essential element in any discussion of drug effects is to understand how the drug acts in the body. Factors involved in the absorption, distribution, metabolism and

elimination of alcohol are considered in Chapter 2 on the clinical pharmacology of alcohol. Genetic differences in alcohol metabolising enzymes are also discussed.

In any forensic inquiry in which alcohol has been implicated as a possible contributing factor, it is critical to establish, with a high degree of accuracy, the concentration of alcohol in the blood at the time the event under investigation took place. Thus, factors important to the accurate analysis of alcohol levels in blood are considered in Chapter 3.

Two key areas of interest to this report are presented in Chapters 4 and 5. Chapter 4 is a review of the literature on the effects of alcohol on cognitive and psychomotor functioning. Chapter 5 considers research on the development of tolerance to the effects of alcohol on these processes. Before acting, a person must first perceive objects and events in the environment, think about the information received, plan a response, and perform controlled movements to carry out the behaviour. The amount of mental processing engaged in at these different stages will depend upon the requirements of the behaviour to be carried out. Alcohol can affect the processing of information at various stages in this chain. The research literature summarised in Chapter 4 discusses these effects of alcohol and considers other important factors which interact with alcohol to modify behaviour.

Any estimation of the acute effects of different blood alcohol concentrations on cognitive and psychomotor functioning must consider the drinking history of the person taking the drug. The effect of a given blood alcohol concentration on an individual who is an inexperienced drinker can be very different from the effect of the same amount of alcohol in someone who has had many years of experience dealing with the intoxicating effects of this drug. The role of tolerance and factors which influence the development and expression of tolerance to the effects of alcohol are reviewed in Chapter 5. Experimental research and clinical reports on tolerance to the intoxicating effects of moderate to very high doses of alcohol are discussed. Other important factors, such as the interaction of alcohol with other drugs present in the body, are also considered briefly in this chapter.

In a consideration of tolerance to the cognitive and psychomotor impairing effects of alcohol, especially to high doses of alcohol, the issue of alcohol dependence is relevant. Much of the research on tolerance to alcohol involves comparisons between groups of individuals classified as light to moderate social drinkers with other individuals who have been diagnosed as alcoholics or as alcohol dependent. Thus, it is important to understand, at least in general terms, how these various classifications are defined. There are medical, psychiatric, and social complications related to alcohol dependence which may also influence behaviour. Cognitive, psychomotor and affective functioning can also be affected if a person is in a state of withdrawal

induced by a sudden, significant reduction in alcohol intake. Thus, this factor should also be considered when investigating behaviours that have occurred while a person is under the influence of alcohol. These and other issues are considered in Chapter 6 on the treatment of alcohol dependence.

Alcohol is known to produce toxic effects on different parts of the body. Of particular interest, in the context of the present report, is the toxic effect of chronic alcohol use on the brain. Chapter 7 reviews the literature on brain damage produced by alcohol. The functional integrity of the brain, in the absence of alcohol, will affect the interpretation of tests which assess the acute effects of a dose of alcohol on cognitive and psychomotor functioning. Clearly, this is another relevant factor in evaluating the mental and behavioural capacity of an individual acting under the influence of alcohol.

Another way in which alcohol can affect mental functioning is through its effects on mood. The emotional state that a person experiences while under the influence of alcohol may importantly influence the type of behaviour in which he/she will engage. Some of the pharmacological and nonpharmacological factors which play an important role in the mood changes observed under alcohol are briefly considered in Chapter 8.

In addition to producing transient changes in emotional state, alcohol can also influence more permanent affective states such as those related to mood disorders (e.g., depression and anxiety). Existing psychiatric conditions can be affected by acute alcohol intoxication and it is reported that a high incidence of psychiatric disorders is found in individuals who have developed a drinking problem or a dependence on alcohol. There is a substantial literature on the relationship between alcohol, alcohol dependence and self-harming behaviours. These issues are considered in Chapters 7 and 8. The issue of incarceration and its association with intoxication and self-harming behaviours is also discussed in these chapters.

In Chapter 10, a particular view of the relevance of empirical research to clinical problems is presented. In this section, some possible shortcomings of applying the scientific method to real-life situations are discussed. Although many of the problems that are outlined in this chapter do occur in clinical research, it would be a mistake to conclude that all research into areas of clinical significance is of poor quality. Knowledge about the capacity to perform tasks under alcohol can be gathered through other methods such as personal experience. One of the major advantages of the scientific method, when it is used appropriately, is that the procedures used to gather information are open to public scrutiny in a way that knowledge gained through personal experience is not.

The preparation of this report was requested by the Royal Commission Into Aboriginal Deaths in Custody. It will be included as one of a series of research reports the Commission may use as resource documents in its deliberations. All contributors to the document were advised that the purpose of the report was to provide an objective framework in which to identify relevant and reliable scientific evidence such as might be useful in a judicial inquiry, and that the report would be used, in the first instance, as a reference source for the Royal Commission.

In all sections of this report efforts were made to discuss research findings which were relevant to possible differences in the effects of alcohol due to population factors such as gender, age and race. Chapter 9 also introduced the importance of possible cultural differences. There are many reports of cultural differences in the ways drugs and alcohol are used and experienced (e.g., Marshall, 1981). A comprehensive consideration of these factors is beyond the scope of this report, but is mentioned here to draw the reader's attention to its potential significance in understanding the effects of alcohol on behaviour.

Although the report is a technical document, its use by the Royal Commission made it important that representatives of the Aboriginal people participate in its preparation. Representatives from the Aboriginal Medical Service in Redfern, New South Wales and the Aboriginal Coordinating Council in Cairns, Queensland were asked to comment on the material presented and to identify and discuss issues which may be of particular relevance to Aboriginal people, culture and health.

In the preparation of the technical chapters (2 to 10), authors were instructed to identify and address key issues in their area in a concise, but comprehensive, manner. The literature reviews are meant to provide an overview of the more important issues rather than a detailed coverage of all published research in a particular area. The reference lists should provide a useful bibliographical resource to those involved in interpreting and providing expert testimony in forensic cases in which alcohol is involved. A collection containing much of the literature cited in this report has been established at the National Drug and Alcohol Research Centre in Sydney.

At the first Committee meeting, the authors of the technical chapters presented outlines of their sections for review and approval by the group. Committee Members who contributed chapters were responsible for the scholarship of their individual sections. The Committee, as a whole, served as a review panel commenting on the clarity, comprehensiveness, and, where appropriate, accuracy of the material presented. All technical sections were reviewed by the Committee during a two-day workshop held at the National Drug and Alcohol Research Centre in Sydney.

The commentary sections (Chapter 11) were prepared after the workshop and did not undergo the same review process as the other sections. Accordingly, they do not reflect a consensus view. None of the papers presented necessarily reflects the policies or programs of the National Drug and Alcohol Research Centre.

Reference

Marshall, M. (1981). "Four Hundred Rabbits": An anthropological view of ethanol as a disinhibitor. In R. Room & G. Collins (Eds.), Alcohol and disinhibition: Nature and meaning of the link (Research Monograph No. 12, pp. 186-204). Rockville, MD: National Institute on Alcohol Abuse and Alcoholism.

Chapter 2. Clinical Pharmacology of Alcohol

John Saunders

1. The fate of alcohol in the body

1.1 The chemistry of alcohol

Alcohol is a simple chemical substance with a molecular weight of 46. It is highly soluble in water but barely so in fat because of its particular chemical properties. It is produced by fermentation of sugars and other carbohydrates which derive from fruits and vegetables including grapes (from which wines and certain spirits are produced) or from cereals (beer and grain spirits). Alcohol is naturally present in very low concentrations in the body (typically less than 0.1 mg/100 ml (0.0001 g%). These concentrations are of no pharmacological significance.

1.2 Absorption

After ingestion, alcohol is rapidly absorbed in the upper gastrointestinal tract, principally in the upper small bowel (80%) and to a lesser extent in the stomach (20%) (Kalant, 1971). As it is water soluble, alcohol is absorbed by passive diffusion and there is no evidence of facilitated or active transport mechanisms. Following absorption, it enters the portal venous circulation, through which it reaches the liver and then the systemic circulation. Typically, the peak blood alcohol concentration (BAC) is reached 30-60 minutes after a single dose of alcohol.

1.3 Distribution

As a water-soluble compound alcohol is distributed into the 'total body water'. This compartment, which is equivalent to the lean body mass, includes such organs as the liver, heart, muscles and brain, and also the blood. The speed with which tissue concentrations of alcohol come into equilibrium with those in blood varies. For the brain it is approximately ten minutes, and for skeletal muscle about one hour (Harger, Hulpieu & Lamb, 1937). Alcohol does not enter fatty tissue, nor the skeleton, to a significant extent.

1.4 Elimination

Alcohol is eliminated from the body principally by metabolism (chemical breakdown) in the liver. In a normal healthy individual, approximately 95% of a dose of alcohol is metabolised here. Approximately 2-3% is metabolised elsewhere in the body (for example in the lung) (Larsen, 1959) and a similar proportion is excreted in urine or sweat, or exhaled in breath. At high BACs, proportionately more alcohol is excreted or exhaled (Kalant, 1971).

The principal pathway by which alcohol is metabolised is oxidation. This is a two stage process which involves, firstly, the conversion of alcohol to acetaldehyde (for a

diagrammatic illustration see Lieber, 1982; Saunders, 1988). Acetaldehyde is a very reactive and toxic substance which reacts with many constituents of the body's tissues, including proteins and structural lipids. It is also responsible for the facial flushing reaction which occurs in Oriental peoples. Acetaldehyde is in turn oxidized to acetate which then participates in a number of subsidiary metabolic reactions, with the production ultimately of carbon dioxide and water (Lieber, 1982). The first metabolic step is catalysed (chemically accelerated) by an enzyme, alcohol dehydrogenase. The second step is catalysed by aldehyde dehydrogenase. Another enzyme system, termed the 'microsomal ethanol oxidizing system', is probably responsible for a small proportion (approximately 3-5%) of the initial oxidation of alcohol; this reaction also results in the production of acetaldehyde. Because of the particular properties of these enzymes the concentrations of acetaldehyde in the liver and other tissues are normally extremely low.

The rate of alcohol elimination in a healthy person is approximately 15 mg/100 ml/hour (0.015 g% per hour), which is equivalent to about 8 g (one standard drink) per hour.

2. Factors influencing blood alcohol concentrations (BACs)

2.1 Introductory remarks

The concentration of alcohol in the blood that is reached after drinking is relatively difficult to predict. Clearly it is related to the amount of alcohol consumed and the time over which drinks are taken. The peak BAC will be lower if drinking takes place over several hours than if the same amount is consumed over a shorter period. The interval since the last drink is also important as the BAC may not have returned to zero. However there are several other factors which influence how alcohol is handled by the body and therefore affect the BAC. These include:

(1) The type of drink consumed.

(2) Whether alcohol is taken on an empty stomach or after a meal.

(3) Body weight and composition.

(4) The state of hydration.

(5) Sex.

(6) The rate of alcohol metabolism.

(7) Previous alcohol and drug intake.

(8) Concurrent disease.

The variation in BACs after a standard (weight-adjusted) dose is considerable. Even in healthy subjects peak BACs can vary two to three-fold after a given dose. Some of the causes will now be explored.

2.2 Factors influencing absorption and distribution

The rate of absorption and therefore the peak BAC are significantly influenced by the alcohol content of the drink taken. Absorption is most rapid when alcohol is ingested as a 20% solution (equivalent to sherry, or spirits diluted 50/50 with water, soda or tonic) (Haggard, Greenberg & Cohen, 1938; Mellanby, 1919). Absorption of diluted drinks such as beer is slower. Drinks with a higher alcohol concentration than 20% tend to inhibit stomach emptying by causing spasm of the pyloric sphincter. Delivery of alcohol to the upper small bowel, the site of most rapid absorption, is therefore delayed and absorption is erratic and prolonged.

The presence of food in the stomach also retards the absorption of alcohol considerably, partly because alcohol is retained in the stomach longer. The type of food seems to make little difference, as the effect has been demonstrated with carbohydrate, fat and protein (Kalant, 1971). When alcohol is taken with a meal, the peak BAC may be less than 20% of that reached if it is taken on an empty stomach (Welling, Lyons, Elliot & Amidon, 1977). Alcohol is absorbed more rapidly after stomach resection or vagotomy (operations commonly undertaken for peptic ulcer disease) (Elmslie, Davis, Magee & White, 1964). Absorption is also enhanced if drugs that relax the pyloric sphincter are taken concomitantly; an example is metoclopramide, an anti-nausea preparation.

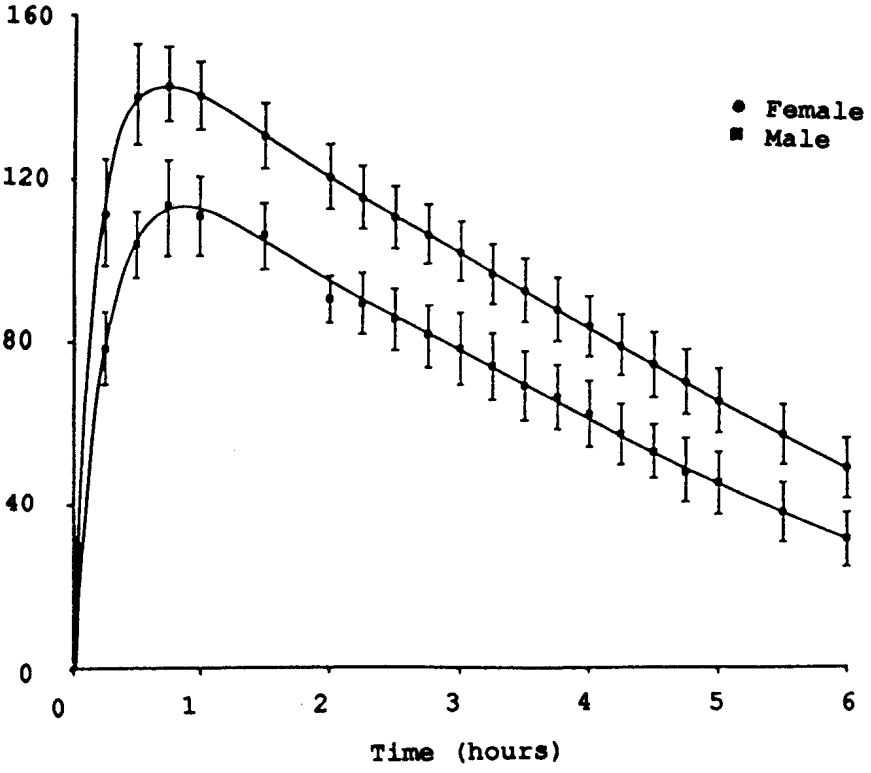
As alcohol is distributed into the lean body mass, blood and tissue concentrations will depend on the size of this compartment and the relative proportion of fat to lean body mass. Clearly, a person weighing 40 kg will have a smaller lean body mass than someone weighing 120 kg. Because fatty tissue constitutes a higher proportion of body mass in women, they have higher BACs after a standard dose of alcohol than men (Figure 1) (Saunders, Davis & Williams, 1981; Marshall, Kingstone, Boss & Morgan, 1983), and therefore higher concentrations of alcohol in the liver and brain. Persons who are obese have higher average BACs after a standard dose than do muscular individuals of the same sex and identical weight. A person who is dehydrated will have a lower than normal body water (and smaller lean body mass). BACs are likely to be higher after drinking than in a normally hydrated individual; no research data are available on this point though.

2.3 Factors influencing the rate of elimination of alcohol

The rate at which alcohol is eliminated from the body in normal, healthy persons ranges from approximately 13 mg/100 ml/hour (0.013 g% per hour) to 20 mg/100 ml/hour (0.020 g%

per hour). This is equivalent to 6 - 10 g of alcohol (or approximately one standard drink) in a healthy 70 kg person. The rate of elimination has a significant heritable component (Vesell, Page & Passananti, 1971) which presumably reflects metabolic factors such as the activities of alcohol dehydrogenase and aldehyde dehydrogenase. The metabolic rate increases by a maximum of 50-70% in chronic heavy drinkers (Kater, Carulli & Iber, 1969; Saunders, 1986)(Figure 2).

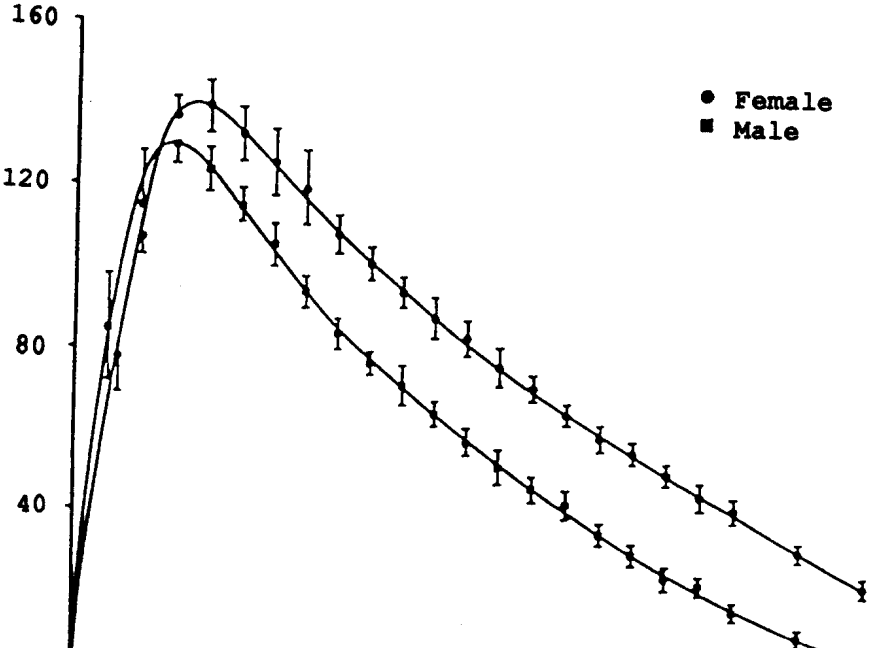
Figure 1: Blood alcohol concentrations after an oral dose of alcohol in healthy subjects.



(Adapted from Saunders (1986) by permission of the author.)

Blood alcohol concentration (mg 100^{-1})

Figure 2: Blood alcohol concentrations after an oral dose of alcohol in patients with minimal changes in liver, fatty liver or fibrosis.



Accordingly, BACs decline more rapidly, as can be seen by comparing Figures 1 and 2. The biochemical basis of this is not fully established. In part it seems to be due to induction of the cytochrome P450-dependent microsomal ethanol oxidising system. Increase in the synthesis of an inducible form of alcohol dehydrogenase is also a possibility.

The increase in the metabolic rate following chronic alcohol consumption is sometimes termed 'metabolic tolerance' and is distinct from, but complementary to, the 'functional tolerance' that occurs as a result of neuroadaptation by the central nervous system. Metabolism of alcohol is also increased to a limited extent by prior medication with sedative drugs which also induce cytochrome P450-dependent enzymes.

The rate of elimination of alcohol is not greatly affected by the dose of alcohol consumed or the BAC, for the enzymes metabolising it are essentially saturated at concentrations above 50 mg/100 ml (0.05 g%). At very high concentrations (above 200 mg/100 ml (0.2 g%)) the rate of elimination is somewhat greater than usual, probably because other alcohol metabolising enzymes are operating and also because a higher proportion is excreted in urine. The rate of metabolism of alcohol is reduced by the presence of serious liver disease. For it to decline substantially below the normal range the liver disease has to be severe enough to cause hepatic decompensation (Lieberman, 1963; Saunders, 1986).

Because of all these factors, prediction of BACs after a given amount of alcohol is not possible with any degree of accuracy. Correspondingly, predicting the amount of alcohol that has been consumed by an individual with a known BAC cannot be done precisely. As a very rough guide, three standard drinks (30 g alcohol) taken over 30 minutes on an empty stomach will result in a peak BAC of approximately 50 mg/100 ml (0.05 g%) one hour after the last drink. However, this might vary from 20 mg/100 ml (0.02 g%) to 80 mg/100 ml (0.08 g%). Twelve standard drinks (120 g alcohol) taken over two hours will result in BACs of approximately 200 - 250 mg/100 ml (0.20 - 0.25 g%) one hour after the last drink. (The formula used to calculate a rough estimate of the BAC is given in Appendix A.)

2.4 Racial variations in the rate of elimination of alcohol

Despite the fact that there are racial variations in the composition of both alcohol dehydrogenase and aldehyde dehydrogenase (see Section 3), there are no significant racial differences in the rate of elimination of alcohol. Of particular relevance to the present report is a study of alcohol elimination by Australian Aboriginals (defined as being of at least 50% pure Aboriginal descent) in comparison with white Caucasian Australians, which was reported by Marinovich and colleagues (1976). They studied 16 Aboriginal men and 12 men of European descent aged between 18 and 48 years. Each was given 1 ml of alcohol per kilogram body weight, and BACs were estimated for up to six hours after

ingestion. The mean rate of elimination for the Aboriginal men was 18 mg/100 ml/hour (0.018 g% per hour), with a range of 13 - 24 mg/100 ml/hour. This compared with a mean rate for European males of 17.7 mg/100 ml/hour (0.0177 g% per hour) with a range of 15-21 mg/100 ml/hour. The study was criticised (Gaa'l, 1976) on the basis of its relatively small sample size and the fact that the Aboriginal group was a heterogeneous (mixed) one. This could conceal important differences in the rate of metabolism amongst different Aboriginal peoples.

Studies among American Indians who also have a high prevalence of alcohol problems, show no differences in the rate of elimination between them and Americans of European descent.

3. Individual and racial variations in alcohol metabolising enzymes

3.1 Alcohol dehydrogenase

Alcohol dehydrogenase and aldehyde dehydrogenase are complex enzymes which consist of a number of isoenzymes. There are five isoenzymes of alcohol dehydrogenase, termed alpha, beta, gamma, pi and chi (Bosron & Li, 1986). The corresponding components of the genome are termed ADH1, ADH2, ADH3, ADH4 and ADH5. The isoenzymes which are principally responsible for the metabolism of alcohol are those derived from alpha, beta and gamma subunits. They have a high affinity for alcohol. The pi isoenzyme probably contributes at high BACs while the chi isoenzymes play a minimal role.

The beta, gamma and chi isoenzymes exist in several forms and this variation has been suggested for many years to be the basis of individual and racial variations in the effects of alcohol. To date, however, there is no convincing evidence that this is the case, even though the biochemical characteristics of the variants differ considerably. A so-called 'atypical' form of alcohol dehydrogenase, which contains beta2 subunits was discovered by von Wartburg and colleagues (1965). The atypical form occurs in approximately 5-10% of Europeans and in upwards of 75% of Oriental races. It is more active in metabolising alcohol at physiological pH. However, the rate of elimination of alcohol is no greater in persons who have the atypical enzyme compared with those with the normal enzyme (Edwards & Price-Evans, 1967). In studies of alcohol metabolism in Oriental subjects Goedde and colleagues (1979) found that the presence of the atypical enzyme did not correlate with flushing or any other idiosyncratic effect of alcohol. No significant associations of either of the two gamma sub-unit variants with adverse responses to alcohol have been reported.

3.2 Aldehyde dehydrogenase

Aldehyde dehydrogenase also consists of several isoenzymes with different chemical and physiological characteristics (Bosron & Li, 1986; Goedde & Agarwal, 1987). The two

principal isoenzymes are ALDHI (sometimes termed E2) and ALDHII (sometimes called E1). ALDHI is found predominantly in the mitochondria (sub-cellular organelles). It has a very high affinity for acetaldehyde and is responsible for the bulk of acetaldehyde metabolism in humans. ALDHII is found in the soluble component of cells, the cytosol. Its affinity for acetaldehyde is lower and it becomes more active when steady state acetaldehyde levels rise as a consequence of alcohol metabolism.

3.3 ALDH deficiency and the alcohol flush reaction

The most significant discovery in the area of alcohol biochemistry in the past decade has been that a deficiency of ALDHI is the basis for the flushing reaction that occurs within a few minutes of ingestion of alcohol in Oriental (Mongoloid) races (Goedde & Agarwal, 1987; Goedde, Harada & Agarwal, 1979). The flushing affects the face and upper trunk and is sometimes accompanied by nausea, palpitations and dizziness. The reaction is essentially the same as the disulfiram-alcohol reaction (Antabuse reaction), though less severe. The enzyme deficiency is found in 25-60% of Chinese, Japanese, Koreans and neighbouring peoples in South East Asia. It is also found in Mongoloid races elsewhere, for example in South American Indians and in a small proportion of the members of North American Indian tribes (e.g., the Sioux). The enzyme deficiency has not been found in Caucasian races - either in white people of European descent or peoples in the Indian subcontinent. Nor has it been found in Negroid races, either American blacks or in African peoples. It is found in a small proportion of people of mixed Oriental-Caucasian race.

3.4 Influence of the flushing reaction on drinking behaviour

The flush reaction has a significant effect on drinking behaviour. It is aversive for most people who experience it, and the alcohol consumption of 'flushers' is generally low. Such persons have to drink alcohol carefully and in small quantities in order not to experience the more unpleasant of the side effects. Only 2% of Japanese alcoholics admitted to a treatment unit had the ALDHI deficiency, in comparison with 41% of a control group of Japanese who had no alcohol problem (Goedde and Agarwal, 1987).

3.5 Studies on alcohol biochemistry in Aboriginal people

Little is known about alcohol dehydrogenase and aldehyde dehydrogenase composition in Aboriginal people. No studies have been published. As part of a larger study on enzyme polymorphisms, Dr Graham Jones (then of the Australian National University) examined ADH and ALDH isoenzymes in a small number of liver specimens. No deficiency of ALDHI was apparent and the isoenzyme composition of both enzymes was broadly comparable to that of white Caucasians (Jones, personal communication, 1988). Examination of genomes coding for ADH and ALDH isoenzymes is currently being undertaken by Dr Sriprakash in Darwin.

If a deficiency of ALDHI was discovered in a larger scale study, its relevance to predisposition to drinking problems or depression is not immediately apparent. Because of the aversive nature of the flush reaction, it is unlikely that persons with the enzyme deficiency would consistently achieve BACs in excess of 0.2 g%, because of the constraining effects of the flushing reaction on continuation of drinking. There is no reported association of depression or suicidal ideation with either the flushing reaction or the enzyme deficiency. Clearly, if persons with the enzyme deficiency consumed large quantities of alcohol, they would have very high blood levels of acetaldehyde which would cause a severe flushing reaction in those individuals; this might be accompanied by cardiac arrhythmias and possibly sudden death. However, an association with hanging seems unlikely.

References

Bosron, W. F., & Li, T.K. (1986). Genetic polymorphism of human liver alcohol and aldehyde dehydrogenase, and their relationship to alcohol metabolism and alcoholism. Hepatology, 6, 502-510.

Edwards, J.A., & Price-Evans, D.A. (1967). Ethanol metabolism in subjects possessing typical and atypical liver alcohol dehydrogenase. Clinical Pharmacology and Therapeutics, 8, 824-829.

Elmslie, R. G., Davis,R. A., Magee, D. F., & White, T. T. (1964). Absorption of alcohol after gastrectomy. Surgery, Gynaecology and Obstetrics, 119, 1256-1258.

Gaa'l, C. L. (1976). Alcoholism in Aborigines. Medical Journal of Australia, 1, 848.

Goedde, H. W., & Agarwal, D. P. (1987). Polymorphism of aldehyde dehydrogenase and alcohol sensitivity. Enzyme, 37, 29-44.

Goedde, H. W., Harada, S., & Agarwal, D. P. (1979). Racial differences in alcohol sensitivity: A new hypothesis. Human Genetics, 51, 331-334.

Haggard, H. W., Greenberg, L. A., & Cohen, L. H. (1938). Quantitative differences in the effects of alcoholic beverages. New England Journal of Medicine, 219, 466.

Harger, R. N., Hulpieu, H. R., & Lamb, E. B. (1937). The speed with which various parts of the body reach equilibrium in the storage of alcohol. Journal of Biological Chemistry, 120, 689.

Kalant, H. (1971). Absorption, diffusion, distribution and elimination of ethanol: Effects on biological membranes. In B. Kissin & H. Begleiter (Eds.), The biology of alcoholism, Vol. 1: Biochemistry (pp. 1-62). New York: Plenum Press.

Kater, R. M. H., Carulli, N., & Iber, F. L. (1969). Differences in the rate of ethanol metabolism in recently drinking alcoholic and non-drinking subjects. American Journal of Clinical Nutrition, 22, 1608-1617.

Larsen, J. A. (1959). Extrahepatic metabolism of ethanol in man. Nature, 184, 1236.

Lieber, C. S. (1982). Medical disorders of alcoholism. Philadelphia: W.B. Saunders.

Lieberman, F. L. (1963). The effect of liver disease on the rate of ethanol metabolism in man. Gastroenterology, 44, 261-266.

Marinovich, N., Larrson, O., & Barber, K. (1976). Comparative metabolism rates of ethanol in adults of Aboriginal and European descent. Medical Journal of Australia, 1 (Suppl. 3), 44-46.

Marshall, A. W., Kingstone, D., Boss, M., & Morgan, M. Y. (1983). Ethanol elimination in males and females: Relationship to menstrual cycle and body composition. Hepatology, 3, 701-706.

Mellanby, E. (1919). Alcohol: Its absorption and disappearance from the blood under different conditions (Medical Research Committee Special Report Series No. 31). London: HMSO.

Saunders, J. B. (1986). Studies in alcoholic liver disease: Its natural history, dtection and pathogenesis. MD Thesis, University of Cambridge, England.

Saunders, J. B. (1988). Medical aspects of management: Basic pharmacology of alcohol and principles of assessment. In J. C. Clarke & J. B. Saunders, Alcoholism and problem drinking: Theories and treatment (pp. 98-123). Sydney and Oxford: Pergamon Press.

Saunders, J. B., Davis, M., & Williams, R. (1981). Do women develop alcoholic liver disease more readily than men? British Medical Journal, 282, 1140-1143.

Vesell, E. S., Page, J. G., & Passananti, G. T. (1971). Genetic and environmental factors affecting ethanol metabolism in man. Clinical Pharmacology and Therapeutics, 12, 192-201.

von Wartburg, J. B., Papenberg, J., & Aebi, H. (1965). An atypical human alcohol dehydrogenase. Canadian Journal of Biochemistry, 43, 889-898.

Welling, P. G., Lyons, L. L., Elliot, R., & Amidon, G. L. (1977). Pharmacokinetics of alcohol following single low doses to fasted and non-fasted subjects. Journal of Clinical Pharmacology, 17, 199-206.

Chapter 3. The Analysis of Alcohol in Blood Specimens

Allan Hodda

1. Introduction

The chemical substance in beverages causing intoxication is called alcohol. This substance is present in fermented beverages, and is derived from microbial action on sugars. The definitive chemical name for this substance is ethanol or ethyl alcohol. The name 'alcohol' is a generic chemical term, and strictly applies to a large number of substances including methanol, ethanol and propanol to name only a few. The term 'alcohol' in this presentation will refer to ethanol unless explicitly qualified otherwise.

The analysis of alcohol in blood and other biological specimens has been carried out for more than a century by chemists using a variety of means to separate the alcohol and then to quantitate it. The analytical techniques have varied considerably during this period and the refinements are too numerous to mention. Suitable reviews of these methods (Harger, 1961; Jain & Cravey, 1972; Dubowski, 1986) may be consulted. Some small laboratories and the widely used Breathalyser rely on the oxidation of alcohol by dichromate under strong acid conditions. This early chemical methodology will be discussed further. All Australian forensic laboratories now use gas chromatography as their principal method of analysis. This method is accepted almost universally as the most effective way to accurately analyse alcohol in blood. Despite its universal acceptance, gas chromatography does not guarantee accuracy unless it is performed by a skilled operator and basic analytical safeguards are followed.

Other methods of analysis are based mainly on alcohol dehydrogenase enzyme reactions and because of their current wide usage, will also be reviewed. The quality of the analysis of blood for alcohol is, of course, compromised if the sample of blood is not representative of the subject's brain blood supply at the time of interest. This aspect of blood alcohol analysis will require detailed exposition.

2. The blood sample

Alcohol is a colourless, odourless, volatile liquid. It is very water soluble and distributes itself throughout the body tissues rapidly and roughly in proportion to the water content of tissues. The mode of collection, preservation and storage of a sample of blood may lead to changes in the blood, per se, and to the volatile and reactive alcohol content of the blood. The likelihood of such effects is remote when recommended practices are followed.

2.1 Sampling

2.1.1 Site sterilization

In the living patient the site (anterior cubital vein) of sampling is usually sterilized prior to withdrawing blood. The use of swabs containing 70% ethanol or 70% isopropanol have been seen as likely sources of error in analysing these samples by a chemical method (since the alcohol and possible contaminants could not be differentiated). The use of gas chromatography allows individual alcohols to be identified and research has shown (Dubowski & Essary, 1983; Ryder & Glick, 1986) that properly conducted sampling does not present a problem. It has been demonstrated that extreme conditions (the disinfectant swab is held in place while the needle is drawn through it) will lead to contamination. Most blood alcohol sampling kits in use in Australia contain either a quaternary ammonium type or povidone iodine type disinfectant.

2.1.2 Sample site

While sampling sites are generally limited in the living patient, the dead body may be widely sampled for blood. The absorption of alcohol into the bloodstream begins in the stomach but alcohol is even more rapidly absorbed from the small intestine. The blood flow serving these areas is carried to the liver initially, then onto the right side of the heart, lungs, left side of the heart and then via the arterial system to other parts of the body including the brain. Obviously a sample taken from a blood source early in this circulatory system will contain higher levels of alcohol or drugs than will the peripheral sampling points. This is particularly the case during the early absorptive stages of alcohol ingestion.

Recent investigations (Jones & Pounder, 1987; Prouty & Anderson, 1987) of alcohol and drug disposition in post mortem blood have reported significant differences in drug levels throughout the body but alcohol levels were relatively constant providing no decomposition or gross trauma case was considered. The use of heart blood or chest cavity blood is often convenient but because of its proximity to the stomach, and possible high alcohol burden, these samples may be contaminated by diffusion or leakage due to injury. It is generally conceded (Budd, 1988) that chest, thorax or pleural blood may give a good estimate of the blood alcohol concentration (BAC), but a peripheral sample is desirable if a truly representative blood alcohol level is sought. The femoral vein is recommended for several reasons. It is sufficiently distant from any source of alcohol that might diffuse from the stomach or intestines. Microbes (see Sections 2.2.3 and 2.3.3), if present, would travel initially from sources such as the intestines via the arteries.

2.1.3 Containers and preservatives

Alcohol is a volatile substance and this volatility is increased with temperature. Losses from blood will occur if containers do not seal properly or if the air volume to blood volume is large in the container. Containers used in

Australia have varied considerably over the last ten years. Glass is preferred but no losses have been noted from properly sealed polyethylene or polycarbonate bottles.

The need for preservatives will be discussed in detail under the heading of alcohol losses and alcohol gains. Sodium fluoride or potassium bifluoride has been used and must be present at a level greater than 0.5% w/v and preferably 1% w/v when the blood sample is mixed in the bottle. Blood will normally clot if an anti-coagulant is not used. Most sample bottles contain an anti-coagulant, commonly potassium oxalate sufficient to produce a concentration of 0.25% w/v when the blood sample is mixed in the container. Lower quantities of these solid additives may result from accidental spillage, at the time of sampling, or poor quality control when blood collection bottles are prepared in the factory.

2.2 Loss of alcohol

While alcohol gain in samples of blood is a matter of some speculation, the loss of alcohol in stored preserved blood has been well documented (Brown, Neylan, Reynold, & Smalldon, 1973; Corry, 1978; Dick & Stone, 1987). The main reasons for loss are: (a) natural diffusion due to the volatility of alcohol and poor container design, (b) enzyme oxidation by blood enzymes and (c) microbial action. All forms of loss are hastened by increased temperature. Bloods stored under refrigerated conditions and preservatised will show negligible loss of alcohol if the container is properly sealed (Somogyi, Buris, & Nagy, 1986).

2.2.1 Containers

The loss of alcohol from blood samples has been demonstrated to be due to physical loss if the container seals are defective. This may be exacerbated by operators not tightening the seal closure. Usually the laboratory will be alerted to this problem on receipt of a sample - due to blood spillage.

2.2.2 Alcohol oxidation

The most common cause of alcohol loss in preserved and non preserved blood samples is the oxidation of alcohol to acetaldehyde. The process has been noted to be hastened if the air to blood ratio in a container is high; it is not dependent on the alcohol concentration and is hastened by increased temperature (Smalldon & Brown, 1973). The rate will vary with certain conditions but has been reported as causing losses of 0.29 mg% per day for samples stored at 22^{o}C, 43 mg% per day at 62^{o}C, and 0.02 mg% per day at 4^{o}C.

2.2.3 Microbiological action

Microbes have the ability to metabolise a wide range of chemical substances as their energy source. A number of different types of microbes are capable of metabolising alcohol although this effect is not noted until the microbes have reached high growth levels. Blood taken from a person is usually sterile, as is the blood from a fresh corpse and if preservatised and refrigerated the loss of alcohol due to microbial action is slight (Corry, 1978). Unpreserved blood,

particularly if unrefrigerated may suffer large alcohol losses from this source. A report of certain species of microbes capable of living in 1% sodium fluoride and metabolising alcohol has been published (Dick & Stone, 1987). These organisms may be inhibited by using sodium fluoride at 2% concentration.

2.3 Gain of alcohol

2.3.1 Contamination

There are several opportunities for the contamination of a blood sample to take place. The more obvious sources of extraneous alcohol have been discussed under 2.1.1 (Site sterilization) and 2.1.2 (Sample site). The tampering with blood specimens, once they have been sealed, is beyond the scope of this paper. The existence of two samples of blood (or even 3 required by the Motor Transport Act 1901, NSW) and a well defined chain of custody makes such criminal action unlikely. Most Coronial sampling for blood alcohol requires only one sample of sealed, preservatised blood to be taken. A further sample, of unpreservatised blood, may be taken for poison analysis and could be used as a reference sample in doubtful cases. The remaining site of contamination is the laboratory. While alcohol standards may be kept in the same laboratory where a specimen is analysed, the transfer of alcohol vapour from the standards to an opened bottle of blood in the laboratory is not credible scientifically.

2.3.2 Biochemical formation

The formation of alcohol and other volatile substances is known to occur naturally in the body (Jones, 1985b). The formation is due to normal metabolic processes in the body and, in the case of metabolic disorders, the levels may be measurable. The level of acetone produced by a diabetic is a matter for concern if measuring low levels of alcohol using an infra-red spectroscopic breath analyser. For a laboratory analysing for alcohol by gas chromatography it poses no problems. The endogenous occurrence of alcohol has been reported but at levels below normal blood alcohol detection limits.

2.3.3 Microbiological formation

This topic has been well researched and apart from decomposing specimens the production of alcohol in preservatised, refrigerated samples does not seem to have been reported (Dubowski & Essary, 1983; Corry, 1978; Dick & Stone, 1987). There is no doubt that various microbes have the ability to produce alcohol in blood specimens, principally by acting on the glucose that may be present. Glucose levels may be quite high - especially if the deceased was subject to shock prior to death. This state will cause the glycogen store in the liver to be mobilised and blood glucose levels to rise. The production of alcohol is not usually observed until bacteria levels are very high, exceeding 10^6 colony forming units (cfu) per ml. The presence of sodium fluoride at levels of 1% will prevent bacterial growth but will not necessarily kill the bacteria.

2.3.4 Intragastrointestinal alcohol fermentation
This unusual condition is recorded for the sake of completeness. It refers to a physiological condition in which certain microbes may ferment carbohydrate material in the bowel and thus produce alcohol. The levels produced can be sufficient to cause intoxication. The syndrome seems to have only been noted in Japan and is most likely to occur in people who have undergone gastrointestinal surgery (Keji et al., 1984).

3. Analytical procedures

3.1 Chemical analysis

The analysis of alcohol by chemical means is no longer used regularly in most major laboratories. The analysis is slow, messy and open to more errors than the currently popular gas chromatographic methods. The procedure was widely used in the 1950s and 1960s and involved the distillation of alcohol from a measured volume of blood. The alcohol solution was reacted with acidified potassium dichromate and then the excess dichromate titrated to establish the amount of ethanol present. A variation of the procedure, popular in Australia, was called the Kozelka and Hine Method and recoveries were claimed to be 96-98% with a standard error of +/- 8% (Harger, 1961).

The chemical change in the alcohol is called oxidation and the procedure is identical to that used in the Breathalyser. In this instrument a measured amount of breath containing a representative amount of alcohol is bubbled through an acidified dichromate solution. The dichromate solution oxidizes the alcohol and is itself reduced. This change in the dichromate solution can be monitored spectroscopically by measuring the change in light energy absorbed by the solution. Visually the yellow potassium dichromate solution will change to a greenish colour.

Analytical methods using dichromate reactions were nominally subject to error in that other oxidizable, volatile substances could also react. These include other alcohols and acetone, but not petrol or hydrocarbons. The method of Kozelka and Hine includes a mercuric chloride trap to remove some compounds - such as acetone.

3.2 Enzymatic analysis

The body and many other organisms can metabolise alcohol with the assistance of an enzyme called alcohol dehydrogenase (ADH). This enzyme catalyses the oxidation of alcohol to a compound called acetaldehyde. This reaction, under controlled conditions, can be monitored spectroscopically to provide quantitative levels of alcohol in blood. Because the method relies on observing the change in the absorption of light (at a particular wavelength) the blood must be either clarified by deproteination or the alcohol distilled off the blood sample. The analytical technique was first researched in the 1950s but mainly enjoyed popularity during the 1960s

and 1970s. The procedure lacks specificity, since other alcohol compounds will react to a lesser extent, and the reagents and conditions of analysis must be carefully controlled. Excellent agreement between ADH and gas chromatographic analysis is possible (Poklis & MacKell, 1979). Despite lack of absolute specificity for ethanol it must be noted that other alcohols would not normally be expected in blood samples - especially at levels necessary to react. This technique will not respond to methanol or acetone and the oxidation of higher alcohols is at a reduced rate compared to ethanol. It is desirable that gas chromatography be also run on the specimens.

A more recent development of the ADH technique is referred to as radiative energy attenuation (REA). It offers excellent correlation with gas chromatography. Decomposed whole blood did cause interference with the earlier iodonitrotetrazolium violet (INT) dye (Caplan & Levine, 1986, 1987).

3.3 Gas chromatographic analysis

3.3.1 Theory

The main technique used for the analysis of alcohol in blood is gas liquid chromatography (GLC or gas chromatography). The procedure is universally accepted and well documented in the literature. Definitive works on the technique and related laboratory procedures for alcohol analysis are available (Dubowski, 1977; Stone, Muirhead, Norris, & Singers, 1980). The technique relies on the general principles of chromatography: a mixture of like compounds may be separated into individual components when passed through a polar inert matrix which preferentially inhibits the compound's passage (usually due to dissimilar polarities of the molecules). It should be noted that the analysis does, in fact, simply rely on the relative retention times of components and does not provide absolute identification. This point will be discussed further under quality assurance.

3.3.2 Direct injection GLC method

There are essentially two methods of applying the GLC to the analysis of alcohol. The direct injection approach is by far the simplest and has been used since the GLC was developed. The sample of blood is diluted with water and injected directly into the gas chromatograph. The advantage is that very little sample handling is required and instrumentation is relatively straightforward. The disadvantage is that the non-chromatographable blood substances will soon deposit at the beginning of the column (due to charring) and may affect the subsequent injections, eventually blocking the column. The latter problem has been overcome by incorporating a precolumn of micro glass beads which serve as a large surface condensation site and can be conveniently removed at suitable intervals, thus leaving the column packing material unaffected.

3.3.3 Head space GLC method

This application of GLC has been widely adopted - particularly in the 1970s and early 1980s. The method requires the blood sample to be placed in a suitable sample

container, which will contain a diluent and sufficient salt (sodium chloride) to saturate the liquid. The container is capped and must then be equilibrated in a constant temperature bath (usually part of the GLC) for at least 45 minutes. This process is to allow the ratio of alcohol in the air space above the sample and the sample solution to reach a constant ratio. The headspace in the container is then sampled and the sample introduced into the GLC for analysis, as previously detailed. The advantage of the technique is that there is very little interference from substances less volatile (than alcohol) and the column is not degraded by the blood constituents. The disadvantages are that there is a need for a dedicated instrument and there are more factors to be controlled, over direct injection GLC (sample temperature, equilibration time, bottle seals and salt levels).

3.3.4 Internal standards

While this is relevant to quality assurance it is appropriate to discuss it now as most GLC methods do incorporate an internal standard. The internal standard is a substance which is added to a sample of the blood prior to analysis to allow for the automatic correction of any analytical or instrumental aberrations. The principle is that a substance, closely related to the substance being analysed, is quantitatively added to the sample and also to any standard solutions that will be run to calibrate the instrument. If a variation to the amount of diluted blood injected occurs from sample to sample, it will not affect the overall quantitation because the relative amounts of the unknown alcohol and the internal standard will vary in the same proportion. In direct injection GLC the most common internal standard is n-propanol and in head space GLC n-propanol, acetonitrile or t-butanol have been used.

4. Laboratory quality assurance

Most modern laboratories have well defined activities and practices that are designed to guarantee the results obtained are reliable. Analytical methods aim to be selective, precise and accurate. The individual means by which this is achieved are called the quality control measures. In Australia, laboratories would be expected to follow the relevant national standard (Standard Association of Australia, 1985).

4.1 Reference material

Most of the analytical procedures discussed do not provide absolute identification and need calibration data. This in turn means that the laboratory must have validated reference material. Ethanol does tend to absorb water vapour and consequently a 100% pure sample may, in time, become contaminated with water. To guard against this laboratories would only purchase small quantities and these must be well sealed after opening. Generally they will be disposed of if any doubt exists about their integrity. While a laboratory will purchase certified reference material it is still

necessary to check the integrity of these samples. This may be done by comparison with an established standard using one of the analytical techniques, by checking the standard spectroscopically (infra-red) or by using a primary type chemical analysis (e.g., dichromate oxidation). The latter procedure, of course, relies on each of the reagents also being validated. Potassium dichromate can be confidently validated. Reference material also refers to the internal standards which may be used in the methods and, since these substances are used comparatively, it is likely that errors would only occur from batch to batch and the reason would be immediately obvious to the laboratory staff.

4.2 Calibration of equipment

Part of any quality assurance program is the regular maintenance and calibration of laboratory equipment. In fact, Section 10 of the National Measurement Act, 1960 requires that "a measurement of a physical quantity" for "any legal purpose" be done with reference to an Australian primary standard or other appropriate reference standard. "Appropriate" refers to standards recognised by the Act. This means that a laboratory must be able to demonstrate that mass measurements are conducted with reference to one of the approved Australian standards of mass.

4.3 Method validation

A laboratory employing a method of analysis should establish the accuracy and precision of the method in the hands of that laboratory's personnel.

4.3.1 Accuracy
This term refers to the laboratory's ability to obtain the correct result within certain tolerance limits. This is done by analysing a range of known standards and assessing the variation of results from the known results.

4.3.2 Precision
This term refers to the laboratory's ability to obtain consistent results. This is normally assessed by performing repetitive analysis on a single sample and assessing the spread of results. It is quite conceivable that a laboratory is precise and yet inaccurate!

4.3.3 Specificity
This problem was mentioned under 3.1, 3.2 and might be more fully discussed at this point. All the methods of alcohol analysis have shortcomings as far as specificity for alcohol. The rationale for accepting this shortcoming is that the incidence of interfering substances may be quite predictable and the incidence of substances other than alcohol in blood at levels sufficient to mislead the analyst would create a noticeable medical condition. The analytical chemist has taken other steps to overcome specificity problems, such as the mercuric chloride trap in the Kozelka and Hine procedure or combining two different techniques to increase confidence in the identification. A highly recommended combination was ADH and gas chromatography. The appeal of gas

chromatography, with the fact it achieves a separation of most volatile compounds, has resulted in most laboratories opting for the analysis of blood samples on two different gas chromatographs using different conditions of separation (column phase or temperature).

4.4 Quality control

This is the part of the quality assurance program which actively checks on the results being produced on a particular sample. The blood samples should be analysed at least in duplicate, and the batch of samples should include several samples independently prepared either within the laboratory or externally. If the results from the controls do not meet the tolerances established for the method, all analysis must be repeated. Further control measures include the repeat analysis of a blood sample analysed by a different operator.

4.5 Proficiency testing

Many large laboratories are able to conduct their own proficiency testing by separately generating either 'known' or 'blind' samples for the blood analysis unit to report on. In Australia the State forensic laboratories have conducted annual blood alcohol analytical trials since the early 1970s (Russell, 1971). Approximately four to six samples of blood of various alcohol levels are sent to each participating laboratory at a declared time each year. The alcohol levels are only known to the laboratory generating the specimens and results must be returned within a stated period of time. While anonymity is maintained a statistical report is prepared on each trial so that laboratories can assess overall performance. These trials have been invaluable in maintaining a high standard of accuracy and precision amongst the State laboratories.

5. Breath analysis

Breath analysis is a non-invasive sampling technique which is widely used for forensic blood alcohol analysis. The analysis is controversial because of the lack of agreement with actual blood alcohol analysis (Knight, 1984). The sources of variation will be briefly discussed.

5.1 Blood: breath ratio

The fundamental of this form of analysis is that there is a constant alcohol ratio between blood in the lungs and the air in contact with the blood. Considerable variations have been found but most instruments have standardised on a ratio of 1:2100 (breath/blood alcohol). Reports continue to contradict this figure, but all support variations amongst the population and even recommend the higher ratio of 1:2300 as being closer to the mean (Jones, 1985a). Reports acknowledge the ratio as generally favouring the subject but suggest legislation should simply define breath alcohol limits (Dubowski, 1986).

5.2 Time of sampling

The sampling of a breath sample is greatly influenced by the phase of alcohol absorption of the subject. If the breath is sampled in the absorptive stage the variation between the result will be significantly higher than the result for a peripheral blood sample (Simpson, 1987b). The reason is due to the circulatory path of blood in the body - discussed in 2.1.2. Some studies also agree that sampling during the post absorptive phase (alcohol level has passed a maximum) can lead to over-estimation of blood alcohol (Simpson, 1987a). In the main, reports (Dubowski, 1986) agree that the breath alcohol result will be an underestimate of the true blood alcohol figure.

5.3 Volume of air sampled

The air in the upper reaches of the lung and upper respiratory tract is not closely in contact with the blood. The ratio quoted is only applicable to air derived from the 'deep lung' or alveoli. It is essential for accuracy that alveoli air be sampled by the instrument. Instruments are designed to discharge non-alveoli air prior to sampling the last portions of breath. Poor sampling techniques may lead to errors - once again the blood alcohol result will be underestimated.

5.4 Temperature effects

The equilibrium of a volatile substance in a liquid and its vapour phase are temperature dependent. A higher temperature will cause a higher level of the liquid, and any volatile constituents, in the air space above it. This also applies to blood and reports that breath control or breathing cool air can cause a lowering of alcohol in the breath (Jones, 1982; Fox & Hayward, 1987) suggest that greater accuracy may be achieved by also monitoring the subject's temperature and the temperature of the expired air.

5.5 Breath alcohol analysis instruments

There are several types of instruments available and all are currently calibrated to calculate blood alcohol using the 1:2100 ratio discussed earlier (5.1). Most modern instruments are using infra-red light absorption as the principle of operation. The more accurate instruments use several wavelengths of infra-red light to allow corrections for artifacts, particularly acetone. The other type of instrument uses a fuel cell which on contact with alcohol produces an electric current. It is relatively insensitive to acetone. The Breathalyser, which is still the legislated instrument of analysis in Australian states, uses the potassium dichromate reaction previously described (see Section 3.1).(Tasmania, Western Australia and South Australia have recently approved infra-red based instruments also.) The instrument contains two equivalent vials of acidified potassium dichromate and a portion of the expired air is

bubbled through one. The colour change between the two vials is measured by difference in light absorption and is equivalent to the alcohol in the breath sample.

6. Conclusion

The analysis of alcohol in blood is a relatively straightforward analytical procedure for a trained analytical chemist using modern instruments. There are opportunities for error but good laboratory practice will guarantee the accuracy of the results. Standards of analysis are high in State Government laboratories, however, there may be some variability in other smaller laboratories. The quality of the results can only be as good as the specimen submitted and poorly presented or poorly sampled specimens, especially at elevated temperatures, can lead to erroneous results. There have been no reports of increased alcohol levels, after sampling into preservatised sample containers, due to microbiological action. If analysis is delayed for weeks, even with fluoride preservative, then alcohol losses may be expected. This may be compounded by poorly sealed containers and, more remotely, by certain microbes. Drugs do not interfere with the analysis of samples.

Blood alcohol and breath analysis results are rarely coincident (Harding & Field, 1987). The use of a factor, in instruments, to calculate blood alcohol results from breath alcohol readings is recognised as a compromise. The factor used (1:2100) is regarded as a lower mean result of many studies and should generally underestimate the true blood alcohol figure. Apart from instrumental errors, breath analysis may be influenced by the alcohol absorptive stage of the subject, temperature of respired air and sampling techniques. The individual physiology of the person may also be a factor. In the hands of a trained operator these factors can be managed and results will be mainly in close agreement or less than the actual BAC.

References

Brown, G. A., Neylan, D., Reynold, N. J., & Smalldon, K. W. (1973). The stability of ethanol in stored blood. Part 1. Important variables and interpretation of results. Analytica Chimica Acta, 66, 271-283.

Budd, R. D. (1988). Validity of post mortem chest cavity blood ethanol determinations. Journal of Chromatography, 449, 337-340.

Caplan, Y. H., & Levine, B. (1986). The analysis of ethanol in serum, blood, and urine: A comparison of the TDx REA ethanol assay with gas chromatography. Journal of Analytical Toxicology, 10, 49-52.

Caplan, Y. H., & Levine, B. (1987). Evaluation of the Abbott TDx-radiative energy attenuation (REA) ethanol assay in a study of 1105 forensic whole blood specimens. Journal of Forensic Sciences, 32, 55-61.

Corry, J. E. L. (1978). Possible sources of ethanol anteand post mortem: Its relationship to the biochemistry and microbiology of decomposition. Journal of Applied Bacteriology, 44, 1-56.

Dick, G. L., & Stone, H. M. (1987). Alcohol loss arising from microbial contamination of drivers' blood specimens. Forensic Science International, 34, 17-27.

Dubowski, K. M. (1977). Manual for analysis of ethanol in biological liquids. (HS 802 208). Washington, DC: U.S. Department of Transportation (NHTSA).

Dubowski, K. (1986). Recent developments in alcohol analysis (review). Alcohol, Drugs and Driving, 2, 13-46.

Dubowski, K. M., & Essary, N. A. (1983). Contamination of blood specimens for alcohol analysis during collection. Abstracts and Reviews in Alcohol and Driving, 4, 3-8.

Fox, G. R., & Hayward, J. S. (1987). Effect of hypothermia on breath-alcohol analysis. Journal of Forensic Sciences, 32, 320-325.

Harding, P., & Field, P. H. (1987). Breathalyzer accuracy in actual law enforcement practice. Comparison of blood and breath-alcohol results in Wisconsin drivers. Journal of Forensic Sciences, 32, 1235-1240.

Harger, R. N. (1961). Ethyl alcohol. In C. P. Stewart & A. Stolman (Eds.), Toxicology: Mechanisms and analytical methods. Vol. II. (pp. 85-151). New York: Academic Press.

Jain, N. C., & Cravey, R. H. (1972). Analysis of alcohol. I. A review of chemical and infrared methods. Journal Chromatographic Science, 10, 257-262.

Jones, A. W. (1982). How breathing techniques can influence the results of breath-alcohol analysis. Medicine, Science, and the Law, 22, 275-280.

Jones, A. W. (1985a). Electrochemical measurement of breath-alcohol concentration: Precision and accuracy in relation to blood levels. Clinica Chimica Acta, 146, 175-183.

Jones, A. W. (1985b). Excretion of low-molecular weight volatile substance in human breath: Focus on endogenous ethanol. Journal of Analytical Toxicology, 9, 246-250.

Jones, G. R., & Pounder, D. J. (1987). Site dependence of drug concentrations in postmortem blood - A case study. Journal of Analytical Toxicology, 11, 191-197.

Keji, H., Asanuma, Y., Yahara, O., Shibue, H., Hisamura, M., Saito, N., Kawakimi, Y., & Murao, M. (1984). Intragastrointestinal alcohol fermentation syndrome: Report of two cases and review of the literature. Journal of Forensic Science Society, 24, 461-471.

Knight, B. (1984). Driving and alcohol: A case report of a biological impossibility. Medicine, Science, and the Law, 24, 271-272.

Poklis, A., & MacKell, M. (1979). Use of gas chromatography and alcohol dehydrogenase in routine procedure for the determination of ethanol in post mortem blood. Journal of Analytical Toxicology, 3, 183-186.

Prouty, R. W., & Anderson, W. H. (1987). A comparison of post mortem heart blood and femoral blood ethyl alcohol concentrations. Journal of Analytical Toxicology, 11, 191-197.

Russell, A. B. (1971). Post mortem blood alcohol survey in some Australian states. Medical Journal of Australia, 2, 948-951.

Ryder, K. W., & Glick, M. R. (1986). The effect of skin cleansing agents on ethanol results measured with the Dupont Automatic Clinical Analyzer. Journal of Forensic Sciences, 31, 574-579.

Simpson, G. (1987a). Accuracy and precision of breath-alcohol measurements for a random subject in the postabsorptive state. Clinical Chemistry, 33, 261-268.

Simpson, G. (1987b). Accuracy and precision of breath-alcohol measurements for subject in the absorptive state. Clinical Chemistry, 33, 753-756.

Smalldon, K. W., & Brown, G. A. (1973). the stability of ethanol in stored blood. Part II. The mechanism of ethanol oxidation. Analytica Chimica Acta, 66, 285-290.

Somogyi, G., Buris, L., & Nagy, F. (1986). Changes in blood alcohol concentration during storage. Blutalkohol, 23, 208-213.

Standard Association of Australia. (1985). Australian standard for good laboratory practice - chemical analysis. Melbourne. (AS2830).

Stone, H. M., Muirhead, J. M., Norris, R. J., & Singers, W. A. (1980). The accuracy, precision and comparison of methods of blood alcohol analysis by gas chromatography (Report CD. 2305). New Zealand: Department of Scientific and Industrial Research.

Chapter 4. The Effect of Alcohol on Cognitive and Psychomotor Functioning

Greg Chesher & Janet Greeley

1. The measurement of cognitive and psychomotor functioning

Virtually everything we do as humans involves some skill. When we talk about measuring psychomotor and cognitive performance we are interested in assessing different types of skills, particularly those employed in receiving and acting on information from the environment.

The basic components of skills are sensation, perception, memory and response. Sensation is the ability to detect objects and events in the environment (i.e., the world in which we live). Perception involves the receipt and recognition of sensory experience; it is the point at which meaning is attached to the sensory experience. Memory involves the storage and retrieval of information. The response component of skill is that segment of behaviour that is observed. From measurements of the response and its characteristics, we may make inferences about what has occurred in the other components of skilled performance. Questions such as: Is the response accurate? How long did it take to be emitted? Can it be modified by feedback? - are aspects of the response which can be used to assess other stages or processes involved in using skills.

No human task involves only one component function, but it is possible to design tasks which primarily emphasise one or another component of function (Fitts & Posner, 1967). The carrying out of any skilled performance involves an organised sequence of activities, some of which can be observed such as movement and speech, and others which are not seen but are presumed to be taking place in the person's mind, such as thinking and remembering. These internal or inferred activities are the cognitive components of skills performance. They are sometimes referred to as central information processing functions.

"Cognition refers to all processes by which sensory input is transformed, reduced, elaborated, stored, recovered and used....Such terms as sensation, perception, imagery, retention, recall, problem-solving and thinking, among many others, refer to hypothetical stages or aspects of cognition" (Neisser, 1967, pp. 4). The cognitive functions underlying behaviour are inferred from tests of performance. Performance is typically assessed as the individual's outcome on some task such as recalling a list of words or solving an arithmetic problem. Psychologists, behavioural pharmacologists, and others who study human behaviour, attempt to break down performance on a task into its component parts to gain a better understanding of how humans process information, and to find out what stages in the processing sequence are disrupted by external manipulations such as the administration of a drug.

In assessing research on the effects of alcohol on cognition, it must be recognized that tasks which are developed to assess cognitive and psychomotor functioning are based on theories of how the various stages and processes of these functions work. As research and knowledge grow, theories change. Therefore, care should be taken in interpreting performance on a given task as being a true reflection of an underlying theoretical process. The validity and reliability of the task as a measure of the process(es) of interest should be established before the task is employed as an assay for drug effects. Unfortunately, this information is not always readily available and is seldom addressed in research papers on drug effects on cognition (Bahrick, 1977).

Another factor which is important in the interpretation of the effects drugs have on behaviour is individual differences in initial non-drugged performance level and in response to drug treatment. For example, the same dose of alcohol may act as a stimulant in one person and a depressant in another. Such variability can lead to an experimental result of no effect if only the average performance of the group of drugged subjects is considered and individual differences are not taken into account. In some studies differences have been reported in the effect of alcohol on performance in individuals who showed poor versus good performance on a task before the drug was administered (Carpenter & Ross, 1965). In some instances these factors can be accounted for by statistical procedures and by manipulations of the experimental design. There are, however, many instances where such factors have not been taken into consideration.

In addition to the validity of the measurement procedures employed, other factors which are not considered to be directly involved in the processing of information can, however, have a significant influence on task performance. For example, motivation to perform well and paying attention to the task at hand both affect performance outcome. The operation of these factors in any test of performance should be taken into consideration where possible.

The majority of individuals tested in studies of the acute effects of alcohol on cognitive and psychomotor functioning are males between the ages of 21 and 35 years, who are in good physical health, and who are classified as light to moderate social drinkers. Thus, research results found in this population may have few implications for populations differing in age, sex, race, fitness, and level of regular alcohol consumption. For instance, the effects of aging on memory performance are thought to parallel those produced by alcohol intoxication (Craik, 1977). Therefore, it might be expected that an older person who is suffering from an age-related memory deficit may show greater impairment by alcohol than someone younger who may have a more intact memory system. Of particular concern, is the observation that the effects of alcohol are quite different in heavy versus light drinkers. This consideration will be emphasised in the sections on tolerance.

2. What kind of a drug is alcohol?

Although alcohol has been used by people for many centuries, we still do not have a full understanding of how it exerts its activity. Alcohol is a relatively simple molecule and unlike most other psychotropic drugs, it does not appear to act on a specific receptor site on the nerve cell. Its activity appears to be non-specific, affecting the fluidity and structure of the nerve cell membrane (and other cell membranes). The consequence of this is that alcohol is capable of exerting an effect on all nerve cells. It is a reasonable assumption to say that the effects of alcohol are potentially very widespread and can involve actions on all nerve cells and tissues of the body.

The absence of clear specificity of action of alcohol, as described above, is to be contrasted with the effects produced by most other drugs. We can take as an example the opioids (heroin, morphine). These drugs exert their activity on the opioid receptors and will therefore act only on those cells which bear these receptors. The opioid receptors are not found in all areas of the brain. Opioid drugs are therefore completely without effect on a great proportion of nerve cells. Despite this apparent lack of receptor specificity, alcohol does exhibit, like all drugs, a dose-dependency in its actions. Although alcohol can be shown to affect many, and very possibly all, physiological systems and behaviours, some systems are peculiarly sensitive to the effects of this drug.

Research over the last twenty or so years has not only provided experimental descriptions of the effects of alcohol, but has more clearly elucidated where in the body's physiological and psychological processes the predominant effects are generated. By examining research data in a somewhat systematic manner, these effects of alcohol will become clear.

3. A review of the literature

The following is a brief review of the research literature on the effects of alcohol on human skills performance and cognitive functioning. By far the greatest proportion of the research effort in the area of skills performance has been directed to the effects on skills related to driving a motor vehicle. Research of this nature was prompted by the epidemiological study of Borkenstein, Crowther, Shumate, Ziel, and Zylman (1964) in which a correlation between blood alcohol concentration (BAC) of the driver and the probability of being involved in a road crash was demonstrated. This and subsequent studies have described a logarithmic increase in the crash incidence at BACs in excess of 0.05 g%. Subsequent reanalysis of these data have indicated that there is an increase in crash incidence at any deviation from zero BAC (Allsop, 1966). It is of interest to note that the original study was made possible by the invention, by Borkentsein, of the "breathalyser" - an instrument to estimate the BAC by the analysis of a sample of expired air.

In the years following the publication of these early reports, research efforts intensified and reached a peak in the 1970s. Studies of the effects of alcohol on human performance skills have been undertaken using a number of techniques, both in the laboratory and on the road. Laboratory studies have chosen tests, which by careful variation of the task presented to the volunteer, have been able to describe, in broad terms, the way alcohol affects human psychomotor skills. This review will outline these findings.

Most of the studies referred to in this review were conducted in the 1960s and 1970s, after Borkenstein's study. Since that time research efforts into the investigation of the effects of alcohol on human psychomotor skills have significantly reduced as interest has turned to drugs other than alcohol.

Most of the reported studies of the effects of alcohol have employed a single acute dose of the drug. No doubt, for reasons of ease in the practical conduct of experiments, the majority of these studies have required the consumption of the required amount of alcohol over a short time period. This pattern of alcohol consumption does not represent the most common pattern of the social use of the drug, but it does maximise the impairment produced from the administered dose.

The duration of drinking is important in determining the effects of alcohol. If we were to conduct a study to examine the effects of a peak BAC of 0.1 g% on the same volunteer tested on two occasions, each with a different duration of drinking, we would see notable differences in the degree of intoxication. For example, if on one occasion the individual must reach this peak within a period of 30 to 40 minutes, and on another occasion within a period of 2 hours, the degree of drunkenness and impairment will be greater when alcohol is consumed at the more rapid rate. When more time has been taken to consume alcohol, although it will require more alcohol to achieve the same BAC, the degree of drunkenness and impairment is less. This is because of the development of acute tolerance (Moskowitz and Burns, 1971). It has been well established, since first described by Mellanby in 1919, that alcohol-induced impairment is greatest during the absorption phase when the BAC is rising, than on the metabolic phase when the BAC is falling. This decline in effect on the falling phase of the BAC curve is described as acute tolerance (see Chapter 5 for further details).

Another factor which will affect the degree of drunkenness of an individual after consuming a specified dose of alcohol is the previous drinking history of that individual. Previous drinking history is related to the degree of tolerance (chronic tolerance). It is important also to realize that alcohol will exhibit cross tolerance to other depressants of the central nervous system, such as other depressant drugs including the benzodiazepines and barbiturates, as well as the volatile solvents (including petrol). This means that

someone who has become tolerant to one of these substances by repeated usage, will also have developed some tolerance to alcohol, although they may never have used alcohol. As will be discussed in Chapter 5, the extent of tolerance to alcohol in some individuals, who are very heavy drinkers, can be quite remarkable.

3.1 Effects on human physical performance

The consumption of alcohol, even at a BAC of less than 0.1 g%, can lead to a deterioration of physical working capacity. Dynamic strength, as measured for example by the ability to jump vertically, is decreased. Similarly, the time taken to sprint over an 80 metre course is significantly increased (Hebbelinck, 1963). The ability to control posture or steadiness on standing is also affected, even by quite low concentrations of alcohol. Thus, the classical picture of the drunken individual is one who stands unsteadily and walks with an even more erratic gait.

3.2 Effects on the transducers - visual and auditory functions

Before information can be processed, we must receive it from the environment. This we do by means of specific transducers to sense visual, auditory, olfactory and tactile stimuli. The greatest research attention has been directed to the effects of alcohol on visual and auditory functions.

Early studies reported contradictory findings regarding the effect of alcohol on visual processes (Carpenter, 1962). The concept of "tunnel vision" as described by Drew (1963) suggested that alcohol induced an impairment in the ability to see objects in peripheral vision. However, later studies have indicated that under alcohol, the disturbance of visual functions is very slight. Wallgren & Barry (1970) concluded that measures of vision were "relatively insensitive to alcohol".

However, if the same measures of vision are examined under conditions in which the subject is required to perform another task and is presented with some information processing requirements, alcohol can be shown to adversely affect performance (Buikhuisen & Jongman, 1972; Hamilton & Copeman, 1970; Huntley, 1970; Moskowitz & Sharma 1974).

To investigate the hypothesis that visual functions are affected by alcohol only when central information processing demands are placed on the subject, Moskowitz and Sharma (1974) asked volunteers to detect signals in peripheral vision under two conditions: when observing a central unblinking light, and when the light was blinking and subjects were required to count the blinks. Only in the latter condition did alcohol, at a BAC of less than 0.05 g%, produce significant deficits in peripheral vision.

When the brain is occupied in performing more than one task and a division of attention is required, one or more of these tasks will suffer impairment. Thus, the effect of alcohol is

not on visual processes per se, but rather on central processing. The earlier reports of alcohol producing "tunnel vision" were really manifestations of this central processing impairment.

Similar effects of alcohol can be demonstrated on auditory functions. When examined singly, alcohol appears to have no effect on auditory tasks. However, if the same task is combined with another and a division of attention is required, alcohol produces significant deficits in performance on the auditory task (Moskowitz & DePry, 1968).

When two activities in a time-shared, divided attention task are combined, there is an overall performance decrement on the combined task under alcohol. Since neither of the two elements of the combined task are particularly sensitive to alcohol when performed alone, the question arises as to how the errors on the combined task will be distributed. In the opinion of Zeidman, Moskowitz, and Nieman (1980) the attitude of the subject really determines which of the tasks will receive the higher priority. In driving, one would expect that the constant demand for attention by the tracking task would overshadow the intermittent demands of the peripheral search and recognition task, so that the latter would more likely demonstrate an impairment.

As an illustration of the last point, a study by Billings, Wick, Gerke, and Chase (1972) which examined the effects of alcohol on flying an aircraft under the influence of alcohol, provides pertinent information. In this study, 16 subjects took off and instrument flew and landed a plane under 4 different doses of alcohol resulting in BACs of 0.0 (placebo) ; 0.04; 0.08 and 0.12 g%. Eight of the pilots were professional and highly experienced and 8 were inexperienced and non-professional. The professional pilots exhibited no impairment of their tracking abilities even at the highest dose. On the other hand, even at the lowest BAC, these pilots committed serious procedural errors which were hazardous to safety. At the BAC 0.12 g% the safety co-pilot had to assume control of the aircraft on 11 occasions in order to prevent an accident. The less experienced pilots exhibited errors in tracking as well as very serious procedural errors. Some of these included loss of control in flight, turns towards on-coming traffic, and landing errors that would have involved striking the ground had the co-pilot not intervened.

The authors commented,

> If we assume that instrument-rated pilots, flying ILS approaches, consider the job of guiding their aircraft to a position from which a visual landing can safely be made as their primary task, then it follows that the other, discrete procedures involved, while no less essential to safe operations, are relegated to a secondary role. The evidence is clear this is in fact the hierarchy which exists. It is equally clear that as pilots are progressively affected by alcohol, they become

> progressively less able to cope with the various facets of their task, and it is the secondary tasks which suffer first and most. (p.)

Alcohol also produces impairment of oculomotor control. At doses as low as 0.05 g% alcohol impairs saccade maximum velocity and response time and the velocity of smooth pursuits (Baloh et al., 1979). Saccadic eye movements are rapid and are made to follow rapidly moving targets; smooth pursuit movements of the eye are slow and continuous and are made to follow slow moving targets. The basic premise of vision is the movement of the eye to the target and a subsequent fixation or dwelling on the target to bring it into the central area of the retina for the best resolution of the image. Without dwelling on the target we cannot "see" and therefore have no visual information to process. By studying eye movements of volunteers before and after alcohol a measurement can be made of the duration of eye fixation and other aspects of eye movements. There are now several studies which have clearly demonstrated that alcohol increases fixation duration (Belt,1969; Beidman & Stern 1976; Moskowitz, Zeidman & Sharma, 1976; Stapleton, Guthrie & Linnoila, 1986; Stern, 1976). In addition to this it has been found that under the influence of alcohol, there is a decrease in the frequency of saccades, an increase in the proportion of long duration fixations, a decrease in large amplitude saccades and a decrease in the peak velocity of saccades (Beidman & Stern, 1976). All of this indicates that over a given time period, less information is being received and therefore being processed. In addition, the visual field is reduced, generally to concentrate on the central field (straight ahead) (Buikhuisen & Jongman, 1972; Schroeder et al., 1974). These data have been interpreted to indicate that the increased fixation time reflects the increased time required to process the data collected during each fixation. The restriction of the field of vision to a central field could be an attempt, under reduced processing abilities, to concentrate attentional resources to where the greatest task demands are perceived to exist.

3.3 Effects on neuromuscular control

One of the very early recognised effects of alcohol is its effect on neuromuscular control. The inability to muster the necessary muscular control to pronounce clearly "British Constitution" has been for many years a widely recognised indicator of some degree of inebriation. Similarly, the Romberg test, which tests the ability to stand perfectly still, especially with eyes closed, reveals the adverse effect of alcohol on neuromuscular co-ordination. Standing steadiness is one of the most sensitive laboratory measures available for alcohol impairment studies (Chesher et al 1984; Linnoila, Dubyoiski et al., 1985). The manner in which alcohol affects standing steadiness can be inferred from findings which indicate that the effect with eyes closed is generally more pronounced than if the subject has eyes open and is fixing attention on a stable point. This suggests that some of the effect is central in origin and depends upon processing of sensory visual information. If tactile input

is significantly attenuated by providing vibration to the feet via the platform on which the volunteer is standing, a similar effect is observed. The attenuation of both visual and tactile sensory input by the methods described increases the sensitivity of the task to the effects of alcohol (Erwin, Linnoila, Hartwell, Erwin & Guthrie, 1986). In short, it seems that the effects of alcohol on standing steadiness are predominantly central in origin, and not an effect on muscular strength.

3.4 Effects on tracking tasks

Another frequently employed laboratory task which involves neuromuscular control is that of tracking. The task of driving a motor vehicle is in simple terms a time-sharing of the tasks of tracking, visual search, and recognition. Tracking is the ability to maintain the vehicle in its appropriate place on the roadway.

Consistent with the findings described above for visual and auditory tasks, simple tracking tasks such as compensatory tracking, are not particularly sensitive to the effects of alcohol if the task is studied in isolation. Moskowitz (1973), Perrine (1973), and Wallgren and Barry (1970) all agreed that unless the subjects are required to perform the tracking task in combination with another task such as to force the division of attention between the two, alcohol does not induce significant impairment until the BAC is in excess of 0.1 g%. On the other hand, when subjects are required to perform a tracking task in the divided attention condition, the task is sensitive to quite low BACs (0.05 g% or less).

3.5 Reaction time

Reaction time is defined as the time expired between the presentation of a signal and the subject's response to that signal. The response is usually the depression of a button. As might be anticipated from the description of the effects of alcohol on other measures which have been described already, effects on reaction time are greater when the task involves increasing degrees of central processing (thinking time). The reaction time measure which requires the least degree of information processing is simple reaction time.

Even with simple reaction time, when the response required is the pressing of a button every time the stimulus is presented, results have varied between no significant effect, to a prolongation of response time after alcohol consumption (Carpenter, 1962; Sutton & Burns, 1971). The reality of the situation is that reaction time is not an accurate description of the tasks as presented, despite their apparent simplicity. Even with simple reaction time studies, factors such as attention and motivation have been recorded as having influenced results. Studies which have been conducted under conditions of high- and low-motivation (the motivation being monetary reward) have indicated differences in the effects of the same concentration of alcohol (Obitz, Rhodes & Creel, 1977). At a BAC of 0.09 g%, a significant prolongation of simple reaction time could be demonstrated under the

low-motivation condition. However, under the high-motivation condition, this dose of alcohol was without effect. These authors attributed the alcohol-induced increase in reaction time under the low-motivation condition to "inattention". The inattention could be overcome by the monetary reward affecting the subjects' motivational arousal level.

Interestingly, in another study (Sutton & Burns, 1971), alcohol was found to impair the reaction times of female volunteers but not that of the males. The authors attributed the difference to the more competitive attitude of the males in the study.

It has also been reported that alcohol increases reaction time in studies where subjects have been required to respond to stimuli whilst performing another task in parallel (Cozad & Zavala, 1973; Rafaelsen, Bech & Rafaelsen, 1973; Rafaelsen, Christup & Bech, 1973). In a study to measure the psychological refractory period, Moskowitz and Burns (1971) used a task which required the subject to respond to two simple reaction time signals, one auditory the other visual. The response was made to the appropriate button for each stimulus. The two stimuli were presented in rapid succession, and if the interstimulus interval was less than 300 milliseconds, the response to the second stimulus was longer than that to the first. The shorter the interstimulus interval, the greater the prolongation of the response to the second stimulus. This increase in reaction time is interpreted to indicate the time for central processing to complete the processing of the information for the first stimulus before processing information for the second. Alcohol further increased the slowing of the reaction time to the second stimulus. That this was due to an effect on central processing was indicated by the finding that the increase in reaction time to the first stimulus after alcohol was only slight.

3.6 Complex reaction time and speed-accuracy trade-off

Some reaction time tasks require a greater component of information processing than those required for the simple task described above. A complex reaction time task presents the subject with a series of stimuli, only one of which is the target to which a response is required. Reaction times in these tasks are longer in view of the central information processing required; the extent of the increase in reaction time is of course dependent upon the degree of difficulty of the processing requirement. In tasks such as these, another component which can confound the assessment of the effect of a drug on reaction time per se is the speed-accuracy trade-off. Some subjects display a willingness to trade off accuracy of response in order to maintain speed of response. This behaviour can therefore provide inaccurate information of the effect of alcohol on a reaction time measure unless care is taken to record accuracy of response as well as its speed (Jennings, Woods & Lawrence, 1976; Shillito, King & Cameron, 1974).

In summary, the confounding influences of motivation and speed-accuracy trade-off can explain much of the inconsistency of findings in the literature as to the effects of alcohol on reaction time measures. Nevertheless, it seems that alcohol does indeed produce a small prolongation of reaction time. This effect is made greater when the information processing demand of the task is made greater.

3.7 The effects of alcohol on learning and memory

It is generally accepted that the acute administration of alcohol can affect cognitive functioning (Lister, Eckardt & Weingartner, 1987). Reports in the research literature show that alcohol impairs: perception (Tyson & Gavard, 1976), learning and memory (Miller, Adesso, Fleming, Gino & Lauerman, 1978), attention (Moskowitz & DePry, 1968), judgement (Maisto, Connors, Ruff & Watson, 1981), and reasoning (Tartar, Jones, Simpson & Vega, 1971). This section will concentrate on aspects of cognitive impairment produced by alcohol which involve disruption of learning, memory and reasoning processes.

The cognitive impairment produced by alcohol is often subsumed under the general classification of the intoxicating effects of alcohol. One of the earliest sources on the relationship between the concentration of alcohol in body fluids and alcoholic intoxication is a monograph by Goldberg (1943). In Goldberg's classic studies he found that there was a linearly increasing relationship between BAC and the amount of impairment in performance shown on tests of motor coordination, sensory perception and intellectual function within a BAC range of approximately 0.04 to 0.12 g%. In other words, as the BAC increased performance became more impaired. He found the threshold for impairment for individuals classified as habitual light drinkers was between 0.05 and 0.09 g% in each test of performance.

There is an extensive research literature consisting of investigations of the effects of alcohol on specific cognitive processes such as the input (acquisition or encoding) and output (retrieval) of information in memory (for reviews see Birnbaum & Parker, 1977; Carpenter, 1962; Lister, Eckardt & Weingartner, 1987; Lister, Weingartner & Linnoila, 1985; Mello & Mendelson, 1978; Ryback, 1971). In general, the results from reviews of this research are inconclusive. Throughout the literature, findings are seldom replicated and, in most experiments, only a single dose of alcohol is used, thus preempting any dose-response determinations.

Nevertheless, some generalizations about the effect of alcohol on cognition can be made. Although the dosages of alcohol employed in these studies are relatively low (BACs range from 0.02 to 0.12 g%), impairment of cognitive functioning is frequently observed. In some instances, however, facilitation of memory and performance has also been found (e.g., Parker, Morihisa, Wyatt et al. 1981) after the consumption of low doses of alcohol. Alcohol is thought to have its greatest effects on the acquisition of new

information and, in particular, on those processes involved in the organisation of information in memory. An extensive literature on the subject of state-dependent learning (see next section) indicates that alcohol can also affect retrieval of information from memory. It is generally accepted that factors such as the nature of the task & Huntley, jr., 1972), the subject's pre-drug performance level (e.g., Lovibond & Bird, 1970), and practice on the task (e.g., Tartar, Jones, Simpson & Vega, 1971) can affect the degree of impairment observed under alcohol. Some of these assumptions have, however, been questioned by other researchers (Maylor & Rabbitt, 1987).

3.7.1 State-dependent learning

In some instances people are unable to retrieve information from memory if they are in a different (drug) state from that in which they learned the information. It has been observed that retrieval of the apparently lost information can be facilitated by reinducing the original drug state. This phenomenon is referred to as state-dependent learning or state-dependent retrieval (Lister et al., 1987).

Although there is a substantial amount of research literature which indicates that state-dependent retrieval exists, it is not a readily predictable phenomenon. Whether or not state-dependent effects are seen, tends to depend upon the type of task used to assess retrieval. If cues are provided during retrieval, such as when the subject is required to say whether he recognises an object as one that has been presented before, state-dependency is eroded (Eich, 1980). Failures to produce state-dependent learning and the occurrence of asymmetrical effects (that is, reports that information learned while sober was retrieved equally well while sober or intoxicated, and that information learned while intoxicated was retrieved better in the intoxicated state) present problems for attributing the state-dependency effect solely to deficits in retrieval processes.

These state-dependent effects may relate to the hedonic or mood state of the person at the time of encoding as well as the hedonic quality of the information being encoded. Research has shown that when people are happy they learn more about positive things and when they are sad they learn more about negative things (Bower, 1981). Alcohol has a biphasic effect on mood, tending to promote elation and positive feelings while the BAC is rising and to induce more negative feelings, such as anger and depression, during the falling phase of the BAC curve. Depending on the mood state the acquisition or retrieval of different types of material may be either facilitated or hindered.

3.8 Conclusions

Alcohol, at relatively low doses, can impair various aspects of cognitive and psychomotor functioning. Earlier reviews of the effects of alcohol on psychomotor performance (Moskowitz, 1973; Moskowitz & Austin,1979; Perrine, 1973; Wallgren & Barry, 1970) have indicated that the primary effects of alcohol are on central nervous system mechanisms involved in

perception and cognition rather than on the "transducers" of sensory input or on the motor effectors of physical performance - the general muscular system. The present review is in general agreement with these earlier reports. Although it has been found that alcohol impairs performance on tasks which assess visual and neuromuscular control, these results are largely due to alcohol's effects on the central information processing mechanisms that control movement rather than a direct effect on muscle action.

Tasks which require a division of attention and rapid responding are very sensitive to impairment by alcohol. An examination of some of the cognitive processes involved in performance indicates that alcohol can affect both the acquisition of new information and the retrieval of stored information. Alcohol appears to disrupt the organisation of information in memory.

References

Allsop, R. E. (1966). Alcohol and road accidents. A discussion of the Grand Rapids study (RRL Report No. 6). Harmondsworth, U.K.: Harmondsworth Road Research Laboratory.

Bahrick, H. P. (1977). Reliability of measurement in investigations of learning and memory. In I. M. Birnbaum & E. S. Parker (Eds.), Alcohol and human memory (pp. 59-71). Hillsdale, NJ: Lawrence Erlbaum Assoc..

Baloh, R., Sharma, S., Moskowitz, H., & Griffith, R. (1979). Effect of alcohol and marijuana on eye movements. Aviation, Space, and Environmental Medicine, 50, 18-23.

Beideman, L., & Stern, J. (1976). Visual search activity and motor performance under alcohol intoxication. In M. Horvath (Ed.), Adverse effects of environmental chemicals and psychotrophic drugs (Vol. 2, pp.). New York: Elsevier.

Belt, B. L. (1969). Driver eye movement as a function of low alcohol concentrations. Colombus, Ohio: Ohio State University, Driving Research Laboratory.

Billings, C. E., Wick, R. L., Gerke, R. J., & Chase, R. C. (1972). The effects of alcohol on pilot performance during instrument flight (Report FAA-AM-72-4). Washington, DC: Federal Aviation Administration.

Birnbaum, I. M., & Parker, E. S. (1977). Acute effects of alcohol on storage and retrieval. In I. M. Birnbaum & E. S. Parker (Eds.), Alcohol and human memory (pp.). Hillsdale, New Jersey: Lawrence Erlbaum.

Borkenstein, J. F., Crowther, R. F., Shumate, R. P., Ziel, W. B., & Zylman, R. (1964). The role of drinking driver in traffic accidents. Indiana University: Department of Police Administration.

Bower, G. H. (1981). Mood and memory. American Psychologist, 36, 129-148.

Buikhuisen, W., & Jongman, R. W. (1972). Traffic perception under the influence of alcohol. Quarterly Journal of Studies on Alcohol, 33, 800-806.

Carpenter, J. A. (1962). Effects of alcohol on some psychological processes. Quarterly Journal of Studies on Alcohol, 23, 274-314.

Carpenter, J. A., & Ross, B. M. (1965). Effect of alcohol on short-term memory. Quarterly Journal of Studies on Alcohol, 26, 561-579.

Chesher, G.B., Bird, K., Crawford, J., Dauncey, H., Nikias, N., & Stramarcos, A. (1984). Further studies in the psychopharmacology of cannabis and alcohol: Acute and chronic effects. New South Wales Drug and Alcohol Authority Research Grant Report Series B84/5.

Craik, F. I. M. (1977). Similarities between the effects of aging and alcoholic intoxication on memory performance, construed within a "levels of processing" framework. In I. M. Birnbaum & E. S. Parker (Eds.), Alcohol and human memory (pp. 9-21). Hillsdale, NJ: Lawrence Erlbaum Assoc Tates.

Drew, G. C. (1963). The study of accidents. Bulletin of the British Psychological Society, 16, 1-10.

Eich, J. E. (1980). The cue-dependent nature of state-dependent retrieval. Memory and Cognition, 8, 157-173.

Erwin, W., Linnoila, M., Hartwell, J., Erwin, A., & Guthrie, S. (1986). Effects of buspirone and diazepam, alone and in combination with alcohol, on skilled performance and evoked potentials. Journal of Clinical Psychopharmacology, 6, 199-209.

Fitts, P. M., & Posner, M. I. (1967). Human performance. Belmont, CA: Brooks/Cole Publishing.

Goldberg, L. (1943). Quantitative studies on alcohol in man: The influence of ethyl alcohol on sensory, motor, and psychological functions referred to blood alcohol in normal and habituated individuals. Acta Physiologica Scandinavica, 5 (Suppl. 16), 1-128.

Hamilton, P., & Copeman, A. (1970). The effect of alcohol and noise on components of a tracking and monitoring task. British Journal of Psychology, 61, 149-156.

Hebbelinck, M. (1963). The effects of a small dose of ethyl alcohol on certain basic components of human physical performance. II. The effect on neuromuscular performance. Archives Internationales de Pharmacodynamie, 143, 247-259.

Huntley, M. S. (1970). Effects of alcohol and fixation-task demands upon human reaction time to achromatic targets in the horizontal meridian of the visual field. Doctoral dissertation, University of Vermont. (University Microfilms No. 3026-B, 31)

Huntley, M. S., Jr. (1972). Influences of alcohol and S-R uncertainty upon spatial localization time. Psychopharmacologia (Berl.), 27, 131-140.

Jennings, J. R., Woods, C. C., & Lawrence, B. E. (1976). Effects of graded doses of alcohol on speed-accuracy tradeoff in choice reaction time. Perception and Psychophysics, 19, 85-91.

Lister, R. G., Eckardt, M. J., & Weingartner, H. (1987). Ethanol intoxication and memory: Recent developments and new directions. Recent Developments in Alcoholism, 5, 111-126.

Lister, R. G., Weingartner, H., & Linnoila, M. (1985). New drugs for reverting ethanol-induced memory impairments. In C. A. Naranjo & E. M. Sellers (Eds.), Research advances in new psychopharmacological teatments for alcoholism (pp. 237-256). Amsterdam: Elsevier.

Lovibond, S. H., & Bird, K. (1970). Effects of blood alcohol level on the driving behavior of competition and non-competition drivers. Paper presented at the 29th International Congress on Alcoholism and Drug Dependence, Sydney, Australia.

Maisto, S. A., Connors, G. J., Ruff, C., & Watson, D. (1981). Alcohol and volitional control on affect/sensation, cognitive and perceptual-motor measures. Current Psychological Research, 1, 235-250.

Maylor, E. A., & Rabbitt, P. M. A. (1987). Effects of practice and alcohol on performance of a perceptual motor task. Quarterly Journal of Experimental Psychology, 39A, 777-795.

Mello, N. K., & Mendelson, J. H. (1978). Alcohol and human behavior. In L. L. Iversen, S. D. Iversen & S. H. Snyder (Eds.), Handbook of psychopharmacology: Vol. 12. Drugs of abuse (pp.235-317). New York: Plenum Press.

Miller, M. E., Adesso, V. J., Fleming, J. P., Gino, A., & Lauerman, R. (1978). Effects of alcohol on the storage and retrieval processes of heavy social drinkers. Journal of Experimental Psychology: Human Learning and Memory, 4, 246-255. @@

Moskowitz, H. (1973). Laboratory studies of the effects of alcohol on some variables related to driving. Journal of Safety Research, 5, 185-199.

Moskowitz, H., & Austin, G. (1979). A review of selected research studies from the last decade on the effects of alcohol on human skills performance. Fort Detrick, MD: U.S. Army Medical Research and Development Command.

Moskowitz, H., & Burns, M. (1971). Effects of alcohol on response on the psychological refractory period. Quarterly Journal of Studies on Alcohol, 32, 782-790.

Moskowitz, H., & DePry, D. (1968). Differential effect of alcohol on auditory vigilance and divided-attention tasks. Quarterly Journal of Studies on Alcohol, 29, 54-63.

Moskowitz, H., & Sharma, S. (1974). Effects of alcohol on peripheral vision as a function of attention. Human Factors, 16, 174-180.

Moskowitz, H., Ziedman, K., & Sharma, S. (1976). Visual search behaviour while viewing driving scenes under the influence of alcohol and marihuana. Human Factors, 8, 417-432.

Neisser, U. (1967). Cognitive psychology. New York: Appleton-Century-Crofts.

Obitz, F. W., Rhodes, L. E., & Creel, D. (1977). Effect of alcohol and monetary reward on visually evoked potentials and reaction time. Quarterly Journal of Studies on Alcohol, 38, 2057-2064.

Parker, E. S., Morihisa, J. M., Wyatt, R. J., Schwartz, B. L., Weingartner, H., & Stillman, R. C. (1981). The alcohol facilitation effect on memory: A dose-response study. Psychopharmacology, 74, 88-92.

Perrine, M. W. (1973). Alcohol influences on driving-related behaviours: A critical review of laboratory studies of neurophysiological, neuromuscular and sensory activity. Journal of Safety Research, 5, 165-184.

Rafaelsen, O. J., Bech, P., & Rafaelsen, L. (1973). Simulated car driving influenced by cannabis and alcohol. Pharmakopsychiatrie Neuro-Psychopharmakologie, 6, 71-83.

Rafaelsen, L., Christup, H., & Bech, P. (1973). Effects of cannabis and alcohol on psychological tests. Nature, 242, 117-18.

Ryback, R. S. (1971). The continuum and specificity of the effects of alcohol on memory. Quarterly Journal of Studies on Alcohol, 32, 995-1016.

Schroeder, S., Ewing, J., & Allen, J. (1974). Combined effects of alcohol with methapyrilene and chlordiazepoxide on driver eye movements and errors. Journal of Safety Research, 6, 89-93.

Shillito, M., King, L., & Cameron, C. (1974). Effects of alcohol on choice reaction time. Quarterly Journal of Studies on Alcohol, 35, 1023-1034.

Sutton, D., & Burns, J. (1971). Alcohol dose effects on feedback maintained simple reaction time. Journal of Psychology, 78, 151-159.

Tartar, R. E., Jones, B., Simpson, C. D., & Vega, A. (1971). Effects of task complexity and practice on performance during acute alcohol intoxication. Perceptual and Motor Skills, 33, 307-318.

Tyson, P. D., & Gavard, M. (1976). Perceptual compensation for the effects of alcohol. Journal of Studies on Alcohol, 37, 1593-1599.

Wallgren, H., & Barry, H. (1970). Actions of alcohol (Vol. 1). New York: Elsevier.

Zeidman, K., Moskowitz, H., & Nieman, R. A. (1980). The effects of alcohol on the driver's visual information processing (Contract No. DOT HS-5-01233). Washington, DC: U. S. Department of Transportation, National Highway Traffic Safety Administration.

Chapter 5. Tolerance to the Effects of Alcohol

Greg Chesher, Janet Greeley & John Saunders

1. Introduction

It is often observed that over repeated bouts of alcohol consumption the effects produced by early experiences with alcohol diminish; that is, tolerance is acquired to the physiological and behavioural effects produced by the drug. Tolerance is defined as a decrease in the magnitude of effect of a given dose of drug over repeated administrations. In order to achieve the magnitude of effect produced by earlier administrations, an increase in dosage is required (Kalant, LeBlanc & Gibbins, 1971).

Tolerance has been differentiated into two basic types: (1) metabolic tolerance, and (2) functional tolerance. Metabolic tolerance is due to physiochemical changes which affect the rate of metabolism and elimination of alcohol from the body. The faster the body gets rid of the alcohol ingested the less drug there is around to have an effect. Functional tolerance (sometimes known as "target organ" or "central nervous system" tolerance) and dependence are responses to repeated alcohol consumption that have both a pharmacological and a behavioural basis. They can be regarded, pharmacologically, as complementary manifestations of a state of neuroadaptation which develops in response to alcohol consumption. Pharmacologically, tolerance is a state whereby a higher blood alcohol concentration (BAC) (and therefore dose) is needed to produce effects that were originally achieved at lower concentrations. Dependence is a state of abnormal function of the body when alcohol is absent and which is normalised when alcohol is present. This is seen characteristically by the appearance of physical withdrawal symptoms (such as tremor and sweating) when BACs are approaching zero in a dependent individual.

2. The biological basis of functional tolerance.

Several mechanisms underlining tolerance and dependence have been postulated and although it is beyond the scope of this review to describe them all, a few brief remarks will be made. Fuller accounts have been provided by Kalant (1988) and Littleton and colleagues (1988).

2.1 Changes in calcium flux

The theory that has gained most credence in recent years is that tolerance and dependence result from changes in neuronal calcium flux. Essentially, acute exposure to alcohol inhibits the influx of calcium into the nerve terminal in response to the arrival of the nervous impulse; this reduces transmission and hence suppresses activity in nerve pathways. Following chronic exposure there is an increase in the number of calcium channels which tends to restore calcium flux to normal (Littleton, Harper, Brennan & Guppy, 1988). This is

the postulated basis of tolerance. When alcohol is no longer present the increased calcium influx due to nerve depolarisation results in an increase in neurotransmission and neuronal activity. It is suggested this results in withdrawal symptoms, indicating the presence of dependence.

2.2 Condensation products

Another theory concerns the generation, as a result of alcohol metabolism, of condensation products with brain amines (Kuriyama, Taguchi & Hashimoto, 1988). These condensation products have been found to have addictive potential. These include tetrohydrobetacarboline (derived from the reaction of acetaldehyde and serotonin), salsolinol (produced from acetaldehyde and dopamine) and tetrahydropapaveroline (produced from the combination of acetaldehyde and a dopamine condensate, DOPAC). There is no known association of any of these compounds with depression or sudden death, and the levels which have been detected in human subjects during alcohol metabolism are considerably lower than are active pharmacologically.

2.3 Serotonin deficiency

There is also some evidence that deficiency of serotonin may contribute to the development of dependence on alcohol. Levels of serotonin in parts of the brain subserving hunger and reward functions are lower in experimental animals who have been selectively bred for preference for alcohol (Lumeng, Murphy, McBride & Li, 1988). Following chronic alcohol consumption brain levels of serotonin are lower in non-selectively bred animals. Increasing brain levels of serotonin by administering a serotonin uptake inhibitor, reduces the spontaneous alcohol consumption of experimental animals. Studies reported in human beings also show that such drugs reduce the alcohol consumption of problem drinkers, both by reducing the number of drinks taken per day and by increasing the number of abstinent days (Naranjo and Sellers, 1988). No specific changes in mood during drinking or abstinence have been reported with these drugs although some (e.g., zimelidine) also have antidepressant properties.

2.4 Monoamine oxidase

Several studies have shown that activities of the enzyme monoamine oxidase are lower in the brain and also in blood platelets from alcohol abusing and dependent persons (von Knorring, Oreland & von Knorring, 1987). This is relevant to the issues discussed in this report because a deficiency of monoamine oxidase is also found in some persons who are depressed (Poirier, Loo, Mitrani et al., 1987). Indeed, it has been suggested that the lowering of monoamine oxidase levels is a particular feature of alcohol dependent persons who experience depression. Studies of racial variations in monoamine oxidase levels are in their infancy. This would be a relevant area to investigate the basis for depression, suicidal ideation and hanging in Aboriginal persons.

3. Drug and alcohol interactions

Alcohol can interact with many drugs, both those taken for medical reasons and those used recreationally. When taken concurrently, alcohol potentiates the action of sedative drugs such as benzodiazepines and barbiturates. The potentiation is caused both by inhibition of the metabolism of drugs by alcohol and also because they share common sites of action in the central nervous system. Chronic alcohol consumption induces a state of metabolic and functional tolerance to many psychoactive drugs and when a chronic drinker is sober, the effective dose of a sedative drug is frequently considerably higher than normal. The metabolic tolerance declines with abstinence over 2-3 weeks; functional tolerance is more long-lasting though adequate serial studies are not available to indicate the exact time course. Tolerance does not offer secure protection against the acute potentiation of a sedative drug's action by alcohol. Alcohol also potentiates the blood sugar lowering effect of insulin and oral hypoglycaemic drugs. A fuller account of drug and alcohol interactions is given in a review by Saunders (1986).

4. Tolerance to the cognitive and psychomotor effects of alcohol

Under the classification of functional tolerance a distinction is often made between acute tolerance (tolerance which is observed within a single drug-taking session) and chronic tolerance (tolerance which is acquired over repeated drug-taking sessions). Each of these will be discussed in connection with tolerance to the effects of alcohol on cognitive and psychomotor functioning.

4.1 Acute tolerance

When alcohol or any drug is taken orally it goes through various processes of being absorbed into the bloodstream and distributed throughout the body. While absorption and distribution are taking place, other processes begin to occur which break down the alcohol and remove it from the body (metabolism and excretion). Thus, the BAC curve has a rising phase which largely reflects the absorption process and a falling phase which predominantly reflects the elimination of alcohol from the body. It has been observed that the effect of a dose of alcohol is often greater on the ascending limb of the BAC curve, than it is at the same BAC on the descending limb of the curve. Such adaptation after an acute or single administration of a dose of alcohol has been observed on a number of cognitive tasks (e.g., recall of material shortly after its presentation, Jones, 1973; a pursuit tracking task, Beirness & Vogel-Sprott, 1984b; coding and standing steadiness, Hurst & Bagley, 1972). However, the extent of the adaptation observed seems to depend upon the nature of the task (Hurst & Bagley, 1972). Hurst and Bagley suggest that reaction time shows least acute adaptation while perceptual-motor skills and cognitive skills such as those involved in coding are more sensitive. This

reduced effect of a given BAC during the elimination of a single dose of alcohol has been referred to as acute tolerance. Some early studies of this effect were criticised for not controlling for differences in the concentration of alcohol as measured in blood and the concentration of alcohol in the brain at the time of testing. However, many later studies have taken this factor into account and still report evidence of the phenomenon (Kalant et al., 1971).

4.2 Chronic tolerance

It is generally accepted that chronic tolerance develops to many of the effects of alcohol, including its effects on cognitive functioning. Although there is a vast literature on the acquisition of tolerance to the physiologic and sensorimotor effects of alcohol in animals, there are few studies which have systematically investigated the effect of tolerance on cognitive impairment produced by alcohol in humans (Poulos, Wolff, Zilm, Kaplan & Cappell, 1981).

Laboratory research on this topic has followed two basic strategies: (1) studies which compare the acute effects of alcohol on heavy versus light drinkers (e.g., Rosen & Lee, 1976), and (2) studies which give repeated administrations of alcohol to the same subjects and compare their performance across trials (e.g., Poulos et al., 1981). Each of these bodies of literature will be discussed below.

4.2.1 Comparisons of heavy versus light drinkers

One of the earliest assessments of chronic tolerance to the cognitive impairing effects of alcohol was conducted by Goldberg (1943). He compared individuals classified as heavy drinkers, moderate drinkers and abstainers on tests of motor coordination, sensory perception, and intellectual function. In all tests he found reduced impairment in the more experienced drinkers when tested across a range of BACs from 0.04 to 0.16 g%. Abstainers and heavy drinkers differed in their response to alcohol by a factor of 1.5 to 2 in BAC. Goldberg's conclusion that the more experienced drinkers had acquired tolerance is compromised, however, as there were no estimates of the individual's responses to alcohol before extended alcohol consumption had occurred. It is therefore possible that the group differences observed were due to an innate difference in sensitivity to alcohol rather than a function of extended experience with the substance. People show tremendous variability in their initial sensitivity to the impairing effects of alcohol (Kalant et al., 1971). It could be argued that the abstainers in Goldberg's study were self-selected on the basis of their profound sensitivity to the impairing effects of alcohol (Goldstein, 1983). This criticism can apply to most studies of this type where groups with different drinking histories are compared.

Investigations of chronic tolerance to the effects of alcohol on memory and other cognitive functions have produced inconsistent findings (for a brief review see Parker, Alkana, Birnbaum, Hartley & Noble, 1974). In their study, Parker et al. compared the performance of 12 hospitalised volunteers, who were diagnosed as chronic alcoholics, with 12@@

nonalcoholics, who were classified as moderate social drinkers, on tasks of digit-span and free-recall of categorized word lists both when sober and after two different doses of alcohol (i.e., 0.67 and 1.33 ml/kg of absolute alcohol; no BACs were reported). Drinking history data from both groups revealed that the average yearly consumption of absolute alcohol for the alcoholic group was 17.2 gallons while for the moderate social drinkers it was 1.5 gallons. Parker and her colleagues found that both groups were impaired by alcohol on both tasks and that there was no evidence of tolerance in the alcoholic group. Recall of categorised lists allowed an assessment of the subjects' ability to organise information information in memory, as well as their ability to recall information. On this task, the alcoholic group performed more poorly than the moderate drinkers, whether sober or intoxicated. The occurrence of such a deficit suggested that some members of the alcoholic group may have been suffering from brain damage, possibly as a result of their many years of heavy alcohol use. It is possible that the presence of neurotoxicity in the alcoholic group acted to cancel out any acquired tolerance to the cognitive impairment induced by alcohol (Poulos et al., 1981). Self-reports by alcoholics have indicated that they experience a loss of tolerance after years of heavy drinking which the alcoholics themselves tend to attribute to organic damage (Jellinek, 1960).

In contrast to the findings of Parker et al. (1974), Mello (1972) found that alcoholic subjects showed no impairment in a recognition task in which they were required to match one of a sample of four pictures with a target picture which had been presented between 0 and 6 minutes previously (delayed matching to sample task). Two groups of alcoholics were compared, those who had a history of 'blackouts' and others who did not. "Blackout" is usually defined as a loss of memory for events that occur during drinking. Although the person may be obviously conscious while these events take place, he or she cannot recall them later. It has been postulated that blackouts can be attributed to an alcohol-induced deficit in short-term memory (i.e., memory for events that occurred in the recent past, usually within an interval of 5-30 minutes) (Lisman, 1974).

The BACs reached by some subjects in this study exceeded 0.20 g%, a level at which most inexperienced drinkers would be grossly intoxicated. Yet some of these alcoholic individuals showed an improvement in performance when compared with their baseline levels of performance while sober. Previous research has shown that at much lower BACs (e.g., 0.07 g%) social drinkers were impaired on a similar task (Carpenter & Ross, 1965). The alcoholic groups tested by Mello were apparently tolerant to such impairing effects of the drug.

Two nonpharmacological factors, manipulated in the Mello study, may have contributed to the high performance levels shown by the alcoholics. First, reinforcement was provided for correct responding, and, second, it was ensured that subjects were paying attention to the target items as they

were presented by requiring subjects to initiate each target presentation. In a later review, Mello and Mendelson (1978) argued that these nonpharmacological factors of attention and motivation are important considerations in any study assessing the effects of alcohol on cognitive performance (see also Section 3.5 of Chapter 4). Deficiencies in either of these factors could result in an over-estimation of the amount of cognitive impairment produced by alcohol. Furthermore, unlike the study by Parker et al. (1974), all participants in this study were examined to ensure the absence of neurological disorders.

Rosen and Lee (1976) compared the performance of alcoholics, heavy drinkers and social drinkers on several cognitive tasks while sober and intoxicated. Subjects were recruited through newspaper advertisements and were classified into the three different drinking categories according to the length of time they had been drinking, the quantities they drank and their self-reports of withdrawal symptoms, blackouts and personal problems attributed to excessive alcohol use. (In the studies described previously, the alcoholic subjects were typically individuals in treatment for their alcohol problems. It may be that these individuals had a longer or heavier drinking history or were more severely affected by alcohol use than the subjects who were classified as alcoholics Rosen and Lee (1976).)

Rosen and Lee found that although the social drinkers showed gross signs of intoxication at BACs of 0.10 g% (e.g., nausea, slurring of speech, poor coordination) and the heavy drinkers and alcoholics showed almost none of these symptoms, all groups were equally impaired on the cognitive performance measures (i.e., digit span - recall of lists of numbers in forward and reverse order; free and cued recall of categorized word lists). All groups showed equivalent performance levels while sober suggesting that the alcoholic subjects did not suffer from any chronic deficits in cognitive functioning.

The discrepancy between the levels of gross signs of intoxication observed and of performance on specific cognitive tasks in the heavy drinkers and alcoholics is a rather paradoxical finding. It is, however, an interesting observation in light of the fact that most of the studies reporting on tolerance to very high BACs rely on gross measures of intoxication (see Section 6.1.3). It suggests that failure to observe gross signs of intoxication in someone who has consumed alcohol does not necessarily entail that the cognitive functioning of that individual is not impaired. Tolerance may develop to these different effects of alcohol at different rates. Also the heavy drinkers and alcoholics in this study would have had much more practice in coping with the gross signs of intoxication than they would have had with the cognitive tasks they were required to perform. No strong conclusions regarding these differences in tolerance to the cognitive and intoxicating effects of alcohol can be drawn from this study since the results are based on a very small sample size of 8 subjects per group.

Therefore, the findings require replication with a larger sample size and with a range of doses of alcohol before any broad generalizations can be made.

In summary, a review of the literature on studies in which experienced and inexperienced drinkers have been tested for their resistance to cognitive impairment by alcohol has revealed no conclusive results. Whether or not tolerance is observed may depend, to a large extent, upon the types of tasks evaluated, the motivation and attention levels achieved in the study, the dose of alcohol employed, and the integrity of the subjects' cognitive functioning capacities in the absence of alcohol.

4.2.2 Studies of acquired tolerance

Another approach to the investigation of tolerance involves an evaluation of changes in the degree of cognitive impairment produced by a given dose of alcohol over repeated exposures to that dose in relatively inexperienced drinkers. In one such study, Poulos et al. (1981) found that over repeated exposures to alcohol, a group of 7 social drinkers showed a significant reduction in memory impairment on a task requiring recall of a categorized list of words. This reduction in impairment over repeated alcohol exposures was interpreted as the acquisition of tolerance. However, the absence of a control group to control for the effect of practice compromises this conclusion (cf. Maylor & Rabbitt, 1987).

Vogel-Sprott (1979) investigated the acquisition of tolerance to a cognitive task (coding) and a motor skills (pursuit rotor) task by examining performance on both the rising and falling limbs of the BAC curve over repeated alcohol exposures. The mean peak BAC achieved in this study was 0.08 g%. The results indicated that coding performance showed both acute recovery and the development of tolerance over drinking sessions, while performance on the pursuit rotor task did not show tolerance. However, impairment on each task was observed at different points on the BAC curve, and this may have implications for the observed differences in acute and chronic recovery. Distinguishing between rising and falling limbs of the curve is clearly an important consideration in the assessment of task differences in sensitivity to alcohol (Vogel-Sprott, 1979).

Again it appears that the task requirements are important factors in the observation of tolerance. Memory impairment showed better adaptation over repeated alcohol trials than did perceptual-motor functioning. However, these studies are based on very small sample sizes and repeated exposures to a single dose of alcohol, therefore, their findings cannot be readily generalised.

5. Learning and tolerance

5.1 Behaviourally-augmented tolerance

A substantial body of research on animals shows that the rate of acquisition of tolerance to motor-impairment produced by alcohol can be influenced by practice on the motor-task during intoxication (LeBlanc, Kalant & Gibbins, 1976; Wenger et al., 1981). The moving belt test is a task commonly applied to assess the motor-ataxia produced by alcohol in rats. It involves having rats walk on a narrow moving belt which is suspended over an electrified grid. If the rat falls off the belt it receives a shock. On initial exposures to alcohol the rat's motor abilities are impaired and it spends substantial amounts of time off the belt. However, with repeated exposures to alcohol the time spent off the belt decreases as tolerance develops. If rats are allowed to practice walking on the moving belt while intoxicated they acquire tolerance more rapidly than rats who receive the same amounts of alcohol and practice on the task, but who never have an opportunity to practice while under the influence of alcohol.

Recent research with humans indicates that practice on a task or even mental rehearsal of the task while intoxicated can increase tolerance (Beirness & Vogel-Sprott, 1984a; Vogel-Sprott & Webster, 1984). In a study by Vogel-Sprott and Webster (1984) social drinkers were required to track a moving light stimulus with a photosensitive stylus while under the influence of alcohol (the average peak BAC was 0.086 g%). Time on target with the stylus was the dependent measure. Over three sessions in which alcohol was consumed, one group was allowed to practice the task, another group was allowed to mentally rehearse and a third group rested. On test the groups who had practiced and mentally rehearsed the task performed better than the group that had rested after alcohol consumption.

In another study subjects were allowed to mentally rehearse the same pursuit rotor task described above either after or before alcohol consumption (Sdao-Jarvie & Vogel-Sprott, 1986). The group that mentally rehearsed the task after alcohol was significantly more tolerant to alcohol impairment than the group who rehearsed before alcohol. Thus, it was concluded that mental rehearsal of a task while intoxicated can facilitate the development of tolerance.

5.2 Conditioned tolerance

Research with animals indicates that tolerance to various physiological effects of alcohol can be modulated by the environment in which the drug is administered (e.g., Crowell, Hinson & Siegel, 1981; Le, Poulos & Cappell, 1979). In a study by Le et al. (1979), a constant dose of alcohol was repeatedly administered to rats in the presence of a distinctive set of environmental cues. Over repeated drug sessions, tolerance developed to the temperature-reducing effect of alcohol such that when the same dose of alcohol was administered it no longer produced a significant reduction in

body temperature. When those rats who showed tolerance to alcohol in the presence of the distinctive environment were given the same dose of alcohol in the presence of a different set of cues, a significant drug effect was observed. Tolerance was only evident in the presence of the environment in which prior drug administrations had taken place. This phenomenon is known as conditioned tolerance and it has been demonstrated consistently by several labs and with other drug effects (for reviews see Goudie & Demelweek, 1986; Hinson & Siegel, 1980; Siegel, 1983). A model based on principles of Pavlovian conditioning has been postulated to account for this effect. According to the model, environmental features repeatedly associated with alcohol-intake come to act as cues or conditional stimuli for alcohol. These cues acquire the capacity to elicit drug-opposite responses which serve to counteract the effects of alcohol. In the example described above, the alcohol-paired cues elicited an increase in body temperature which presumably summated with the temperature reducing effect of alcohol to produce tolerance (i.e., a diminished drug effect).

Several studies have provided evidence for conditional tolerance to alcohol when its effects on physiological, cognitive and psychomotor functions have been assessed in human subjects (Annear & Vogel-Sprott, 1985; Beirness & Vogel-Sprott, 1984a; Newlin, 1986; Shapiro & Nathan, 1986). In the studies by Vogel-Sprott and her colleagues two nonpharmacological factors were manipulated: the environment in which alcohol was given and whether practice on the task while intoxicated was allowed. These studies found that both practice under intoxication, and giving alcohol in the presence of an environment where it was expected, enhanced tolerance.

Other studies have found mixed results where conditioning factors influence tolerance to some effects of the drug but not others (e.g., Shapiro & Nathan, 1986). The phenomenon of conditioned tolerance requires further investigation before its significance to real-world demonstrations of tolerance can be predicted.

6. Tolerance to high concentrations of alcohol

As indicated earlier, the information concerning the effects of alcohol on skills performance has been derived from studies using healthy volunteers, in most cases young university students, who were given an acute dose of alcohol to drink in a relatively short time period. The effects reported were therefore usually studied at the peak or the rising phase of the BAC curve after one dose of alcohol. The peak BACs reached were seldom greater than 0.1 g%.

Most of the information concerning the gross behavioural effects of alcohol has also been collected from the proportion of the population who could be regarded as "social drinkers". The descriptions of gross behavioural effects of alcohol have therefore been perpetuated and descriptions such as that illustrated below have been accepted as the norm.

The descriptions provided in the table below apply to most of the population of social drinkers and describe the effects at the peak of intoxication after drinking has stopped and before the BAC curve begins to decline. They may not apply to those individuals who are regular drinkers and who have developed a degree of tolerance to alcohol.

TABLE I

EFFECTS OF ALCOHOL AT VARIOUS BLOOD CONCENTRATIONS

BAC	Likely effects (during upswing of BAC curve)
0.03	Feeling of well-being and relaxation. More talkative. Slightly slower reactions.
0.05	Euphoria. Increased self-confidence. Impairment of attention, judgement and psychomotor reactions. Increased risk of accidents.
0.075	Disinhibition. Garrulousness. Further loss of judgement and coordination. Nausea.
0.10	Emotional instability. Argumentativeness. Impaired balance. Onset of ataxia (staggering gait). Slurred speech. Dizziness. Desire to sleep when not in company.
0.15	Confusion and disorientation. Apathy. Drowsiness and sleep. Decreased pain sensation. Worsening ataxia.
0.20	Stupor. Inertia. Inability to stand or walk. Vomiting. Incontinence.
0.30	+ Coma. Anaesthesia. Hypothermia. Possible death from respiratory paralysis, aspiration of vomit.

Note that the effects are typical of those experienced by non-tolerant drinkers. When tolerance has developed the effects on behaviour and coordination may not be seen until the BAC is three to four times higher. Note also that dysphoria, apathy and nausea are commonly experienced at BACs below 0.075 g% during the downswing of the BAC curve.

Note.

From Alcoholism and problem drinking: Theories and treatment (p.87) by J.Christopher Clarke with John B.Saunders, 1988, Sydney: Pergamon Press (Australia). Copyright 1988 by J.Christopher Clarke with John B.Saunders. Reprinted by permission.

Some of the variables which render the above descriptions of behaviour and subjective states somewhat inaccurate at the various BACs have been discussed already. These include the rate of drinking- with slower drinking time, behavioural effects at a given BAC tend to be less due to the development of an acute tolerance. Another, and a more important confounding factor is associated with the drinking history of the individual, and the degree of chronic tolerance to the effects of alcohol. It is clear that a proportion of the population of alcohol consumers will be significantly tolerant to the behavioural effects of the drug, and for whom the behavioural effects at BACs indicated above will not necessarily apply.

There is a lamentable lack of experimental studies which have examined the effects of high concentrations of alcohol on human skills performance. There are obvious practical and ethical reasons why this is so. A search of the research literature found that the only studies which have addressed the question of the effects of very high BACs on cognitive functions in humans have been those by Mendelson and Mello (1966) using alcoholic volunteers. The study employed operant conditioning techniques to examine drinking behaviour of alcoholic men. The operant conditioning consisted of requiring the subject to perform a number of continuous tasks in order to receive reinforcement of either alcohol or money. The tasks required a degree of complex cognitive skills. The prediction was that if the alcohol reinforcement was made contingent upon the successful performance of a complex discrimination task, a subject who became increasingly inebriated would not be able to sustain his initial performance. The four volunteers were chronic alcoholics with histories of alcoholism of some 20 to 23 years duration. At the time of the study they had been abstinent for a period of between 10 and 60 days. Two tasks were studied, each requiring the subject to operate an ignition switch to begin the experiment and to use a selector switch to choose whether alcohol (delivered directly into a glass) or money was required as the reinforcer. One task required the subject to press a button for reinforcement which was delivered according to one of four schedules. (1) When on a fixed ratio schedule the subject had to press the key repeatedly a specified number of times (60, 120, 240 or 360) to get the reinforcement. (2) When on a fixed interval schedule of reinforcement only the first response which occurred after a specified interval (1, 2, or 3 minutes) had expired was reinforced. (3) During the extinction schedule no responses were reinforced, therefore responding should have decreased. (4) On the differential reinforcement of no response schedule reinforcement was given only if a response was made after an interval of 30 seconds in which no button pressing had occurred. As each schedule was introduced it was signalled by the illumination of one of four different coloured lights.

The second task employed a console rather like an amusement park driving simulator in which the subject was required to turn an ignition key to begin the experiment, keep the vehicle on the road (a tracking task), obey traffic signals,

and stop or turn. Points were scored for performance and reinforcement was delivered on a fixed ratio schedule for accumulated points.

Although two different procedures were employed, several consistent features were observed in patterns of drinking and in the subjects' responses to alcohol. All subjects achieved high BACs (between 0.150 and 0.300 g%) within the first 24 hours of the study. Despite these continuous high BAC levels, the subjects only exhibited mild signs of intoxication characterised by a tendency to become more talkative and boisterous, without concomitant slurring of speech, ataxia or gross disturbances of behaviour. No subject became markedly intoxicated with severe ataxia, dysarthria and stupor. No subject proved unable to work in the operant task because of his intoxication.

6.1 What is the incidence of high order tolerance to alcohol?

The alcoholic subjects studied by Mendelson and Mello (1966) exhibited a remarkable degree of tolerance to alcohol. An unknown factor, however, is the actual incidence of tolerance of this order within the general community. There has been remarkably little research effort to address this question. The information currently available can be considered under three classifications of data:

(a) The collection of data concerned with enforcement of the drink driving laws. Some of these data provide some idea of the proportion of alcohol consumers within the population who have developed a high degree of tolerance. However, the populations studied are selective because they have been derived from drivers or pedestrians involved in an accident. They are not necessarily representative of the general community of alcohol consumers.

(b) Behavioural tests developed to assess the degree of intoxication by gross clinical measures rather than by the measurement of breath (or blood) alcohol concentration. This has been necessary in some countries (for example, Finland and in some States of the United States of America) for various legal reasons. In collecting data for these tests some data which indicate the proportion of high BACs within the test population are available as well as the ability to recognise impairment by the behavioural tests.

(c) Clinical reports of patients who survived surprisingly high concentrations of alcohol. These reports present a clinical picture which describes gross behavioural signs.

6.1.1 Evidence from BAC data collected for traffic law enforcement

A report from the Council of Scientific Affairs, Division of Personal and Public Health Policy of the American Medical Association (Council Report, 1986) includes an analysis of data derived from seven studies, which indicates the BAC and the identification of "drunkenness". A total of 13,230 cases were accumulated and the following outline given.

BAC (g%)	Percentage considered to be "drunk"
0.00 - 0.05	0 to 10
0.051 - 0.10	14 to 68
0.101 - 0.15	41 to 93
0.151 - 0.20	83 to 97
0.201 - 0.25	93 to 100
0.251 - 0.30	94 to 100
0.301 - 0.35	96 to 100
0.351 - 0.40	93 to 100
0.401	99 to 100

Data from Council Report, 1986

It is to be noted that in the BAC of greater than 0.20 g% there are, within each subclassification, between 1 and 7% of cases which were not considered to be "drunk". The criteria for drunkenness are not provided in the report.

Australian data from the New South Wales Police Department (Moynham, 1988) reported on the analysis of blood samples taken under the Motor Traffic Act. Samples were from drivers of motor vehicles or pedestrians. From 1st January to 31st December 1987, there were 532 persons whose blood had been taken following a motor accident and whose BAC was 0.300 g% or greater. These persons made up 2.6% of the total blood samples collected which had a BAC of 0.05 g% or greater. These people, having been involved in a road accident, were either drivers or pedestrians. Whilst we know little of their psychomotor skills, we do know they had sufficient coordination to drive (however unsafely) a motor vehicle or, as pedestrians, to attempt to cross a road (and come into contact with a vehicle). What is more, these people were involved in an accident. There may be others who attain these BACs but are not involved in accidents.

6.1.2 The development of behavioural tests to assess BAC
For various legal reasons, studies have been conducted to examine the correlation between signs of intoxication and the actual BAC. Some studies were conducted in order to develop a practical instrument for law enforcement where, for legal reasons, a breathanalysis cannot be conducted at the roadside (Burns, 1985; Penttila, Tenhu & Kataja 1971 ; Teplin & Lutz, 1985). Others have examined the ability of individuals with varying degrees of training, to identify drunkenness, either in themselves (Russ, Harwood & Geller, 1986) or in others stopped whilst driving a motor vehicle (McGuire, 1986). According to McGuire, only somewhere in the the order of 20% of drivers whose BAC was at or over 0.1 g% were identified by observation. The study involved 1,115 drivers in a roadside survey.

Finnish data (Penttila, Tenhu & Kataja, 1971) generated in a study of 6,839 cases in the development of a test battery to determine drunkenness in drivers (Field Sobriety Tests; FST), also provide information as to the distribution of BAC of drivers among this sample. This study also gives some

indication of the ability of the FST to detect impairment. The cases were examined by 16 participating physicians and 99% of cases were examined at the request of the police; almost without exception, these referrals were for suspected impaired driving or for accidents. The sample was therefore selected from drivers and those who were conscious and able to drive a vehicle, however unsafely. The clinical assessment was performed within 1-2 hours of admission in 64.5% of cases and within 2-5 hours in 12% of cases. Therefore, most subjects were assessed during the elimination phase of the BAC curve, when impairment would have been reduced because of acute tolerance.

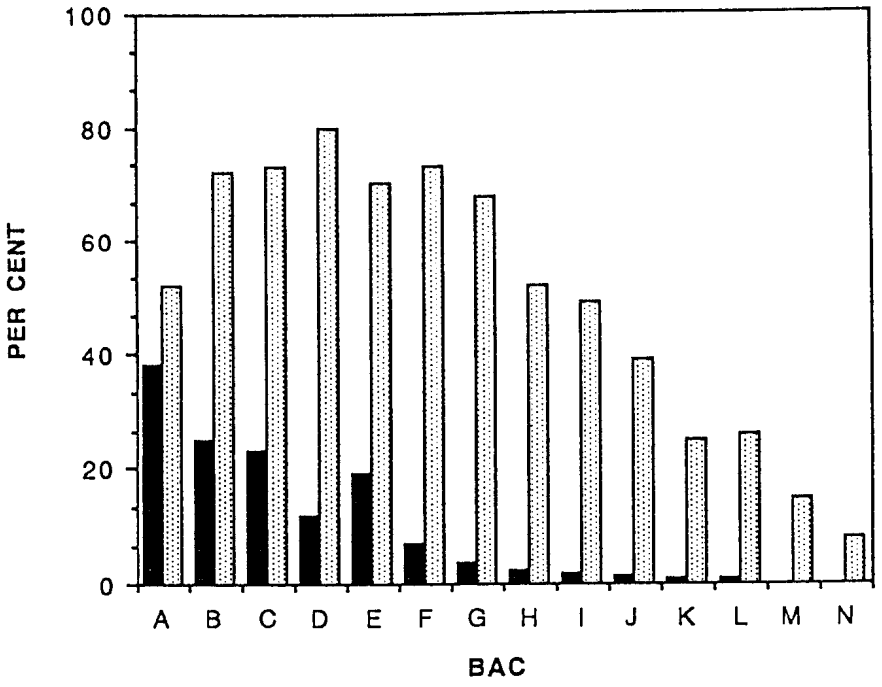
The distribution of BACs recorded followed a normal distribution curve with peak values lying between 0.161 and 0.180 g%. At the time of examination, BAC of zero was recorded in 254 persons. Forty-five of the sample had BACs in excess of 0.30 g% and the highest recorded was 0.37 g%. In the sample there were 1,842 persons who had a BAC in excess of 0.20 g%. Of these, only 11 were classified as being sober by the test battery, and presumably exhibited at this BAC a high degree of tolerance. The distribution of classification of grades of drunkenness (no; mild; moderate; strong) across the range of BACs is shown in Figure 5.1 (next page). Here it will be noted that at the lower BACs there has been a decided error in the over-estimation of the degree of drunkenness - or of the BAC.

Gross performance measures were used: smell of alcohol; colour of face; pulse rate; walking tests with eyes open and closed; gait in turning; Romberg's test with eyes open and closed; finger-finger test; match test; speech and behaviour; relaxation of inhibitions; pulling oneself together; counting backwards test; and orientation as to time.

A brief description of the match test will be useful to give the reader some idea of the types of behaviours being assessed. The match test required the subject to collect matches from the floor: "Four matches are thrown on the floor in an area of approximately 0.5 meter diameter from which the subject collects them with one hand and, at the same time, places them on the table." (Penttila et al. 1971, p.). The grades of response for each test were defined; for example, for the match test a grade of 1 was given for fumbling in collecting ; 2 if the subject keeps dropping the matches or leaves some of them on the floor; 3 if attempts are failures, throwing the matches around. Each subject was given a final numerical error score derived from the scores received on all of the tests.

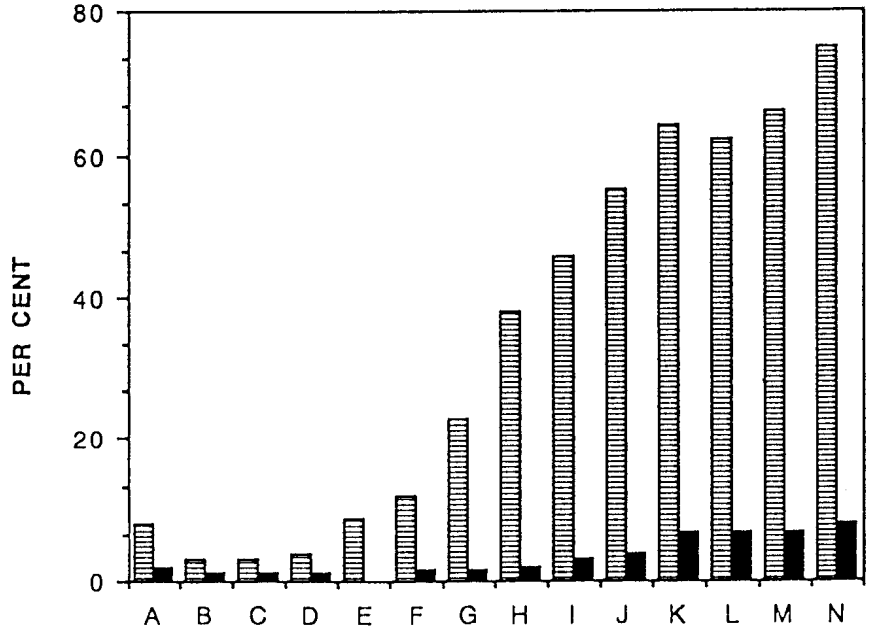
There was a large degree of error with these fairly gross tests of impairment. The research group has subsequently pursued the concept of clinical testing and have recommended a shortened battery of performance tests which nevertheless are still of gross measures (Penttila, Tenhu & Kataja, 1974).

THE FINAL EVALUATION OF THE STATE OF INTOXICATION AT THE VARIOUS BLOOD ALCOHOL CONCENTRATIONS



NO
LITTLE (MILD)

KEY TO BAC (g%)
A = 0.00 - 0.015
B = 0.016 - 0.06
C = 0.061 - 0.08
D = 0.081 - 0.1
E = 0.101 - 0.12
F = 0.121 - 0.16
G = 0.161 - 0.18
H = 0.181 - 0.2
I = 0.201 - 0.22
J = 0.221 - 0.24
K = 0.241 - 0.26
L = 0.261 - 0.3
M = > 0.3



MODERATE
STRONG

Independently, a group in the United States (Burns 1985) has examined a series of Field Sobriety Tests and have recommended a standardised three test battery: horizontal gaze nystagmus; the walk and turn test; and the one-leg stand test (Burns & Moskowitz, 1977; Tharp, Burns & Moskowitz, 1981).

6.1.3 Clinical reports of patients who survived extremely high levels of alcohol

These are clinical curiosities, usually described as single cases, for example, such as the case reported by Hammond, Rumack and Rodgerson (1973). The case was of a woman, suspected of drunken driving and a hit-run incident, who was admitted to hospital in a comatose state with a BAC of 0.78 g%. Three hours after admission her BAC was 0.52 g% and she was fully conscious and able to relate her drinking history.

A truly extreme case was that described by Johnson, Noll and Rodney (1982) of a patient admitted with a BAC of 1.51 g%. The patient on admission was described as "..agitated and slightly confused but alert, responsive to questioning and oriented to person and place (though unclear as to time), neurological examination showed slightly depressed reflexes only. Rest of general physical examination was normal" (p. 1394). These cases make medical history.

Clinical descriptions of selected, or sequential cases admitted to casualty have indicated the incidence within emergency admissions of extremely high BACs in patients with a high degree of alcohol tolerance (Davis & Lipson, 1986; Lindblad & Olsen, 1976; Perper, Twerski & Wienand, 1986; Urso, Gavaler & Van Thiel, 1981). Unfortunately, the description of the degree of tolerance and of the psychomotor skills of the patients have been of gross measures only.

A study by Urso et al. (1981) reported on all patients admitted to the emergency room who fulfilled two criteria: (a) admitted to having consumed alcohol and (b) who were deemed non-intoxicated by the physician. This diagnosis depended on the patient being:

> 1) ambulatory; 2) alert and capable of passing a simple mental status examination consisting of demonstrating orientation for person, place and time correctly following a simple three-step command, and being able to perform simple calculations (i.e. make change) ; 3) have no obvious neurologic abnormality; and 4) be deemed responsible for self (i.e. the subject could be released without delay and without the need to have a third party accept responsibility for the subject. (p.1054)

Over the period of the study, 86 subjects conformed to the inclusion criteria including the behavioural description. Ten refused to have their BAC measured and 11 had a zero BAC. Of the remaining 65, the range of BACs was 0.12 to 0.54 g% with a mean of 0.268 g%.

Davis and Lipson (1986) described the degree of impairment of alcoholic patients attending the detoxification unit of the Royal Prince Alfred Hospital, Sydney. The assessment was made by the physician and graded according to the scale, 0 = apparently unaffected; 1 = altered mood or behaviour; 2 = confused; 3 = drowsy; 4 = stuporous; 5 = comatose. An assessment was made of 32 ambulatory patients, 17 of whom exceeded a BAC of 0.3 g% and yet had little clinical evidence of intoxication (grades 1 to 3; only 1 patient was graded as 3).

A similar series of alcoholic patients presenting at a detoxification centre in Pittsburgh USA was described by Perper, Twerski and Wienand (1986). One hundred and ten cases were assessed using measures of impairment of vision; pupillary size; speech; verbal comprehension; coordination; ability to undress. The measure of coordination was based on the ability of the subject to follow directions, move arms and hands when so instructed, hold the thermometer, get up on a carriage and so forth. The degree of impairment was graded as slight, moderate, or severe.

Of the 110 admissions, 87 (80%) had positive BACs, with a range of values between 0.015 and 0.444 g%. Fifty-four cases had BAC values of 0.20 g% or higher, and, of these, 13 (24%) showed no sign of clinical intoxication. Of 26 subjects with BACs of 0.30 g% or higher, the following number of subjects were normal in the clinical signs indicated :

Speech - 11; Verbal Comprehension - 8
Gait - 6; Pupils and Vision - 11; Coordination - 2.

From the available clinical data it is apparent that there is a considerable number of people who fail to show obvious signs of drunkenness even though they have very high BACs. To these published reports, there are many other anecdotal reports made by physicians involved in the treatment of alcoholism; in many of these the physicians testify to the fact that the patients drove themselves to the clinic and successfully made the necessary manoeuvres to park the car in the car park (e.g., Linnoila, personal communication). Although this group represents a relatively small proportion of the drinking population it is nevertheless numerically quite large.

The measures of performance skills described in all of the studies reviewed above are of gross behaviour or clinical signs, many of which are quite subjective. There is a notable lack of information in the literature as to the effects on more complex measures of human psychomotor performance at these very high BACs.

7. Conclusions

"The phenomenon of acquired tolerance to alcohol....has been systematically studied for at least 50 years, [yet] many questions remain about the causes, consequences, and modifying factors that affect tolerance ...." (Kalant, 1988, p. 27). The literature reviewed in this section on the effects of alcohol on cognitive functioning demonstrates some of the complexities implied in Professor Kalant's statement.

It is well recognized that alcohol can impair cognitive functioning in humans. It is also evident that tolerance develops to some of these effects. In most of the controlled laboratory studies where an attempt is made to objectively quantify performance on cognitive and psychomotor tasks, the subjects are inexperienced drinkers who are given relatively low doses of alcohol (BACs between 0.02 and 0.12 g%). The applicability of these findings to individuals with extensive drinking histories and who are found to have very high BACs is extremely limited.

The majority of reports in which the effects of very high BACs have been assessed rely on relatively subjective observer ratings of gross behavioural changes in the intoxicated individual. These reports are the major available reference source for making judgements about the degree of tolerance that can be acquired to the intoxicating effects of very high doses of alcohol (greater than 0.20 g%). There are insufficient data in these studies to evaluate sex and racial differences adequately. At these high BACs there appears to be no major effect of age, at least when considering adults as opposed to children. In general, experienced drinkers (in particular, individuals classified as alcoholics) show profound degrees of tolerance to the obvious intoxicating effects of alcohol (e.g., slurred speech, impaired gait, coordination, and verbal comprehension). However, this may not mean that they are unimpaired in their ability to perform tasks involving fine coordination, complicated reasoning and rapid judgement.

References

Annear, W. C., & Vogel-Sprott, M. (1985). Mental rehearsal and classical conditioning contribute to ethanol tolerance in humans. Psychopharmacology, 87, 90-93.

Beirness, D., & Vogel-Sprott, M. (1984a). Alcohol tolerance in social drinkers: Operant and classical conditioning effects. Psychopharmacology, 84, 393-397.

Beirness, D., & Vogel-Sprott, M. (1984b). The development of alcohol tolerance: Acute recovery as a predictor. Psychopharmacology, 84, 398-401.

Burns, M. (1985). Field sobriety tests: An important component of DUI enforcement. Alcohol, Drugs and Driving, 1, 21-25.

Burns, M., & Moskowitz, H. (1977). Psychophysical tests for DWI arrest. (DOT-HS-802-424) Washington, DC: National Highway Traffic Safety, US Department of Transportation.

Carpenter, J. A., & Ross, B. M. (1965). Effect of alcohol on short-term memory. Quarterly Journal of Studies on Alcohol, 26, 561-579.

Clarke, J. C. & Saunders, J. B. (1988). Alcoholism and problem drinking: Theories and treatment. Sydney: Pergamon Press.

Crowell, C. R., Hinson, R. E., & Siegel, S. (1981). The role of conditional drug responses in tolerance to the hypothermic effects of alcohol. Psychopharmacology, 73, 51-54.

Council on Scientific Affairs (American Medical Association) (1986). Alcohol and the driver. Journal of the American Medical Association, 255, 522-527.

Davis, A. R., & Lipson, A. H. (1986). Central nervous system tolerance to high blood alcohol levels. Medical Journal of Australia, 144, 9-12.

Goldberg, L. (1943). Quantitative studies on alcohol in man: The influence of ethyl alcohol on sensory, motor, and psychological functions referred to blood alcohol in normal and habituated individuals. Acta Physiologica Scandinavica, 5 (Suppl. 16), 1-128.

Goldstein, D. B. (1983). Pharmacology of alcohol. New York: Oxford University Press.

Goudie, A. J., & Demellweek, C. (1986). Conditioning factors in drug tolerance. In S. R. Goldberg & I. P. Stolerman (Eds.), Behavioral analyses of drug dependence (pp. 225-285). London: Academic Press.

Hammond, K. B., Rumack, B. H., & Rodgerson, D. O. (1973). Blood ethanol: A report of unusually high levels in a living patient. Journal of the American Medical Association, 226, 63-64.

Hinson, R. E., & Siegel, S. (1980). The contribution of Pavlovian conditioning to ethanol tolerance and dependence. In E. Rigter & R. Crabtree (Eds.), Alcohol tolerance and dependence (pp. 179-197). Amsterdam: Elsevier.

Hurst, P. M., & Bagley, S. K. (1972). Acute adaptation to the effects of alcohol. Quarterly Journal of Studies on Alcohol, 33, 358-378.

Jellinek, E. M. (1960). The disease concept of alcoholism. New Brunswick, NJ: Hillhouse Press.

Johnson, R. A., Noll, E. C., & Rodney, W. M. (December, 1982). Survival after a serum ethanol concentration of 1 1/2%. Lancet, 1394.

Jones, B. M. (1973). Memory impairment on the ascending and descending limbs of the blood alcohol curve. Journal of Abnormal Psychology, 82, 24-32.

Kalant, H. (1988). Alcohol tolerance, dependence and withdrawal: An overview of current issues. Australian Drug and Alcohol Review, 7, 27-34.

Kalant, H., LeBlanc, A. E., & Gibbins, R. J. (1971). Tolerance to, and dependence on, some non-opiate psychotropic drugs. Pharmacological Reviews, 23, 135-179.

Kuriyama, K., Taguchi, J., & Hashimoto, T. (1988). Interactions of salsolinol and betacarbolines with cerebal GABA/benzodiazepine receptor complex: Alteration during alcohol dependence. Australian Drug and Alcohol Review, 7, 43-49.

Le, A. D., Poulos, C. X., & Cappell, H. (1979). Conditional tolerance to the hypothermic effect of alcohol. Science, 206, 1109-1110.

LeBlanc, A. E., Kalant, H., & Gibbins, R. J. (1976). Acquisition and loss of behaviorally augmented tolerance to ethanol in the rat. Psychopharmacology, 48, 153-158.

Lindblad, B., & Olsson, R. (1976). Unusually high levels of blood alcohol? Journal of the American Medical Association, 236, 1600-1602.

Lisman, S. A. (1974). Alcoholic "blackout": State dependent learning. Archives of General Psychiatry, 30, 46-53.

Littleton, J. N., Harper, J. C., Brennan, C. H., & Guppy, L. J. (1988). Adaptation in membrane Ca++ channels as a basis for alcohol-related pathology. Australian Drug and Alcohol Review, 7, 35-38.

Lumeng, L., Murphy, J. M., McBride, W. J., & Li, T. K. (1988). Behavioural and biochemical correlates of alcohol drinking preference: Studies on the selectively bred P and NP rats. Australian Drug and Alcohol Review, 7, 17-20.

Maylor, E. A., & Rabbitt, P. M. A. (1987). Effects of practice and alcohol on performance of a perceptual motor task. Quarterly Journal of Experimental Psychology, 39A, 777-795.

Mello, N. K. (1972). Behavioral studies of alcoholism. In B. Kissin & H. Begleiter (Eds.), The biology of alcoholism. Vol. 2: Physiology and behavior (pp. 219-291). New York: Plenum Press.

Mello, N. K., & Mendelson, J. H. (1978). Alcohol and human behavior. In L. L. Iversen, S. D. Iversen & S. H. Snyder (Eds.), Handbook of psychopharmacology: Vol. 12. Drugs of abuse (pp.235-317). New York: Plenum Press.

Mendelson, J. H., & Mello, N. K. (1966). Experimental analysis of drinking behavior of chronic alcoholics. Annals of the New York Academy of Sciences, , 828-845.

McGuire, F.L. (1986). The accuracy of estimating the sobriety of drinking drivers. Journal of Safety Research, 17, 81-85.

Moynham, A. (Assistant Director, Police Medical Services, N. S. W.). (1988). (Letter to Mr P. Robinson, State Crown Solicitors Office).

Naranjo, C. A., & Sellers, E. M. (1988). Serotonin uptake inhibitors attenuate ethanol intake in humans. Australian Drug and Alcohol Review, 7, 109-112.

Newlin, D. B. (1986). Conditioned compensatory response to alcohol placebo in humans. Psychopharmacology, 88, 247-251.

Parker, E. S., Alkana, R. L., Birnbaum, I. M., Hartley, J. T., & Noble, E. P. (1974). Alcohol and the disruption of cognitive processes. Archives of General Psychiatry, 31, 824-828.

Penttila, A., Tenhu, M., & Kataja, M. (1971). Clinical examination for intoxication in cases of suspected drunken driving. An evaluation of the Finnish system on the basis of 6839 cases. Finland: Statistical and Research Bureau of Talja: Iso Roobertinkatu 20 Helsinki.

Penttila, A., Tenhu, M., & Kataja, M. (1974). Examination of alcohol intoxication in cases of suspected drunken drivers. II. A mathematical analysis of the relationship between the results of clinical examination and blood alcohol. Finland: Liikenneturva, Iso Roobertinkatu 20, 00120 Helsinki 12.

Perper, J. A., Twerski, A., & Wienand, J. W. (1986). Tolerance at high blood alcohol concentrations: A study of 110 cases and review of the literature. Journal of Forensic Sciences, 31, 212-221.

Poirier, M. F., Loo, H., Mitrani, N., Benkelfat, C., Askienazy, S., & Le Fur, G. (1987). Platelet MAO activity in clinical subtypes of depression and DST suppression. Acta Psychiatrica Scandinavica, 75, 456.

Poulos, C. X., Wolff, L., Zilm, D. H., Kaplan, H., & Cappell, H. (1981). Acquisition of tolerance to alcohol-induced memory deficits in humans. Psychopharmacology, 73, 176-179.

Rosen, L. J., & Lee, C. L. (1976). Acute and chronic effects of alcohol use on organizational processes in memory. Journal of Abnormal Psychology, 85, 309-317.

Russ, N.W., Harwood, M.K., & Geller, E.S. (1986). Estimating alcohol impairment in the field: Implications for drunken driving. Journal of Studies on Alcohol, 47, 237-240.

Saunders, J. B. (December, 1986). Drugs and alcohol: Their interactions and how to avoid them. Current Therapeutics, 31-53.

Sdao-Jarvie, K., & Vogel-Sprott, M. (1986). Mental rehearsal of task before or after ethanol: Tolerance facilitating effects. Drug and Alcohol Dependence, 18, 23-30.

Shapiro, A. P., & Nathan, P. E. (1986). Human tolerance to alcohol: The role of Pavlovian conditioning processes. Psychopharmacology, 88, 90-95.

Siegel, S. (1983). Classical conditioning, drug tolerance, and drug dependence. In Y. Israel, F. B. Glaser, H. Kalant, R. E. Popham, W. Schmidt, & R. G. Smart (Eds.), Research advances in alcohol and drug problems (Vol. 7, pp. 207-246). New York: Plenum Press.

Teplin, L.A., & Lutz, G.W. (1985). Measuring alcohol intoxication: The development, reliability and validity of an observational instrument. Journal of Studies on Alchol, 46, 459-466.

Tharp, V. K., Burns, M., & Moskowitz, H. (1981). Psychophysical tests for DWI arrest (Contract DOT-HS-8-01970). California: Southern California Research Institute.

Urso, T., Gavaler, J. S., & Van Thiel, D. H. (1981). Blood ethanol levels in sober alcohol users seen in an emergency room. Life Sciences, 28, 1053-1056.

Vogel-Sprott, M. D. (1979). Acute recovery and tolerance to low doses of alcohol: Differences in cognitive and motor skill performance. Psychopharmacology, 61, 287-291.

Vogel-Sprott, M., & Webster, E. R. (1984). Mental rehearsal of a task under ethanol facilitates tolerance. Pharmacology, Biochemistry & Behavior, 21, 329-331.

von Knorring, L., Oreland, L., & von Knorring, A. L. (1987). Personality traits and platelet MAO activity in alcohol and drug abusing teenage boys. Acta Psychiatrica Scandinavica, 75, 307.

Wenger, J. R., Tiffany, T. F., Bombardier, C., Nicholls, K., & Woods, S. C. (1981). Ethanol tolerance in the rat is learned. Science, 213, 575-577.

Chapter 6. Treatment of Alcohol Dependence

F. Harding Burns

1. Identification of tolerance and dependence

Laboratory animals and humans quickly learn to self-administer many drugs, including alcohol (Jaffe, 1985). A hazard of these drugs is that some individuals eventually develop dependence. Physical dependence refers to an altered physiological state produced by the repeated administration of a drug, which necessitates the continued administration of the drug to prevent the appearance of withdrawal or abstinence symptoms.

Physical dependence is induced when a high blood alcohol concentration (BAC) is maintained. Humans can metabolise alcohol at a rate of 15 mg/100 ml per hour. This is equivalent to approximately 8 g of alcohol (one standard drink). When this rate is exceeded, dependence may be induced in a few days. Withdrawal phenomena occur with abstinence, or even after a relative decrease in BAC (e.g., from 0.30 to 0.10 g%). This may occur with changes in drinking pattern over a 24 hour period, or a decrease in total daily intake.

Tolerance develops after the repeated administration of a given dose of the drug produces a decreased effect, or when increasingly larger doses must be administered to obtain the effect observed with the original dose. Individuals tolerant to alcohol may show cross tolerance to sedatives and anaesthetics. For tolerant individuals, however, there is no marked elevation of the lethal dose, so that respiratory depression may occur with severe alcohol intoxication. At higher BACs the effects of other sedatives and hypnotics may be additive (Dorian et al., 1983).

In patients with drinking problems, the clinical manifestations of tolerance and dependence generally occur together. To establish tolerance the patient would have to have established high BACs, and this may cause dependence. In exceptional cases, however, the manifestations of tolerance may occur without those of dependence, and, vice versa, the patient may be dependent without clinical signs of tolerance. In some patients tolerance is lost at a late stage in their drinking career.

1.1 Physical and psychological dependence

In 1965 the World Health Organisation proposed that the term 'dependence' replace the commonly used terms 'addiction' and 'habituation'. Dependence was defined as:

> "A state, psychic, sometimes also physical, resulting from the interaction between a living organism and a drug, characterised by behavioural and other responses that always include a compulsion to take the drug on a continuous or periodic basis, in order to experience its

> psychic effects, and sometimes to avoid the discomforts of its absence". (Royal College of Psychiatrists, 1986, p. 52)

A distinction was then made, in the same document, between psychological dependence and physical dependence. Psychological dependence was defined as:

> "A condition in which a drug produces a feeling of satisfaction, and a psychic drive that requires periodic or continued administration of the drug to produce pleasure and avoid discomfort." (Royal College of Psychiatrists, 1986, pp. 52-53).

Physical dependence was defined as:

> "An adaptive state that manifests itself by intense physical disturbance when the administration of the drug is suspended." (Royal College of Psychiatrists, 1986, p. 53).

1.2 Alcohol dependence syndrome

Rather than attempt to sharply distinguish between psychological and physical dependence, a clinical syndrome, the alcohol dependence syndrome (or other drug dependence syndrome) was proposed (Edwards, Gross, Keller, Moser & Room, 1977). This is a clinical syndrome description, not an explanation in biological terms of the elements of the syndrome. The syndrome may exist in degrees, it is not an 'all or none' phenomenon. The age at which the dependence syndrome presents is often in the mid 40's for men and women (Royal College of Psychiatrists, 1986), but it is being seen with increasing frequency at an earlier age. It may develop over six months, and can be diagnosed by the application of clinical questionnaires (e.g., CAGE, MAST (Saunders, in press)).

The syndrome can be identified by seven clinical conditions and signs:

a) a subjective awareness of a strong desire to drink

b) narrowing of the drinking repertoire (i.e., as dependence proceeds the drinking schedule becomes stereotyped)

c) primacy of drinking over other activities (i.e., it becomes a dedicated occupation)

d) altered tolerance to the drug; the tolerance usually increases in the early stages of a drinking career, but declines in the later stages of dependence

e) repeated withdrawal symptoms; these are mild in early cases (e.g., 'bad nerves', shakiness, sweating, and nausea). In extreme withdrawal delirium, tremor, and seizures may occur. Any circumstance which suddenly leads to a reduction of intake or complete abstinence may precipitate withdrawal

symptoms. Admission to hospital, or arrest and imprisonment are typical circumstances in which delirium or fits may develop

f) relief of withdrawal symptoms by further drinking, and

g) reinstatement of the drinking pattern after abstinence; this may occur rapidly and explosively in the severely dependent, but more gradually in the less dependent.

1.2.1 Alcohol-related disability

An important corollary of the alcohol dependence syndrome is the consideration of alcohol-related disability (Edwards et al., 1977). An alcohol-related disability is an impairment in the physical, mental, or social function of the individual, which may be inferred as being at least in part due to alcohol. Persons who experience impairment or disability related to alcohol may or may not also be suffering from the dependence syndrome. Dependence may advance, but the disabilities may not develop at the same pace.

Constitutional and genetic factors may predispose to alcohol -related disability (Pols & Hawks, 1987), and the development of the alcohol dependence syndrome. Females are more susceptible than males because of lower muscle bulk and distribution of body water. Males with a family history of other male alcohol dependence are more prone to develop severe and early dependence.

The patterns of consumption and the associated use of other drugs (e.g., tobacco and analgesics), may also predispose to alcohol-related damage. Alcohol-related liver disease has been shown to be due to both volume and duration of alcohol consumption, even at levels of 40-60 gms per day. The situation in which alcohol is consumed (e.g., work, car driving, or water environment), may also predispose to mortality and damage.

1.3 Cross dependence

One sedative drug may suppress the manifestations of physical dependence produced by another, and help maintain the physically dependent state (Jaffe, 1985). The degree of cross dependence between drugs is due more to similarities in pharmacological effects than to chemical similarities. In general, most sedatives and hypnotics show a reasonable degree of cross dependence with each other, and with alcohol and barbiturates. Withdrawal symptoms occurring after the use of drugs having a long half life (e.g., diazepam) are generally less severe, though more protracted. This is the basis for the use of diazepam in the treatment of alcohol withdrawal. Patients who have developed physical dependence may more easily tolerate the central nervous system effects of higher BACs (Pols & Hawks, 1987).

2. The pattern of drinking in Australia

The pattern of drinking is determined by the interaction of social and cultural factors, attitudes, availability, and the laws. Drinking is learned in the pattern of family drinking. In New South Wales (Baker, Homel, Flaherty & Trebilco, 1987), 91% of males and 86% of females have already consumed alcohol at some time by the age of 17. By mid-adolescence more than half had consumed alcohol within the month before interview, and up to a half had consumed to intoxication at least once a fortnight. Aboriginal school children also start drinking at an early age (Williams, 1987). This study reported that drinking had started at the age of 9, a substantial number were drinking regularly by the age of 13, and schoolchildren had ready access to alcohol either from their family or from the community. Although this study may not be representative of the drinking patterns of Aboriginal children throughout New South Wales or other states, Kamien (1986) gives a similar account. Hamilton (1985) has reported on the drinking of school populations and their families in Mount Isa and Frankston, and the pattern there is consistent with these findings.

2.1 Alcohol consumption

The Commonwealth Department of Health Survey (1987) of the population 14 years and over showed that only 12% of males and 19% of females did not drink. Australia still ranks 13th in the world in per capita consumption of alcohol (9.4 litres per year), the highest in the English speaking world. Between 1976 and 1986 there has been a marked increase in the proportion of alcohol consumed as table wine, and a decline in the consumption of beer. Pols and Hawks (1987) have described the Australian norms and attitudes as toleration of heavy drinking, reinforced by peer group pressure. There are social and other rewards for drinking which also reinforce drinking behaviour.

2.2 Prevalence of risky drinking

Overall 64% of males and 42% of females in Australia drink weekly, or more often. Given the early age at which drinking starts, the patterns of drinking in school children, and the overall patterns of regular drinking in adults, the risks of dependence and alcohol-related disabilities are apparent. The National Heart Foundation (1983) reported that the drinking behaviour of 9.5% of men and 5.8% of women placed them in the medium to very high risk group. In another study of alcohol consumption in a work place group in New South Wales (Schlosser & McBride, 1984), 20% of the workforce were in the problem to high risk category. The population at an alcohol clinic in Melbourne (Wilkinson, Santamaria & Martin, 1969) had started to drink regularly in their late teens (around the age of 18), excessively by their mid-20s (around age 26), and presented to the clinic around the age of 45. They had had a period of moderate drinking lasting 7 years, and a period of excessive drinking averaging 16 years. The women started to drink later, and drank excessively for a shorter period before presenting for help at about the same

age as the men. By the time they presented to the clinic men were consuming 220 ± 100 g alcohol daily, and the women were consuming 155 ± 55 g alcohol per day. In Sydney 80% of patients referred to the alcohol clinic at a general hospital (Burns, 1980) were drinking more than 120 g a day. In the general wards of the same hospital (Williams, Burns & Morey, 1978) the drinking habits of 30% of males and 9% of females placed them at risk.

3. Alcohol withdrawal

The development of alcohol withdrawal varies from patient to patient, and even from one occasion to another for the same patient. It depends upon the duration and intensity of the preceding alcohol consumption, the severity of dependence, and the patient's use of other sedative drugs. The withdrawal symptoms are likely to occur from 6 to 24 hours after the last drink. Mild reactions which may last up to 48 hours include insomnia, irritability, tremor, and sweating. Purposeful behaviour directed towards getting another drink, or a sedative, may occur. Hyperreflexia may be present. Hallucinosis may occur during acute intoxication, or as acute alcoholic hallucinosis during withdrawal. (Chronic alcoholic hallucinosis refers to auditory hallucinations occurring in the absence of withdrawal, and persisting for periods of up to months. The diagnostic problem here is to differentiate between alcoholic hallucinosis and paranoid schizophrenia.) Phenothiazines and butyrophenones may have anti-hallucinatory activity in withdrawal, and in alcoholic hallucinosis; however, they may lower the seizure threshold (Devenji & Saunders, 1986; Saunders, in press).

In severe withdrawal the earliest symptoms progress to auditory, visual, and tactile hallucinations, seizures, or delirium. The likelihood of developing severe withdrawal symptoms is increased when the patient has concurrent infections or medical problems, a previous history of seizures or delirium, and after more frequent and heavier drinking. Stress responses during withdrawal include tachycardia, sweating, hypertension, and fever. Delirium tremens has a significant morbidity and mortality. Mortality risk for severe withdrawal is less than 5% (Schuckit, 1987).

Seizures during withdrawal are typically grand mal (major fit), non-focal, one or two in number, and occur 12-60 hours after the last drink. The timing of seizures may be altered by polydrug use (e.g., alcohol and benzodiazepines). Seizures require treatment if they are repeated, continuous, or life threatening. There is no certainty about the therapeutic and prophylactic value of phenytoin for alcohol withdrawal seizures. For most patients benzodiazepines alone are suitable. Patients with a previous history of fits, or idiopathic epilepsy, will be given anti-convulsants, continued for a variable period depending on the eventual diagnosis (e.g., organic nervous disease). Anti-convulsants will usually not be continued for alcohol withdrawal seizures alone. For alcohol/narcotic withdrawal, or alcohol/benzodiazepine withdrawal, regimens using diazepam

alone or diazepam and clonidine are safe and effective. For alcohol/barbiturate withdrawal, phenobarbitone may be the drug of choice.

4. Alcohol-related medical problems

The medical problems associated with alcohol and drug use are numerous and often complex. Patients need to be carefully assessed, as associated medical illness may alter the approach to detoxification, as well as demand particular medical or surgical managements in a medical setting. Many of the medical disorders are thought to be related to multiple nutritional deficiencies. These are, in turn, the result of the capacity of alcohol to depress appetite while supplying calories, without vitamins, minerals, or amino acids. Such disorders include peripheral neuropathy, pellagra, nutritional amblyopia, and Wernicke-Korsakoff syndrome. The Wernicke components (confusion, ataxia, and ophthalmoplegia) may go unrecognised in subclinical form. In the most severe form the mortality may be as high as 20% (Victor & Martin, 1987). In Australia Wernicke's encephalopathy is underdiagnosed and the incidence appears to be increasing (Harper, 1983). Korsakoff psychosis will be manifested by severe recent memory impairment and confabulation. Thiamine deficiency has a prominent role in the aetiology of Wernicke-Korsakoff syndrome. The Wernicke components usually respond promptly to thiamine, the Korsakoff components less so. The injudicious use of glucose or dextrose, either orally or intravenously without thiamine supplementation, may precipitate or aggravate the Wernicke-Korsakoff syndrome in a recovering or withdrawing patient. In patients with long standing alcohol abuse chronic organic brain syndromes varying from minimal cognitive and intellectual impairment through to severe dementia may make assessment difficult. There is now both good scientific and popular support for thiamine supplementation in food and alcoholic beverages (Royal Australasian College of Physicians, 1988).

Surgical conditions are more likely to occur in the alcoholic population than in controls (e.g., subdural haematoma after trivial head injury). Other disorders, such as alcoholic liver disease, alcoholic cardiomyopathy, skeletal muscle disease, pancreatitis, and alcoholic cerebellar degeneration are thought to be due to a direct toxic effect of alcohol itself. Some of these disorders may either fully or partially resolve with abstinence. Sudden deaths in middle aged males may have resulted from a long history of harmful drinking, even though definite pathology was not demonstrable post mortem (Petersson, 1988). Possible reasons for sudden death might be a seizure, hypoglycaemia, or cardiac arrhythmia. The use of the common laboratory markers MCV and GGT to identify patients with harmful consumption (Saunders, in press) should be better understood and more readily applied.

5. Treatment

5.1 Proclaimed places

Many people sober up and withdraw from alcohol at home or in other places, either on their own, or cared for by relatives or friends. The proclaimed place (New South Wales Bureau of Crime Statistics and Research, 1980) was designed to provide care during the initial period of recovery from drinking. A police cell is not designed to provide a proper standard of shelter and care for sobering up.

5.2 Detoxification

Detoxification is the management of withdrawal so that the symptoms of withdrawal are minimised and the risk of delirium tremens is reduced. Detoxification can be carried out at home or in a hospital, either in a medical or non-medical setting (Burns, Flamer, Morey & Novak, 1983). Patients who have no history of severe withdrawal or signs of severe withdrawal or medical illness, who have a suitable home environment and no access to alcohol, are suitable for home care. For milder cases non-medical units are suitable as anxiety, agitation, and even hallucinations are lessened or prevented by supportive care in a calm environment. Psychiatric symptoms such as depression may be merely part of withdrawal, and not necessarily attract ongoing psychiatric treatment (Blankfield, 1986). Pedersen (1988) has shown that non-medical detoxification is a safe and resource efficient treatment for patients assessed as appropriate. The non-medical unit will be staffed by non-medical personnel.

Patients who have a history of severe withdrawal, the signs of marked withdrawal, medical or psychiatric disorders, or unfavourable domestic situations, should be admitted to care. Medical detoxification requires medical and surgical staff, and careful monitoring of the withdrawal state.

5.3 Post detoxification treatment

Basic assumptions about treatment include the need for assessment, identification of goals, continuing care and relapse management, and the availability of detoxification services (Royal College of Psychiatrists, 1986). Special treatment modes using either inpatient or outpatient care, may be applied in particular cases. Patients with long standing alcohol-related disabilities including brain damage, and who have severe dependence, may require nursing home or hostel accommodation, or even long term placement in psychiatric hospitals (Saunders, in press).

The view that the only acceptable goal is total abstinence for alcoholics (patients with moderate to severe dependence) is the key note of the Alcoholics Anonymous philosophy, and current among many therapists. As an alternative to total abstinence, the place of controlled drinking for problem drinkers has been debated for 25 years, and fully reviewed by Heather and Robertson (1980).

5.4 Outcomes

Long term studies of the natural history of alcoholism (Vaillant et al., 1983), and long term outcome studies after treatment (Edwards, Duckitt, Oppenheimer, Sheehan & Taylor, 1983) have shown that few patients with severe long standing dependence will be able to return to asymptomatic drinking. Patients with severe symptomatic dependence, however, often achieve stable abstinence. Edwards et al. (1983), followed a group of 68 'alcoholic' males and found that after 10 years 8 were successful social drinkers. These patients never had severe dependence.

Overall, 40% of the patients followed by Edwards et al. (1983) were doing well at 10 years. Polich, Armor and Braiker (1981) reported on a 4 year follow up of patients in eight alcohol treatment centres in the U.S.A.. While 7% abstained for less than 1 year, and 21% abstained for 1 year or more, only 7% of the total sample abstained for the whole of the 4 year follow up period. Eighteen per cent of the group were drinking without problems. Those with dependent symptoms at 18 months (even with low levels of dependence), however, were more likely to experience adverse consequences of drinking, for example, continued dependence, health problems, and even death. There was no difference in outcome between inpatient and outpatient treatments at four years. Patients under the age of 40 with low levels of dependence on admission, who were also engaged in non-problem drinking, had lower relapse rates than those who abstained.

6. Early intervention

Studies of minimal intervention for patients early in their drinking careers have yielded promising results (Chick, Lloyd & Crombie, 1985; Kristenson, Ohlin, Hulten-Nosslin, Trell & Hood, 1983; Wallace, Cutler & Haines, 1988). Chick et al. (1983) showed that brief intervention for problem drinkers in medical wards resulted in a reduced alcohol consumption after 12 months. This assumes that intervention will be more effective early in the drinking career. Babor, Ritson and Hodgson (1986) concluded that low intensity brief interventions had much to recommend as initial approaches to problem drinking in primary care settings. Heather (1988) argued that intervention with controlled drinking as the goal had important potential applications at each level of prevention.

7. Summary

(i) Tolerance and dependence are related phenomena, and can be identified by a clinical examination.

(ii) The alcohol dependence syndrome is described.

(iii) Drinking patterns in the Australian community place many persons at risk of developing tolerance and dependence.

(iv) Requirements for the management of sobering up and withdrawal are discussed.

(v) The presence of dependence is an important factor determining the goal and outcome of treatment.

(vi) Alcohol-related disabilities also determine the setting in which treatment is carried out, and may influence the immediate and long term outcome.

References

Babor, T. F., Ritson, E. B., & Hodgson, R. J. (1986). Alcohol related problems in the primary health care setting: A review of early intervention strategies. British Journal of Addiction, 81, 23-46.

Baker, W., Homel, P., Flaherty, B., & Trebilco, P. (1987). The 1986 survey of drug use by secondary school students in New South Wales. Sydney: New South Wales Drug and Alcohol Authority.

Blankfield, A. (1986). Psychiatric symptoms in alcohol dependence: Diagnostic and treatment implications. Journal of Substance Abuse Treatment, 3, 275-278.

Burns, F. H. (1980). An alcohol clinic in a general hospital. Paper presented at the Autumn School of Studies on Alcohol and Drugs, St. Vincents Hospital, Melbourne.

Burns, F. H., Flamer, H. E., Morey, S., & Novak, H. (1983). The Royal Prince Alfred Hospital detoxification unit. Australian Alcohol and Drug Review, 2, 23-25.

Chick, J., Lloyd, G., & Crombie, E. (1985). Counselling problem drinkers in medical wards: A controlled study. British Medical Journal, 290, 965-967.

Commonwealth Department of Health. (1987). Alcohol: The facts. Canberra: Australian Government Publishing Service.

Devenyi, P., & Saunders, S. (1986). Physicians handbook for medical management of alcohol and drug related problems. Toronto: Addiction Research Foundation.

Dorian, P., Sellers, E. M., Reed, K. L., Warsh, J. J., Hamilton, C., Kaplan,H. L., & Fan, T. (1983). Amitriptyline and ethanol: Pharmacokinetic and pharmacodynamic interaction. European Journal of Clinical Pharmacology, 25, 325-331.

Edwards, G., Duckitt, A., Oppenheimer, E., Sheehan, M., & Taylor, C. (1983, July 30). What happens to alcoholics? Lancet, 269-271.

Edwards, G., Gross, M. M., Keller, M., Moser, J., & Room, R. (1977). Report of a WHO group of investigators on criteria for identifying and classifying disabilities related to alcohol consumption. In G. Edwards, M. M. Gross, M. Keller, J. Moser & R. Room (Eds.), Alcohol-related disabilities (WHO Offset Publication No. 32, pp. 5-22). Geneva: World Health Organization.

Hamilton, M. (1985). The social environment perspective: Young people's drinking. Paper presented at the Autumn School of Studies of Alcohol and Drugs, St. Vincents Hospital, Melbourne.

Harper, C. (1983). The incidence of Wernicke's encephalopathy in Australia - a neuropathological study of 131 cases. Journal of Neurology, Neurosurgery and Psychiatry, 46, 593-598.

Heather, N. (1988). The role of controlled drinking goal in the prevention of alcohol problems. Paper presented at the 8th Annual Conference of the Australian Medical and Professional Society on Alcohol and Drugs, Hobart.

Heather, N., & Robertson, I. (1980). Controlled drinking. London: Methuen.

Jaffe, J. (1985). Drug addiction and drug abuse. In A. G. Gilman, L. S. Goodman, T. W. Rall & F. Murad (Eds.), The pharmacological basis of therapeutics (7th ed., pp. 532-581). New York: Macmillan Publishing Company.

Kamien, M. (1986). Alcohol and drug problems in minority groups. Australian Drug and Alcohol Review, 5, 59-61.

Kristenson, H., Ohlin, H., Hulten-Nosslin, M., Trell, E., & Hood, B. (1983). Identification and intervention of heavy drinkers in middle-aged men : Results and follow up of 24-60 months of long term study with randomised controls. Alcoholism: Clinical and Experimental Research, 203-209.

New South Wales Bureau of Crime Statistics and Research (1980). Intoxicated persons statistical collection. August, 1980.

Pederson, C. M. (1988). Non-medical alcohol detoxification six years on. Paper presented at the 8th Conference of the Australian Medical and Professional Society on Alcohol and Drugs.

Petersson, B. (1988). Analysis of the role of alcohol immortality, particularly sudden unwitnessed death, in middle aged men in Malmo, Sweden. Alcohol and Alcoholism, 23, 259-263.

Polich,. M., Armor, D. J., & Braiker, H. B. (19) The course of alcoholism four years after treatment. New York: Wiley and Sons.

Pols, R., and Hawks, D. (1987). Is there a safe level of daily consumption of alcohol for men and women? Canberra: Australian Government Publishing service.

Royal Australasian College of Physicians (1988). Filer 1416.

Royal College of Psychiatrists. (1986). Alcohol: Our favourite drug. New report on alcohol and alcohol-related problems from a special committee of the Royal College of Psychiatrists. London: Tavistock Publications.

Saunders, J. B. (in press). The management of alcohol problems. In P. J. B. Beumont, R. Hampshire & R. Meares (Eds.), An Australian textbook of psychiatry. Blackwell.

Schlosser, D., & McBride, W. (1984). Estimating the prevalence of alcohol and other drug related problems in industry. Sydney: New South Wales Drug and Alcohol Authority.

Schuckit, M. (1987). In Harrison's principles of internal medicine (11th ed., p. 2110). McGraw-Hill.

Vaillant, G. E., et al (1983). Prospective trial of alcoholism treatment eight year follow up. American Journal of Medicine, 75, 455.

Victor, M., & Martin, J. B. (1987). In Harrison's principles of internal medicine (11th ed., p. 2002). McGraw-Hill.

Wallace, P., Cutler, C., & Haines, A. (1988). Randomised controlled trial of general practitioner intervention in patients with excessive alcohol consumption. British Medical Journal, 297, 663-668.

Wilkinson, P., Santamaria, J., & Martin, D. (1969, May 17). Epidemiology of alcoholism : Social data and drinking patterns on a sample of Australian alcoholics. Medical Journal of Australia, 1020-1025.

Williams, A. T., Burns, F. H., & Morey, S. (1978). Prevalence of alcoholism in a Sydney teaching hospital : Some aspects. Medical Journal of Australia, 2, 608-611.

Williams, M. (1987). The 1986 survey of drug and alcohol use by Aboriginal school students in New South Wales. Sydney: N.S.W. Aboriginal Education Consultative Group.

Chapter 7. Alcohol-Related Brain Damage

Martin Jackson

1. Introduction

Acute alcohol intoxication is characterised by slurred speech, nausea, dizziness, unsteadiness of gait and changes in behaviour and can progress to stupor and coma. The effects of chronic alcohol abuse (> 130 g per day) on the brain are well documented (Butters & Cermak, 1980; Parsons & Leber, 1982; Ryan & Butters, 1980; Tarter, 1973, 1980; Walsh, 1983, 1985) and have been divided into two major syndromes:

i. The Korsakoff amnesic syndrome
ii. Non-amnesic cognitive deficits

Both syndromes of deficits frequently occur in the same individual; however, the two syndromes of impairment are often dissociated and it is generally accepted that they probably arise from two different aetiologies.

2. The Korsakoff amnesic syndrome

The Korsakoff amnesic syndrome is thought to be caused by thiamine deficiency resultant from poor nutrition. The amnesic syndrome may develop with or without a prior florid episode of Wernicke's encephalopathy. Neuropathological evidence suggests that the crucial structures of the brain involved are the mamillary bodies and the dorso-medial nucleus of the thalamus (Victor, Adams, & Collins, 1971).

The clinical features of the Korsakoff amnesic syndrome are as follows:

i. The person's immediate memory span and their ability to interact in their immediate environment is intact.

ii. The person experiences a profound difficulty learning new material. It is a general amnesic syndrome and occurs for all types of material presented through any sense modality. Information which appears to have been learned cannot be remembered after only a short period of time. As nothing new is being learned, an increasing period of anterograde amnesia develops and the person may appear to be stuck in time.

iii. The person has a retrograde amnesia characterised by difficulty with spontaneous recall of past events. The amount of information the person can remember decreases as questions move closer in time to the present and much more information can be obtained by direct questioning, rather than by free recall.

Information loses time tags and the person has difficulty remembering when events have occurred and their temporal relations to other events. Memories of childhood and early adult life are often less affected.

iv. The person may be confused and disorientated and may be confabulating. Confabulation has been defined by Berlyne (1972) as "a falsification of memory occurring in clear consciousness in association with an organically derived dementia" (p.). Confabulation may be present in the acute amnesic state, but it is rarely a picture of the chronic amnesic state.

v. Many aspects of learned behaviour, such as speech, language and well practised skills, are preserved. The person will be able to cope adequately in old and familiar surroundings, but will get into difficulties in unfamiliar surroundings where they need to learn new information.

vi. The person may exhibit a lack of spontaneity, together with a blunting of affect. This symptom is usually seen in severely damaged alcoholics and can lead to the diagnosis of a burnt-out alcoholic or alcoholic dementia.

2.1 Behavioural features of the Korsakoff amnesic syndrome

i. Mild impairment
The person may have noticed some minor problems with memory, particularly if they are in a demanding job. No one else is likely to notice that anything is wrong.

ii. Moderate impairment
Forgetfulness will have been noticed by the person and by others. They will be experiencing difficulties at work and may be failing badly in a high powered job. They will have considerable difficulty learning new material, but they will usually be able to eventually do so after considerable repetition.

iii. Moderate/severe impairment
The person will be unable to hold down anything but the simplest of jobs unless they have been in it for years. They may still be able to learn some new material, but this will be very limited.

iv. Severe impairment
The person will be unable to hold down any sort of job. They will usually be disorientated in time and place and may confabulate. The person may be able to repeat immediately what you have said to them but will forget it after only a short period of time, maybe only a few minutes.

3. Non-amnesic cognitive deficits

The second major set of cognitive changes that are seen in alcoholics are the non-amnesic cognitive deficits. As well as occurring commonly with the Korsakoff amnesic syndrome, these cognitive deficits also occur in alcoholics who have adequate nutrition and have not developed an amnesia (Lynch, 1960). The cause of these deficits is still unknown, but evidence suggests that they may be due to direct neurotoxic effects of alcohol or its metabolic products on the brain, rather than to thiamine deficiency resulting from poor nutrition (Harper, 1986, 1988). They are commonly reported in the cases of 'well nourished alcoholics'. The area of the brain which is reported to be impaired is the anterior frontal lobes. Neuropathological evidence is now available which shows a large loss of neurons in the anterior frontal lobes when compared to other areas of the brain (Harper, 1988). The psychological functions of concern here are general intelligence and adaptive behaviour.

3.1 General intelligence

It is well reported that alcoholics generally do not show a decline in IQ scores on standard intelligence tests (Parsons, 1974). These data have been used to support arguments that there is not a generalised intellectual deterioration. However, standard IQ tests have been shown to be relatively insensitive to detecting subtle cognitive changes or changes in the person's ability to adapt to new situations.

3.2 Adaptive behaviour

For behaviour to be effective a person must be able to create goals and intentions, form plans of action, carry out and monitor ongoing actions, analyse the effectiveness of an action and make any necessary adjustments. Chronic alcohol abuse causes a gradual destruction of this adaptive behaviour process (Ron, 1977). The person experiences difficulties with planning and organisation, they are unable to recognise the errors they are making and they are unable to learn from them. The person has difficulty conceptualising and forming anticipatory goals. Their thinking becomes inflexible and they cannot follow logic or see more than one point of view. A gradual lack of awareness of these deficits develops. The person does not perceive that anything is wrong and therefore, having inappropriate feedback, they lack self criticism and concern about the consequences of their actions. Their behaviour becomes inappropriate. The person may also experience a blunting of affect and a lack of initiative and spontaneity. This inability to adapt to new situations has been called an adaptive behaviour syndrome (Walsh, 1985).

New learning and memory can also be affected, but in a different way to the Korsakoff amnesic syndrome (Walsh, 1985). The person has difficulty learning new information because of their inefficient planning and organisational abilities; there is a disruption of the intellectual process

of organisation for the purpose of committing material to memory. Whilst this person has difficulty learning there is relatively little loss of material after delay. These people often complain of having memory problems. Since there is preservation of old skills, general intelligence, remote memories and generally intact social skills, the person may appear to be normal at a conversational level, especially if they are in a familiar environment such as their home. However, once placed in an unfamiliar environment they lack the adaptive skills necessary to be able to cope. For the well concealed alcoholic this may not occur until there is a major change in life, such as job loss and trying to find re-employment.

3.3 Behavioural consequences of frontal lobe damage

i. Mild impairment
The person may have noticed problems with organisation, particularly in a high powered job, although they are more likely to report having a memory problem. It is unlikely that any of these problems would be noticed by anyone else.

ii. Moderate impairment
The person experiences difficulties solving new problems and making new plans. They are liable to get flustered and anxious when presented with problems. The person may be aware of what is needed and may be able to tell you what the problem is, but is unable to do anything about it. The person may seem a bit concrete in their thinking and their disorganisation may be apparent to others.

iii. Moderate/severe impairment
The person is unable to plan or organise anything complex. They do not learn from their mistakes and keep repeating them no matter how catastrophic the results. They have poor insight and appear very concrete and rigid. They need structure provided by others to be able to survive and this is often provided by a family member or friend to compensate for the disability.

iv. Severe impairment
The person will lack insight, and be concrete and rigid in their thinking. They often perseverate and have major problems shifting from one idea to another. They may be unable to organise themselves even in simple routine tasks. They need their lives to be completely structured and supervised by others. The person may also have difficulties with disinhibition and amotivation.

4. Social drinking

The previous sections illustrate the cognitive impairment which occurs with heavy chronic drinking. In recent years it has been suggested that light drinking, as little as two or

three drinks per occasion, causes cognitive impairment. (Cala et al., 1983; Parker & Noble, 1977, 1980; Parsons, 1986). The type of impairment is similar to that found in well nourished chronic alcoholics but is milder in severity. Thus, people have claimed that almost any intake of alcohol may result in measurable brain damage.

Common estimates of safe drinking levels in males are between 40 to 50 g per day (four to five standard drinks) and 20 to 30 g for women. Most research has relied on small, but significant, negative correlations between drinking and test variables to support its claims. However, the correlations rarely account for more than 4% of the variance, which renders them practically meaningless. Attempts to replicate these findings have not been convincing and a comprehensive review of the social drinker literature found no evidence for low levels of alcohol consumption causing cognitive impairment (Bowden, 1987; Bowden, Walton & Walsh, 1988). This result has been supported by Waugh, Jackson, Fox, Hawke and Tuck (in press) who found no evidence of cognitive impairment in healthy male subjects drinking up to 80 g of alcohol per day. However, Waugh et al. found mild impairment of the frontal lobe type in subjects drinking between 80 and 130 g of alcohol per day. These latest two studies lead to the conclusion that small levels of alcohol intake probably do not cause cognitive impairment and strengthen the hypothesis that cognitive impairment may be due to a threshold effect rather than a dose-related effect.

5. Recovery of function

Neuroradiological studies have found reversibility of brain shrinkage following prolonged periods of abstinence from alcohol (Carlen, 1979). Younger alcoholics and those with the shortest drinking history showed the greatest degree of improvement.

Improvement of cognitive function during the first two weeks of abstinence is well established (Allen, Faillace, & Wagman, 1971; Burdick, Johnson, & Smith, 1970).

Partial improvement in the cognitive functioning of alcoholics has been reported after intervals of six months to four years abstinence (Schau, O'Leary, & Chaney, 1980). However, little or no recovery in cognitive function after prolonged periods of abstinence has been found by others (Yohman, Parsons, & Leber, 1984). Jackson, Fox, Waugh, and Tuck (1987) found that alcoholics with evidence of neurological disease (cerebellar ataxia and peripheral neuropathy) had more severe cognitive impairment than alcoholics without evidence of neurological disease. Furthermore, once there is evidence of neurological disease the probability of recovery of cognitive function is reduced.

6. Other medical problems

The Regional Brain Damage Unit at Royal Prince Alfred Hospital in Sydney is currently conducting a retrospective study of 340 consecutive patients referred for suspected alcohol-related brain damage. Wernicke's encephalopathy (4%) and Korsakoff syndrome (9%) were rarely diagnosed. Symptoms of alcohol-related neurological disease were found in just under half of the subjects (cerebellar ataxia 47%, neuropathy 40%). Other common medical problems were a past history of head injury (26%), liver disease (19%), and seizures (12%). Other neurological diseases were found in 20% of the patients and other medical problems were found in 32% of the patients. Males drank at higher levels than females and were more likely to have cerebellar ataxia and a past head injury. Older subjects drank at the same level as younger subjects, but were far more likely to have ataxia, neuropathy, seizures, Korsakoff syndrome, liver disease, a history of head injury and other medical problems. These figures indicate that a large proportion of the alcoholic population is likely to have one or more significant medical problems. Another important finding from the study is that the vast majority of patients had significant cognitive impairment (95%). This indicates that cognitive impairment is likely to occur well before any clinically significant medical symptoms of alcohol-related problems arise.

7. Sex differences

Sex differences have been found in the rate of ethanol metabolism and evidence suggests that there are differences between males and females in susceptibility to the toxic effects of ethanol (Acker, 1986; Jacobson, 1986).

Most neuropsychological research has used male alcoholics as subjects. Studies which have investigated differences in performance between male and female subjects tend to report that male alcoholics perform more poorly than female alcoholics (Fabian, Parsons, & Sheldon, 1984). However, the male subjects have usually drunk a significantly larger amount of alcohol for a significantly longer period of time and no attempts have been made to control for these variables. Therefore, the findings in this area are inconclusive.

8. Effects due to ageing

Grant, Adams and Reed (1984) suggested that ageing- and alcohol-related neuropsychological deficits could interact in the following ways:

i. Older alcoholics have had more time to drink and might suffer more of the cumulative effects of ethanol.

ii. The ageing brain may be more sensitive to ethanol neurotoxicity.

iii. The ageing brain might take longer to recover from an alcoholic insult.

iv. Prior alcoholism might advance the effect of ageing, whether a person continues to drink or not.

Research in this area has also been inconclusive. Some research has found that younger alcoholics are not impaired on cognitive tasks (Eckardt, Ryback, & Pautler, 1980). Other studies have found impairment on learning tasks (Bowden, 1988). Page and Cleveland (1987) found that age and alcohol consumption were both significant factors on test performance, but did not interact significantly. Reviews by Grant (1987) and Ryan and Butters (1984) concluded that current research does not favour a premature ageing explanation.

9. Conclusions

Chronic alcohol abuse can lead to two syndromes of cognitive impairment: 1. The Korsakoff amnesic syndrome. 2. An adaptive behaviour syndrome. Both syndromes of deficits occur frequently, but can be disassociated and probably arise from two different aetiologies. The Korsakoff amnesic syndrome is characterised by intact immediate memory, a profound difficulty learning new information, poor recall of recent events, confabulation and lack of spontaneity. The adaptive behaviour syndrome is characterised by poor planning and organisation, concrete and inflexible thinking, lack of insight, inappropriate behaviour and a lack of self-criticism. Whilst it has been suggested that drinking even as little as two or three drinks per day can cause cognitive impairment, recent evidence suggests that this is not the case. The latest research suggests that there is likely to be a threshold effect for alcohol-related brain damage rather than a dose effect relationship.

Improvement in cognitive functioning over the first two weeks of abstinence has been well established, but conflicting evidence exists as to the possibility of further improvement over a longer period of time. Age and the absence or presence of alcohol-related neurological disease are the important factors associated with recovery of function; improvement is more likely if the person is young and has no evidence of alcohol-related neurological disease. Although it has been shown that sex differences exist in the rate of ethanol metabolism and susceptibility to the toxic effects of ethanol, no conclusive evidence exists as to whether males or females are more prone to incurring alcohol-related cognitive impairment. Further well controlled research is required in this area.

Little evidence exists that there is a significant interaction between alcohol consumption and age, with both young and old alcoholics being impaired on tests of cognition. Recent reviews have concluded that current research does not favour a premature ageing explanation.

Head injuries, liver disease, cerebellar ataxia and peripheral neuropathy are the most common medical complications associated with alcoholism. However, a large proportion of alcoholics have other medical and neurological disorders. Older alcoholics are far more likely to exhibit symptoms of alcohol-related neurological disease, as well as other medical problems. Cognitive impairment is found in nearly all cases referred for suspected brain damage and this indicates that cognitive impairment is likely to result before any neurological or clinically significant medical symptoms of alcohol-related problems arise. Therefore, it is too late to wait until a medical problem arises until some intervention is attempted for a person with an alcohol problem.

References

Acker, C. (1986). Neuropsychological deficits in alcoholics: The relative contributions of gender and drinking history. British Journal of Addiction, 81, 395-403.

Allen, R. P., Faillace, L. A., & Wagman, A. (1971). Recovery of memory functioning in alcoholics following prolonged intoxication. Journal of Nervous and Mental Disease, 158, 417-423.

Berlyne, N. (1972). Confabulation. British Journal of Psychiatry, 120, 31-39.

Bowden, S. C. (1987). Cerebral deficits in social drinkers and the onus of proof. Australian Drug and Alcohol Review, 6, 87-92.

Bowden, S. C. (1988). Learning in young alcoholics. Journal of Clinical and Experimental Neuropsychology, 10, 157-168.

Bowden, S. C., Walton, N. H., & Walsh, K. W. (1988). The hangover hypothesis and the influence of moderate social drinking on mental ability. Alcoholism: Clinical and Experimental Research, 12, 25-29.

Burdick, J. A., Johnson, L. C., & Smith, J. W. (1970). Measures of change during alcohol withdrawal in chronic alcoholics. British Journal of Addiction, 65, 273-280.

Butters, N., & Cermak, L. S. (1980). Alcoholic Korsakoff's syndrome. New York: Academic Press.

Cala, L. A., Jones, B., Burns, P., Davis, R. E., Stenhouse, N., & Mastaglia, F. L. (1983). Results of computerised tomography, psychometric testing and dietary studies in social drinkers, with emphasis on reversability after abstinence. Medical Journal of Australia, 2, 264-269.

Carlen, P. L. (1979). Reversible effects of chronic alcoholism on the human central nervous system: Possible biological mechanism. In A. Wilkinson (Ed.), Cerebral deficits in alcoholism (pp. 107-119). Toronto: Addiction Research Foundation.

Eckardt, M. J., Ryback, R. S., & Pautler, C. P. (1980). Neuropsychological deficits in alcoholic men in their mid-thirties. American Journal of Psychiatry, 137, 932-6.

Fabian, M. S., Parsons, O. A., & Sheldon, M. D. (1984). Effects of gender and alcoholism on verbal and visual-spatial learning. Journal of Nervous and Mental Disease, 172, 16-20.

Grant, I. (1987). Alcohol and the brain: Neuropsychological correlates. Journal of Consulting and Clinical Psychology, 55, 310-324.

Grant, I., Adams, K. M., & Reed, R. (1984). Ageing, abstinence and medical risk factors in the prediction of neuropsychologic deficit among long-term alcoholics. Archives of General Psychiatry, 41, 710-718.

Harper, C. (1986). Pathological changes in alcoholic brain shrinkage. Medical Journal of Australia, 144, 3-4.

Harper, C. (1988). Brain damage and alcohol abuse: Where do we go from here? British Journal of Addiction, 83, 613-615.

Jackson, M., Fox, G. A., Waugh, M., & Tuck, R. R. (1987). A follow-up study in alcoholics with and without neurological disease. Australian Drug and Alcohol Review, 6, 71-76.

Jacobson, R. (1986). The contribution of sex and drinking history to the CT brain scan changes in alcoholics. Psychological Medicine, 16, 547-559.

Lynch, M. J. G. (1960). Brain lesions in chronic alcoholics. Archives of Pathology, 69, 342-353.

Page, R. D., & Cleveland, M. F. (1987). Cognitive dysfunction and ageing among male alcoholics and social drinkers. Alcoholism: Clinical and Experimental Research, 11, 376-384.

Parker, E. S, & Noble, E. P. (1977). Alcohol consumption and cognitive functioning in social drinkers. Journal of Studies on Alcohol, 38, 1224-1232.

Parker, E. S., & Noble, E. P. (1980). Alcohol and the ageing process in social drinkers. Journal of Studies on Alcohol, 41, 170-178.

Parsons, O. A. (1974). Brain damage in alcoholics: Altered states of consciousness. Alcohol Technical Reports, 2, 93-105.

Parsons, O. A. (1986). Cognitive functioning in sober social drinkers: A review and critique. Journal of Studies on Alcohol, 47, 101-114.

Parsons, O. A., & Leber, W. R. (1982). Alcohol, cognitive dysfunction and brain damage. In Biochemical and health consequences of alcohol use (Alcohol and Health Monograph No. 2). Washington, DC: U.S. Government Printing Office.

Ron, M. A. (1977). Brain damage in chronic alcoholism: A neuropathological, neuoradiological and psychological review. Psychological Medicine, 7, 103-112.

Ryan, C., & Butters, N. (1980). Further evidence for a continuum of impairment encompassing male alcoholics, Korsakoff patients and chronic alcoholic men. Alcoholism (NY), 4, 190-197.

Ryan, C., & Butters, N. (1984). Alcohol consumption and premature ageing: A critical review. In M. Galanter (Ed.), Recent developments in alcoholism. New York: Plenum Press.

Schau, E. J., O'Leary, M. R., & Chaney, E. F. (1980). Reversability of cognitive deficit in alcoholics. Journal of Studies on Alcohol, 41, 733-740.

Tarter, R. E. (1973). An analysis of cognitive deficits in chronic alcoholics. Journal of Nervous and Mental Disease, 157, 138-147.

Tarter, R. E. (1980). Brain damage in chronic alcoholics. In D. Richter (Ed.), Addiction and brain damage (pp. 267-297). London: Croom Helm.

Victor, M., Adams, R. D., & Collins, G. F. (1971). The Wernicke-Korsakoff syndrome. Davis, Philadelphia.

Walsh, K. (1983). Alcohol related brain damage: An hypothesis. Australian Drug and Alcohol Review, 2, 84-85.

Walsh, K. (1985). Understanding brain damage. Edinburgh: Churchill Livingstone.

Waugh, M., Jackson, M., Fox, G. A., Hawke, S. M., & Tuck, R. R. (in press). Neuropsychological performance in neurologically normal social drinkers. British Journal of Addiction.

Yohman, J. R., Parsons, O. A., & Leber, W. R. (1984). Lack of recovery in male alcoholics' neuropsychological performance one year after treatment. Alcoholism (NY), 9, 114-117.

Chapter 8. The Effects of Alcohol on Mood

Greg Chesher & Janet Greeley

1. Introduction

It is suggested by some that alcohol's most alluring feature is its ability to alter one's emotional state. Indeed, several theories of why people drink are based on the presumed mood-altering effects of alcohol (e.g., tension-reduction - Cappell & Greeley, 1987; stress response dampening - Sher, 1987). Although it is an accepted fact that alcohol influences mood, it is also well recognized that the assessment of changes in emotional state is difficult. By virtue of the very subjective nature of emotional states, it is necessary to rely upon self-report measures to evaluate them. Most studies that have examined changes in mood induced by the intake of alcohol have employed self-report scales such as the Mood Adjective Checklist. Some researchers have questioned the appropriateness of such scales in the measurement of drug-induced changes in mood (Lindman, 1985; Sjoberg, Svensson & Persson, 1979).

The relationship between alcohol use and affective or mood disorders such as depression is discussed briefly in Chapters 9 and 10. This chapter concentrates on research investigating immediate, short-term changes in mood state induced by acute alcohol consumption. The direction and magnitude of the changes in mood induced by alcohol are influenced by a number of pharmacological and nonpharmacological factors. Some of these are considered briefly here.

2. Pharmacological factors

Robbins and Brotherton (1980) claim there is almost a total lack of agreement among studies on the effects of alcohol on mood. This may be accounted for, in part, by a number of methodological issues that are often neglected in studies on the effects of alcohol on mood. For example, the BAC is often not assessed at the time of mood measurement (Robbins & Brotherton, 1980). Also, researchers seldom assess whether the person's BAC is on the ascending or descending limb (see Chapter 5, Section 4.1) of the BAC curve at the time of assessment (Freed, 1978). Both of these factors are important in the interpretation of the results.

Early research has shown that there are significant differences in mood on the ascending and descending limbs of the BAC curve (Kelly, Myrsten, Neri & Rydberg, 1970). Increased elation is reported on the ascending limb and reduced elation on the descending limb. It is possible that some of the confusion in the literature on the effects of alcohol on mood may be accounted for by researchers having assessed mood at different phases of the BAC curve.

Freed (1978) states that "[a]lcohol is at least a biphasic drug" (p. 177). This means that while you may see an increase in positive mood states at low doses, at high doses more negative mood states may be observed. A greater variety of doses need to be assessed to gain a better understanding of the dose-response relationship. Thus, some studies may report positive effects of alcohol on mood while others report negative effects. Without information about the BACs achieved in the different studies, these results are of little value.

The amount of experience an individual has had with alcohol use can influence the mood changes produced by this drug. The effects of alcohol on emotional states in both experienced and inexperienced drinkers will be considered briefly in Section 5.

3. Nonpharmacological factors

A number of nonpharmacological factors can influence the effect a dose of alcohol will have on mood. One such factor is the setting in which drinking takes place. Lindman (1982) found that drinking in the company of other people produced an increase in euphoria, while drinking alone did not increase euphoria, and was perceived as aversive.

Few studies have examined sex differences in the effects of alcohol on mood. In one study by Sutker, Tabakoff, Goist and Randall (1983), no differences were reported between the sexes in mood, perceived intoxication or physical sensations produced by alcohol, even though the females in this study achieved a higher BAC than the males. In a later study, Sutker, Goist, Allain and Bugg (1987) found that there was a sex difference in ratings of anxiety and depression when females consumed alcohol at the early follicular phase of the menstrual cycle. The effect was, however, a complex function of sex, limb of the BAC curve, and dose.

In a review on the psychological benefits of moderate alcohol consumption, Baum-Baicker (1985), referred to several other factors that reportedly affect the degree to which alcohol changes mood. These included: personality, race (Kastl, 1969), the degree of stress present (Levenson et al., 1980), and expectations. Expectations about alcohol will be considered further in Section 4.1.

4. Mood states in social drinkers

Wallgren and Barry (1970) reviewed studies which measured the effects of various doses of alcohol on nondependent human volunteers. Alcohol predominantly produces pleasant and exhilarated moods characterised by friendly behaviour with boisterous conversation at an increased volume. There is also a decrease in anxiety and feelings of tension. This is in contrast to what appears to be the change in mood state of alcohol dependent subjects (Mendelson & LaDou, 1964; Mendelson & Mello, 1966).

In a recent study involving normal volunteers (Chesher, Dauncey, Crawford & Horn, 1986), bipolar, horizontal analogue scales were used to assess several dimensions of mood states, before and after the consumption of alcohol and marijuana. The volunteers were asked to place a mark on a horizontal line to indicate where their present mood fitted between the poles of moods described by the adjectives at either end of the line. The study involved the assessment, on a blind basis, of four dosage conditions of alcohol (0; 0.25; 0.5 and 0.75 g/kg). The results were analysed to examine the linear trend across doses; that is to say, the dose-response relationship. Significant dose-dependent effects were recorded on measures which indicated significant changes in states described as 'alertness' (alert, clear-headed, attentive, bright), 'sociability' (sociable, involved, concerned, talkative) and 'self-assurance' (assertive, brave, confident, self assured). Alcohol produced a dose-dependent decrease in the 'alertness' scale and increases in the 'sociability' and 'self-assurance' scales. These effects are in accordance with earlier mood evaluations with alcohol (Wallgren & Barry, 1970). In this study, subjects were anticipating a dose of alcohol, but did not know that different dosage levels were to be used or that there was a placebo condition. There were between 4 and 8 subjects in the laboratory on each experimental session. This study did not involve the collection of data to assess subject's expectancies.

4.1 The effect of expectations about alcohol

Research has shown that what a person expects the effects of alcohol to be, can significantly influence the effect they report experiencing after drinking alcohol, or even after being led to believe that they have consumed alcohol. A study by Sher (1985) was designed to examine subjective effects of alcohol. The BAC achieved in this study was approximately 0.1 g%. The goal of the study was to examine the joint effects of setting and individual differences in the strength of alcohol expectancies on the subjective effects of alcohol. All subjects believed they were getting alcohol, though some, in fact, received a placebo drink. Subjects were required to drink alcohol in a setting where they were either alone or in a group of 3 to 5 subjects. The authors found:

> that the effect of alcohol on the subjective state is highly dependent on a number of factors including the time elapsed since the beverage consumption (i.e., the BAL curve), setting and individual differences in alcohol expectancies....The relative importance of each of these factors, and their interactions appears to be specific for the type of alcohol effect under consideration. For example, the effect of alcohol, relative to placebo, on the dimension of Pleasure appears to be short lived in comparison with the effects of alcohol on various measures of perceived physical sensations.

> Similarly, mood, particularly Pleasure and Dominance, appears to be more dependent on setting than are perceived physical sensations. (p.)

Significant Setting x Strength of Expectancy interactions were noted. The authors attributed this finding to the consideration that the expectancies held by most social drinkers are relatively specific to group situations.

4.2 Aggression

It is well documented that alcohol consumption is often associated with crimes of violence (Lanzcron, 1963; Pernanen, 1976; Roslund & Larson, 1979). It is also widely reported that alcohol use is associated with social violence. For example, Hobart (1978) found that the with economic development in the Northwest Territories in Canada the per capita alcohol consumption rose along with the number of criminal offences. Within recent years the association of alcohol consumption and violence among spectators at sporting events has received widespread media coverage, particularly in Great Britain. The volume of evidence linking alcohol with aggression or violence has led to an assertion that a pharmacological property of alcohol may serve as a releaser of aggressive behaviour. Unfortunately, as indicated by earlier reviewers of the subject (Carpenter & Armenti, 1972; Wallgren & Barry, 1970) and a review of the association between alcoholism and violence (Coid, 1982) there is a shortage of experimental evidence which examines the relationship between the dose of alcohol ingested and the measure of aggression recorded.

The ability to study aggression in the laboratory is also a confounding issue; most studies have used an experimental paradigm which involves an assessment of the willingness of an individual to deliver electric shocks to another person (Buss, 1961). In many cases the volunteer does not see the 'confederate' (i.e., the individual they believe they are supposed to shock) and in most cases there is none. The correlation between aggression as measured in this experimental paradigm and aggression in the 'real world' is difficult to assess. What has emerged from this research, however, is a picture which indicates that whilst alcohol is associated with aggression, the association is not a direct cause-effect pharmacological response. Interactions with perceived threat or provocation, as well as the expectancy of having consumed alcohol have been demonstrated to be important determinant factors in alcohol and aggression. These findings are well described in the studies of Lang, Goeckner, Adesso and Marlatt (1975). These workers employed a complex design to control for expectancy effects in a study of aggressive behaviour in 96 male undergraduate students who were social drinkers. Expectancy was controlled by issuing placebo or alcohol such that some volunteers received alcohol, expecting placebo; others expecting placebo received alcohol, whilst others received what they were expecting (told) they would receive. Following beverage administration half of the subjects were provoked to aggress by exposing them to an insulting confederate (who was to

receive the 'shock') whereas the control subjects experienced a neutral interaction. The only significant determinant of aggressive behaviour was the expectation factor. Only the subjects who believed they had consumed alcohol exhibited an increase in aggression, whether the beverage contained alcohol or did not. However, the subjects who received alcohol did show a significant increase in a reaction time measure, regardless of the experimental condition. Provocation to be aggressive was also a significant determinant of aggression, but it was independent of the beverage conditions.

Expectancy also may be associated with the findings first reported by Takala, Pihkanen and Markkanen (1957) that aggression was greater when subjects had consumed spirits than after drinking beer; though the BACs were similar. A later study by Pihl, Smith and Farrell (1984) confirmed the observation and reported that subjects who consumed beer or believed that they were consuming beer, were significantly less aggressive than subjects who had consumed distilled spirits or believed that they were consuming distilled spirits. Takala et al. attributed the difference in aggression after alcohol versus distilled spirit consumption to different congener content of the two beverages. The findings of Pihl et al., however, suggested that the results could be accounted for by differences in expectations about the effects of beer versus spirits. Perhaps the perception or expectancy of drinking spirits was considered as more serious drinking than the consumption of beer; the latter being regarded as a more sociable drink! Pihl et al. also pointed out that an explanation of the results based on the congener content of the beverages was not viable since beer has a proportionately higher congener content than distilled spirits.

Yet another dimension of the role of alcohol in aggressive behaviour has been suggested by Pihl, Zeichner, Niaura, Nagy and Zacchia (1981), who examined the role of attribution. They considered that some individuals attributed their loss of control over their emotions to their state of intoxication rather than to themselves. Their results argue the point that both expectancy and attribution in the intoxicated state contribute to aggression under alcohol. Clearly, this fascinating, complex and socially important interaction between alcohol and aggression requires, and will encourage, further research.

4.3 Risk-taking behaviour

A related behaviour change induced by alcohol, and one possibly related to aggression, is risk-taking. Research into the effects of alcohol on risk-taking has been prompted by the role of the drug in road crashes. Barry (1973) has suggested that alcohol reduces perceived fear, and increases assertiveness. The combination of these two effects may result in increased risk-taking behaviour. It is difficult to replicate risk-taking behaviour in the laboratory, but one example of this effect of alcohol has been described by the studies of Dott (1974), Beck et al. (1973), and Rafaelsen,

Christrup, Bech, and Rafaelson (1973). Using a driving simulator and a simulated risk paradigm with an overtaking task, these authors showed that subjects were more prepared to engage in a risky overtaking task when under the influence of alcohol than when sober.

5. Mood effects in experienced drinkers

The amount of prior experience a person has had with the consumption of alcohol can influence the mood effects produced by the drug. Some studies have reported that more frequent drinkers derived more positive mood effects from alcohol than did people who rarely drank alcohol (Smith, Parker & Noble, 1975; Persson, Sjoberg & Swensson, 1980). The effect alcohol has on the emotional state of individuals diagnosed as alcoholics, however, is quite different from that seen in moderate social drinkers (Freed, 1978). Both alcoholics and non-alcoholics may anticipate an increase in positive mood states after drinking alcohol. Although the non-alcoholic may achieve an initial increase in elation, the alcoholic often does not. Alcoholics often report an increase in negative mood states such as dysphoria, depression and anxiety as a consequence of alcohol consumption (Freed, 1978; Martorano, 1974; McNamee, Mello & Mendelson, 1968). This is not always the case, however. In a study by Vanicelli (1972), half of the alcoholic subjects tested showed an increase in anxiety after alcohol while the other half showed a lowered anxiety score. This split did not occur in the depression measure. The mean depression score was higher after alcohol than before drinking.

It has been suggested that alcohol-induced mood changes may carry over into the non-intoxicated state. Many alcoholics report negative mood states when they enter treatment or when they have been recently detoxified (Lubman, Emrick, Mosimann & Freedman, 1983). It is not known whether this negative affect is a result of excessive alcohol use or perhaps a factor which may have contributed to the development of alcohol-related problems. A study by Birnbaum et al. (1983) suggested that there was a carryover of mood effects from the intoxicated to the sober state in a group of female social drinkers which could be altered by having the women abstain from their usual pattern of consumption over a six-week period. This was an interesting, though preliminary, finding which deserves further investigation.

6. Conclusion

In summary, it is clear that alcohol is capable of affecting a person's mood. The change in mood observed after alcohol consumption depends on numerous factors aside from the dose of the drug administered at the time of mood assessment. Some of these other factors include: the phase of the BAC curve on which mood is assessed, the amount of experience the person has had with alcohol consumption, the person's expectations about the effects of alcohol, and the setting in which alcohol is consumed. There are numerous complex interactions among these variables which are often difficult to interpret.

References

Barry, H. (1973). Motivational and cognitive effects of alcohol. Journal of Safety Research, 5, 200-221.

Baum-Baicker, C. (1985). The psychological benefits of moderate alcohol consumption: A review of the literature. Drug and Alcohol Dependence, 15, 305-322.

Bech, P., Rafaelson, L., & Rafaelson, O.J. (1973). Cannabis and alcohol effects on estimation of time and distance. Psychopharmacologia, 32, 373-381.

Birnbaum, I. M., Taylor, T. H., & Parker, E. S. (1983). Alcohol and sober mood in female social drinkers. Alcoholism: Clinical and Experimental Research, 7, 362-368.

Buss, A. H. (1961). The psychology of aggression. New York: Wiley.

Cappell, H., & Greeley, J. (1987). Alcohol and tension reduction: An update on research and theory. In H. T. Blane & K. E. Leonard (Eds.), Psychological theories of drinking and alcoholism (pp. 15-54). New York: The Guilford Press.

Carpenter, J., & Armenti, N. (1972). Some effects of ethanol on human sexual and aggressive behaviour. In B. Kissin & H. Begleiter (Eds.), The biology of alcoholism (Vol. 2, pp.). New York: Plenum.

Chesher, G. B., Dauncey, H., Crawford, J., & Horn, K. (1986). The interaction between alcohol and marijuana. A dose dependent study of the effects on human moods and performance skills (CR 40). Federal Department of Transport, Federal Office of Road Safety.

Coid, J. (1982). Alcoholism and violence. Drug and Alcohol Dependence, 9, 1-13.

Dott, A. B. (1974). Effect of marijuana on aggression and risk acceptance in an automotive simulator. Clinical Toxicology, 7, 289-293.

Freed, E. X. (1978). Alcohol and mood: An updated review. International Journal of the Addictions, 13, 173-200.

Hobart, C. W. (1978). Economic development, liquor consumption and offence rates in North-west territories. Canadian Journal of Criminology, 20, 259-278.

Kastl, A. J. (1969). Changes in ego functioning under alcohol. Quarterly Journal of Studies on Alcohol, 30, 371-380.

Kelly, M., Myrsten, A., Neri, A., & Rydberg, V. (1970). Effects and after-effects of alcohol on physiological and psychological functions in man - a controlled study. Blutalkohol, 7, 422-436.

Lang, A. R., Goeckner, D. J., Adesso, V. J. & Marlatt, G. A. (1975). Effects of alcohol on aggression in male social drinkers. Quarterly Journal of Studies on Alcohol, 84, 508-518.

Lanzkron, L. (1963). Murder and insanity: a survey. American Journal of Psychiatry, 119, 754-758.

Levenson, R. W., Sher, K. J., Grossman, L. M., Newman, J., & Newlin, D. B. (1980). Alcohol and stress response dampening: Pharmacological effects, expectancy, and tension reduction. Journal of Abnormal Psychology, 89, 528-538.

Lindman, R. (1982). Social and solitary drinking: Effects on consumption and mood in male social drinkers. Physiology and Behavior, 28, 1093-1095.

Lindman, R. (1985). On the direct estimation of mood. Perception and Psychophysics, 37, 170-174.

Lubman, A., Emrick, C., Mosimann, W. F., & Freedman, R. (1983). Altered mood and norepinephrine metabolism following withdrawal from alcohol. Drug and Alcohol Dependence, 12, 3-13.

McNamee, H. B., Mello, N. K., & Mendelson, J. H. (1968). Experimental analysis of drinking patterns of alcoholics: Concurrent psychiatric observations. American Journal of Psychiatry, 124, 81-87.

Martorano, R. D. (1974). Mood and social perception in four alcoholics. Quarterly Journal of Studies on Alcohol, 35, 445-457.

Mendelson, J., & LaDou, J. (1964). Experimentally induced chronic intoxication and withdrawal in alcoholics. II. Psychophysiological findings. Quarterly Journal of Studies on Alcohol, 2, 39.

Mendelson, J. H., & Mello, N. K. (1966). Experimental analysis of drinking behavior of chronic alcoholics. Annals of the New York Academy of Sciences, , 828-845.

Pernanen, K. (1976). Alcohol and Crimes of violence. In B. Kissin & H. Begleiter (Eds.), Social aspects of alcoholism (pp. 351-444). London: Plenum Press.

Persson, L., Sjoberg, L., & Svensson, E. (1980). Mood effects of alcohol. Psychopharmacology, 68, 295-299.

Pihl, R. O., Smith, M., & Farrell, B. (1984). Alcohol and aggression in men: A comparison of brewed and distilled beverages. Journal of Studies on Alcohol, 45, 278-282.

Pihl, R. O., Zeichner, A., Niaura, R., Nagy, K., & Zaccia, C. (1981). Attribution and alcohol-mediated aggression. Journal of Abnormal Psychology, 90, 468-475.

Rafaelson, L., Christrup, H., Bech, P., & Rafaelson, O.J. (1973). Effects of cannabis and alcohol on psychological tests. Nature, 242, 117-118.

Robbins, B. J., & Brotherton, P. L. (1980). Mood change with alcohol intoxication. British Journal of Social and Clinical Psychology, 19, 149-155.

Roslund, B., & Larson, C. A. (1979). Crimes of violence and alcohol abuse in Sweden. International Journal of the Addictions, 14, 1103-1115.

Sher, K. J. (1985). Subjective effects of alcohol: The influence of setting and individual differences in alcohol expectancies. Journal of Studies on Alcohol, 46, 137-146.

Sher, K.J. (1987). Stress response dampening. In H. T. Blane & K. E. Leonard (Eds.), Psychological theories of drinking and alcoholism (pp. 227-264) New York: The Guilford Press.

Sjoberg, L., Svensson, E., & Persson, L. O. (1979). The measurement of mood. Scandinavian Journal of Psychology, 20, 1-18.

Smith, R. C., Parker, E. S., & Noble, E. P. (1975). Alcohol and affect in dyadic social interaction. Psychosomatic Medicine, 37, 25-40.

Sutker, P. B., Goist, K. C., Allain, A. N., & Bugg, F. (1987). Acute alcohol intoxication: Sex comparisons on pharmacokinetic and mood measures. Alcoholism: Clinical and Experimental Research, 11, 507-512.

Sutker, P. B., Tabakoff, B., Goist, K. C., & Randall, C. L. (1983). Acute alcohol intoxication: Mood states and alcohol metabolism in women and men. Pharmacology, Biochemistry and Behavior, 18 (Suppl. 1), 349-354.

Takala, M., Pihkanen, T. A., & Markkanen, T. (1957). The effects of distilled and brewed beverages: A physiological, neurological and psychological study (Publication No. 4). Helsinki: Finnish Foundation for Alcohol Studies.

Vanicelli, M. L. (1972). Mood and self-perception of alcoholics when sober and intoxicated. I. Mood change. II. Accuracy of self-prediction. Quarterly Journal of Studies on Alcohol, 33, 341-357.

Wallgren, H., & Barry, H. (1970). Actions of alcohol (Vol. 1). New York: Elsevier.

Chapter 9. Suicide, Alcohol, Incarceration and Indigenous Populations: A Review

Ernest M. Hunter

1. Introduction

The absolute nature of the act of suicide suggests that its analysis should be unhindered by the confusions surrounding investigations of morbidity or mortality from natural causes. Unfortunately this is not so and several issues must be addressed at the outset. An attempt at self harm suggests an intent to die, but clearly suicidal people represent a heterogeneous group (Linehan, 1986). Indeed, within Western societies, there are clear differences in terms of age and sex distribution between suicide attempters (parasuicides) who tend to be young and female (Morgan, Burns-Cox, Pocock & Pottle, 1975) and those who successfully kill themselves, who are more commonly older and male (Barraclough, Bunch, Nelson & Sainsbury, 1974). Men are also more likely to use violent and immediately lethal means, and to have a history of substance abuse (Rich, Ricketts, Fowler & Young, 1988).

While there have been increases in suicides especially among young males reported from Great Britain (McClure, 1984a; 1984b; Murphy, Lindsay & Grundy, 1986), the United States (Murphy & Wetzel, 1980), and Australia (Dorsch & Roder, 1983; Goldney & Katsikitis, 1983), this increase has been from a baseline of low rates in the younger age-groups several decades ago, so that the contribution of youth suicide is still small in terms of overall rates (Robins & Kulbock, 1986).

The act of committing suicide thus must be seen in relation to other behaviours including suicidal ideation, parasuicide, ludic suicide equivalents, and deliberate self harm. Even those who succeed in taking their lives may represent more than one population capable of being differentiated clinically (Overstone & Kreitman, 1974). These complexities are reflected in problems with suicide statistics including the question of how to deal with deaths listed as unknown, or suggestive accidents (McClure, 1984a; Sainsbury & Jenkins, 1982). The idiosyncracies of coroners' reporting practices must be recognised especially since the distortion in suicide rates across different societies is not uniform (McCarthy & Walsh, 1975), being influenced not only by means, but by age (Walsh, Walsh & Whelan, 1975). Holinger, Offer & Ostrov (1987) suggest that underreporting may result in United States' suicide rates being two to three times lower than the real figures. These problems become further magnified in moving into a cross-cultural context where the very definition of suicide becomes problematic (Burton-Bradley, 1985).

Such confusion calls for a working definition. Schneidman (cited in Pfeffer, 1986), while explicitly addressing suicide in the West, offers the following:

> suicide is the conscious act of self-induced annihilation, best understood as a multidimensional malaise in a needful individual who defines an issue for which the suicide is perceived as the best solution. (p. 342)

This avoids the thorny question of whether suicide necessarily implies 'mental illness' or 'derangement' presented by altruistic or institutionalized suicides such as *seppuku* (Fuse, 1980).

2. Suicide and indigenous populations

There is an extensive cross-cultural literature on suicide, however this report focuses on indigenous populations of the Pacific and Pacific rim.

2.1 North America

By far the most intensively examined group (and the biggest) are native Americans (including American Indians and natives of Alaska and Canada). The annotated bibliography of Peters (1981) lists 65 citations between 1930 and 1980. Although suicide appears to have been rare among pre-contact native Americans, it did occur, with higher rates among women (Pine, 1981). This is no longer the case. The suicide rates among young males between 15 and 25 years of age have tripled from 4.5 per 100,000 in 1958 to 12.1 per 100,000 in 1982 (May, 1987). There are, however, wide variations across differing groups with annual rates of between 8 and 120 per 100,000 (Shore, 1975; Shore & Manson, 1983). The concern regarding adolescent Indian suicide resulted in the setting up of suicide prevention centres on various reservations from the early 1970s (Shore, Bopp, Waller & Dawes, 1972). Alcohol abuse (seen as a normative pattern among youth and young adults) is implicated in 75% of accidents, 80% of suicides and 90% of homicides, as well as the significant excess of court appearances during adolescence (Yates, 1987). May (1987) notes that youth suicides cluster in certain tribes, and identifies certain characteristics of contemporary Indian suicide generally. Those committing suicide are commonly young males, women by contrast now have particularly low rates in most tribes. Highly lethal or violent means are typical. In addition "tribes with loose social integration which emphasizes a high degree of individuality, generally have higher suicide rates than those with tight integration (which emphasizes conformity)" (p.). Finally, areas experiencing rapid social and economic change have higher rates of suicide. Berlin (1985) suggests that these are areas where a breakdown in traditional structures has occurred. Intergenerational tensions accompanying the stress of acculturation may lead to family dysfunction. The resultant instability of parental figures, and consequently multiple caretakers, appears associated with higher suicide rates (Berlin, 1985; Shore et al., 1972). Similar findings are reported from Alaska (Kost-Grant, 1983) and Canada (Tonkin, 1984).

2.2 Micronesia

Perhaps the most dramatic examples of 'epidemic' suicide are found in Micronesia (Hezel, 1976; Rubinstein & King, 1981). Rubinstein (1983) analysed over 300 suicides (nearly all hangings) occurring over a 20 year period from all areas of Micronesia. Annual rates increased from 0 to 4.5 per 100,000 for females between 1960-63 and 1976-79, and from 6.4 to 49.5 per 100,000 for males during the same period. The islands of Truk and the Marshalls bore the brunt of this loss, with young males being overrepresented. At a time when the U.S. suicide rates for males aged 15-19 and 20-24 years were 12.8 and 27.4, respectively, the corresponding rates from the Marshall Islands were 158 and 174, and from Truk 243 and 255 per 100,000. Rubinstein (1983) indicates the emergence of a "suicide subculture" described as "a set of coherent meanings which organize, provide significance for, and contribute to the dynamics and frequency of adolescent suicide" (p. 658).

To hang oneself, for a young male Micronesian, became an increasingly frequent response to personal problems that on the surface appeared trivial. On a deeper level, this option reflected post-war social changes that placed particular stress on young males. There was, in particular, a disruption of the traditional institutions and rituals (such as the lineage men's house) that regulated and controlled a period of intense tension. Such institutions were important as an organising force for adolescent socialisation and the formation of a social identity. Furthermore, a climate of increasing economic and political dependence on the United States had occurred in conjunction with an increasing demand for modern consumer goods, with profound disruption of traditional occupational and social roles.

In addition to suicide, alcoholism and violence emerged as a consequence of this role confusion (Marshall, 1979). Hezel (1984) investigated the 129 suicides that occurred on Truk between 1971 and 1983. Half of all the suicide victims had consumed alcohol prior to the act. Along with Rubinstein (1983) he identified the peri-urban or rural fringe as having the highest suicide rates, those groups living in areas where social change was occurring most rapidly. Most of the suicides were associated with a disruption of a significant interpersonal relationship, usually family members with authority over the victim.

An investigation of 31 of 61 suicides occurring since the Second World War on the island of Palau in the south-west of Micronesia (Polloi, 1985) also focuses on endemic stress accompanying a breakdown of traditional bonds. All bar one were male, 75% single, and most had grown up during the period of American administration, in contrast to their parents who had lived under the Japanese. Thirty-six percent of these suicides were known to have been drinking.

2.3 Western Samoa

In Western Samoa, as in Micronesia, suicide is not a new phenomenon, however epidemic increases in both regions were contemporaneous. In a population of around 160,000, there were 237 suicides from 1970 to 1981 (Oliver, 1985) and 106 between 1981 and 1983 (Bowles, 1985). The increase in suicides was predominantly among young males who died as a result of paraquat ingestion. A 'blocked opportunity model' (MacPherson & MacPherson, 1985), focusing on youth alienated from the traditional values of the society, yet still constrained by an entrenched traditional power structure, was suggested as a structural interpretation of these events. The increased desires and expectations of young Samoans that have arisen during the development of the last decades (the involvement of New Zealand and the United States being central), have not been accompanied by opportunities for social mobility.

Intergenerational conflict is again offered as an explanation in a cultural model utilising the concept of *musu* (White, 1985), which "represents a culturally defined way of feeling and acting in response to conflict with someone in authority, especially parents, towards whom one owes love and respect and should not express anger" (p. 6). The role of alcohol in the Western Samoan suicides is not discussed.

2.4 Melanesia

The enormous ethnic variation in Melanesia makes generalizations problematic. However, in contrast to the above regions, there is a tradition and social patterning of suicide with young women at greatest risk (White, 1985). Marital conflict and the consequences for intergroup relationships appear central. There are, however, exceptions in which males dominate, including the Bimin-Kuskusmin of Papua New Guinea (Poole, 1985) and the Kandrian people of New Britain (Hoskin, Friedman & Cawte, 1969). While female suicides in the former appear traditionally culturally patterned, emergent patterns of male suicide involve young men who are relatively socially isolated, and who are experiencing a sense of failure in a group where male roles are clearly defined. In the Kandrian situation, the suicides are interpreted in terms of endemic stress resulting from social fragmentation due to an unusual degree of chronic tribal conflict coupled with a paradoxical emphasis on the value of close interpersonal ties.

2.5 Fiji

The suicide rate in Fiji is reported to be higher among young women (and older men) than men. However, the majority of these deaths were Indian, and may reflect the position and status of women in the Indian family (Haynes, 1984). White (1985), in a comment relevant to Aboriginal suicide, states that the increases in the Pacific:

> are only one part of a broad change which is occurring in patterns of mortality and morbidity in the Pacific and developing world generally....The specter of infectious

> diseases as the most serious threat to health is rapidly being replaced by lifestyle-related disorders such as diabetes, hypertension, malnutrition, alcoholism and death or injury resulting from violence. (pp. 1-2)

2.6 Australia

In terms of Aboriginal suicide the information is far more sparse with, until recently, most reports being anecdotal. Cleland (1962) recorded a possible suicide attempt in the Northern Territory in the 1940s and a suicide of an Aboriginal prisoner some 20 years earlier in South Australia. Cawte (1972) described one suicide and a number of attempts among the Kaiadilt people who had been transferred from Bentnick to Mornington Island following a period of devastating environmental pressures. Jones (1971; 1972), and Jones and Horne (1973) reported on surveys conducted in the Kimberley region of Western Australia among populations that were mainly 'traditional'. In each of these the absence of suicide was noted; however, the authors commented on reports of three suicides and one attempted suicide during the previous 40 years among Aboriginals who had given up tribal life. They indicated that while "suicide has not yet been found in the tribal state" death from 'boning' may be understood as a suicide equivalent. Berndt and Berndt (1985) likewise suggested that both deliberate self-exposure to danger and loss of will to live on learning that one has been selected as a victim of sorcery may be the nearest approximations to suicide in traditional situations. An important component of the potent fatalism engendered by sorcery appears to have been the withdrawal of support by the social network on learning that an individual had been identified as a victim (Eastwell, 1982).

Reid (1983) listed several suicide attempts precipitated by loss among the Yolngu of Arnhem Land, and suggested that risk taking behaviour may represent a ludic suicide equivalent. Eastwell (1979; 1988) studying the same population, reported two suicides over a 30 year period in a population of about 5,000 Aboriginals. Both victims were males, one was recovering from delirium tremens, and the other considered himself to have been the victim of sorcery, but who also appeared to suffer from episodes of alcohol-induced jealousy. Eastwell also speculated on the relationship of suicide to alcohol-related instances of males placing themselves in harm's way. Brady (1988) recorded one alcohol-associated suicide by shooting by an Aboriginal person in Tennant Creek. It was noted that this incident took place in the context of increasing rates of alcohol-related deaths from external causes for both Aboriginals and non-Aboriginals.

Reports of parasuicide are likewise scanty, and come from urban settings. Burvill (1975) reported on 4 males and 14 females admitted to hospital in Perth in 1971-1972. Three of the four males were less than 25, and all of the females were between 15 and 44 years. Kamien (1978) described four serious suicide attempts in Bourke in the early 1970s, all of which were associated with alcohol.

Hunter (1988a) collected data on a total of 21 suicides of Aboriginals from the North-West of Western Australia. The average age in this group was 23 years. Of these 21 suicides, 17 occurred since 1980 and 10 since 1985, with two having died in custody. An examination was made of 12 male and 2 female suicides of individuals in or from the Broome shire (an area of particularly rapid social change). Relevant findings for the males included an overrepresentation of partial descent Aboriginals (all save two); a common history of disorders of ideation and perception associated with alcohol, including both recurrent delirium tremens and a chronic and recurrent form of hallucinosis; and a history of a recent loss, specifically the breakup of a relationship or altercation with a partner. Thus suicides in the Kimberley are an emergent phenomenon, and must be seen in the context of a rising proportion of deaths from external causes. Material based on a review of all recorded deaths in the Kimberley between 1957 and 1986 showed that deaths from external causes (motor vehicle accidents, accidents, homicide, and suicide) was steady at between 2% and 5% of all non-infant deaths until the early 1970s (a period of rapid and massive demographic shifts and social changes in the Kimberley including citizenship rights and access to alcohol). From that time the proportion has been climbing steadily, reaching 15% of female and 25% of male deaths between 1982 and 1986 (Hunter, in press).

Recently presented material (Hunter, 1988, September) based on ongoing research in the Kimberley is of relevance. Initial data from the first 230 of a random sample of 630 Aboriginals from throughout the region demonstrated that, to date, all those who reported past suicide attempts, and the majority of those with histories of suicidal ideation, fell in the groups of heavy regular drinkers. These groups contained the majority of individuals with histories of incarceration, unstable relationships, and disorders of ideation and perception. Furthermore, these regular heavy drinkers admitted to more depressive and anxiety symptoms. Chegwidden and Flaherty (1977) reported that 27% of a sample of 55 alcohol-dependent Aboriginals in a Sydney detoxification program admitted to previous suicide attempts, supporting the notion that these individuals make up a particularly vulnerable subgroup.

2.7 Summary

The general impression is that suicide, as we have defined it, was at least very rare among traditional Aboriginal populations, and remains so among tradition-oriented groups. As with the other indigenous populations reported on, the suicides as yet appear to cluster in areas of rapid social change. The emergence of suicide has occurred against a backdrop of major increases in deaths from external causes generally, and similarly appears to have an association with alcohol. The group at greatest risk are young males of partial descent, and commonly with a history of a recent disruption of a relationship. Issues of acculturation

stress, identity, and the psychological sequelae of cultural exclusion (Brody, 1966) must be addressed in a more comprehensive analysis.

3. Suicide and alcohol

The relationship between alcohol and suicide is clear, in the sense that the research evidence supports a connection, but complex, in terms of the nature of that association. A dilemma arises as there are a number of conditions (such as depression and certain personality disorders) in which both suicide and alcoholism are found, and as both are conditions with a high prevalence, their coexistence is to be expected in some cases. Indeed one position states that alcoholism represents a form of slow suicide (Menninger, 1938). Additionally, research in alcoholism suffers from the same problems of definition and analysis as have been outlined for suicide earlier. In the previous section, alcohol has appeared repeatedly in the reports of suicide among indigenous groups. It also figures as a prominent precursor of depression in many of these groups including the Hopi (Manson, Shore & Bloom, 1985) and Australian Aboriginals (Cawte, 1988).

3.1 Suicide rates among alcoholics

The research on suicide and alcoholism has taken two courses. The first is an examination of the rate at which individuals identified as alcoholics subsequently suicide. Kessel and Grossman (1961) followed up two series of 131 and 87 chronic alcoholics in Edinburgh and found that 8% and 7% (all males) killed themselves within 1 and 11 years of discharge, with the ratio of observed to expected deaths being 86:1. Adelstein (1976) examined the mortality of alcoholics identified through a mental health registry and hospital records in London. There was an excess of mortality, particularly in the young, due to violent deaths. They estimated the observed to expected ratio for deaths from suicide to be 22 for males and 23 for females in the alcoholic populations sampled. Miles (1977), in a review of research into conditions predisposing to suicide, found similar findings, predicting that 15 percent of alcoholics would die by suicide. He comments on the overlap with depression, and points out that, in contrast to depression, where suicide tends to be an early event following initial diagnosis, suicide in alcoholism appears to occur late, often for males, following a loss.

3.2 Rates of alcoholism in deaths by suicide

The second approach is to ascertain what proportion of suicide victims could be diagnosed as alcoholic. An Edinburgh study (McCulloch, Philip & Carstairs, 1967) demonstrated that of 20 suicides who were known to have presented previously with suicide attempts, all were 'characterized by impulsiveness and habituation to drink or drugs' (p. 318). Barraclough et al. (1974) investigated the past psychiatric history of 100 suicides in southern

England. Fifteen of these were identified as alcoholic, four having had a history of delirium tremens, with nine also being depressed. The male to female ratio was 4:1, suicide usually being a late event. 'The most obvious clinical features were the combination of severe alcohol addiction and depression occurring in a recently disturbed domestic and social setting' (p. 185).

In a review of the research in the field Goodwin (1979) records that in 11 studies the rates of alcoholism among suicides varied from 6% to 47%, with the proportion of alcoholics subsequently committing suicide varying between 6% and 21% in six studies. Suicide victims from this group were more frequently male. Suicide tended to occur in the middle years. Goodwin suggested that this was a period of their lives when alcoholics may be more vulnerable to the impact of loss.

In the most recent review Frances, Franklin and Flavin (1986) stated that:

> alcohol has been found to be associated with 50% of suicides and to increase the risk of suicidal behavior both for alcoholic and nonalcoholic populations. Between 5% and 27% of all deaths of alcoholics are caused by suicide. The incidence of alcoholism among persons who commit suicide ranges from 6% to 30% in different studies with approximately 20% most frequently cited. Lifetime risk for suicide is 1% in the general population, 15% for major affective illness, and 15% for alcoholism. (p. 316)

Alcoholic depressed males again emerge as being most vulnerable, and the authors present two clinical pictures similar to Berglund et al., the first associated with intoxication, aggression, anger and hyperactivity, the second following mounting depression accompanying chronic intoxication.

3.3 Suicide, alcoholism, and BAC

An examination of the blood alcohol concentrations (BACs) of 55 Swedish suicides who had previously been diagnosed as alcoholic (Berglund, Krantz & Lundqvist, 1987) showed differences based on the symptom ratings at the time of initial diagnosis (mean time to suicide was five years). Those described as 'brittle and sensitive' tended to have high post mortem alcohol levels, whereas those with initial ratings of dysphoria (depression) tended to have low levels. The latter group also had more frequent histories of previous suicide attempts, thus supporting the idea that they were an affective subgroup. A recent loss was reported in 36% of all suicides.

3.4 Alcoholism and psychiatric disorders

A consistent association between alcoholism and depression emerges, and as already mentioned, the population of individuals suffering from affective disorder has a 15% lifetime mortality from suicide (Guze & Robins, 1970). In a

large prevalence survey in New Haven (Weissman, Myers & Harding, 1980), of those diagnosed as being alcoholic, 70% had a history of another psychiatric diagnosis at some point in their lives - major depression in 44%, minor depression in 15%, and personality disorder in 17%. Among those considered to have both alcoholism and major depression, depression was the major diagnosis in 60%. A study using interview and self rating scale instruments (Lippmann, Manshadi, Christie & Gultekin, 1987) found between 40% and 60% of 40 alcoholics in a treatment program to be depressed. Gibson and Becker (1973) administered self-report measures of depression to alcoholic inpatients during withdrawal and found rapid improvement which they attributed largely to a reduction in somatic symptoms. Affective improvements appeared to be maintained among those who remained abstinent (Pettinati, Sugerman & Maurer, 1982).

The immediate effect of intoxication on affect is itself complex, with moderate drinkers experiencing more relief from depressive symptoms than excessive drinkers in a laboratory situation (Mayfield, 1968a). Furthermore, for alcoholics, low levels of intoxication were associated with relief of such symptoms, but at higher levels this effect was negated, "the difference between the response of alcoholics and non-alcoholics becomes greater as the alcoholics reach higher levels of intoxication" (Mayfield, 1968b, p. 319).

Schuckit (1983) reported that a third of 233 male primary alcoholics in a Veteran's Administration study had significant affective disorder. In a later paper (1986), he identified five factors contributing to the frequent diagnostic confusion:

> 1) alcohol can cause depressive symptoms in anyone, 2) signs of temporary serious depression can follow prolonged drinking, 3) drinking can escalate during primary affective episodes in some patients, especially during mania, 4) depressive symptoms and alcohol problems occur in other psychiatric disorders, and 5) a small proportion of patients have independent alcoholism and affective disorder. (p. 141)

Schuckit concluded that while depression was common in the lives of alcoholics, particularly for men, alcoholism usually preceded the depression. These depressions were usually transitory, and for those with concurrent alcoholism and depression, the primary diagnosis (defined chronologically) was generally alcoholism. Reviewing the genetic research on alcoholism and depression, he concluded that there was little evidence to support a genetic linkage between the two disorders. Nevertheless, their concurrent existence, regardless of primary diagnosis, did appear to increase the risk for suicide.

3.5 Psychobiology of suicide

It is worth noting that much of the research at present is focusing on the psychobiology of suicide, in particular the role of serotonin in violent suicide (Mann, Stanley, McBride & McEwen, 1986), however the relationship of this to the neurobiology of depression is unclear (Van Praag, Plutchik & Conte, 1986), and the role of alcohol is yet to be investigated.

3.6 Suicide and interpersonal loss

In many of the foregoing studies, a history of an interpersonal loss has been suggested as increasing vulnerability to suicide, particularly for males. In an investigation of 50 alcoholic suicides (Murphy, Armstrong, Hermele, Fischer & Clendenin, 1979), 56% were found to have a history of a definite affective disorder, and 26% were found to have experienced a significant interpersonal loss within six weeks of their deaths. These included 5 of 11 considered to have uncomplicated alcoholism and 6 of 28 with a definite secondary affective disorder. The authors report that there was, surprisingly, little relationship between affectional loss and the presence or absence of secondary depression. Thus, that group of alcoholics who committed suicide in the absence of secondary depressive symptoms appeared more likely to have experienced a loss. The nature of the interaction between these two factors in the context of alcoholism remains unclear.

Among the Aboriginal suicides described by Hunter (1988a), all the males who were not alcoholic, and all alcoholics for whom there was no history of disorders of ideation and perception, had experienced a recent disruption of an important relationship.

3.7 Alcohol withdrawal and psychotic symptomatology

Of relevance to suicide in the Australian Aboriginal population is the relationship with psychotic symptomatology. It is well known that alcohol can cause disorders of ideation and perception as a result of withdrawal, which does not necessarily require abstinence (Kanzow, 1986). Patterns of acute and chronic auditory hallucinosis may occur with prolonged drinking (Pearce, 1977), and may proceed to a chronic form indistinguishable from schizophrenia (Alpert, 1985). Hays and Aidroos (1986) found schizophrenics with a history of alcohol abuse to have clinical pictures characterised by earlier hallucinosis with prominent visual components. In a comparison of depressed and non-depressed hospitalised alcoholics, Cadoret and Winokur (1974) found an excess of alcoholic paranoia in the non-depressed males. In the same study, the depressed males were reported to have both more frequent and more serious suicide attempts. The authors speculate on the internalisation (depression) versus projection (paranoia) of aggression and guilt.

The relationship of suicide to psychosis was examined among 134 suicides in St Louis (Robins, 1986), in which 25 (19%) were psychotic at the time of suicide. Thirty-three of the suicides were alcoholic, among whom five (15%) were psychotic, all male.

There is evidence that psychotic syndromes associated with alcohol are increasing among Aboriginals of Northern Australia. The Northern Territory Department of Health (1986) reports a five-fold increase in the standardised hospital separation rates for the diagnosis of 'alcoholic psychosis' between 1977 and 1982 for Aboriginal males. While this may in part reflect changed diagnostic practices, other groups (non-Aboriginals and Aboriginal females) do not show the same increases. In addition, those being so labelled are younger by more than a decade than their white counterparts. As noted earlier (Hunter, 1988a), young Aboriginal males with histories of either recurrent delirium tremens or hallucinosis are overrepresented among Kimberley suicides.

3.8 Diagnosis of psychiatric conditions

Diagnostic confusion likewise occurs in other psychiatric conditions associated with impulsivity, recklessness, unstable relationships, and drinking, in which suicidal behaviour is not uncommon. Schuckit (1973) examined the relationship with sociopathy and suggested they are two overlapping entities. There appears to be a subgroup of male alcoholics who demonstrate particularly severe alcoholism associated with sociopathic behaviour in which patrilineal familial transmission occurs (Cloninger, Bohman & Sigvardsson, 1981; Frances, Timm & Bucky, 1980).

Nace, Saxon and Shore (1983) identified 12.8% of 94 alcoholic patients as having borderline personality disorders, of whom 60% had histories of suicide attempts, compared to only 13.6% of non-borderline alcoholics. The former group also had higher rates of motor vehicle accidents and other accidents. From an alternate perspective, those individuals with borderline personality disorder, and concurrent affective disorder and substance use disorder, had a higher rate of serious suicide attempts (Fyer, Frances, Sullivan, Hurt & Clarkin, 1988).

3.9 Summary

There appears to be a relationship between alcohol and suicide. For alcoholics, this is at least in part mediated through the development of affective disorder which is usually secondary to the alcoholism. Affective symptoms tend to abate quickly with cessation of drinking, the immediate effect of alcohol on affect appearing to be worse with both chronicity of drinking and increasing intoxication. As suicide is a late event among alcoholic individiuals whose primary diagnosis is alcoholism, it appears to be associated with chronicity of drinking and affective symptoms. At greatest risk are those male alcoholics who are concurrently depressed and who have experienced recent object loss.

This picture is consistent with the findings emerging from work in the Kimberley. In addition, of particular relevance to this group, is the development of psychotic symptomatology in association with heavy drinking, a pattern that is increasing in Northern Australia, and which is associated with suicide among Aboriginals in the Kimberley region.

Individuals with certain personality patterns characterized by impulsiveness and unstable interpersonal relationships have a higher likelihood of suicidal behaviour with alcohol, particularly if affective symptoms are present, and these individuals are likely to be heavily intoxicated at the time of death.

4. Incarceration and suicide

Up until 1985 the rate at which Aboriginals committed suicide while in prison custody was similar to that of incarcerated non-Aboriginals (Hatty & Walker, 1986). Of the 100 deaths in custody reported to the Royal Commission by February 1988, 26 were reported to be by hanging, however more than half of these (14) occurred in 1987, raising the question of the influence of media coverage (Biles, 1988). Twenty of these deaths by hanging occurred in police cells, a location which has been focused upon in the international literature on suicide. Almost all of the victims were intoxicated at the time of death.

4.1 A review of the international literature

Suicide in custody was the subject of a recent paper by this author (Hunter, 1988b), and thus the following section quotes extensively from the literature reviewed therein.

> Hayes (1983) investigated 419 suicides occurring in county jails and police lock-ups throughout the United States during 1979. The suicide rate in this setting was 16 times the national average. Of these, 75% were under the age of 32, 73% were arrested for non-violent crimes, 30% for alcohol/drug related offences with 40% under the influence of alcohol, 9% drugs, and 11% both, at the time of incarceration. The majority died between midnight and 8.00 a.m., and 26% of the total group (50% of those under the influence at the time of arrest) died within the first three hours. Hanging was the cause of death for 96%, and 68% of the suicides occurred while the individual was in isolation, with 49% of these occurring within the first 12 hours.
>
> Copeland (1984) analyzed 229 deaths in custody (including police cells, prisons, and hospitals) in Dade county (Miami) between 1956 and 1982. Suicides accounted for 20%, 87% being by hanging, most typically being a younger white male. A further 16% died of 'accidents' with 50% of these being

cranio-cerebral trauma. However police lockup or equivalent was the 'place of incident prior to demise' in 78%.

Jordan, Schmeckpeper and Strope (1987) investigated 17 suicides occurring in Oklahoma jails between 1981 and 1983. Of this small sample all were male, the mean age being 29.3 years. Alcohol related offences accounted for 62% with death occurring typically soon after confinement, in the early morning hours.

Of 56 hangings between 1979 and 1984 occurring both in and out of custody in Fulton county Georgia (Guarner & Hanzlick, 1987) 27% occurred in jail, constituting 93% of male jail suicides. Of these, most occurred 'within a day or so of incarceration' with detectable blood alcohol in 40% (versus 34% of the total sample). Forty percent of the jail suicides had been arrested for crimes against persons.

The Jail Suicide Prevention Information Taskforce (National Center on Institutions and Alternatives, 1987) identifies seven predisposing factors: 1) Recent excessive drinking and/or drug use; 2) Recent loss of stabilizing resources; 3) Severe guilt or shame over offence; 4) Same-sex rape or threat of such; 5) Current mental illness; 6) Poor physical health or terminal illness; 7) Approaching an emotional breaking point. They further suggest seven high risk periods: 1) The first 24 hours; 2) Intoxication/ withdrawal; 3) Trial and sentencing hearings; 4) Impending release; 5) Decreased staff supervision; 6) Weekends and holidays; 7) Bad news from home.

From Great Britain a report by HM Chief Inspector of Prisons (1984) analyzed 169 suicides occurring between 1972 and 1982 (this report entirely from prisons). While 40% of these were 'lifers' (who themselves represent only 5% of the general prison population) 45% were on remand and 30% had only minor charges. The suicide rate overall was four times that in the general population, with two-thirds demonstrating a past psychiatric history and 50% having a history of self harm or suicidal ideation.

A special area for concern is juvenile suicide in custody. The series of Hayes included 15 juveniles, of whom 12 died in isolation, with only five of the total charged with serious crimes. Six died within 14 hours of incarceration. The paradox that isolation is used for the 'protection' of juveniles in adult detention centres is noted by Hayes and also by Flaherty (1983) who compared 21 suicides of minors in adult detention centres across the United States with six occurring in juvenile centres during 1978. The suicide rate for those dying in adult centres was some 3.5 times that of the general population which itself

> was twice as high as the rate in the juvenile centres. Seventeen of these minors died while in isolation. (p. 275)

4.2 Deaths in custody in Australia

A report on deaths in custody in Western Australia between 1980 and 1987 (Bowley, 1988), stated that 20 were by suicide, including 6 Aboriginals and 14 non-Aboriginals. Of the Aboriginal suicides five were male and five occurred in lock-ups. Four of seven Aboriginals and six of fourteen non-Aboriginals died within 24 hours of confinement, five of the total within four hours. Of these, four had 'extremely high' blood alcohol levels, no information being available on the fifth. Nearly two thirds of those dying by suicide "showed some indication of psychological disturbances" (p. 11), however the nature of the disturbances is not elaborated on. No information is given on whether these individuals were isolated at the time of death, or on what proportion of the Aboriginal suicides were partial descent.

In the paper by Hunter (1988b), of 100 Aboriginal lock-up inmates interviewed, 25 reported past suicidal ideation, and 15 previous attempts (12% of the males, 30% of the females). These proportions are not typical of the wider Aboriginal population (as identified by ongoing work by this writer in the random sample survey mentioned previously), but it is typical of a subgroup, those with patterns of heavy regular alcohol consumption.

Lock-ups in the Kimberley are largely filled from these groups - with Aboriginals on 'drunk' charges. For August 1988, out of a lockup population of 1391, 1288 (92.6%) were Aboriginal, with 686 (49.3% of the total) being there for intoxication (Police Department of Western Australia, personal communication). Thus at least 50% (probably many more including alcohol related offences) are intoxicated. These individuals are furthermore frequently arrested, in the study by Hunter (1988b) up to eight times in two months.

Thus members of a heavy drinking subgroup within the Aboriginal population of the region characterised by more frequent histories of disordered ideation and perception, as well as more frequent histories of suicidal ideation and attempts, are being repeatedly incarcerated. These lock-ups draw from an already vulnerable population.

The question remains whether incarceration, particularly incarceration in isolation, exacerbates those conditions predisposing to suicidal behaviour. There is no research on this issue per se. Those individuals experiencing hallucinations and frightening paranoid ideation (an example is recounted in Hunter, 1988b) describe an intensification of these dysphoric experiences when isolated.

4.4 Summary

Suicides in custody occur more commonly in police cells, with the victims often young males arrested on minor offences, who die typically by hanging within a few hours of arrest, often in isolation. The most consistently mentioned feature that emerges is alcohol, in particular being intoxicated at the time of arrest. Aboriginal suicides in custody conform broadly to this picture. An important consideration is that members of a particularly vulnerable section of the Aboriginal population are being repeatedly incarcerated. Certain alcohol related experiences found more commonly among these individuals are associated with suicide outside of custody, and anecdotally appear worsened by the experience of isolation in custody.

5. Conclusion

The interplay between four variables - ethnicity, alcohol, incarceration, and suicide - has been posed in terms of the three combinations which have been explored above. The short answer as to whether a relationship in each case exists - is yes, however to rest on that naive analysis may be to have scored a direct hit on a windmill. Suicide, incarceration, and alcoholism have all increased among people of Aboriginal descent in the relatively recent past. They are overdetermined and dynamically interrelated, being epiphenomena reflective of forces at a far more fundamental level of Aboriginal existence. As such they require a social and historical analysis. We should perhaps be less concerned with asking are they related than demanding why are they related now? In the Aboriginal Australias a constellation of transformations has converged on a few decades of rapid social change, which have among other things seen the breakdown of traditional social structures and controls, linked with increasing dependency on an ambivalently perceived majority culture, and sudden easy access to alcohol (Albrecht, 1974). In particular Aboriginal males have suffered through an undermining of their traditional economic and sacred roles, and latterly (in the north) their place in the pastoral industry. If the broader understanding of the above issues based on an interrogation of the dynamics and consequences of change is not sought, the conclusions drawn, like the windmills, will be without substance.

References

Adelstein, A., & White, G. (1976). Alcoholism and mortality. Population Trends, 6, 7-13.

Albrecht, P. G. (1974). The social and psychological reasons for the alcohol problem among Aborigines. In B. S. Hetzel, M. Dobbin, L. Lippmann, & E. Eggleston (Eds.), Better health for Aborigines? Report of a national seminar at Monash University (pp.36-41). St Lucia: University of Queensland Press.

Alpert, M. A. (1985). Alcohol withdrawal syndromes: Recognition and pathogenesis. Missouri Medicine, 82, 79-83.

Barraclough, B., Bunch, J., Nelson, B., & Sainsbury, P. (1974). A hundred cases of suicide, clinical aspects. British Journal of Psychiatry, 125, 355-73.

Berglund, M., Krantz, P., & Lundqvist, G. (1987). Suicide in alcoholism: A prospective study of 55 cases with autopsy findings. Acta Psychiatrica Scandinavica, 76, 381-385.

Berlin, I. N. (1985). Prevention of adolescent suicide among some native American tribes. In S. C. Feinstein, M. Sugar, A. H. Esman, J. G. Leary, A. Z. Schwartzberg, & A. D. Sorosky (Eds.), Adolescent psychiatry: Vol. 1. Developmental and clinical studies (pp. 77-93). Chicago: University of Chicago Press.

Berndt, R. M., & Berndt, C. H. (1985). The world of the first Australians. Sydney: Allen & Unwin.

Biles, D. (1988). Research paper no. I: Preliminary analysis of current data base. Unpublished manuscript, Royal Commission into Aboriginal Deaths in Custody, Canberra.

Bowles, J. R. (1985). Suicide and attempted suicide in contemporary Western Samoa. In F. X. Hezel, D. H. Rubinstein, & G. H. White (Eds.), Culture, youth and suicide: Papers from an East-West Center Conference (pp. 15-35). Honolulu, Hawaii: University of Hawaii Press.

Bowley, M. (1988). Western Australian deaths in custody (Aboriginal and Caucasian) 1980-1987. Unpublished manuscript, Police Department of Western Australia, Research and Statistics Section, Perth.

Brady, M. (1988). When the beer truck stopped: Drinking in a Northern Australian town. Darwin: Australian National University North Australia Research Unit.

Brody, E. B. (1966). Cultural exclusion, character and illness. American Journal of Psychiatry, 122, 852-858.

Burton-Bradley, B. G. (1985). Overview: Transcultural psychiatry in Papua New Guinea. Transcultural Psychiatry Research Review, 22, 1-15.

Burvill, P. W. (1975). Attempted suicide in the Perth statistical division, 1971-1972. Australian and New Zealand Journal of Psychiatry, 9, 273-279.

Cadoret, R., & Winokur, G. (1974). Depression in alcoholism. Annals of the New York Academy of Sciences, 233, 34-39.

Cawte, J. (1972). Cruel, poor and brutal nations. Honolulu: University of Hawaii Press.

Cawte, J. (1988). The devil's dozen: 13 Aboriginal depressions. Aboriginal Health Worker, 12, 19-28.

Chegwidden, M., & Flaherty, B. J. (1977). Aboriginal versus non-Aboriginal alcoholics in an alcohol withdrawal unit. Medical Journal of Australia, 1, 699-703.

Chief Inspector of Prisons. (1984). Suicides in prison: A report. London: Home Office.

Cleland, J. B. (1962). Disease in the Australian native. Journal of Tropical Medicine and Hygiene, 65, 95-105.

Cloninger, C. R., Bohman, M., & Sigvardsson, S. (1981). Inheritance of alcohol abuse: Cross-fostering analysis of adopted men. Archives of General Psychiatry, 38, 861-868.

Copeland, A. R. (1984). Deaths in custody revisited. American Journal of Forensic Medicine and Pathology, 5, 121-124.

Dorsch, M. M., & Roder, D. M. (1983). A comparison of Australian suicide rates in 1969-73 and 1976-80. Australian and New Zealand Journal of Psychiatry, 17, 254-257.

Eastwell, H. D. (1979). The state of risk for psychiatric illness among the tribes of North Australia. Unpublished M.D. thesis, University of New South Wales, Sydney.

Eastwell, H. D. (1982). Voodoo death and the mechanism for dispatch of the dying in East Arnhem, Australia. American Anthropologist, 84, 5-18.

Eastwell, H. D. (1988). The low risk of suicide among the Yolngu of the Northern Territory: The traditional pattern. Medical Journal of Australia, 148, 338-340.

Flaherty, M. G. (1983). The national incidence of juvenile suicide in adult jails and juvenile detention centers. Suicide and Life-Threatening Behavior, 13, 85-94.

Frances, R. J., Franklin, J., & Flavin, D. K. (1986). Suicide and alcoholism. In J. J. Mann, & M. Stanley (Eds.), Psychobiology of Suicidal Behavior, Annals of the New York Academy of Sciences, 487, 316-326.

Frances, R. J., Timm, S., & Bucky, S. (1980). Studies of familial and nonfamilial alcoholism: I. Demographic studies. Archives of General Psychiatry, 37, 564-566.

Fuse, T. (1980). Suicide and culture in Japan: A study of seppuku as an institutionalized form of suicide. Social Psychiatry, 15, 57-63.

Fyer, M. R., Frances, A. J., Sullivan, T., Hurt, S. W., & Clarkin, J. (1988). Suicide attempts in patients with borderline personality disorder. American Journal of Psychiatry, 145, 737-739.

Gibson, S., & Becker, J. (1973). Changes in alcoholic's self-reported depression. Quarterly Journal of Studies on Alcohol, 34, 829-836.

Goldney, R. D., & Katsikitis, M. (1983). Cohort analysis of suicide rates in Australia. Archives of General Psychiatry, 40, 71-74.

Goodwin, D. W. (1979). Alcoholism and heredity: A review and hypothesis. Archives of General Psychiatry, 36, 57-61.

Guarner, J., & Hanzlick, R. (1987). Suicide by hanging: A review of 56 cases. American Journal of Forensic Medicine and Pathology, 8, 23-26.

Guze, S. B., & Robins, E. (1970). Suicide and primary affective disorder. British Journal of Psychiatry, 117, 437-438.

Hatty, S.E., & Walker. J. R. (1986). Deaths in Australian prisons. Canberra: Institute of Criminology.

Hayes, L. M. (1983). And darkness closes in.... A national study of jail suicides. Criminal Justice and Behavior, 10, 461-484.

Hayes, R. H. (1984). Suicide in Fiji: A preliminary study. British Journal of Psychiatry, 145, 433-438.

Hays, P., & Aidroos, N. (1986). Alcoholism followed by schizophrenia. Acta Psychiatrica Scandinavica, 74, 187-189.

Hezel, F. X. (1976). Tragic end of troubled youth. Micronesian Reporter, 24, 8-13.

Hezel, F. X. (1984). Cultural patterns of Trukese suicides. Ethnology, 23, 193-206.

Holinger, P. C., Offer, D., & Ostrov. E. (1987). Suicide and homicide in the United States: An epidemiologic study of violent death, population changes and the potential for prediction. American Journal of Psychiatry, 144, 215-219.

Hoskin, J. O., Friedman, M. I., & Cawte, J. E. (1969). A high incidence of suicide in a preliterate primitive society. Psychiatry, 32, 200-210.

Hunter, E. M. (1988a). On Gordian knots and nooses: Aboriginal suicide in the Kimberley. Australian and New Zealand Journal of Psychiatry, 22, 264-271.

Hunter, E. M. (1988b). Aboriginal deaths in custody: A view from the Kimberley. Australian and New Zealand Journal of Psychiatry, 22, 273-282.

Hunter, E. M. (1988, September). Broken bottles, broken dreams: Evolving psychosocial consequences of alcohol in an isolated Aboriginal population. Paper presented at the annual conference of the Australian Medical Association, Cairns.

Hunter, E. M. (in press). Changing mortality patterns in the Kimberley. Aboriginal Health Project Information Bulletin.

Jones, I. H. (1971). Stereotyped aggression in a group of Australian Western Desert aborigines. British Journal of Medical Psychology, 44, 259-65.

Jones, I. H. (1972). Psychiatric disorders among aborigines of the Australian Western Desert (II). Social Science and Medicine, 6, 263-267.

Jones, I. H., & Horne, D. J. (1973). Psychiatric disorders among aborigines of the Australian Western Desert: Further data and discussion. Social Science and Medicine, 7, 219-228.

Jordan, F. B., Schmeckpeper, K., & Strope, M. (1987). Jail suicides by hanging: An epidemiological review and recommendations for prevention. American Journal of Forensic Medicine and Pathology, 8, 27-31.

Kamien, M. (1978). The dark people of Bourke: A study of planned social change. Canberra: Australian Institute of Aboriginal Studies.

Kanzow, W. T. (1986). The clinical stages of alcoholic delirium and their therapeutic significance. Acta Psychiatrica Scandinavica, 73 (Suppl. 329), 124-128.

Kessel, N., & Grossman, G. (1961). Suicide in alcoholics. British Medical Journal, 2, 1671-1672.

Kost-Grant, B. L. (1983). Self-inflicted gunshot wounds among Alaskan natives. Public Health Reporter, 98, 72-78.

Linehan, M. M. (1986). Suicidal people: One population or two. In J. J. Mann, & M. Stanley (Eds.), Psychobiology of Suicidal Behavior, Annals of the New York Academy of Sciences, 487, 16-33.

Lippmann, S., Manshadi, M., Christie, S., & Gultekin, A. (1987). Depression in alcohlics by the NIMH-Diagnostic Interview Schedule and Zung Self-Rating Depression Scale. International Journal of the Addictions, 22, 273-281.

MacPherson, C., & MacPherson, L. (1985). Suicide in Western Samoa: A sociological perspective. In F. X. Hezel, D. H. Rubinstein, & G. H. White (Eds.), Culture, youth and suicide: Papers from an East-West Center Conference (pp. 36-73). Honolulu, Hawaii: University of Hawaii Press.

Mann, J. J., Stanley, M., McBride, A., & McEwen B. S. (1986). Increased serotonin2 and '-adrenergic receptor binding in the frontal cortices of suicide victims. Archives of General Psychiatry, 43, 954-959.

Manson, S. M., Shore, J. H., & Bloom, J. D. (1985). The depressive experience in American Indian communities: A challenge for psychiatric theory and diagnosis. In A. Kleinman, & B. Good (Eds.), Culture and depression: Studies in the anthropology and cross-cultural psychiatry of affect and disorder (pp.331-368). Berkeley: University of California Press.

Marshall, M. (1979). Weekend warriors: Alcohol in a Micronesian culture. Palo Alto: Mayfield Press.

May, P. A. (1987). Suicide and self-destruction among American Indian youths. Journal of the National Center for American Indian and Alaskan Mental Health Research, 1, 52-69.

Mayfield, D. G. (1968a). Psychopharmacology of alcohol: I. Affective change with intoxication, drinking behavior and affective state. Journal of Nervous and Mental Disease, 146, 314-321.

Mayfield, D. G. (1968b). Psychopharmacology of alcohol: II Affective tolerance in alcohol intoxication. Journal of Nervous and Mental Disease, 146, 322-327.

McCarthy, P. D., & Walsh, D. (1975). Suicide in Dublin: I. The under-reporting of suicide and the consequences for national statistics. British Journal of Psychiatry, 126, 301-308.

McClure, G. M. G. (1984a). Recent trends in suicide among the young. British Journal of Psychiatry, 144, 134-138.

McClure, G. M. G. (1984b). Trends in suicide rates for England and Wales 1975-80. British Journal of Psychiatry, 144, 119-126.

McCulloch, J. W., Philip, A. E., & Carstairs, G. M. (1967). The ecology of suicidal behaviour. British Journal of Psychiatry, 113, 313-319.

Menninger, K. (1938). Man against himself. New York: Harvest.

Miles, C. P. (1977). Conditions predisposing to suicide: A review. Journal of Nervous and Mental Disease, 164, 231-246.

Morgan, H. G., Burns-Cox, C. J., Pocock, H., & Pottle, S. (1975). Deliberate self-harm: Clinical and socio-economic characteristics of 368 patients. British Journal of Psychiatry, 127, 564-574.

Murphy, E., Lindesay, J., & Grundy, E. (1986). 60 years of suicide in England and Wales: A cohort study. Archives of General Psychiatry, 43, 969-976.

Murphy, G. E., Armstrong, J. W., Hermele, S. L., Fischer, J, R., & Clendenin, W. W. (1979). Suicide and alcoholism: Interpersonal loss confirmed as a predictor. Archives of General Psychiatry, 36, 65-69.

Murphy, G. E., & Wetzel, R. D. (1980). Suicide risk by birth cohort in the United States, 1949-1974. Archives of General Psychiatry, 37, 519-523.

Nace, E. P., Saxon, J. J., & Shore, N. (1983). A comparison of borderline and nonborderline alcoholic patients. Archives of General Psychiatry, 40, 54-56.

National Center on Institutions and Alternatives. (1987, summer). Jail suicide update: Training. Alexandria, Virginia: Jail Suicide Prevention Information Taskforce.

Northern Territory Department of Health. (1986). Health indicators in the Northern Territory. Darwin: Government Printer.

Oliver, D. (1985). Reducing suicide in Western Samoa. In F. X. Hezel, D. H. Rubinstein, & G. H. White (Eds.), Culture, youth and suicide: Papers from an East-West Center Conference (pp. 74-87). Honolulu, Hawaii: University of Hawaii Press.

Overstone, I. M. K., & Kreitman, N. (1974). Two syndromes of suicide. British Journal of Psychiatry, 124, 336-345.

Pearce, J. M. S. (1977, August). Neurological aspects of alcoholism. British Journal of Hospital Medicine, 132-142.

Peters, R. (1981). Suicidal behavior among native Americans: An annotated bibliography. White Cloud Journal, 2, 9-20.

Pettinati, H. M., Sugerman, A. A., & Maurer, H. S. (1982). Four year MMPI changes in abstinent and drinking alcoholics. Alcoholism: Clinical and Experimental Research, 6, 487-494.

Pfeffer, C. R. (1986). Suicide prevention: Current efficacy and future promise. In J. J. Mann, & M. Stanley (Eds.), Psychobiology of Suicidal Behavior, Annals of the New York Academy of Sciences, 487, 341-350.

Pine, C. J. (1981). Suicide in American Indian and Alaskan native tradition. White Cloud Journal, 2, 3-8.

Polloi, A. H. (1985). Suicide in Palau. In F. X. Hezel, D. H. Rubinstein, & G. H. White (Eds.), Culture, youth and suicide: Papers from an East-West Center Conference (pp. 125-138). Honolulu, Hawaii: University of Hawaii Press.

Poole, F. J. P. (1985). Among the boughs of the hanging tree: Male suicide among the Bimin-Kuskusmin of Papua New Guinea. In F. X. Hezel, D. H. Rubinstein, & G. H. White (Eds.), Culture, youth and suicide: Papers from an East-West Center Conference (pp.152-181). Honolulu, Hawaii: University of Hawaii Press.

Reid, J. (1983). Sorcerers and healing spirits: Continuity and change in an Aboriginal medical system. Canberra: Australian National University Press.

Rich, C. L., Ricketts, J. E., Fowler, R. C., & Young, D. (1988). Some differences between men and women who commit suicide. American Journal of Psychiatry, 145, 718-722.

Robins, E. (1986). Psychosis and suicide. Biological Psychiatry, 21, 665-672.

Robins, L. N., & Kulbok, P. A. (1986). Methodolgical strategies in suicide. In J. J. Mann, & M. Stanley (Eds.), Psychobiology of Suicidal Behavior, Annals of the New York Academy of Sciences, 487, 1-15.

Rubinstein, D. H. (1983). Epidemic suicide among Micronesian adolescents. Social Science and Medicine, 17, 657-665.

Rubinstein, D. H., & King, J. (1981). Suicide: An act of impulse, an act of despair. Pacific Magazine, 6, 28-30.

Sainsbury, P., & Jenkins, J. S. (1982). The accuracy of officially reported suicide statistics for purposes of epidemiological research. Journal of Epidemiology and Community Health, 36, 43-48.

Schuckit, M. A. (1973). Alcoholism and sociopathy: Diagnostic confusion. Quarterly Journal of Studies on Alcohol, 34, 157-64.

Schuckit, M. A. (1983). Alcoholic patients with secondary depression. American Journal of Psychiatry, 140, 711-714.

Schuckit, M. A. (1986). Genetic and clinical implications of alcoholism and affective disorder. American Journal of Psychiatry, 143, 140-147.

Shore, J. H. (1975). American Indian suicide: Fact and fantasy. Psychiatry, 38, 86-91.

Shore, J. H., Bopp, J. F., Waller, T. R., & Dawes, J. W. (1972). A suicide prevention center on an Indian reservation. American Journal of Psychiatry, 128, 1086-1091.

Shore, J. H., & Manson, S. (1983). American Indian psychiatric and social problems. Transcultural Psychiatry Research Review, 20, 159-180.

Tonkin, R. S. (1984). Suicide amongst British Columbian adolescents. Journal of Adolescent Health Care, 5, 172-177.

van Praag, H. M., Plutchik, R., & Conte, H. (1986). The serotonin hypothesis of (auto)aggression: Critical appraisal of the evidence. In J. J. Mann, & M. Stanley (Eds.), Psychobiology of Suicidal Behavior, Annals of the New York Academy of Sciences, 487, 150-167.

Walsh, B., Walsh, D., & Whelan, B. (1975). Suicide in Dublin: II. The influence of some social and medical factors on coroners' verdicts. British Journal of Psychiatry, 126, 309-312.

Weissman, M. M., Myers, J. K., & Harding, P. S. (1980). Prevalence and psychiatric heterogeneity of alcoholism in a United States urban community. Journal of Studies on Alcohol, 41, 672-681.

White, G. M. (1985). Suicide and culture: Island views. In F. X. Hezel, D. H. Rubinstein, & G. H. White (Eds.), Culture, youth and suicide: Papers from an East-West Center Conference (pp. 1-14). Honolulu, Hawaii: University of Hawaii Press.

Yates, A. (1987). Current status and future directions of research on the American Indian child. American Journal of Psychiatry, 144, 1135-1142.

Chapter 10. Alcohol, Suicide and Self-Harm: A View on the Relevance of Empirical Research to Clinical Situations

John Ellard

1. Introduction

My role as a clinical psychiatrist will be twofold: firstly, and more importantly, I shall endeavour to consider the relevance and usefulness of empirical research when it is directed at clinical questions; secondly, I shall indicate those papers which may be relevant. I intend to show that even though a literature on this topic exists, it might not be significant. Also, a distinction is to be made between critical thought and following the usual conventions of research.

2. Problems of the empirical approach

It is often assumed that the 'scientific' way of investigating complex behaviour is to divide it into fragments with the intention of so limiting the number of variables that they can be controlled. Those fragments are then researched and subsequently recombined into the complex behaviour first encountered, in the belief that the results obtained in this way can be applied to it. To do this is to exhibit faith, and not logic, for there is no proof of the validity of such a view. The attributes of people are not logically independent, but interactive; furthermore there may be no specifically identifiable entities as required by research of this kind.

Some of the problems the literature presents to the clinician are discussed in the following sections.

2.1 Meaning

Much clinical work is a pursuit of meaning. Consider two suicides. The first is that of a middle aged medical practitioner who learns that she is suffering from widespread cancer, and that it is very probable that she is about to become quadriplegic. She enjoys some of her best champagne and then injects herself with a lethal dose of morphine. The second is that of a young woman who encounters a crisis, gets drunk and then - ambivalently - takes a very large dose of paracetamol. She is not aware that it can cause liver necrosis, but it does. Both are unconscious on admission to hospital, with identical blood alcohols, which a researcher duly notes.

The clinician is concerned with the meaning of their actions. The search for meaning is a complex topic and if pursued firmly can take one back to a consideration of Platonic essences. Empirical research throws little light on the meaning of individual acts. Similar issues arise when one considers different populations. The suicide of an adherent

of Bushido will have a different meaning to the suicide of a member of the Society of Jesus; Suttee will be different again.

2.2 Limiting the number of variables

As stated above, there are many questions about how valid it may be to adopt this as a general method of enquiry. Problems remain even when an apparently suitable variable can be identified. For example, if one places pilots in a Link trainer it is possible to show that there is a linear correlation between a rising blood alcohol concentration and the errors made (Henry, Davis, Engelken, Triebwasser, & Lancaster, 1974). If one now considers flying *in vivo*, and adds things such as the experience and anxiety of the pilot, the amount of cloud and turbulence, the proximity of mountains and other aircraft, the complexity of the instrumentation and the pilot's familiarity with it, and many other factors which are known to be connected with crashes, then it is difficult to say more than it is unwise to drink and then fly and that the degree of unwisdom is a function of the amount drunk. There can be few who did not know this before the experiment was performed, and their total knowledge about *in vivo* situations will be much more useful than the research results.

In short, the search for experimental precision may produce quantitative results, but clinical triviality.

2.3 The unsatisfactory nature of some procedures

2.3.1 Questionnaires

Every day the clinical psychiatrist sees patients who begin by asserting one thing and who then, on reflection, assert something very different. That process of reflection may extend over weeks or months. While questionnaires may be reliable, there are serious doubts about the validity of the information obtained in this way. There are also questions of meaning. Many patients are uncertain about the meaning of words used in questionnaires. Such confusions may not be merely questions of definition; for example, many patients initially describe themselves as depressed, and then observe that their behaviour reflects anger, rather than depression.

The difficulties and inaccuracies which can surround the use of questionnaires are exemplified in Kortmann and Horn's (1988) examination of the performance of the WHO-designed Self Reporting Questionnaire when used in Ethiopia. It is intended to act as a culturally independent psychiatric case finding instrument. Some of their findings were:

* 40% of the 'yes' answers were ruled invalid.
* the lowest scores were given by psychotic patients.
* the highest scores were given by malingerers.

They also observed that the cut-off point of significance varied widely: someone giving four 'yes' answers in the Sudan was classified as psychiatrically ill, while someone answering 'yes' ten times in Columbia was classified as healthy.

2.3.2 Retrospective data

Since it takes a long time for many of the clinical processes to unfold it is common to use retrospective enquiry as a means of acquiring data. Such studies need critical examination, for they are often associateed with error. Some of the errors are:

* The observed group may not be representative of the total relevant population.
* It is difficult to obtain accurate data about the prior characteristics of the experimental group.
* It is difficult to set up a proper control group.
* Recall bias is likely - people who acquire a chronic disease are more likely to examine their past for possible causes.
* Embarrassment, shame or public duty will influence the reports given by the subjects.
* The symptoms of the disease in question may cause the sufferer to react (e.g., take more Aspirin), which consequently may emerge as a possible cause of the disease.
* The act of supervision may determine the result (e.g., women taking oral contraceptives need to see their doctors for prescriptions, and therefore symptoms such as headache are more likely to be recorded).

2.3.3 Dredging for correlation
An array of data is assembled to test one or more hypotheses. When this is done, the array is further inspected for correlations not anticipated when the experiment was designed. If a sufficiently large number of correlations is examined, it is very probable that some of them will achieve statistical significance as an artefact of statistical method.

2.3.4 The clinically important data are ignored
Empirical research is generally concerned with things done to a person; the clinician is generally interested in things done by a person. Even when the researcher is inclined to deny it the intentions, powers and interests of those participating are relevant. One knows that a psychologist is researching pigeons, rather than the pigeons researching the psychologist, only by a consideration of such motives, and not by counting the pecks made by the pigeons nor hand movements made by the psychologist.

2.3.5 Technical problems in laboratory research
This issue is discussed extensively by Champion (1978). Some of the problems are:

* inconsistent use of technical terminology within the ranks of laboratory investigators.

* inconsistent use of terminology when results move from the laboratory to the clinical field.

* inadequate description of procedures, making the results difficult to interpret.

* use of procedures in the control group which make it 'active', that is, it is no longer a valid control group.

2.3.6 Statistical errors
While the clinician claims no statistical expertise, he or she is aware that statistical errors are common. Thus in Davies' (1987) survey of 29 articles applying statistical methods to data generated by human subjects in the Australian and New Zealand Journal of Psychiatry and in the American Journal of Psychiatry, 23 papers contained at least one error, and ten categories of error occurred.

2.3.7 The poor quality of some clinical research
An important and commonly researched matter is the treatment of depression. In order to research the research into this topic the relevant studies reported up to June 1982 were assembled ("A Treatment Outline," 1983). Four hundred and sixty-four papers were self described as controlled trials. On inspection 264 were rejected, 29% because the data were insufficient to provide a quantifiable result, 32% for not being properly controlled, 25% for having non-depressed subjects and 14% for other deficiencies. Only 57% of the studies offered criteria for the principal diagnostic decisions made.

The median period of observation in the accepted studies was four weeks; only one in eight had observations made over a period of three months or more. From his daily work the clinician knows that it may take many weeks before he or she can be confident that a patient's depression has departed. Indeed, there is often an initial transient period of improvement due to powerful non-specific influences. To accept that as an important and lasting change within the patient is to fall into error. In short, of the original 464 papers, a clinician might turn to 25 with some interest, but even then other problems remain. For example, one paper reported excellent results obtained by using medications long since discarded as quite useless.

2.3.8 Other failures in technique
In 1984 Dr. Shapiro of the University of California, Los Angeles, used the Freedom of Information Act to obtain the results of 964 routine audits of research carried out by the Food and Drug Administration, USA between 1977 and 1983

(Sutherland, 1984). Only 16% had no deficiencies. Forty-eight percent had deficiencies related to consent, but there were other matters more relevant to this review. Twenty-three percent failed to adhere to protocol and 22% had inaccurate, unavailable or unbelievable records. Detailed investigations resulted in the disqualification of 33 investigators and the restriction of 10. It was observed that the rate of investigator delinquency was rising, not falling. Shapiro attributed what he found to four main motivating factors:

* economic fraud - getting funds and doing no work
* arrogance - disregarding rules
* academic fraud - altering results to achieve publication.
* incompetence.

2.3.9 Summary

In short, the clinician, well aware of the subjective and pragmatic way in which he has to proceed, is not disposed to accept the findings of empirical research at face value, nor as necessarily relevant to his every day work. It may help to examine a particular clinical problem in a little more detail.

3. Research into dangerousness

Suicidal behaviour is a special case of dangerous behaviour. Dangerousness has been researched many times, for if dangerous behaviour could be predicted then some aspects of social policy could be based upon such predictions, and sentencing could be given a more objective basis.

Some results of this research have been:

* there is no agreed definition of dangerousness. For example, the research did not consider such very dangerous people as manufacturers who continued to use asbestos at a time when its dangers were known. Similarly drunken and dangerous drivers were not considered in the relevant papers.
* one can determine the results by choosing the population. Thus if one takes a very large random sample of people, there will be so few dangerous members in it that a prediction of non-dangerousness is very likely to be correct. If those already convicted are examined, attempts at predicting dangerousness produce a large number of false positives.
* studies of personality help very little.
* the presence of mental illness is of little significance.

* procedures which diminish dangerous behaviour in one environment have no effect in another. Therapeutic community style units in prisons diminish violence in prisons, but have no effect on post-release behaviour. The social ambience is more important than factors within the individual.

* a simple actuarial measure - the number of previous convictions for violence - provides the best prediction for future violence.

* even if one could predict dangerousness there are problems of policy and morality one cannot solve empirically.

4. Some notes on the published literature

4.1 The relationship between alcohol and criminal conviction

A measure of the variation which can occur from place to place is to be found in "Alcohol in Australia" (1979) which records that in 1972 in magistrates' courts convictions for drunkenness ranged from 4.5/1000 of the whole population in the ACT to 91.9/1000 in the Northern Territory. Approaching the question from another direction Gorta and Hunter (1985) record that during the period of their survey more Aborigines (47%) than non-Aborigines (30%) admitted to having a drinking problem before coming to prison (more non-Aborigines (24%) than Aborigines (11%) admitted to a drug problem). Subsequent changes in legislation have meant that no later figures are available.

Two other surveys are of interest. McLean (1988) administered the Michigan Alcoholic Screening Test (MAST) to 129 male and 102 female prison inmates in New Zealand. Using the usual norms, 71% of the males and 75% of the females were classified as 'alcoholic'; revised norms still classified 50-60% of the population as 'alcoholic'.

White and Boyer (1985) administered a shortened version of the same test to all sentenced men and women admitted in 1983 to Tasmania's only prison at Risdon. The shortened test indicated alcoholism in 43.5% of the prisoners and an additional 15.2% were classified as possibly alcoholic. Two hundred and sixty-eight male prisoners (60.9%) had drink driving convictions.

Allowing for problems of definition and many other compounding factors it is reasonable to conclude that a significant relationship exists between excessive alcohol consumption and imprisonment.

4.2 The relationship between alcohol and suicide

Miles (1977) reviewed the literature to that time concerned with conditions contributing to suicide. He presented (p. 234) a table of 15 studies reporting the incidence of suicide

in alcoholic populations; the figures range between 3% and 56%. Miles' own estimate was that perhaps 15% of all alcoholics will suicide, but it is my opinion that one could choose a figure to suit one's purpose.

Lindsay (1978) analysed 26 Australian studies of attempted suicide and 12 of completed suicide published in the preceding decade. Excessive drinking and suicide were frequently associated (male 13-52% and female 9-16%). These estimates are not too large, for Burvill's (1971) analysis of 114 West Australian suicides in the year 1967, using figures derived from Coroner's records, showed that 14% of male suicides and 9% of female suicides reveal clear evidence of alcoholism or heavy drinking, "although in most cases the records did not contain sufficient information on this point" (p. 40).

It is reasonable to conclude that a significant relationship exists between excessive alcohol consumption and suicide.

4.3 The relationship between alcohol and depression

It is a matter of common observation that many people who drink to excess become depressed - as many as one in two or three (Schuckit, 1983). It should be noted that this may not be much higher than the figure for a Sydney middle class population, in which while 2% of men and 7% of women reported frequent prolonged dperession, five times that number reported frequent brief depression (Reynolds & Rizzo, 1979).

Sometimes the reasons for depression are self evident (the physical and social consequences of alcoholism can be very burdensome). Sometimes there is a family history of depression, raising the question of a primary biological affective disorder manifesting itself partly as alcoholism. One can be miserable because one drinks, or drink because one is miserable - or both. Enquiry is made difficult by many alcoholics being addicted to other substances which also affect mood. Thus in the San Diego Study (Rich, Fowler, Fogerty & Young, 1988) of 204 consecutive suicides 101 were substance abusers and of these only 12 restricted themselves to alcohol alone, and 12 to other substances alone. Schuckit's (1986) survey makes it clear that little more has been done than noting the association and listing the possibilities.

There is ample evidence to demonstrate that both alcoholism and depression are familial, and that genetic factors are important. No evidence, however, has been produced to demonstrate a close linkage between the two (Spring & Rothgery, 1984). In any case no one knows what it is that is inherited that predisposes to alcoholism and it is emerging that clinically similar depressive disorders represent several genotypes (Pauls, 1988). An excellent review of current information concerning the genetics of alcoholism is given by Goodwin (1988).

4.4 The relationship between alcohol and anxiety disorders

It has long been observed that a person suffering from an anxiety disorder is at increased risk of becoming dependent on sedatives and alcohol. Judd, Burrows, and Hay (1987) provide an extensive review of the literature relevant to this observation and of the more general issues concerning the genetics of the anxiety disorders and the wider relationships between those disorders, alcoholism, and depression. It is possible that any one may be a cause or effect of any of the others, directly or recursively. Nutt (1988) provides another review and an extended critical bibliography.

4.5 The relationship between alcohol, prisons and suicide

Once more there are difficulties about the significance of the data. For example, in 1953 Partridge suggested that in the United Kingdom more men and women suicide after committing murder than reach Broadmoor gaol after being convicted of murder. Twelve years later, West (1965) published a thorough study of murder followed by suicide which described the several populations involved.

There are also large differences in suicide rates among prison systems. The figures of Hatty and Walker (1986) quote prison suicide rates for the United States, Austria, Denmark and Belgium as respectively three times, twenty times, twenty-six times and forty-seven times the relevant community figures. Their own research, conducted on official correctional statistics, provides an up to date review of Australian data; there is no mention of alcohol in their study presumably because there was none in the correctional data.

Clark (1985) reported the figure for New South Wales as 90:100,000 inmates/year (eight times the general rate) until 1983, when it rose to 400:100,000; a similar rise occurred in Victoria (500:100,000). He stated that most suicides were male, relatively older than the general population, serving a long sentence for a violent crime and often from another culture. A significant number were psychiatrically ill; there was often a clear precipitant.

Suicide rates also differ between prisons and police cells. Hayes (1983) reported that individuals held in police lock-ups and county jails in the USA were sixteen times more likely to suicide as individuals in the general population; the lock-up and jail rate is more than five times the reported prison rate.

4.6 Suicide, hopelessness and loss

It has long been recognised clinically that there is an association between believing that one has no future, hopelessness, and suicide. Investigations performed on people who have harmed themselves but survived (Dyer & Kreitman, 1984; Melges & Weisz, 1971) support such a view.

In the case of alcoholics, loss of an important relationship within six weeks or less of death has been found to be an important predictor of suicide (Murphy, Armstrong, Hermele, Fischer & Clendennin, 1979).

4.7 Summary

In summary of sections 4.1 - 4.6 it is reasonable to conclude that people who drink heavily have an increased chance of receiving a prison sentence, and that on the basis of their increased proclivity to suicide outside prison that tendency may have remained with them in prison and have been exacerbated by the experience of conviction and imprisonment. Moreover, alcoholics who suffer the loss of an important relationship are more likely to suicide.

5. Conclusion

There is a relationship between alcohol, imprisonment, suicide, depression and anxiety. The exact nature of the relationship is unclear, and in any event is likely to vary substantially from case to case, and population to population.

There is at present no satisfactory scientific method of studying the significance and meaning of the acts of individuals in a social context, and it is unlikely that more satisfactory methods will become available in the foreseeable future.

References

A treatment outline for depressive disorders. The quality assurance project (1983). Australian and New Zealand Journal of Psychiatry, 17, 129-146.

Burvill, P. H. (1971). Suicide in Western Australia, 1967: An analysis of coroner's reports. Australian and New Zealand Journal of Psychiatry, 5, 37-44.

Champion, R. A. (1978). Some problems in the translation from laboratory research to behaviour therapy research. In J. P. Sutcliffe (Ed.), Conceptual analysis and method in psychology (pp. 147-158). Sydney: Sydney University Press.

Commonwealth Department of Health. (1979). Alcohol in Australia. Canberra: Australian Government Publishing Service.

Davies, J. (1987). A critical survey of scientific methods in two psychiatric journals. Australian and New Zealand Journal of Psychiatry, 21, 367-373.

Dyer, J. A. T., & Kreitman N. (1984). Hopelessness, depression and suicidal intent in parasuicide. British Journal of Psychiatry, 144, 127-133.

Goodwin, D. W. (1988). Genetics of alcoholism. Current Opinion in Psychiatry, 1, 317-322.

Gorta, A., & Hunter R. (1985). Aborigines in NSW prisons. Australian and New Zealand Journal of Criminology, 18, 26-40.

Hatty, S. E., & Walker, J. R. (1986). A national study of deaths in Australian prisons. Canberra: Australian Institute of Criminology.

Hayes L. M. (1983). And darkness closes in: A national study of jail suicides. Criminal Justice and Behaviour, 10, 461-484.

Henry, P. H., Davis, T. Q., Engelken, E. J., Triebwasser, J. H., & Lancaster, M. C. (1974). Alcohol-induced performance decrements assessed by two Link trainer tasks using experienced pilots. Aerospace Medicine, 45, 1180-1189.

Kortmann, F., & Horn, T. H. (1988). Comprehension and motivation in responses to a psychaiatric screening instrument. British Journal of Psychiatry, 153, 95-101.

Judd, F. K., Burrows G. D., & Hay, D. A. (1987). Panic disorder: Evidence for genetic vulnerability. Australian and New Zealand Journal of Psychiatry, 21, 197-208.

Lindsay, J. S. B. (1978). Australian suicidology. Australian and New Zealand Journal of Psychiatry, 12, 175-180.

McLean, A. (1988). Screening for alcoholism in New Zealand prison inmates. Australian and New Zealand Journal of Criminology, 21, 45-47.

Melges, F. T., & Weisz, A. E. (1971). The personal future and suicidal ideation. Journal of Nervous and Mental Disease, 153, 244-250.

Miles, C. P. (1977). Conditions predisposing to suicide. Journal of Nervous and Mental Disease, 164, 231-246.

Murphy, G. E., Armstrong, J. W., Hermele, S., Fischer, J. R., & Clendenin, W. W. (1979). Suicide and alcoholism. Archives of General Psychiatry, 36, 65-69.

Nutt, D. J. (1988). Anxiety and alcohol. Current Opinion in Psychiatry, 1, 330-334.

Partridge. R. (1953). Broadmoor: A history of criminal lunacy and its problems. London: Chatto and Windus.

Pauls, D. L. (1988). Genetics of bipolar affective disorders. Current Opinion in Psychiatry, 1, 41-45.

Reynolds, I., & Rizzo, C. (1979). Psychosocial problems of Sydney adults. Sydney: Health Commission of New South Wales.

Rich, C. L., Fowler, R. C., Fogarty, L. A., & Young, D. (1988). San Diego - suicide study. Archives of General Psychiatry, 45, 589-592.

Schuckit, M. (1983). Alcoholic patients with secondary depression. American Journal of Psychiatry, 140, 711-714.

Schuckit, M. A. (1986). Genetic and clinical implications of alcoholism and affective disorder. American Journal of Psychiatry, 143, 140-147.

Spring, G. K., & Rothgery, J. M. (1984). The link between alcoholism and affective disorders. Hospital and Community Psychiatry, 35, 820-823.

Sutherland, R. (1984, October 22). Doctors' misconduct jeopardises drug trials. Australian Doctor, pp. 22-23.

West, D. J. (1965). Murder followed by suicide. London: Heineman.

White, R., & Boyer, K. (1985). Alcoholism amongst the Tasmanian population: Research note. Australian and New Zealand Journal of Criminology, 18, 109-114.

Chapter 11: Part A. Commentary on the other sections of the report raising matters of relevance to Aboriginal people, culture and health

Patricia Fagan and Pat Swan

Aboriginal Medical Service Redfern, New South Wales
An Aboriginal Community Controlled Health Service

1. Introduction

In this section, we will outline the background to our involvement in this project, review briefly and comment on some of the findings of the literature concerning alcohol use and Aboriginal people, and comment on other sections of the report, in so far as they have an impact on Aboriginal people, our culture and health.

These issues are pertinent to the interpretation of the report's findings and relevant to the Royal Commission's concerns.

1.1 Background to this report

The background to our contribution to this report is an issue of relevance for Aboriginal people and is worthy of consideration by the Royal Commission with regard to the effective functioning of its research section.

We had difficulty in establishing clearly the definition for an appropriate Aboriginal contribution to the report. The difficulty arose partly out of the essentially technical nature of the subject matter, but more importantly it resulted from the inappropriateness of the terms of reference initially proposed for the Aboriginal contribution.

The National Drug and Alcohol Research Centre (N.D.A.R.C.) had been asked to contribute a paper to shed light on an area of relevance to the Royal Commission. In an attempt to be comprehensive, the issue of "alcohol and its effects on Aboriginal society", was included in the proposal, and this was accepted by the Royal Commission.

The Aboriginal Medical Service believes that this report is not the appropriate framework within which to comprehensively review "alcohol use and its effects on Aboriginal society".

We feel strongly that the N.D.A.R.C., expert as it is in drug and alcohol research, is not the appropriate body to be directing a review of alcohol and its effects on Aboriginal communities. Such a central and complex issue should not be covered in ten pages and tacked onto the end of a technical report. In addition, the modus operandi of the Working Party is not acceptable to Aboriginal research.

That the initial terms of reference outlined for the proposal were approved by the Royal Commission reflects a need for it to more clearly define its policies and guidelines so that its own research and that of outside consultants is better directed. The N.D.A.R.C. needed assistance in defining the nature of the relationship between the contents of the report and the needs of the Royal Commission.

Aboriginal people recognise that the philosophy, and hence the policies that guide the work of the research section are critical to the effectiveness of the Royal Commission. The Royal Commission's conclusions will be largely shaped by the direction of its research.

Therefore, its research must address the relevant issues and be conducted in a manner that is acceptable to the Aboriginal community. We do not want to see the mistakes of the past repeated.

2. Alcohol and Aboriginal people

The excessive use of alcohol is a major health and social problem in the wider Australian community. This is recognised by the National Campaign Against Drug Abuse and is supported by an examination of hospital records in every Australian state. Discussions about the "Aboriginal alcohol problem" should be conducted in the light of this reality.

However, most Aboriginal people agree that the health of too many Aboriginal people, and the lives of their families and communities are being damaged and often dominated by alcohol. Many communities believe alcohol is the most significant issue (health and social) facing them in the 1980s (Simmons, 1988).

2.1 Use of alcohol in Aboriginal communities

2.1.1 Surveys

The extent to which alcohol is used by Aboriginal people has been documented and quantified in a few communities. It is a mistake to generalise from the findings in one community to Aboriginal people nationwide. There is enormous variation in the extent to which alcohol is a part of Aboriginal community life. Many Aboriginal communities particularly in Central and Northern Australia are alcohol-free.

There is variability in the definitions employed in different surveys and the reliability of the estimates is unknown.

In Bourke, Kamien (1978) reported that of those surveyed in 1971, 90% of the Aboriginal men and 29% of the women over the age of 21 consumed alcohol, while in the same town, Harris, Sutherland, Cutter and Ballangarry (1985) reported that in their sample 69% of men and 45% of women drank. Note that these figures include people who may have one drink, once or twice each year.

In 1971, 53% of the men and 3% of the women drank more than 80 gm/day; in 1985, 28% of the men and 5% of the women drank more than 80 gm/day.

A comparison of the findings of these two surveys is consistent with the suggestion that more women and more young people drank alcohol in 1985 than in 1971.

A Survey of Drug Use Patterns in Northern Territory (N.T.) Aboriginal Communities (1986) found that 40% of the adults drank alcohol and 72.7% of these consumed more than 60gm/day. In the N.T., over 75% of non-Aboriginal adults drink alcohol, 13.6% of them more than 60gm/day.

In summary, while fewer Aboriginal adults may consume alcohol, among those that do, consumption is reported to frequently be at a level considered harmful (more than 60 gm/day).

In addition, there is a suggestion that significantly fewer Aboriginal men drink alcohol now than 15-20 years ago, and that those that do, drink less.

2.1.2 Features of alcohol use

Hazardous use of alcohol among Aboriginal people is described by many as a group phenomenon rather than being due to individual psychological disturbance (Beck, 1985). It is often recognised as "environmental" rather than "chronic" in nature (Larsen, 1979; Kamien, 1978). As such, Aboriginal people who drink heavily have a better prognosis than non-Aboriginal people who drink the same quantities. They are more likely to cease drinking altogether if the environment is conducive, for example, if they get employment. In the 1986 Drug Use Patterns in N.T. Aboriginal Communities Survey, 25.6% of those who did not currently drink, drank in the past.

2.1.3 Abstinence

Complete abstinence from alcohol is common in Aboriginal *communities, and it may be becoming more common, among the* men at least. In Bourke in the 1970s, Kamien reported that 10% of men and over 70% of women did not drink; in 1985, in Bourke, Harris reported that 31% of the men and 55% of the women were tee-totallers. More recently, in 1986 in the same survey in the N.T., 60% of Aboriginal adults were reported to not drink. This compares to 25% of non-Aboriginal adults in the N.T..

2.1.4 Alcohol and health problems

The effects of alcohol on health are mitigated by a good diet and the absence of other health problems. Among Aboriginal drinkers a poor diet and the prevalence of other health problems, for example, liver disease from hepatitis B, respiratory disease, diabetes, and hypertension (high blood pressure), may exacerbate the ill-effects of alcohol on health, both acutely and chronically.

2.2 Understanding the use of alcohol in Aboriginal communities

2.2.1 Aboriginal views

There is a burgeoning literature by psychologists, anthropologists and social workers that attempts to understand the high prevalence of a hazardous level of alcohol use in Aboriginal communities. Aboriginal people, too, have spoken and written on the subject. Some Aboriginal people emphasise the individual disease concept of alcoholism (Bryant, 1987; Carroll & Wilson, 1984; Hunt, 1984; Potter, 1985; Sigston, 1984), while at the same time incorporating a community approach to rehabilitation, for example, the Aboriginal concept of Alcoholics Anonymous (AA). However, there is a consensus in the Aboriginal community that understands the 'alcohol problem' from a community perspective, as a symptom, ultimately, a symptom of dispossession, alienation and discrimination.

We believe that the excessive use of alcohol must be seen and dealt with as a community problem rather than solely as an individual's pathology. We feel that the recognition of this is crucial to the development of a logical approach to prevention for our future generations. As is pointed out in a report prepared for the National Aboriginal and Islander Health Organisation (NAIHO):

> European professionals are absorbed in the concept of alcoholism as individual pathology, and this obscures the real issues. A move must be made (away) from the disease concept of alcoholism if change and solutions are to be forthcoming. (p. 5)

So, while recognising the need for acceptable and accessible facilities for detoxification, and the vital role that AA may play in rehabilitation for some, Aboriginal people are concerned with prevention - the implementation of long-term solutions that involve structural changes in the wider society that will empower Aboriginal people and address the underlying causes, dispossession and discrimination - Land Rights.

2.2.2 Academic views

Among the academics, single-factor cause/effect explanations for heavy alcohol use are almost universally rejected. There is now the recognition of a complex and dynamic situation in which many factors - historical, legislative, political and social - interact to produce a social milieu in some Aboriginal communities in which alcohol figures prominently.

The central role of covert and overt discrimination against Aboriginal people by the dominant non-Aboriginal society is undenied (Larsen, 1979; Hazlehurst, 1986).

Importantly, the more positive and functional role that alcohol plays in contemporary Aboriginal society is recently being acknowledged. Sharing alcohol promotes sociability and solidarity. It provides a place, an activity and a purpose. Bain (1974) considers "the culture of alcohol consumption as

a substitute for the traditional ritual life". Brady and Palmer (1984) are said to describe alcohol use as "a conscious and determined statement of an alternative to compliance with the surrounding power structure" (Hazelhurst, 1986, p. 212).

2.2.2.1 Genetic differences?
There is no evidence in support of the hypothesis that there is a genetic basis underlying the supposed differences in the effect of alcohol on Aboriginal and non-Aboriginal people. A view persists in 'white' Australian minds, however, that such differences exist. As Kamien (1978, p. 145) points out "the implication of the genetic view is that if Aborigines cannot hold their liquor, they are an inferior species to the white man, who can". Indeed, some authors suggest that it would be more appropriate to look for a biochemical difference that enables Aboriginal people to drink more than non-Aboriginal people with impunity (the next day) (Healy, Turpin & Hamilton, 1985).

2.2.3 Alcohol, drugs and human rights
Not only did the 'white man' introduce alcohol to Aboriginal people, but he then used it as a weapon of oppression and control, and as a symbol of his superiority. As writers including Barber, Punt and Albers (1988) illustrate, legislation regarding alcohol and other recreational drugs (e.g., opium) has been used against Aboriginal people for generations, making access to alcohol a political issue and a symbol of Aboriginal civil rights more generally. As Malezer (1980, p.) puts it "citizenship in the white man's society was often explained to the black man as 'having the right to go into the pub and have a beer like any other man'".

Temporarily overcoming powerlessness, social frustration, anxiety, boredom, low self-esteem, and escape from a harsh reality are cited by many authors as factors underlying alcohol consumption (Hazlehurst, 1986; Larsen, 1980; Stanley, 1981).

3. Alcohol, Aboriginal people, non-Aboriginal people and the law

While alcohol does pose real problems for many Aboriginal communities, non-Aboriginal stereotypes about the use of alcohol persist and feed the problem for Aboriginal people. Having introduced alcohol to us and then using it as a weapon against us, non-Aboriginal people now blame us for our poverty, unemployment and other problems, wrongly attributing the cause of all these ills to our 'abuse' of alcohol, discounting their contribution and responsibility.

The "Aboriginal alcohol problem" that the non-Aboriginal community perceives and acts on, is fed by racism and is based on economic, social and aesthetic concerns, not the well-being of the Aboriginal community (Brady, 1984; Simmons, 1988).

3.1 Legislative responses

The non-Aboriginal response to the situation is legislative and discriminatory and it is designed to hide the problem rather than solve it. This is well illustrated in the N.T. where strictly enforced dress regulations keep most drinking outlets "white", leaving Aboriginal people little option but to drink outside or in crowded, furnitureless surroundings with people they would otherwise avoid.

While this situation is evident in Alice Springs (Simmons, 1988), Brady (1984) comments similarly on Tennant Creek:

> [The] variety of [drinking] establishments enables the Europeans to sort themselves into chosen social and class groupings, so that they can avoid encounters with certain others (e.g., management and workers, police and public). Aboriginal people do not have the same range of (drinking) environments at their disposal. This means that grievances, annoyances and disputes may not be dissipated by a distribution between premises (i.e., in controlled, bounded environments rather than the open air) as they are with Europeans. (p. 45)

The N.T. Summary Offences Act (Section 45D) Two Kilometre law introduced in 1983, which prohibits the consumption of alcohol in a public place within two km of licensed premises, keeps the streets 'nice', 'white' and non-threatening for tourists, while increasing the chance that Aboriginal drinkers will come into conflict with the police, and encouraging them to drink in the camps, thus causing further disruption to their families.

3.2 Social repercussions

Where facilities for Aboriginal drinkers are inadequate and unpleasant, drinking becomes an outdoor, street or park activity, as well as a group activity. Aboriginal drinkers are thus more readily exposed to police attention, and more often apprehended for drinking offences.

In 1981 23.6% of the N.T. population was Aboriginal. At the same time 50% of the prison population was Aboriginal and more than 80% of the Aboriginal people in gaols were convicted of alcohol-related offences (D.A.A., 1983; ABS, 1982-1984).

Whereas an intoxicated non-Aboriginal person is likely to be driven home, an intoxicated Aboriginal person is liable to spend the night in police cells and/or be charged with an offence.

This over-representation of Aboriginal people in prisons, the majority of whom are convicted of alcohol-related offences, is not peculiar to the N.T.; it is repeated throughout the Australian states.

4. Aboriginal people, alcohol, suicide and custody

Alcohol appears frequently in the case histories of those who have died in custody. The person may have been "locked up" because he/she was drunk or because they committed an offence while intoxicated.

As Drs Ellard and Hunter point out, there is an association between the heavy use of alcohol and imprisonment, and between these two and depression and suicide. There is a relationship between Aboriginal people and a high rate of incarceration often associated with intoxication.

So we have a situation where an Aboriginal person with inadequate nutrition and probably some significant health problems (diabetes, heart disease, hypertension), struggling with a reality that creates mental health problems (e.g., anxiety and depression) is more likely to drink alcohol to excess, more likely to come to police attention, and more likely to go to gaol when he does. Once in custody, he is more likely to suffer discrimination and outright racism in the hands of his captors, more likely to be sick, less confident to ask for help, and those in charge are less likely to be sympathetic. It is inevitable that the experience of incarceration (and isolation) exacerbates the "pain" in this situation.

The case histories of those who have died in custody reveals that many of the deaths occur within an hour or two of being arrested. Withdrawal symptoms or delirium tremens are unlikely to have dominated the clinical picture surrounding these deaths. Other factors must be operating.

Many intoxicated Aboriginal people are admitted to hospitals and detoxification units or are cared for in private homes when "drying out" or sobering up. Suicides in these situations are rare. It would seem that the environment, for example, police lock-ups/gaols, is a major factor in these deaths in custody.

5. Comments on existing treatment and prevention programs

5.1 Treatment

There are many conflicting concepts held by 'white' professionals about the causes and treatment of 'alcoholism'. These 'experts' are the same people who attempt to judge Aboriginal drinking from within their own confused and conflicting framework.

Most treatment regimes are based on the disease model of alcoholism and the success of these programs to date has been measured in 'white' terms not on the basis of the needs of the Aboriginal clients. Wilson's review of treatment programs (1986) demonstrates this clearly. Two examples of where failure to meet other's expectations resulted in the

loss of funding to alcohol treatment services for Aboriginal people were the FOWAARD program in Darwin and the Aboriginal Drug and Alcohol Services in Alice Springs.

Resource providers (e.g., Department of Aboriginal Affairs) do not have insight into the social and cultural factors that are associated with alcohol use in Aboriginal communities and therefore often provide only enough resources to treat one symptom of a multi-dimensional problem.

Aboriginal communities are not homogeneous. Consequently, there cannot be an across-the-board single solution to substance abuse or a "package deal" that can be applied nationwide. Each and every community needs to look at the causes and solutions taking into account individual pathology, physical, social, psychological and cultural factors.

To date, the only rehabilitation facilities have been "all-in-together-blacks", for example, people with Korsakov's syndrome, chronic alcoholism, delirium tremens, young problem drinkers etc, all in together (Wilson, 1986). Unfortunately, even these services are inadequately resourced. Resources for education, and for programs that address the total needs of the client population and of Aboriginal health workers in this field, have been denied and are urgently needed.

The motivation for government action comes from intermittent political pressure, rather than from a commitment to effective long-term solutions for future generations. Quick, expedient gestures for Aboriginal problems are sought by government, and the commitment lasts only until media attention has eased or until the next election at best. Two examples of media-provoked action were: the response to petrol sniffing in a South Australian community which followed the televising of a documentary on the Channel 9 program 60 Minutes; and the improvement of living conditions for the people of Toomelah following the reporting of the findings of Justice Einfeld. Although requests for assistance to deal with these problems had been made long before these programs came to air, a response was not forthcoming until they were widely publicised.

5.2 Prevention

The same principle of local community involvement and control applies to the development of prevention strategies. Any strategy aimed at reducing the excessive use of alcohol by Aboriginal people must address:

- the economic and political issues that govern Aboriginal disadvantage, and
- the regulation of Aboriginal daily life with regard to the use of alcohol, as evidenced by the discriminatory legislation in force in the N.T.(Simmons, 1988).

6. Comments on other sections of the report

6.1 Clinical pharmacology of alcohol (J. Saunders), The analysis of alcohol in blood specimens (A. Hodda), Effects of alcohol on cognitive and psychomotor functioning (G. Chesher & J. Greeley), Tolerance to the effects of alcohol (G. Chesher, J. Greeley & J. Saunders)
We need only make a few points on these papers:

6.1.1 With regard to the search to identify genetic differences between races in the metabolism or other effects of alcohol:

- this endeavour diverts attention and resources away from the real issues, that is, the causes of alcoholism. We feel the resources would be better used in a preventive program that addresses the underlying needs.

- the definition of Aboriginality that would be used in such studies has little to do with the accepted concept of Aboriginality. The accepted concept is: "An Aboriginal or Torres Strait Islander is a person of Aboriginal or Torres Strait Islander descent who identifies as Aboriginal or Torres Strait Islander and is accepted as such by the community with which he/she is associated" (Castles, 1987, p. 1).

- the findings would be of limited value for Aboriginal health.

6.1.2 It is important that the stated Blood Alcohol Concentrations (BACs) are reliable, whether the specimen is collected in far western N.S.W. or in Sydney, and whether it be an ante mortem or a post mortem specimen. We support the recommendations of this report that are designed to improve the quality of the specimen and ensure the quality of the result obtained.

6.1.3 While chronic tolerance is known to occur, its nature and degree is less well documented. There is little evidence available that allows one to predict how probable it would be for an experienced drinker to perform a given task in a certain situation (for example, in a police lock-up), at a BAC known to render inexperienced drinkers unconscious.

6.2 Treatment of alcohol dependence (H. Burns)

In this paper on the treatment of alcohol dependence Dr Burns raises a number of issues relevant to Aboriginal people and those caring for intoxicated Aboriginal persons.

In Section 1: Tolerance to alcohol may develop in an experienced drinker so that a higher BAC is necessary to produce signs of intoxication than in the inexperienced drinker. However, there is no marked elevation in the lethal dose, so that respiratory depression may occur in a tolerant individual who has a high BAC but who is not displaying the

degree of intoxication that one would normally expect. The danger of respiratory depression may be greater in the presence of concomitant respiratory/cardiovascular disease, as is common in relatively young Aboriginal adults.

In Section 2: The evidence regarding the drinking patterns in Aboriginal communities is patchy, especially with regard to young people. The results of the survey cited (Williams, 1987) suggest that alcohol is used by 80-90% of the children (or their friends) and that its use starts in 13% at less than nine years of age.

However, the validity of the survey method used is questionable and leaves the results open to considerable measurement error. It is difficult to know how generalisable the findings are. In addition, as Dr Ellard states, the motivation of researchers demands attention when evaluating the findings of research.

It would be interesting to compare the drinking habits of Aboriginal and non-Aboriginal school children of the same age (year 6-10), but such a survey has not been done.

In Section 3: It is important to note that a severe withdrawal reaction from alcohol is life-threatening. Seizures which are prolonged and/or repeated can also be life-threatening. These are medical emergencies that require immediate hospitalisation.

In Section 4: The list of medical problems associated with alcohol use and which are not uncommonly seen in Aboriginal patients includes: recurrent seizures, mild and severe chronic organic brain syndromes, head injuries (including chronic subdural haematoma), alcoholic liver disease (including acute alcoholic hepatitis, cirrhosis), alcoholic cardiomyopathy, peptic ulcer disease, pancreatitis and peripheral neuropathy.

As the study at the Regional Brain Damage Unit at Royal Prince Alfred Hospital, Sydney, (cited by Mr Jackson in his paper (Other Medical Problems)) indicated, the prevalence of other medical problems in patients being assessed for suspected alcohol brain damage is high. This is likely to be the case in a population of Aboriginal people who drink heavily.

In Sections 5.1 and 5.2: While detoxification/sobering-up from alcohol does not always need to occur in a medical setting, it is important that the environment is supportive, that the patient is observed by a responsible carer and that if necessary, transfer to a medical setting can take place. Few police lock-ups fulfil these criteria.

In any event, it is definitely not appropriate that people undergo detoxification or sobering up in custody if:

- they show signs of the development of a severe alcohol withdrawal reaction or of affective mental disturbance

- the cause of their physical and mental state (e.g., ataxia, aggression, confusion) is uncertain
- other significant illness co-exists (e.g., diabetes, acute infection).

6.3 Alcohol, Suicide and Self-Harm: The relevance of Empirical Research to Clinical Situations (J. Ellard)

This paper constitutes an important caveat for researchers, clinicians and lawyers who may be seeking to find the answers to clinical problems in the laboratory or in the findings of empirical research.

The paper identifies many of the pitfalls inherent in all empirical research, and it touches on some important issues that arise in cross-cultural clinical research - the "meaning" attached to the findings of research (2.1), and the validity of the instruments of measurement (2.3.1) when applied across cultures.

An important tool in clinical research is language - verbal communication. How do researchers know if their communication is successful across the language and cultural gulf that exists between Aboriginal and non-Aboriginal? How is the validity of their measurements based on verbal communication checked?

However, cross-cultural research, in particular Aboriginal research has other difficulties which remain largely ignored by researchers.

6.3.1 Issues in cross-cultural research
Every approach to "inquiry" (to research) is based on a set of assumptions. The framework, the cultural and philosophical value system within which the research is conceived, designed and conducted comes into question in the cross-cultural situation, and it deserves examination.

Clinical research into Aboriginal people has until now remained almost entirely in the hands of non-Aboriginal researchers. The dominant culture has thus had a monopoly on research. It has the economic and political power, the academic skills and the resources. The model within which research is conducted reflects the values of the dominant culture.

The researchers define the problem to be explored and they frame the questions to be asked from their own cultural perspective. Very few recognise the assumptions implicit in that process, and in Aboriginal research, very few take the trouble to define the problem from the Aboriginal community perspective.

6.3.2 Cultural Influences on Psychiatric Research.
When speaking about psychiatric categories of diagnosis, Kleinman (1977) comments that the search for the same disorder in different cultures is not a useful basis for

cross-cultural research, as "by definition it will find what is universal and miss what does not fit in its tight parameters" (p. 4).

Further, Kleinman (1980) points out that:

> culture probably has its most profound and difficult to assess influence on psychiatry through the elaboration of conceptualisations of mental illness and psychiatric care that parade as value-neutral science but in fact represent a cultural construction of social reality that is only in part empirical, but also an admixture of professional ideology and shared cultural bias. (p. 12)

This aspect of cultural psychiatry is virtually ignored by researchers. Psychiatric research is culturally based and ideologically biased. Researchers, like psychoanalysts can go only as far as "their own complexes and internal resistances permit" (Freud).

These factors have an impact on the conclusions researchers reach, and help to explain why mental health/illness research has had little success in exploring, in usefully describing and defining the Aboriginal situation.

6.3.3 Requirements for Aboriginal research

Aboriginal research, rather than reflecting the fancy of the individual researcher, needs to become problem oriented and Aboriginal people should be defining the problems. Then causes will be better understood and the way towards treatment and prevention made clear.

The politics of research in Aboriginal communities deserves the attention of those involved. Research is frequently imposed on Aboriginal communities and they have no control over it. It rarely provides any benefit to the Aboriginal participants and is in fact used in a detrimental manner by the media and governments. Consequently, researchers are often blocked by communities, and their results are not necessarily valid.

Aboriginal communities demands that it is able to actively participate in the research process, that it be kept fully informed and that they have some "say" in how research findings are publicised and used. They insist that research be Aboriginal community controlled. Then it will be relevant and of benefit to that community. The recommendations of the 1986 Workshop into Research Priorities To Improve Aboriginal Health, in particular, the recommendations pertaining to the ethics, funding and organisation of research should be implemented. (A copy of these are included in Appendix B.)

6.4 Suicide, Alcohol, Incarceration, and Indigenous Populations: A Review (E. Hunter)

The inclusion of this section is a result of the initial terms of reference for the report being accepted by the Royal Commission. From an Aboriginal perspective, to have a

section on "suicide among indigenous people, that in particular focuses on Aboriginal people and suicide" without referring to equally relevant issues such as the brutality experienced within our prison system, is unbalanced.

While we recognise that suicide in custody is an important issue for the Royal Commission, when considered in isolation in the context of this one research report, it suggests a distorted perception of deaths in custody - that all the deaths are suicide.

6.4.1 In this paper, suicide among Aboriginal people is argued to be a recent phenomenon that is increasing in the Kimberley region of W.A., and that should be understood in the context of an increase in the proportion of deaths from external causes (accidents, homicide, suicide etc).

While this may be the case in the Kimberley, it would be unwise to extrapolate to the rest of Australia. There is little information from other regions to confirm or contrast with these findings.

In the past, suicide, as Dr Hunter describes, has been an unusual phenomenon in Aboriginal Australia. Some recent studies suggest that this is still the case (Plant, 1988, Eastwell, 1988, Gray & Hogg, 1988).

The significance for Aboriginal communities elsewhere, of the cluster of suicides in the Kimberley on which the author reports is not yet clear. In this context, it should be noted that a death that is called "suicide" is not always suicide. The Royal Commission was established to clarify the truth surrounding some deaths by "suicide", and, at the time of writing, it has not announced any of its findings.

Mortality studies generally report deaths from external causes as a significant proportion of Aboriginal mortality: Crotty & Webb N.T., 1956-1959: 12% (considerably greater than the 2-4% reported here for the Kimberley in the same period); Moodie's for different states in the 1960's ranged between W.A., 3.5% (consistent with Dr Hunter's findings) and N.S.W. 11.5%; Plant N.T., 1979-1983: 17.3%; Gray & Hogg N.S.W., 1983-1987 18%.

While the figures are suggestive, these studies cannot be compared. There is insufficient information on the basis of which to decide whether in Aboriginal Australia generally, external causes of death are increasing as a proportion of total deaths. Longitudinal studies, preferably prospective are needed to determine this.

6.4.2 The author demonstrates a clear relationship between alcohol and depression, alcohol and suicide, alcohol and incarceration and all these and indigenous people. This is useful documentation for the purposes of the Royal Commission.

However, he relates his findings of an increase in suicides and in deaths from external causes to the increased availability of alcohol for Aboriginal people in the Kimberley. From an Aboriginal perspective, this is not a helpful analysis. The logic directs our attention to the easy availability of alcohol, as the cause. As a preventive measure, some may consider the reintroduction of prohibition (as Spencer (1988) appears to suggest) directed at stemming alcohol consumption by Aboriginal people, but authorised and enforced by non-Aboriginal people. Rather than preventing deaths in custody, we believe this would serve to exacerbate the underlying causes. Alcohol is not a cause - it is a symptom.

6.4.3 The paper identifies a vulnerable sub-group of young Aboriginal men of "partial descent" who are at special risk of self-harm and who are being repeatedly incarcerated.

An Aboriginal description would be: a young, Aboriginal male, alienated from his cultural tradition, with a history of generations of loss, existing in an environment (Broome) where discrimination is evident, imprisonment a major possibility, with the knowledge that he is at the very bottom of the social, economic and political hierarchy.

We disagree strongly with the view that holds that the young men who are at risk of dying in custody are a particularly abnormal sub-group of Aboriginal people. Aboriginal suicide in custody should not be understood simply as the manifestation of individual psychopathology. If suicide is increasing, those that die represent the tip of the iceberg of Aboriginal community distress.

As Kleinman (1988) reports:

> Much of the anthropological work on suicide has indicated that it may (and often does) occur in individuals without mental illness who are under great social pressure. (p. 44)

Suicide, like alcoholism, is a symptom of a community problem for which the dominant non-Aboriginal society must take some responsibility - colonisation, dispossession, continuing oppression, and covert and overt discrimination against Aboriginal people are the responsibility of all Australians.

The individual, clinical view of the situation presented in this paper absolves the wider non-Aboriginal community. It discounts the reality of the continuing oppression of Aboriginal people. It makes the dominant culture feel comfortable and relieves the non-Aboriginal community of any requirement to examine the impact their laws and power structures have on the health and well-being of Aboriginal people.

The term "partial descent" is offensive to most Aboriginal people and it is not a useful concept. We are either Aboriginal or we are not. The increased stresses that Dr

Hunter associates with "partial descent" are in fact stresses associated with lifestyle and disruption of culture. These are the relevant factors, not degree of "descent".

6.4.4 In his conclusion, the author states that a social and historical analysis is required to gain a deeper understanding of the phenomena he describes. He goes further to say that "if a broader understanding ... is not sought, the conclusions drawn...will be without substance". We agree.

The paper's investigation is individually and clinically oriented. Having no social and historical analysis, it takes no account of the subjugation of Aboriginal people by non-Aboriginal society. Consequently, it fails to shed any light on causes or to point the way for solutions. Its thrust is to shift attention away from the political and social realities of Aboriginal life, to absolve the non-Aboriginal community and to firmly locate the problem in individual Aboriginal people.

7. Conclusions

It has been recognised that cultural destruction has been the major contributing factor to the present predicament of Aboriginal people. Though recognition of this fact has been acknowledged, it has never been accompanied by the provision of the necessary resources to develop the essential community programs.

Rather, our people have been incarcerated in gaols to give the impression of concern for their safety. If they survive gaol, they are then subject to the "revolving door" recidivism of that system.

Our people endure the most severe social pressures with the least resources to protect them.

Other important points are:

1. Aboriginal people recognise that alcohol is a significant problem in many Aboriginal communities.

2. There is a consensus in the Aboriginal community that Aborigines regards alcohol as a symptom - a symptom of dispossession, discrimination.

3. In the past, suicide has been an unusual phenomenon in Aboriginal Australia.

4. There is insufficient information available on the basis of which to decide whether suicide and external causes of death are increasing as a proportion of total Aboriginal mortality nationwide.

5. Every death that is labelled as "suicide" is not necessarily suicide. The Royal Commission into Aboriginal Deaths in Custody was established to clarify the cause of death in some of these cases of "suicide".

6. Suicide, when it does occur, may be more a sign of intolerable social pressure than individual psychopathology, especially in a community where suicide is not culturally sanctioned (eg. Aboriginal).

8. Recommendations

General

1. That the Research Section of the Royal Commission define its policies and develop explicit guidelines that will assist its own research and give direction to consultant bodies in the preparation of research reports.

2. That discussions about Aboriginal people and alcohol be conducted in the light of the reality that the excessive use of alcohol is a major health and social problem in the wider non-Aboriginal society.

Alcohol Rehabilitation Programs

3. That more resources be provided to adequately fund and staff alcohol rehabilitation programs.

4. That treatment programs for alcohol rehabilitation be based on a broader philosophy than the disease model. They need to be community-controlled and be flexible in their service provision - so they can respond to the real needs of their client population. These needs include social, cultural, educational and emotional.

Alcohol Prevention Programs

5. That resources be provided for education of Aboriginal Health Workers in the specialist area of alcohol and drugs.

6. That prevention strategies address the economic and political issues that govern Aboriginal disadvantage.

7. That legislation impacting on the use of alcohol by Aboriginal people be reviewed (eg. Two Kilometre Law in the N.T. and other local government ordinances).

Alcohol Withdrawal and Custody

8. That as a general principle, it is inappropriate that people undergo detoxification/sobering up in Police lockups or gaols.

9. That people in custody who show signs of the development of a severe alcohol withdrawal reaction or of affective mental disturbance be immediately transferred to a medical setting.

10. That if the cause of "intoxication" is uncertain, or if other significant illness coexists, the person be immediately transferred to a medical setting.

Research in Aboriginal Communities

11. That the ideological and cultural model within which Mental Health research is conducted be examined so that crosscultural research may begin to reflect the reality of the community being researched, rather than the values of the researchers.

12. That Aboriginal research be community-controlled. The community should be given the opportunity to actively participate in the design and conduct of the research. They should be kept fully informed of its progress, and they should have control over how findings are publicised and used.

13. That Aboriginal people benefit from research that is conducted in their communities. Research should be problem oriented and questions should be framed by the community so that findings may be relevant to the community's needs. Aboriginal people should have the opportunity to acquire skills from researchers during the research process.

14. That the recommendations of the 1986 Workshop into Research Priorities To Improve Aboriginal Health be implemented.

15. That Aboriginal people be given the opportunity to provide the Royal Commission and non-Aboriginal Australia with the social, cultural and historical analysis which will put issues like suicide into their proper perspective.

References

Bain, M. S. (1974). Alcohol use and traditional control in Aboriginal society. In S. Hetzel, M. Dobbin, L. Lippmann & E. Eggleston (Eds.), Better health for Aborigines?: Report of a national seminar at Monash University (pp. 45-52). St. Lucia: University of Queensland Press.

Barber, J., Punt, J., & Albers, J. (1988). Alcohol and power on Palm Island. Australian Journal of Social Issues, 23, 87-101.

Beck, E. (1985). The enigma of Aboriginal health. Canberra: Australian Institute of Aboriginal Studies.

Brady, M. (1984). A study of the impact of alcohol use on the town of Tennant Creek, N.T.. Darwin: Northern territory Department of Health, Drug and Alcohol Bureau.

Bryant, V. (1987). Female alcoholism. Aboriginal Health Worker, 11, 20-21.

Carroll, J., & Wilson, B. (1984). Bennelong's Haven celebrates its tenth anniversary. Aboriginal Health Worker, 8, 25.

Castles, I. (1987). Census of population and housing, 30 June, 1986 Census86 - Aboriginals and Torres Strait Islanders: Australia, States and Territories. (Catalogue No. 2499.0) Canberra: Australian Bureau of Statistics.

Crotty, J., & Webb, R. (1960, September 24). Mortality in Northern Territory Aborigines. Medical Journal of Australia, 2, 489-492.

Eastwell, H. (1988). The low risk of suicide among the Yolngu of the Northern Territory: The traditional Aboriginal pattern. Medical Journal of Australia, 148, 338.

Gray, A., & Hogg, R. (1988). Aboriginal mortality in far western N. S. W., 1983-1987. New South Wales Department of Health.

Harris, M., Sutherland, D., Cutter, G., & Ballangarry, L. (1987). Alcohol related hospital admissions in a country town. Australian Drug and Alcohol Review, 6, 195-198.

Hazlehurst, K. M. (1986). Alcohol, outstations and autonomy: An Australian Aboriginal perspective. Journal of Drug Issues, 16, 209-220.

Healy, B., Turpin, T., & Hamilton, M. (1985). Aboriginal drinking: A case study in inequality and disadvantage. Australian Journal of Social Issues, 20, 191-208.

Hunt, H. (1984). Alcoholism among Aboriginal people. Aboriginal Health Worker, 8, 11.

Hunt, H. (1986). Alcoholism and its treatment in Aboriginal communities. In J. Santamaria (Ed.), Proceedings of seminars and scientific sessions, 1986 Autumn School of Studies on Alcohol and Drugs (pp. 113-122). St. Vincent's Hospital, Melbourne. Melbourne: Melbourne Department of Community Medicine.

Kamien, M. (1978). The dark people of Bourke: A study of planned social change. Canberra: Australian Institute of Aboriginal Studies.

Kleinman, A. M. (1977). Depression, somatatization and the new cross-cultural psychiatry. Social Science and Medicine, 11, 3-10.

Kleinman, A. M. (1980). Major conceptual and research issues for cultural (anthropological) psychiatry. Culture, Medicine and Psychiatry, 4, 3-13.

Kleinman, A. M. (1988). Rethinking psychiatry, from cultural category to personal experience (Chapter 3). New York: The Free Press.

Larsen, K. S. (1979). Social crisis and Aboriginal alcohol abuse. Australian Journal of Social Issues, 14, 143-160.

Larsen, K. S. (1980). Aboriginal group identification and problem drinking. Australian Psychologist, 15, 385-392.

Malezer, L. (1981). The impact of drugs and alcohol use on Aboriginals: Alcohol prevention. In Man, drugs and society - current perspectives. Proceedings of the First Pan Pacific Conference on Drugs and Alcohol (pp.150-151). Canberra, Australia. Canberra: Australian foundation on Alcoholism and Drug Dependence.

Moodie, P. (1973). Aboriginal health. Canberra: Australian National University Press.

National Aboriginal and Islander Health Organisation. (1984). Report on petrol sniffing on Groote Eylandt, N.T..

Plant, A. J. (1988). Aboriginal mortality in the Northern Territory, 1979-1983. MPH Thesis, University of Sydney, Sydney.

Potter, L. (1985). Alcohol and drug use in a changing society. In K. Larkins, D. McDonald & C. Watson (Eds.), Proceedings of the Second National Drug Institute (pp.). Darwin, N.T.. Canberra: Alcohol and drug Foundation.

Recommendations: Workshop on Research Priorities into Aboriginal Health (1986, November). NH&MRC and Menzies Foundation, Alice Springs, N.T.

Sigston, R. (1984). Helping alcoholics and their families. Aboriginal Health Worker, 8, 15.

Simmons, L. (1988). Availability of alcohol in Alice Springs: The grog story. Discussion paper for Central Australian Aboriginal Congress, 1988.

Spencer, D. (19). Transitional alcoholism - The Australian Aboriginal model. In Technical Information Bulletin on Drug Abuse (no. 79, pp.15-23). Canberra: National Drug Abuse Information Centre.

Stanley, V. (1981). From Aboriginal culture to urban society. In Man, drugs and society - current perspectives. Proceedings of the First Pan Pacific Conference on Drug and Alcohol (pp.154-155). Canberra, Australia. Canberra: Australian Foundation on Alcoholism and Drug Dependence.

Watson, C. (1987). A survey of drug use patterns in Northern Territory Aboriginal communities: A preliminary report on the Top End and Katherine region communities - 1986. Darwin: Northern Territory Department of Health and Community Services, Alcohol and Drug Bureau.

Williams, M. (1987). Survey of drug and alcohol use by Aboriginal school students in N.S.W. Sydney: N.S.W. Aboriginal Educational Consultative Group

Wilson, W. (1986). An analysis of Federal Government action on the provision of effective preventive, treatment, and rehabilitative services for Aboriginal alcohol abusers. Canberra: Department of Aboriginal Affairs.

Chapter 11: Part B. Commentary on the Report

Jean Jans in Collaboration with
the Aboriginal Coordinating Council in Queensland

1. Introduction

The literature looks at the effects of alcohol on cognitive, psychomotor and affective functioning. Individual experts have developed specific papers covering 8 different aspects of the effects of alcohol on areas of which they have both scientific knowledge and by nature of their work have experience in presenting such evidence with clarity and conviction, albeit from their perspective.

As a person of Aboriginal descent, on the expert committee I represented the Aboriginal people on isolated communities in the North; with the full support of the Aboriginal Coordinating Council (A.C.C) on the proviso that copies of reports from this committee would be made available to the A.C.C. as a record of the process of consultation which took place, and that of a ready reference for their future use in line with the work of the Commission.

The topics covered were:

1. The Clinical Pharmacology of Alcohol.
2. The Analysis of Alcohol in Blood.
3. The Impairment of Cognitive and Psychomotor Functioning by Alcohol.
4. Tolerance to the Cognitive and Psychomotor Impairing Effects of Alcohol.
5. Alcohol Dependence and its Treatment.
6. Alcohol Related Brain Damage.
7. The Relationship between Suicide and Alcohol.
8. The Relevance of Empirical Research to Clinical Situations.

1.1 The Clinical Pharmacology of Alcohol

Dr John Saunders in this paper made an interesting point in stating that no differences have been found in the rate of elimination of alcohol between Australian Aboriginal people and non-Aboriginal people (white Caucasians). It is interesting that knowledge is available on the elimination process for Aborigines and not on other aspects of alcohol metabolism and possible differences between Aboriginal and non-Aboriginal (white) people.

The following quote from "Four Hundred Rabbits" - an Anthropological View of Ethanol as a Disinhibitor, a paper by Mac Marshall (1981), is relevant here.

> The Problem of Possible Differences in Biological Sensitivity to Ethanol - I would not be true to anthropology if I did not raise an issue which troubles the sociocultural learning camp, though it by no means negates the main tenets of this position. This is the whole lively - and at times highly emotional - matter of possible differences in biological sensitivity to ethanol among human breeding populations, ethnic groups, and the like.
>
> Spurred on by a controversial paper by Fenna et al. (1972), a growing number of human geneticists, biological anthropologists, and physiologists have conducted studies over the past 7 or 8 years in an effort to determine whether "the firewater myth" and related ideas have any basis in biological fact. My understanding of the literature is that the results to date remain equivocal, with no clear pattern having emerged from the various investigations that have been published. Those wishing a brief introduction to the pertinent literature are referred to Hanna (1976, 1977), Heath (1974b), Wolff (1972,1973), Zeiner (1978), Zeiner and Paredes (1978), and Zeiner et al. (1976,1977).
>
> The relevance of this ongoing debate for the issue at hand has to do with certain assumptions that underlie MacAndrew and Edgerton's (1969) review of the cross-cultural evidence against disinhibition; namely, they assume alcohol and its metabilites to have the same pharmacological and biophysiological effects on ALL human beings. For example, they state that "if alcohol were a 'superego solvent' for one group of people due to its toxic action, then this same disinhibiting effect <u>ought</u> to be evident in <u>all</u> people" (1969, p.36, emphasis in original). Furthermore, their effort (1969, p.87) to dismiss the genetic differences argument by depicting Ifaluk drinking as atypical of other Carolinian atoll communities cannot be sustained on closer inspection of the relevant historical and ethnographic evidence (see, e.g. Marshall 1976, p.115, fn. 12; Marshall and Marshall 1975, pp. 449 - 450.)
>
> Ultimately it seems to me, the sociocultural learning hypothesis will have to be brought into accord with any general patterns that derive from studies on ethnic differences in sensitivity to beverage alcohol before we will be in a position to understand the relative contributions on nature and nurture to this fascinating conundrum. (pp. 198-199)

Similar responses have been reported for American Indians but it is of note that Orientals have a very low alcoholism rate while Indians who have a similar physical reactivity have a very high alcoholism rate. This

> suggests that both cultural and psychological, as well as physical factors, need to be considered. (Kahn 1981, p. 171)

1.2 The Analysis of Alcohol in Blood

The author of this paper, Alan Hodda, points out the procedure of analysis; he also emphasises the difference in the quality of the results of analysis if samples are poorly presented.

This raises the question that unless there are accurate processes of analysing, or in fact collecting samples, the whole exercise is wasted.

In isolated Aboriginal communities, where there is no ready access to laboratories, the quality of specimens collected may prove invalid. Most laboratories with experienced staff are in large mainland cities and towns. Those Health Staff who are based on isolated communities are distanced from the experience skills and access to skills that their city counterparts would have.

The analysis of alcohol in the blood is an important aspect of the overall picture which has real implications for Aboriginal people in isolated areas, in terms of quality control and the priority given to the care of blood specimens for analysis of alcohol in blood.

1.3 Impairment of Cognitive and Psychomotor Functioning by Alcohol

This paper highlights the recognition that acute intoxication by alcohol can impair cognitive and psychomotor functioning across a range of doses and in both experienced and inexperienced drinkers. It also points out both the pharmacological and non-pharmacological factors involved.

The authors focused importance on the impairment by alcohol on people generally, rather than just on one group. I found this very helpful for the purposes of this report, because broad research facilitates the determination of possible causes rather than obscures them by a narrow focus. It also gives room for other issues to be taken into account. On the other hand, it raises the question of what efforts have been made to focus this research on Aboriginal people in the normal course for ready reference by Aboriginal workers in their efforts to determine intervention processes for their respective communities.

1.4 Tolerance to the Cognitive and Psychomotor Effects of Alcohol

It is very difficult to find literature based on this subject concerning isolated Aboriginal people. The literature on Aboriginals and alcohol tends to highlight issues such as patterns of drinking, comparative drinking with other races, general effects of alcohol, and sociological aspects of drinking, rather than on empirical research in clinical situations. I believe it is a valid question to ask why not?

1.5 Alcohol Dependence and its Treatment

This paper highlights different treatment methods for people who are physically dependent on alcohol. One of the key issues, as I see it, for isolated Aborigines is that it states that "[a] safe and caring environment should be provided for individuals sobering up from alcohol intoxication".

The paper, I feel, discusses treatment as a tool understood by the carers, as it should be. It is important to make this comment to draw attention to the fact that treatment and the understanding of what treatment is required may be confined to centres where stimulation from exposure and practice of the principles of treatment for alcohol dependent persons is a regular occurrence.

Those who are confronted with the need, for example, for detoxification intervention in an isolated situation, and who have manpower to implement this intervention, may not necessarily understand the principles involved, or have the means to put it into effect.

"We cannot disguise the fact that most of our suicides in custody have been related to alcohol" (Powder and Law, 1987, p. 3).

I agree with the argument that "intervention with controlled drinking as the goal has important potential applications at each level of prevention".

We need to understand more about how Aboriginal people perceive alcohol, and its use and abuse, prior to responding, rather than responding in a reactionary way with inappropriate interventions.

1.6 Alcohol Related Brain Damage

The absence of reporting on, and specific studies of, alcohol-related brain damage in Aboriginal people, makes it difficult to look at the total effect of alcohol on Aborigines. This leaves researchers and others to draw conclusions from literature dealing with such things as patterns of drinking and the sociological effects of alcohol.

It would be useful to determine whether differences in susceptibility to brain damage exist between binge and steady drinkers.

1.7 The Relationship between Suicide and Alcohol-Suicide and Indigenous Populations

Since the term "Aboriginal" embraces a multiplicity of types, backgrounds, and levels of advancement, there can never be one cause or one answer. Each community requires the skills, knowledge and understanding to identify its own problems and

to use its own resources effectively to deal with those problems with approaches that best fit their local needs and conditions.

The erosion of traditional culture and its effect on Aboriginal people is widely recorded. Attempts are being made to piece together important aspects of the culture by both Aborigines and governments. I perceive the problem now as not in the fact that the culture has been lost, but rather in the fact that elder members of the community see the regaining of it as the answer to their present situation, whilst younger members do not see it as having any relevance. This results in a dilemma - confusion on the part of the young as to whether they should follow the lead backwards of their elders, or find their answers in modern standards, with many resolving this conflict by lapsing into substance abuse, mainly alcohol - resolving/avoiding.

Another aspect of suicide is that of belief in "Spirits". "In relation to the suicide committed we came across the impact of Aboriginal 'spirit' who came from the Northern Territory and informed people that someone will die in the cells. So when the suicide happened no one was very surprised. This was the community where the impact of the spirit was very strong and authorities should take note of this" (Powder and Law 1987, p. 14).

1.8 The Relevance of Empirical Research to Clinical Situations

I would agree with the points made in 8.2 of the conclusions.

2. Conclusions

Taking into account the important clinical effects of alcohol on cognitive, psychomotor and affective functioning presented, it is important to encompass the various social courses and look at the dynamics of the relationships between all of these factors including :

1. The relationship between the police and Aboriginal people.

2. The sociological aspects of drinking including :

   - overcrowding of homes
   - unemployment
   - in some cases, transitional changes from one culture to the next
   - the lack of educational achievement
   - in many cases, discrimination
   - the question of who is in control of the community
   - skills training

I would like to elaborate on these two factors of social courses which have a bearing on community members' self perceptions in relation to their level of alcohol consumption, and its link with where both healthy and unhealthy Aboriginal people are at within their communities.

2.1 Police/Aboriginal Relations:

On isolated Aboriginal communities there are two tiers of police enforcement. The philosophy of this needs to be understood by everyone - prior to addressing the value and contribution both made in an enclosed environment.

Training:

The State Police have the statutory authority which gives them the responsibility for all legal matters in society where they have a legitimate organization from which to work. They are the recognized legal authority because of the training and ultimate qualification they received as members of the State Police Force, principally because of their legal authority.

Queensland Aboriginal Community Police - on the other hand have no such authority, no training, no legitimate organization to turn to. They have the added difficulty of enforcing the law on their own people, of being the 'go between' in terms of assisting the State Police to understand their own people, and yet have no equivalent studies for qualifications to further their own careers. Their duties often conflict within this dual role.

The responsibility often lies with the State Police to provide "in service" training to Aboriginal Community Police as they work side by side at the community level, whereas the State Police have a State accepted curriculum, taught within the academically accepted framework prior to qualifying for that ultimate title of "police officer".

This further exacerbates the issue of Police/Aboriginal relationships - when you have Aboriginal Police/Aboriginal relationships developing - the authority line still lies with the legally appointed statutory police, with the Community Aboriginal Police still powerless to change the situation where it really counts. It leaves them and their people at a confused level; which causes more divisions.

2.2 Who Is In Control of the Community at Community Level:

Goodwill, Respect and Practise:

There doesn't seem to have been, since the destruction of Aboriginal Society, a method of teaching and learning which really enhances local customs, traditions and beliefs.

- Outsiders look down on the beliefs as being unscientific and for the most part worthless in a changing world.

- They tend to glamourize Aboriginal customs and traditions as being the only way of dealing with issues.

- Or they don't recognize Aboriginal customs, lores, traditions at all.

In reality, Aboriginal ways, like non Aboriginal ways, have strengths and weaknesses. People who are trainers, whether they are in the Administrative, Community Development or Health area, need to help Aboriginal people to look carefully and critically at both the Aboriginal ways and the non Aboriginal ways in order to avoid what is detrimental and preserve what is best in each. Emphasis must be placed on what is best. This way ensures that Aboriginal people can increase their confidence in their own knowledge; increase their experience and ability to meet the needs themselves. They will be more prepared to enrich their own culture, by learning new ones. Of the three mistakes made in teaching about traditions, the first is the worst. There is the real danger of those community Aboriginal people already involved in positions to become full of new ideas such that they lose respect for their own traditional laws. When this happens they tend to make their own people feel small and ashamed: as a result people lose their confidence, and divisions occur within.

The trainer should emphasise first and always, what is valuable in Aboriginal tradition. This person has a large area of responsibility as advisor (it is a 'power position') of exploring ways that build on Aboriginal beliefs rather than ignoring or rejecting them. This should not be a position of power but one of someone who guides, with the ultimate aim of "doing themselves out of a job well done".

The trainers, irrespective of what Department they belong to, when based on Aboriginal communities, tend to have as a priority their Department's policy to follow. This is important, but should be carried out in terms of <u>why</u> they are based on an Aboriginal community - functioning within the structure, and recognizing and drawing on resources available to that community. The trainer should be more concerned with the process of learning being passed on.

Some examples of the contents in having <u>systems</u> to follow when an outside system is brought in by a trainer to a community are: "making sure the mail is collected at 9 a.m."; "stopping for lunch at 12 m.d."; "finishing work at 5 p.m." This rigid approach is perhaps suited to a town or city which requires such a system to operate due to its effect on the wider community. On a small isolated Aboriginal community, one needs to take into account the different environmental and social structures, rather than try to create an unnatural community.

In an era of deeds of grant in trust (such as occurs on Aboriginal Communities in Queensland) which means power is given to that Aboriginal Council to make their own decisions for the community - is the good will, high respect and practised skills included in this new responsibility? Did the trainer leave, when it was the right time to go? Will the community and its people progress to an accomplished independence because of the special skills the outsider was able to pass on to that community?

The trainer should be more concerned with the process of a total learning being passed on, rather than about just the job getting done. The trainer must be alert to the need to explain why the system works in a particular way as distinct from simply saying this is the way it must be done. Community Aboriginal people in turn will thus have the confidence to question such systems and their relevance for the community. This process of teaching and learning facilitates community growth and enhances the development of <u>self</u> responsibility.

3. Communication

Communication skills are essential components of any training program. The ability to transfer these skills is a skill within itself, and must be a prerequisite for an advisor/trainer to work on Aboriginal communities.

The art of communication is a learned skill, which I believe has implications when appointing outside advisors to Aboriginal communities. They need to have communication skills to work effectively with Aboriginal people on isolated communities and the people in turn must have a willingness to learn for any change to occur.

Here I am not just talking about people talking to each other, but about the processes of communication which have implications for positive lifestyle and behavioural changes, and the development of people within their community.

One illustration of effective communication is that it is a strategy which allows people to <u>raise</u> issues, and <u>work</u> through them, rather than <u>ignore</u> them; it can prevent people looking for alternative ways (usually alcohol) to deal with situations of conflict.

<u>Who Communicates With Whom?</u>
<u>Is there expectation of feedback?</u>

Often the usual flow of communication is between outsiders and council. The missing link is the flow of communication to the people, which means there is no information received, no feedback is expected, and decisions are made on an uninformed basis.

Here is an obvious gap between the decision makers and the community members, which causes distrust, and a feeling of "lack of ownership" of community issues both negative and positive.

Quality communication, when provided, works towards demystifying issues of concern to everybody thus giving people choices, and allowing them to question things such as: why systems are put in place and why their contribution plays an important role in the failure/success of their community. Communication is a two way process and effective strategies are needed to make this a reality.

1. Each community needs to find its own solutions to its own problems. There are no easy or "universal" answers that can be brought in from outside.

2. Human factors (more than technical ones) are what make community activities fail or succeed.

3. To serve those whose needs are greatest, community programs must make every effort to help the weak gain and keep control, not just control of wider political efforts, but of their own personal lives.

It is also important to recognize that Aboriginal communities are within themselves institutions. This has required from people who live on them massive efforts of adjustment. This causes great tensions which can be very difficult for people to handle. This situation is not easy for outsiders to understand.

References

Barber, J. G., Punt, J., & Albers, J. (1988). Alcohol and power on Palm Island. Australian Journal of Social Issues, 23, 87-101.

Beyond these walls - Report of the Queensland Violence Task Force - October 1988, to the Honourable Peter McKechnie, M.L.A., Minister for Family Services and Housing.

Brady, M., & Palmer, K. (1984). Alcohol in the outback: Two studies of drinking. Darwin: Australian National University North Australia Research Unit.

Johnstone, D. (1986). Women's business - Report of the Aboriginal women's task force.

Kahn, M. W. (1981). Basic methods for mental health practitioners. Cambridge, MA: Winthrop Publishers.

Marshall, M. (1981). "Four Hundred Rabbits": An anthropological view of ethanol as a disinhibitor. In R. Room & G. Collins (Eds.), Alcohol and disinhibition: Nature and meaning of the link (Research Monograph No. 12, pp. 186-204). Rockville, MD: National Institute on Alcohol Abuse and Alcoholism.

Powder and Law (1987). Report on the incidence of rising suicides by Aborigines on Queensland communities whilst in custody.

Watson, C. (1987). A survey of drug use patterns in Northern Territory Aboriginal communities: A preliminary report on the Top End and Katherine region communities - 1986. Darwin: Northern Territory Department of Health and Community Services, Drug and Alcohol Bureau.

Chapter 12. Conclusions and Recommendations

A. General Conclusions

The following conclusions were reached by the Expert Committee responsible for the preparation of this report. The conclusions are based upon information presented in the individual papers prepared by the Committee Members, and upon discussions of these papers which took place at the workshop of the 13th and 14th of November, 1988.

1. The clinical pharmacology of alcohol:

1.1 Alcohol is rapidly absorbed from the gastrointestinal tract and peak blood alcohol concentrations are reached 30-60 minutes after a single dose. Alcohol is distributed into body water and eliminated principally by metabolism in the liver, at a rate of approximately 0.015 g% per hour, which is equivalent to 8 grams (1 standard drink) per hour in a healthy 70 kg person. The rate of alcohol metabolism is increased 50-70% in alcohol-dependent persons.

1.2 Many factors influence the blood alcohol concentrations reached after drinking. They include the time over which drinks are consumed, the interval since the last drink, the type of drink consumed, the presence of food in the stomach, body weight and composition, sex, the rate of metabolism, previous alcohol and drug use, and concurrent disease. Predicting what blood alcohol concentration will be reached after drinking cannot be done precisely. As an approximate estimate, 12 standard drinks consumed over two hours will result in a blood alcohol concentration of 0.20-0.25 g% one hour after the last drink.

1.3 The composition of the principal alcohol-metabolising enzymes varies from person to person. A deficiency of a component of aldehyde dehydrogenase is found in 25-60% of Oriental (Mongoloid) races and causes a flushing reaction sometimes accompanied by nausea, palpitations and dizziness after alcohol is consumed. This reaction tends to limit the alcohol consumption of the affected individuals.

1.4 There is no known difference in the distribution of alcohol or in its rate of elimination between Australian Aboriginal people and white Caucasians. It is not known whether aldehyde dehydrogenase deficiency occurs in Aboriginal people, or whether other aspects of alcohol metabolism differ between Aboriginal and white people.

2. The analysis of alcohol in the blood:

2.1 The analysis of alcohol in blood is a relatively straightforward analytical procedure. There are opportunities for error but good laboratory practice should guarantee the accuracy of the results. Standards for analysis in State government laboratories are high. Unless proper quality assurance procedures are followed, the accuracy of a laboratory's results cannot be assured.

2.2 The quality of the results from an analysis of alcohol in blood specimens can only be as good as the specimens submitted. Poorly presented or poorly sampled specimens can lead to erroneous results.

2.3 The integrity of post mortem blood specimens may be compromised by delays in sampling and sample delivery as well as exposure to elevated temperature.

2.4 Breath analysis will, in general, be in close agreement or slightly less than blood analysis for alcohol, when sampled at approximately the same time.

3. The impairment of cognitive and psychomotor functioning by alcohol:

3.1 It is well recognised that acute intoxication by alcohol can impair cognitive and psychomotor functioning across a range of doses and in both experienced and inexperienced drinkers.

3.2 Most studies of the acute effects of alcohol on cognitive and psychomotor functioning have been carried out with young healthy volunteers and acute doses of alcohol to produce blood alcohol concentrations less than 0.15 g%. In these subjects, impairment can be detected in the laboratory at doses as low as 0.02 g%.

3.3 Whether impairment of cognitive and psychomotor functioning is observed following alcohol consumption and the degree of impairment may vary as a function of pharmacological and nonpharmacological factors such as:

A. Pharmacological factors

- dose of alcohol
- phase of the blood alcohol concentration curve
- speed and pattern of alcohol consumption
- prior experience with alcohol (tolerance) and other drugs with which alcohol is cross-tolerant
- presence of other drugs in the body

B. Nonpharmacological factors

- motivation to perform the task
- attention to the task
- previous experience with the task
- previous experience with the task while

under the influence of alcohol
- presence of brain damage
- the nature of the task
- the environment in which alcohol is consumed

3.4 Estimations of the level of intoxication produced by an acute dose of alcohol, such as those presented in the table by Clarke & Saunders (see p. 53), are valid for most people. However, these estimations do not apply to regular, heavy drinkers in whom a variable degree of tolerance may exist.

3.5 The performance of a complex task is the product of an interaction among cognition, psychomotor functioning, and affective state. The evidence available indicates that each of these components contributes to behaviour and that each can be affected by alcohol. The interactions among these components of behaviour are complex. The degree of impairment or change produced by alcohol varies with the blood alcohol concentration and with the amount of practice an individual has had performing the task both while sober and while under the influence of alcohol. No information is available on the effects of alcohol on specific tasks such as knot-tying and hanging.

4. Tolerance to the cognitive and psychomotor impairing effects of alcohol:

4.1 Chronic tolerance to cognitive and psychomotor impairment produced by alcohol does occur but there is variation in the degree of tolerance which develops to different functions or behaviours.

4.2 Several theories of neuroadaptation have been proposed to explain functional tolerance and physical dependence. (These are briefly discussed in Section 2, Chapter 5).

4.3 The following factors influence tolerance:

- the quantity and frequency of alcohol consumption throughout a person's drinking history
- a person's initial sensitivity to the effects of alcohol
- time since the last drinking episode
- practising a behaviour while intoxicated
- recognised versus different environments

4.4 Largely for ethical reasons, there is little experimental evidence on the effects of blood alcohol concentrations exceeding 0.15 g%. However, there are documented reports of individuals showing remarkable degrees of tolerance as measured by the absence of readily observable signs of intoxication at blood alcohol concentrations exceeding 0.25 g%.

4.5 Little is known about the distribution of various degrees of tolerance to alcohol amongst the population of alcohol consumers.

5. Alcohol dependence and its treatment:

5.1 Alcohol dependence has been defined as a clinical syndrome consisting of the following common elements: a) a subjective awareness of the [compulsion] to drink; b) narrowing of the drinking repertoire - the more dependent, the more stereotyped the drinking schedule; c) primacy of drinking over other activities - a dedicated occupation; d) altered tolerance to the drug with tolerance increasing during the early stages of the drinking career but declining in the later stages of drinking; e) repeated withdrawal symptoms which may vary in intensity; f) relief of withdrawal symptoms with further drinking; and g) reinstatement of the drinking pattern after abstinence (Edwards et al., 1977; World Health Organization Memorandum, 1981).

Please refer to the distinction made between psychological dependence and physical dependence in Section 1.1 of Chapter 6.

* The word compulsion is to be replaced in the definition of dependence to be given in the next version of the International Classification of Diseases 10 (ICD10) which will be published in 1991.

5.2 The relationship between tolerance and physical dependence is far from completely understood. It is generally thought that dependence rarely exists without tolerance. Sometimes, however, it has been found that the clinical manifestations of dependence are observed without concomitant evidence of tolerance in heavy drinkers, who are in the later stages of an extended drinking history.

5.3 People who are physically dependent on alcohol are likely to develop withdrawal symptoms 6-24 hours after ceasing or substantially reducing alcohol consumption. Withdrawal symptoms include: tremor, sweating, and agitation. The withdrawal syndrome may progress to delirium tremens which encompass confusion, hallucinations, and sometimes seizures. Seizures can occur both in simple withdrawal and in delirium tremens. This is a potentially life-threatening condition and requires urgent medical treatment.

5.4 A safe and caring environment should be provided for individuals sobering up from alcohol intoxication. It should be noted that it is difficult to predict who will sober up in an uncomplicated fashion and who will require monitored detoxification.

5.5 For those individuals who are physically dependent on alcohol, proper detoxification facilities should be made available. Detoxification can be undertaken in both medical and nonmedical settings. The intoxicated individual should be placed under the observation of a person who is experienced in detecting signs which may

require medical intervention. In the event that medical complications may arise, facilities for medical consultation should be made available when undertaking nonmedical detoxification.

5.6 A range of treatment options should be made available for individuals who present with alcohol-related problems.

6. Alcohol-related brain damage.

6.1 Chronic alcohol abuse can lead to two syndromes of cognitive impairment: 1. The Korsakoff amnesic syndrome. 2. An adaptive behaviour syndrome.

6.2 Whilst it has been suggested that drinking alcohol, even as little as two or three drinks per day, can cause chronic cognitive impairment, recent evidence suggests that this is not the case. The latest research suggests that there is a threshold effect for alcohol-related brain damage rather than a dose-response relationship. The precise levels of consumption that cause chronic cognitive impairment are not known. The National Health and Medical Research Council estimate that the probability of experiencing harmful medical effects from alcohol increases at consumption levels which exceed 60 grams of alcohol (6 standard drinks) per day for males and 40 grams of alcohol (4 standard drinks) per day for females.

6.3 In people recovering from alcohol-related brain damage, improvement in cognitive functioning over the first two weeks of abstinence has been well established, but conflicting evidence exists as to the possibility of further improvement over a longer period of time.

6.4 Although sex differences exist in the rate of alcohol metabolism and susceptibility to the toxic effects of alcohol, no conclusive evidence exists as to whether males or females are more prone to incurring alcohol-related cognitive impairment. There have been no reports on racial differences in susceptibility to chronic cognitive impairment by alcohol. There have been no studies explicitly assessing gender or racial differences in this area.

6.5 Little evidence exists that there is a significant interaction between alcohol consumption and age with respect to chronic cognitive impairment by alcohol. Both young and old alcoholics are found to be impaired on tests of cognition.

6.6 Cognitive impairment is likely to result from chronic alcohol consumption before clinically significant medical problems such as liver disease occur, therefore, it is too late to wait until a significant medical problem arises before some intervention is attempted.

6.7 There have been no studies to determine whether differences in susceptibility to brain damage exist between binge and steady drinkers.

7. The relationship between suicide and alcohol:

7.1 Suicide and indigenous populations:

7.1.1 Suicide is increasing across a number of cultures, including prominantly youth of indigenous populations in North America and the Pacific basin. All of these groups are experiencing a variety of stresses associated with rapid social change. It is important to look at the sources of these stresses and to address the underlying causes.

7.1.2 Most increases in suicide have involved young males and alcohol is frequently implicated.

7.1.3 Among Australian Aboriginals suicide was, and still is, rare in traditional communities. In the Kimberley region of Western Australia, suicide has increased among young adult males who live in areas of rapid social transformation. Psychotic experiences related to alcohol use, and recent loss have been associated with these deaths. The anxiety accompanying alcohol-related disorders of ideation and perception appear to be worsened by isolation (as in incarceration).

7.1.4 Suicide must be interpreted in the wider context of deaths from external causes which are increasing in many of these indigenous cultures. There is only limited evidence that this may be also occurring in Australian Aboriginal society.

7.2 Suicide and alcohol:

7.2.1 There appears to be a relationship between suicide and alcohol mediated, in part, through the development of secondary affective disorders. This tends to be an event which occurs late in a person's drinking history.

7.2.2 At greatest risk are young males who are dependent on alcohol, are concurrently depressed, and have suffered a recent loss of a significant interpersonal relationship.

7.2.3 Preliminary findings from research in progress suggests that a similar pattern is emerging in the Kimberley region of Western Australia. (Psychotic symptomatology associated with heavy drinking is increasing, and both are associated with suicide among Aboriginals in the Kimberley region).

7.3 Suicide and incarceration:

7.3.1 Suicide during incarceration occurs both in prisons and police cells. Features of the populations at greatest risk for suicide include: young adult males, recently arrested, often placed in isolation, and most commonly intoxicated upon arrest.

7.3.2 Preliminary research indicates that a similar situation exists in the Kimberley region of Western Australia. It should be noted that a vulnerable section of the Aboriginal community (i.e., individuals more frequently experiencing features associated with suicide outside of custody) are being repeatedly incarcerated.

7.3.3 The intersection of three phenomena, alcohol intoxication, self-destructive behaviour, and frequent incarceration in Aboriginal society, is manifested prominantly in areas of rapid social change which have seen the breakdown of traditional and stable transitional (e.g., the pastoral industry) social structures. It should be noted that the three phenomena discussed here are but a few among many variables such as - lifestyle, culture, family history, social and political conditions - that interact to produce behaviour. Only a culturally sensitive, socio-historical analysis can address these issues adequately. Violent deaths, suicide, and alcoholism are epiphenomena reflecting profoundly more complex issues at a deeper level of Aboriginal experience.

8. The relevance of empirical research to clinical situations.

8.1 Although there is a relationship between alcohol, imprisonment, suicide, depression and anxiety, the exact nature of that relationship is unclear.

8.2 There is at present no satisfactory scientific method of studying the significance and meaning of the acts of individuals in a social context, and it is unlikely that more satisfactory methods will become available in the forseeable future.

B. Recommendations

The following recommendations were reached by the Expert Committee responsible for the preparation of this report. The recommendations were drafted on the final day of the workshop after all papers had been presented and discussed.

Recommendations for research:

1. It is not possible to specify with any degree of certainty the probability that a given individual will show tolerance to the intoxicating effects of alcohol or the degree of any such tolerance. It was recommended that a large scale study be undertaken to determine the distribution of various degrees of tolerance to the cognitive and psychomotor impairing effects of alcohol across a wide range of levels of alcohol consumption throughout the general community. The findings from such a study would have important implications for forensic and medical practice.

   This study would not involve the administration of alcohol to sober subjects but would rely on the assessment of individuals who are found to be intoxicated upon admission to detoxification units, hospitals, sobering up facilities or during random breath testing by the police.

2. Given that a search of the relevant literature revealed only one study that investigated the metabolism of alcohol by Australian Aboriginal people, it was proposed that a study should be undertaken to investigate this topic. If differences were found in the way alcohol is metabolised by people of Australian Aboriginal descent and by people of European Caucasian descent, then this would have implications for evaluating tolerance and dependence to alcohol in this population and may, in turn, influence the development of pharmacological and other treatment options, as well as prevention options, for alcohol-related problems.

   The point was made, with regard to this proposed study and others which aim to investigate racial differences, that it would first have to be established how Aboriginal was to be defined. Such studies may not have implications for the Australian Aboriginal community as a whole. Please see the recommendations included in Chapter 11: Part A for a further discussion of this issue.

3. It was stated that any research that pertains to Aboriginal people should include Aboriginal people in its design and implementation, and should provide an opportunity for transfer of skills to Aboriginal people. Researchers should be referred to the document which arose from the conference held at Alice Springs in November, 1986 on "Research Priorities to Improve Aboriginal Health" (see Appendix B).

4. It was recommended that research is needed to investigate possible differences in the propensity towards alcohol-related brain damage between binge drinkers and steady drinkers and between males and females. There is a paucity of research on these subjects, yet results from such investigations would have important health and forensic implications.

5. It was recommended that more research should be undertaken to investigate differences due to gender in the other areas related to alcohol use discussed in this report, for example, sex differences in mood changes and cognitive and psychomotor impairment produced by alcohol.

6. During the the workshop the phenomenon of sudden, unexplained death related to alcohol consumption was discussed. A recommendation was made that this was an important area for research as a significant number of the reported deaths in custody appeared to fit this description. By gaining a better understanding of how these sudden deaths come about, we may be in a better position to take steps to prevent their occurrence.

7. It was recommended that a review be undertaken of the effectiveness and efficiency of programs that provide an alternative to incarceration in police lock-ups as facilities for the management of intoxicated individuals.

Other recommendations:

1. The Committee recognised that a number of its recommendations might require that service and educational provisions be expanded. It was noted that two essential factors must be considered in implementing such changes: (1) There must be ongoing dialogue between the communities involved and the service providers such as police, drug and alcohol workers, and medical personnel. (2) To develop effective services, adequate resources are required. The provision of such resources should be a matter of some considerable priority to the government.

2. An educational package should be developed to train people such as police and staff at casualty and detoxification units to assess the level of intoxication by alcohol of individuals entering into their care or protection. Relatively inexpensive modern technologies could be employed to measure the range of blood alcohol concentration into which the individual falls as well as a behavioural assessment package to evaluate the general intoxication level. In addition to providing the care-givers with useful information about the individuals under their care, this exercise could also provide useful data on levels of intoxication and blood alcohol concentrations shown by individuals being dealt with in these various facilities.

3. Appropriate service provisions for sobering up and detoxifying should be provided for individuals who have been apprehended by the police. Criteria should be established to determine whether these intoxicated individuals require close monitoring or medical assistance. For example, individuals presenting with a blood alcohol concentration exceeding a specific range might be recommended for proper detoxification facilities. It is recognised that it may be difficult to provide appropriate facilities in some remote areas of the country but efforts should be made to identify practical ways in which existing situations could be improved.

4. Analytical laboratories which analyse samples for forensic enquiry should be involved in quality assurance trials of an equivalent standard to those employed by government laboratories.

5. Standards for pathology sampling should be regularly assessed and raised to a high level, since the quality of the sample is critical to the whole analysis process.

APPENDIX A

Equation to estimate blood alcohol concentration

The formula for calculating BAC in g% is as follows: the average rate of alcohol metabolism which is estimated at 7.5 grams/hour is multiplied by the time over which alcohol was consumed. This value is subtracted from the grams of alcohol consumed. The value from this subtraction is then divided by the person's body weight in grams multiplied by a constant (6.8 for males and 5.5 for females).

$$\frac{[(\text{grams of alcohol}) - (7.5\ \text{g/h} \times \text{time (h)})]}{[(\text{body weight (kg)}) \times (6.8 \text{ for males or } 5.5 \text{ for females})]}$$

It should be noted that this formula is used to calculate a very rough estimate of BAC. There is a high degree of individual difference among people's capacity to metabolise alcohol, as was noted in Chapter 2 on the Clinical Pharmacology of Alcohol. One should <u>not</u> presume that this formula will give an accurate assessment of actual BAC.

APPENDIX B

RECOMMENDATIONS ARISING FROM THE WORKSHOP ON PRIORITIES TO IMPROVE ABORIGINAL HEALTH

ALICE SPRINGS, 26-28 NOVEMBER, 1986

Ethical Aspects of Research in Aboriginal Health

1. That ethical guidelines for health research involving Aborigines be established.

2. That these ethical guidelines be established by a forum of Aboriginal people, representative of Aboriginal communities and community controlled organisations throughout Australia within 6 months.

3. That this independent forum of Aboriginal people receive the necessary funding to meet several times so as to effectively establish the guidelines.

4. That this forum use as a basis for these ethical guidelines the following: Central Australian Aboriginal Congress guidelines; Aboriginal Health Organisations (SA) guidelines; Relevant sections of the NH & MRC ethical guidelines; RADGAC guidelines; Other relevant guidelines, e.g., Australian Society of Linguists; NAIHO Evaluation Papers.

5. That the close relationship between ethical guidelines and criteria for funding research projects be recognised and that the health research involving Aborigines be very heavily weighted towards problems as perceived by Aboriginal communities.

6. That this Workshop nominate an appropriate Aboriginal person to convene the forum. (Mr. Shane Houston was nominated).

7. That there be Aboriginal representation on the NH & MRC Ethics Committee. The following have indicated their willingness to accept nominators: Dulcie Flower; Shane Houston; Michael Mace; Grace Smallwood.

Principles for the Funding and Organisation of Research

8. The highest funding priority must be given to health and mental health problems which Aborigines identify as most deserving.

9. Money for research, development and training for public health should be given for improving Aboriginal health.

10. Money should also be ear-marked for Aboriginal women's health.

11. All money for research and development training in Aboriginal health should be subject to approval by a representative Aboriginal forum, possibly to be a role for NAIHO.

12. The Aboriginal Health Research and development and Teaching Forum be an assessor for all grant proposals.

13. For the Interim Public Health Research and Development Fund, applications should be assessed on their significance to Aboriginal people and Public Health.

14. In assessing the scientific merit of proposals, the Fund should recognise;

(a) the methods used in applied and social research, especially those relating to health programs and health care;

(b) a range of perspectives and methods for research, especially the growth in stakeholder or community-controlled research.

15. The proposed Aboriginal Forum needs to have resources. It could be granted a proportion of the Public Health Research and Development Fund.

(a) National Aboriginal data;

(b) Data available to Aboriginal/Torres Strait Island Communities to enable priorities.

16. That there should be Aboriginal involvement in Aboriginal research:

(a) setting priorities;

(b) methods;

(c) ethical issues; and

(d) implementation.

17. That there should be an Aboriginal/Torres Strait Island Health Research and Development Fund with contributions from NH & MRC, BADGAC,DAA and the Australian people.

18. NH & MRC and other agencies consider ways of greater funding of Aboriginal Women's health care and MCH research and supply information on how to apply successfully.

19. Research must be organised in such a way that:

(a) local communities are involved in every step of research design and execution;

(b) members of local communities become competent to perform such studies on their own in the future, such as the example demonstrated by the group in Brisbane reported by Bill Lowah.

20. That all information about grants for research, development and community-based education, especially from the Better Health Commission, the new Public Health Research and Development Fund, and NH & MRC be distributed to Aboriginal organisations, and that the Commonwealth Health Department provide assistance to groups preparing submissions for funds. 21. Every research proposal involving the study of an Australian community should contain a list of skills and knowledge that shall be transmitted to community members as a process of that research. The merit of this listing of skills shall be considered integral to the overall evaluation of the quality of the proposal.

22. Research being undertaken in Aboriginal communities be given priority over individual research.

23. Aborigines be given access and support for gaining research skills to enable the various levels of necessary research to be undertaken.

24. Large scale treatment or prevention programs, e.g. hepatitis B, be evaluated in pilot projects before general implementation.

25. Researching to improve the health of Aboriginal people continues to be recognised as needing continuing support.

26. Future workshops of this nature involved Aboriginal people, firstly in consultation, organisation and implementation. Each AMS should have at least one delegate and information should be distributed for community discussion.

27. Funding for such delegates be readily provided.

28. Priority will be given to research in fields that are seen by Aboriginal communities themselves as being of practical importance, special emphasis placed on evaluation or application of knowledge derived from earlier research.

29. Research workers known to have confidence of Aboriginal communities be asked to assist in developing a research program by which NH & MRC and other research funding agencies be guided.

30. The necessity for longitudinal studies, community based.

31. The essential role of control groups in studies was stressed, e.g. age and time matched.

APPENDIX C

Suggested References on Expert Testimony

The following references are relevant to two aspects of expert testimony:

When expert evidence is admissable and how it is admissable:

Byrne, D.M. (1986). Cross on evidence. (3rd Australian edn.) Sydney: Butterworths.

Freckleton, I.R. (1983). Opinion evidence: Evidence reference research paper No. 13. Sydney: Australian Law Reform Commission.

Freckleton, I.R. (1987). The trial of the expert: a study of expert evidence and forensic experts. Melbourne: Oxford University Press.

Practical hints to experts and what experts should expect:

Frumer, R.L., & Minzer, M. (1973). Examination of medical experts. New York: Mathew Bender.

Shayne, N.T. (1980). Medical evidence: Litigation course handbook No. 155. New York: Practicing Law Institute.

Glossary of Technical Terms

Alcohol dehydrogenase: an enzyme found in the liver and in many other living cells, such as yeast, which causes alcohol to be converted to acetaldehyde. The reaction is temperature and pH dependent.

Ambulatory: able to walk

Amotivation: lack of initiative and spontaneity with a blunting of affect.

Anterograde amnesia: loss of impairment of memory concerning events occurring after the trauma or disease that caused the condition.

Ataxia: incoordination of voluntary movement.

Barbiturates: a class of sedative-hypnotic drug and a potent central nervous system depressant.

Benzodiazepines: A class of anxiolytic drug - that is, one used to reduce anxiety and in some cases induce sleep.

Binocular: relating to the use of both eyes

Binocular fusion: the ability of the brain to receive two images one from each eye, yet to perceive it as on image.

Butyrophenones: a class of neuroleptic or antipsychotic drugs.

Cardiomyopathy: disease of the cardiac muscle.

Central processing: a general term to indicate processes which take place in the central nervous system (i.e., the brain and spinal cord).

Cerebellar ataxia: loss of muscular coordination as a result of disease in the cerebellum.

Cystosol: the soluble component of a cell.

Depressant: a drug which lowers the functional activity of the body.

Deproteination: causing protein to precipitate out of solution. Usually can be achieved by adding a suitable chemical such as trichloro-acetic acid.

Disinhibition: problems suppressing/inhibiting irrelevant responses, or of selecting the appropriate response from several possible choices.

Duction: a term to indicate how far one can converge or diverge ones eyes whilst still maintaining fusion (the perception of one image).

Dysarthria: difficulty in articulating speech.

Epilepsy: A condition characterized by episodic seizures due to changes in neurological function. Epilepsy may be described as idiopathic where there is no neurological cause or permanent loss of function. Seizures may be generalized (non-focal), or focal where the disorder arises from a localized area of the brain.

Enzyme: A chemical substance which will catalyse a biochemical reaction but not be altered itself.

Gas Chromatograph: the instrument used for this analysis is now very sophisticated and is often capable of automatically processing up to 100 samples under the control of a microprocessor or a computer. The essential component in the GLC is the column in which the separation is achieved. The column has a continuous flow of gas passing through it and has an injection port (usually a silicon rubber seal) through which samples may be accurately introduced into the gas flow and thence the column. The next most important part of the instrument is the oven which is designed to very accurately control the temperature surrounding the column. Temperature variations will affect the precision of the chromatography. Finally the instrument must have a device to detect the components coming off the end of the column. The most widely used detector for alcohol analysis is a flame ionization detector. The effluent gas passes into a small flame fueled by hydrogen gas and having a controlled electrical potential across the flame. As the components emerge from the column and enter the flame the number of ionic species in the flame will be changed and this will alter the current flow in this part of the electronic circuit (a Wheatstone bridge circuit). These changes are measurable and may be amplified. The changes are recorded on a chart recorder or some other electronic devise (including integrators and computers). The emergence of a substance always takes the form of a triangular peak, with the apex of the peak being used to record the retention time.

Gas Chromatography: The technique relies on the general principles of chromatography: a mixture of like compounds may be separated into individual components when passed through and inert matrix which preferentially inhibits the compounds passage (usually due to dissimilar polarities of the molecules). In the case of a GLC the matrix is usually a temperature stable wax or resin and the mixture is carried past this matrix (liquid phase) by an inert gas (e.g. nitrogen) held at a high temperature to stop condensation. the result is that each component will be separated from other members of the mixture at the end of the GLC column containing the separating matrix. In practice complete separation may not be achieved if the mixture is sufficiently complex and of very similar compounds. In the case of blood alcohol analysis this is rarely the case since the substances

of similar volatility that might reasonably be expected in a sample of human blood can be separated. It should be noted that the technique does, in fact, simply rely on the relative retention times of components and does not provide absolute identification. This point will be discussed further under quality assurance.

Genome: part of a gene which codes for a particular enzyme or other protein.

GGT: gamma-flutamyltransferase (or gamma-glutamyl transpeptidase). A liver enzyne, elevated serum levels of which may be caused by heavy alcohol consumption.

Hepatic decompensation: signs of failure of the liver to maintain its normal function; such signs include accumulation of fluid, haemorrhage and coma.

Hyperreflexia: exaggeration of the deep tendon reflexes.

Hypnotics: a class of drugs which produce drowsiness and facilitate the onset and maintenance of sleep.

Isoenzyne: a subunit or variant of an enzyme.

IQ "Intelligence Quotient": the score on some intelligence tests which relates mental age (the age level at which a child performs on a test) with chronological age.

Lipids: a group of organic compounds, otherwise known as fats.

MCV - Mean Corpuscular Volume -: the average volume of the red blood cell, expressed in cubic microns. The MCV may be elevated in 40-60% of cases of hazardous or harmful alcohol consumption.

Mitochondria: specialized parts of a tissue cell which provide the principal energy source of the cell.

Neuroadaptation: the neuronal changes associated with both physical dependence and tolerance.

Neurons (nerve cells): the basic units of the nervous system.

Normal distribution: a frequency distribution in which the values or scores group around the mean. The greatest frequency of scores is at the mean with the frequencies of other scores trailing off on either side of the mean.

Nutritional amblyopia: dimness of vision or partial loss of sight due to a vitamin or other dietary deficiency (for instance, when alcohol is ingested instead of food).

Opthalmoplegia: paralysis of ocular movement.

Pancreatitis: inflamation of the pancreas.

Pellagra: a skin condition caused mainly by niacin deficiency.

Peripheral neuropathy: deterioration of the nerves of muscle and sensation in the hands and feet.

Phenothizaines: a class of drugs with antipsychotic properties.

Phenytoin: an anticonvulsant drug used in the treatment of epilepsy.

Phoria: resting state deviation of both eyes which will still maintain fusion (the perception of a single image).

Platelets: disc-shaped cells which initiate blood clotting.

Portal venous circulation: the blood vessels which drain the gastrointestinal tract (stomach and bowel) and spleen, and supply the liver.

Psychotropic: a drug which affects the mind.

Pyloric sphincter: the sphincter joining the lower region of the stomach with the duodenum of the small intestine.

REA analysis: the procedure depends on the formation of a dye due to the ADH oxidation of alcohol. The dye produced, causes a quenching of the fluorescence of an inert dye (usually fluorescein) also present and this quenching is proportional to the amount of alcohol present. The system is provided as a commercial kit and auto analyzer instrument (Abbott TDx Analyzer).

Receptor site: a specific site (a chemical group) on a cell or enzyme on which a drug exerts its activity.

Romberg's test: the patient is standing with feet together and eyes closed and note is taken of the extent of body sway.

Sedatives: drugs which decrease activity, moderate excitement and calm the recipient.

Spectroscopic analysis: a technique which relies on the property of many substances to absorb light at particular wavelengths. The change in absorption of light, at a particular wavelength, can then be related to the amount of the substance present, (e.g., alcohol absorbs light in the infra-red wavelengths).

Standard drink: 8-10 grams of absolute alcohol. Also known as one unit. One unit is approximately equivalent to 2 middies of low alcohol beer, 1 middy of ordinary beer, 4 ounces (120 ml) of table wine, 2 ounces (60 ml) of fortified wine, or 1 ounce (30 ml) of spirits.

Stomach resection: operative removal of the stomach.

Subdural haematoma: a clot or collection of blood on the surface of the brain, and under the outermost membrane covering the brain (dura). Alcoholics and elderly persons may be at risk of subdural haematoma after minor head trauma.

Systemic circulation: the circulation of blood through the arteries, capillaries and veins, from and back to the heart.

Thalamus: a region of the brain, one major function of which is to act as a relay station for incoming sensory stimuli.

Tracking task: a task which requires the subject to continuously monitor and move the position of an object in relation to another (e.g., maintaining of a moving motor car on the road is a tracking task).

Transducers: a device which converts signals from one physical form to another. For example, the eye converts the signal of light reflecting from environmental structures into nervous signals which are transmitted to the brain for interpretation.

Visual acuity: a term to indicate the sharpness of vision.

Wernicke's Encephalopathy: a condition characterized by clouding of consciousness, disorientation, difficulty in understanding, muscular weakness, and ataxia. Acute severe thiamine deficiency is generally recognized as the cause. The symptoms usually are ataxia, ocular symptons (opthalmaplegia), and mental confusion (disorientation, confabulation).