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TRAFFIC ACCIDENT RESEARCH UNIT

ALCOHOL, DRUGS AND ACCIDENT RISK

DEPARTMENT OF MOTOR TRANSPORT NEW SOUTH WALES

The Traffic Accident Research Unit was established within the Department of Motor Transport, New South Wales, in May 1969 to provide a scientific approach to the traffic accident problem.

This paper is one of a number which report the results of research work undertaken by the Unit's team of medical, statistical, engineering and other scientists and is published for the information of all those interested in the prevention of traffic accidents and the amelioration of their effects.

W Bruden

Commissioner.

ALCOHOL, DRUGS AND ACCIDENT RISK

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NOVEMBER, 19

Contents

Abstract

The investigations detailed in this report represent a systematic attempt to study the interactive effects of single therapeutic doses of drugs and social doses of alcohol on human sensory, cognitive and motor performance. It was considered likely that the information obtained would be of predictive value in the assessment of the hazards of drug-alcohol involvement in the driving situation.

The effects of alcohol in doses of 0.54, 0.75 and 1.0 g/kg, which induce peak blood alcohol concentrations of about 0.06%, 0.09% and 0.11% respectively, were found to impair the performance of subjects in a dose-dependent manner. A high dose of alcohol was utilised where antagonism of the drug on effects of alcohol might be expected to occur. The two lower doses which induced lesser impairment, were used in investigations where either synergism or an additive effect was anticipated.

The drugs studied were of two types:

(a) Those which are taken to counteract the inebriate effects of alcohol, and

(b) Those for which there is clinical evidence of an interactive effect.

1. Alcohol and Fructose

The interactive effects of fructose and dextrose (1.2 g/kg, orally) and alcohol (1.0g/kg) were compared in order to determine whether fructose had some special ability to influence the way in which a person handles alcohol. Although slightly lower blood alcohol peaks were attained when fructose and dextrose were taken after alcohol than when alcohol was given alone, this effect was attributed to a delay in gastric emptying. The rate of alcohol metabolism was not affected. The sensory, cognitive and motor performance of the subjects was not significantly affected.

2. Alcohol and Caffeine

Decaffeinated coffee containing caffeine (300 mg/kg) taken after alcohol (0.75 g/kg) did not induce the "sobering-up" effect which is commonly believed to occur. Caffeine did improve reaction time when these were impaired by alcohol, however.

3. Alcohol and Antihistamines.

The effects of two antihistamines, dexchlorpheniramine (Polaramine, 4 mg/70 kg) and meclastine (Tavegyl, 1 mg) were investigated alone and in combination with alcohol. Although a general synergistic effect between dexchlorpheniramine and alcohol (0.75 g/kg) was not observed, a delayed recovery from the effects of alcohol occurred in most tests. Meclastine did not significantly modify the effects of alcohol (0.54 g/kg).

4. Alcohol and Tranquillizers.

Diazepam (Valium, 10mg) alone was found to have a slight depressant effect on psychomotor performance but significant synergistic interactions occurred with alcohol at two dose levels (0.54 and 0.75 g/kg). Chlordiazepoxide (Librium) was found to have variable effects. A low dose (20 mg) of chlordiazepoxide did not modify the effects of alcohol (0.75 g/kg) to any great extent. A higher dose of chlordiazepoxide (40 mg), however, although having some detrimental effect on performance per se, antagonised some but not all of the depressant effects of alcohol.

1. General Introduction

Widespread drug use in the industralised urban affluent societies has seemingly become part of the changing life style of these communities. This is evident in that psychotropic drugs are widely prescribed and other drugs such as antihistamines, and the minor analgesics etc, which are readily available without a prescription, are often taken to excess [1,2,3,4,5,6]. It is not surprising therefore, that mixed intoxication with social euphoriants, such as alcohol and marijuana also frequently occurs.

Currently, literature reviews on alcohol, prescription and over - the-counter drug use in both North America and Europe suggest that:

(1) Between 35% and 50% of the general population risk driving after drug use at least once a year.

(2) A conservative estimate of at least 7% of drinking drivers have also taken a psychotropic drug.

(3) Between 11% and 15% of accident-involved drivers had taken a psychotropic drug prior to their accident.

(4) Psychotropic drugs are most likely to be found in fatally injured drinking drivers. These drugs do not present a substitute for alcohol but an additional element frequently found in combination with alcohol.

(5) At least some psychomotor impairment in drivers with low or non-existent blood alcohol levels may be attributed to the use of psychotropic drugs. This implies that some highly impaired drivers are likely to be missed by present alcohol screening procedures[3].

In Australia, information on drug usage in the population is scanty and research on the effects of individual drugs and their combination with alcohol on human performance has been minimal. Questionnaire surveys in Sydney [7,8], and Canberra[9] indicated that about 11% of the population were daily users of analgesics. One Sydney study[7] showed that about 10% of males and 24% of females were taking sedatives and tranquillizers, whilst the Canberra survey[9], revealed that about 3% of males and 7% of females were receiving daily medication with these drugs.

Although the number of people who risk driving while on psychotropic or other drug medication in Australia has yet to be determined, some insight into the problem can be gained from the findings of Milner[10]. As a result of his study on 4584 general practice and psychiatric patients in Perth, Western Australia, it was estimated that of the 15% of the patients for whom psychotropic drugs were prescribed, 57% of the men and 35% of the women would be likely to both drink and drive whilst receiving medication. It is interesting to note that of the 10,000 drivers chosen at random from a sample of drivers who were breathalyzed in New South Wales during the period 1972-1973, over 25% admitted to have been taking drugs concurrently[11]. A classification was made of the drugs said to have been taken from which it is evident (Table 1) that the non-narcotic analgesics and tranquillizers were most frequently involved. Valium alone accounted for nearly 50% of the tranquillizers used, although this figure perhaps only reflects the market share for this compound.

Studies of drug usage to date have given some indications of the nature of the drug-alcohol-driving problem. The seriousness of the problem besetting students of traffic safety is camouflaged by the lack of really adequate information on the effects and risks involved. In an attempt to shed light on this problem, a programme was instigated to investigate the effects of prescribed and over-the-counter drugs, alone and in combination with alcohol, on the performance of subjects in a number of tests of sensory, cognitive and motor function related to driving skills.

The information derived from such a study was considered likely to serve two basic purposes:

(1) It will provide traffic safety information to the public in general and especially to those persons prescribing the drugs in general. Havard[12] has stated that, unless he is told to the contrary, the physician should assume that all patients over the age of 17 years are liable to drive a motor vehicle.
(2) It will serve as a data bank which can be used to aid and guide the formulation of traffic safety strategies and countermeasures.

This paper reports the findings obtained in a baseline study of the effects of alcohol and the effects of fructose, dextrose, caffeine, meclastine (Tavegyl), dexchlorpheniramine (Polaramine), diazepam (Valium), and chlordiazepoxide (Librium) taken alone and together with alcohol. Subsequently, it is proposed to produce reports dealing with the interactive effect of disodium cromoglycate (Intal), allopurinol (Zyloprim), cannabis (marijuana), Contac 500, paracetamol and aspirin with alcohol.

Table 1

Drug taking in individuals subjected to breathanalysis in N.S.W. (1972-73)

Drug Class*	Percentage Distribution of Drug Users (%) (n = 2539)
Analgesics	19.7
Tranquillizers	19.2
(Valium)	(9.0)
Anti-inflammatory agents	11.0
Antibiotics	9.4
Bronchial spasm relaxants	8.3
Non-prescribed vitamins	6.4
Sedatives and hypnotics	6.2
Cardiac reactants	5.7
Decongestants, expectorants	3.0
Antihistamines	2.4
Drugs acting on the nasopharynx	1.2
Anticonvulsants	1.1
Drugs used in the control of diabetes	1.1
Drugs used in the control of obesity	1.1
Iron and erythropoietic preparations	1.0
Anti-depressants	0.9
Stimulants	0.9
Diuretics	0.7
Antimalarials	0.7

* Classifications according to the M.I.M.S. (Monthly Index of Medical Specialities) therapeutic index.

2. General Method

The subjects were University students of both sexes aged between 18 and 30 years.

The two experimental designs utilized in these studies had either dependent or independent controls. In the dependent control experiments every subject received each of two treatments according to a Latin square design (Fig. 1). Where independent controls were used, each subject was randomly assigned to one of the four groups and received only one of the four treatments.

The tests utilized for performance measurement and the methods used for collection and analysis of blood for blood alcohol concentration have been described elsewhere.[13]

Day No.	Group	Treatment	
1.	A	Alcohol + Drug	Independent Control
	B	Alcohol + Drug Placebo	
	C	Alcohol Placebo + Drug	
	D	Alcohol Placebo + Drug Placebo	
2.	A	Alcohol + Drug Placebo	Dependent Control
	B	Alcohol + Drug	
	C	Alcohol Placebo + Drug Placebo	
	D	Alcohol Placebo + Drug	
3.	A	Alcohol Placebo + Drug	
	B	Alcohol Placebo + Drug Placebo	
	C	Alcohol + Drug	
	D	Alcohol + Drug Placebo	
4.	A	Alcohol Placebo + Drug Placebo	
	B	Alcohol Placebo + Drug	
	C	Alcohol + Drug Placebo	
	D	Alcohol + Drug	

Figure 1 : The types of experimental design utilized in the studies.

3. Baseline Study : Alcohol - Determination of the dose-effect relationship for alcohol.

3.1 Introduction

Literature reviews dealing with the effects of alcohol on human performance suggest that impairment may be observed in many of the tests which purport to assess intellectual, perceptual and motor functions [14,15,16,17,18]. Impairment of human performance induced by alcohol may be seen at blood alcohol concentrations as low as 0.02%[14]* In general, it has been concluded that impairment is highly probable at blood alcohol concentrations between 0.05% and 0.08%[15,16], but it should also be noted that not all persons actually exhibit a significant decrement in performance. The differences in the degree of impairment of individual subjects at a given blood alcohol concentration have been variously attributed to factors such as motivation, personality, drinking experience, the nature of the task and sleep deprivation[16,17].

It should also be noted that in epidemiological studies of traffic accidents, a majority of young drivers involved in alcohol-related accidents was found to have a blood alcohol concentration of less than 0.10% [19,20,21]. It was also found that in N.S.W. between 1972 and 1973, a large proportion of young drivers who were breathalyzed had alcohol concentrations of less than 0.08% (Fig. 2)[11]. The data *per se* seems to suggest that young drinking drivers are more liable to involvement in traffic infringements than their older counterparts within this relatively low blood alcohol concentration range. Consequently, it seemed meaningful to use young people as subjects in these investigations.

In the experiment reported here, the effects of three doses of alcohol (0.54, 0.75 and 1.0 g/kg) were assessed. These doses were found to induce peak blood alcohol concentrations of approximately 0.06%, 0.09% and 0.11% respectively. The rationale for the choice of these doses was two-fold:

*Blood alcohol concentrations here refer to grams of alcohol per 100 millilitres of blood and are expressed as percentages. For example, 0.08 gm per 100 ml is expressed as 0.08%.

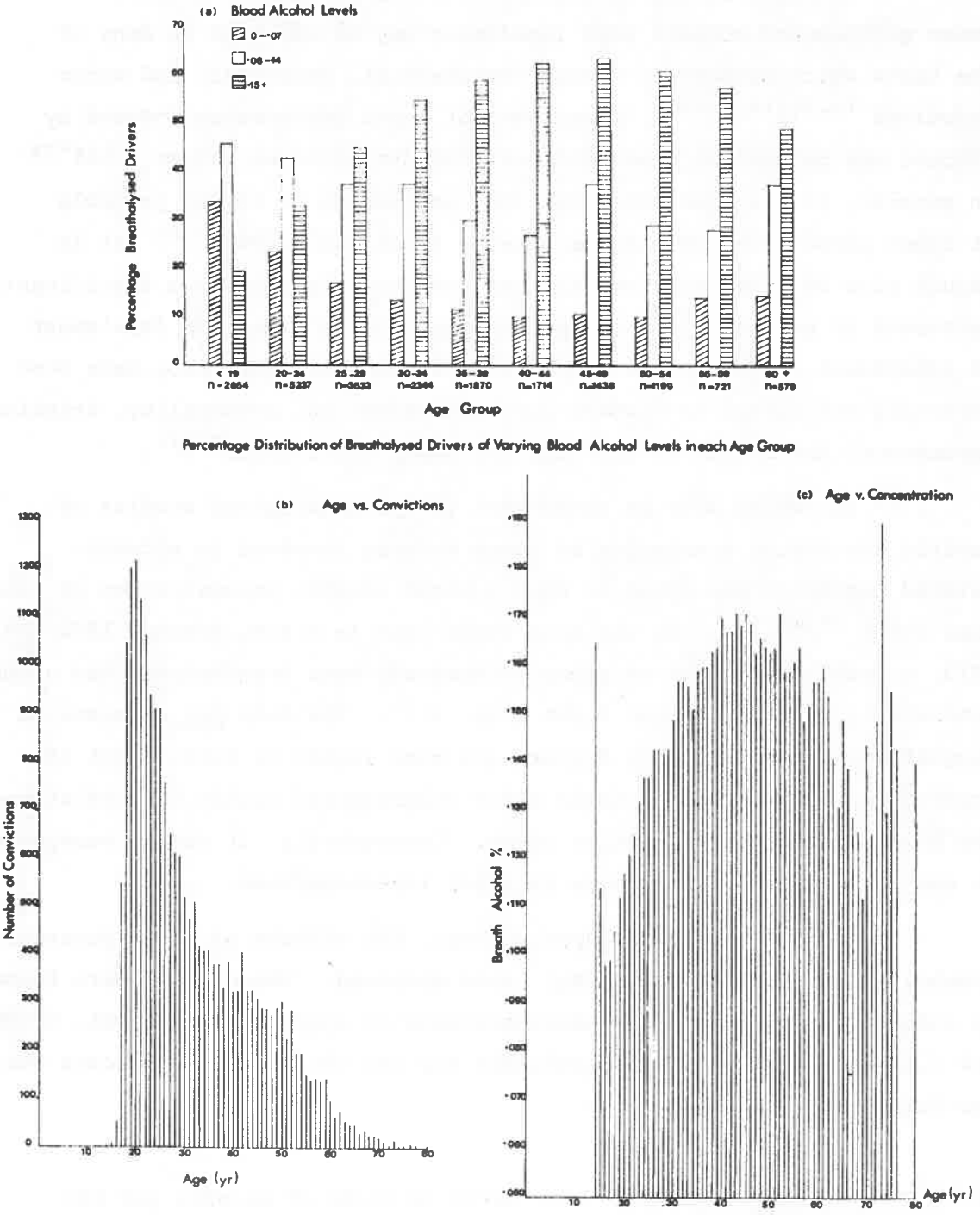

Figure 2: Breathalyzed Drivers in N.S.W. (1972-1973)

1. To determine suitable alcohol doses for use in drug-alcohol interaction investigations.
2. To assess the effects of alcohol on human performance on the battery of tests paying particular attention to blood alcohol concentrations within the vicinity of the N.S.W. legal drink-drive limit (0.08%).

3.2 Procedure

Twelve subjects attended the laboratory on four different occasions and received alcohol (0.54 g/kg, 0.75 g/kg and 1.0 g/kg) and a placebo drink, in random order according to a Latin square design. Capillary blood samples, taken from the finger tip were used for the measurement of whole blood alcohol concentrations.

3.3 Results and discussion

In general, the results from the battery of tests have indicated that the sensory, cognitive and motor functions of young adults were impaired when they ingested alcohol (0.54, 0.75 and 1.0 g/kg) and that the impairment was dose related. These doses of alcohol induced peak blood alcohol concentrations of about 0.06%, 0.09% and 0.11 % respectively. The degree of impairment caused by 1.0 g/kg of alcohol was shown to be highly significant in almost all of the tests. With the other two doses, 0.75 and 0.54 g/kg, although impairment was apparent in some of the tests, statistical significance from control values was not consistently obtained. The most likely explanation of this finding is that individual differences in acute adaptation to the effects of alcohol occured[14,15] . This phenomenon deserves further investigation. In an extrapolation to the driving situation, it was considered that ingestion of a dose of 1.0 g/kg alcohol by a young adult was likely to result in sufficiently severe CNS depression to constitute a major hazard should he or she attempt to drive a motor vehicle. The effects of lower doses were more difficult to pinpoint but the differences in the degree of impairment and acute adaptation which occurred in tests of intellectual and motor function were considered likely to present further dangers which warrant systematic exploration.

The results of this study were considered to provide the basis for a screening procedure to investigate the effects of drugs in combination with alcohol. Since highly significant effects occurred in most of the tests after administration of the high dose of alcohol, it was clear that this dose was inherently unsuitable for use in investigations of the interactive effects of drugs which might be expected to synergise with alcohol. The high and intermediate doses could be employed more usefully in situations where antagonism to the depressant effects of alcohol is likely. Conversely, the low dose, which induced a lesser degree of impairment, was considered suitable for the investigation of the interactive effects of drugs where synergism might be expected.

4.The Interaction Between Alcohol, Fructose and Dextrose

4.1 Introduction

It is desirable in many instances for both medical and social reasons to be able to increase the rate of disappearance of alcohol from the body. In consequence many compounds have been tested in attempts to achieve this aim. However, these attempts have mostly resulted in failure, and of the agents tried, only fructose has been consistently claimed to be effective. Fructose is, in fact, widely sold for Breathalyzer-beating purposes.

The findings for fructose have been extremely variable although some of the variation can be easily attributed to differences in experimental design. Evidence of increased rate of alcohol metobolism induced by an oral dose of 132 g and an intravenous dose of 1 g/kg of fructose was first reported by Stuhlfauth and Neumaier[22]. Pletscher found that rate of alcohol metabolism was increased by 70% after intravenous infusion of 1-2 g/kg/hour of fructose[23]. Other workers, although reporting a fructose-induced increase in the rate of alcohol metabolism have not encountered such marked effects.

4.2 Procedure

A modification of the usual procedure was necessary in this experiment in order to include dextrose as a control. Thus, each of the 12 subjects received the following treatments in random order according to a Latin square design (dependent controls):-

Alcohol + Fructose
Alcohol + Dextrose
Alcohol + Placebo
Placebo + Placebo

The subjects received either fructose (1.2 g/kg) or dextrose (1.2 g/kg) as a 50% w/v solution or a placebo which was coloured yellow with "Lemon Yellow 111" (Alfred Lawrence Australia) (0.5% w/v) and flavoured with Essence of Lemon B.P.C. (0.2% v/v). The placebo drink was sweetened with saccharin and was identical in colour. To eliminate possible behavioural effects which might stem from discrimination between

the drinks, the subjects were told they were receiving different concentrations of fructose on each occasion.

In order to reduce the effect of the concentrated hexose solutions on the absorption of alcohol, they were administered immediately after the first post-alcohol test run (40 minutes after beginning to drink). Since venous blood samples were necessary for determination of the biochemical data consequently, venous blood alcohol measurements were also made.

4.3 Results and discussion

Subjects who received either fructose or dextrose exhibited a lower peak blood alcohol concentration. There were no significant differences in the rates of decline of the blood alcohol concentration, however. The most likely explanation to this finding is that both fructose and dextrose delayed the absorption of alcohol since it has been shown that dextrose slows gastric emptying time in man [24].

The results obtained in the function tests show that, in general, the subjects performed slightly better when they had alcohol and either of the hexoses than when they received alcohol alone. The improvement in performance seemed to correlate with the slightly lower blood alcohol concentration of the two hexose groups.

It must be stated however, that for fructose to have any appreciable effect in reducing the blood alcohol concentration in the body system, large amounts would have to be taken. The high cost and the unpleasant side effect of abdominal cramps would be of little value to the ambulant drinker. Perhaps the intravenous administration of fructose might be of more use in a hospital situation for treatment of severe alcohol overdosage.

Thus, in the area of traffic safety, the amount of fructose in commercially prepared packages sold in many of Sydney's public bars is too small to have any significant effect on the blood alcohol concentration of drink-drivers who attempt to beat the Breathalyzer. It is naive and dangerous to encourage such attempts because impairment of human abilities induced by alcohol at blood alcohol concentration of 0.10% and higher is explicit and the risk of being involved in an accident is at least five times greater than it is to a sober motorist [25].

5. Alcohol And Caffeine

5.1 Introduction

It is a common belief that coffee is able to antagonise the intoxicating effect of alcoholic beverages and it has been tacitly assumed that this antagonistic effect is due to the CNS stimulant effect of caffeine per se. Considering the almost universal acceptance of this belief and the widespread use by the general public of a sobering cup of coffee before the drive home, it is surprising that comparatively little well-controlled research has been performed in this area.

5.2 Procedure

The procedure adopted aimed for as close an approximation to "normal" social drinking conditions as possible, both in terms of the relative concentrations of alcohol and caffeine administered and in the subjects'"knowledge" that they were having alcohol and coffee. They were, of course, ignorant as to the treatment groups to which they were assigned. Immediately after drinking alcohol (0.75 g/kg) the subjects received decaffeinated coffee containing caffeine (300 mg/70kg). The placebo was decaffeinated coffee. An independent control design was used utilizing 17 subjects per group.

5.3 Results and Discussion

The results showed that caffeine had no significant effect on the blood alcohol concentrations attained.

Caffeine appeared to antagonise the alcohol-induced increase in reaction times. These were the classical results which we might have expected and were in close agreement with the findings of Carpenter[26]. However, this effect did not occur in the other function tests.

Caffeine alone had no significant effect on psychomotor performance except in the standing steadiness (eyes open test).

It is perhaps pertinent to speculate on the meaning of these results to the person who has been drinking, realises he is intoxicated and uses a caffeinated beverage (e.g. coffee) to sober up before he drives his car. Legally, it does not help, because caffeine is unlikely to have

an effect on the blood alcohol concentration attained. In addition, there may be an inherent and unsuspected danger in the consumption of an allegedly sobering cup of coffee since caffeine tends to improve reaction time after its lengthening by alcohol and a driver may be expected to feel more alert and perhaps notice an apparent reduction in his drunkenness. He would, however, remain unaware of a continuing impairment of his perceptual and motor functions which would affect his driving - search, identification and prediction, and control - decision making and execution components of his driving skill.

6. Interaction Between Alcohol and Antihistamines.

6.1 Introduction

The antihistamines are a chemically diverse group of drugs which are used to treat a wide range of clinical conditions, such as insect bites, motion sickness and allergic reactions. Many antihistamines induce sedation, and are used clinically for this purpose[27]. Perhaps as a consequence of the sedation which occurs with the older members of the group, all antihistamine preparations sold "over the counter" to the general public in the U.K. must bear cautionary labelling, warning patients not to drive or operate machinery if they become affected by drowsiness. Although there have been numerous warnings in the literature on the interaction of antihistamines with alcohol[28], there is remarkably little definitive information available on the effects of such interaction.

6.2. Procedure

The procedure used in this experiment was essentially the same as described in the "general method". The drugs, dexchlorpheniramine (4mg/70kg) and meclastine (1 mg), were administered orally one hour prior to alcohol. In the case of dexchlorpheniramine, a Latin square design was used (n = 12). For the meclastine experiment, independent controls were used and there were 20 subjects in each group.

The alcohol dose was different in the two experiments. For the dexchlorpheniramine study, 0.75g/kg of alcohol was used, and 0.54g/kg was utilized in the meclastine study. Capillary blood was used to estimate the blood alcohol concentration in the meclastine experiment and in the dexchlorpheniramine experiment, venous blood was used to estimate blood alcohol concentration, glucose and lactate levels.

6.3. Results and Discussion

(a) Dexchlorpheniramine.

The prior administration of dexchlorpheniramine did not modify the blood alcohol concentrations attained.

Dexchlorpheniramine was found to impair performance on the Vienna Determination Apparatus and to decrease standing steadiness.

In all of the tests except manual dexterity (threading beads) dexchlorpheniramine delayed recovery time from alcohol-induced impairment. It is possible that a slow biological handling of the drug occurred and if the premedication time had been longer, more obvious synergism with alcohol might have been seen.

(b) Meclastine

Meclastine did not modify the blood alcohol concentration attained.

In general, meclastine did not modify psychomotor performance when taken alone. Similarly, when meclastine was taken in conjunction with alcohol (0.54g/kg) no significant modification of psychomotor performance induced by alcohol was observed. There was no overall trend towards greater impairment as seen with dexchlorpheniramine.

7. The Interaction of Alcohol with Benzodiazepine Tranquillizers

7.1 Introduction

In recent years,the consumption of tranquillizers has increased enormously although the indications for their use are usually non-specific and poorly defined. The two most widely prescribed members of this group in Australia, chlordiazepoxide (Librium) and diazepam (Valium) both have intrinsic CNS depressant activity and are therefore both potential depressants of psychomotor functions,even when taken in normal therapeutic doses. Thus, it might be predicted from first principles, that interaction of the benzodiazepines with another CNS depressant, alcohol, would produce at least additive effects. The benzodiazepine tranquillizers are commonly taken with alcoholic beverages as can be seen from an examination of drug involvement in N.S.W. breathalyzed drink-driving population where the tranquillizers form the largest group (51.7 per 1000). This would appear to provide an adequate "raison d'etre" for a study of the effects of these drugs on psychomotor functions both alone and in combination with alcohol.

7.2 Procedure

The procedure was essentially the same as described in "general method". Drugs or placebos were given one hour prior to the ingestion of alcohol. The following drug combinations were used:-

1. Alcohol (0.75g/kg) + diazepam (10mg). (N = 14 per group)
2. Alcohol (0.75g/kg) + chlordiazepoxide (20 mg). (N=19 per group)
3. Alcohol (0.54g/kg) + diazepam (10 mg). (N = 14 per group)
4. Alcohol (0.54g/kg) + chlordiazepoxide (40 mg). (N = 15 per group).

Venous blood samples were used to determine blood alcohol concentrations.

7.3 Results and Discussion

There were slight differences observed between the individual effects of chlordiazepoxide and diazepam on human performance. When the drugs were given in combination with alcohol, these differences were greatly magnified.

(a) Chlordiazepoxide

The low dose of chlordiazepoxide (20 mg) when administered alone had little effect on performance. With the high dose (40 mg) however, impaired performance was observed which was consistent with the expected effects of a minor tranquillizer. Although chlordiazepoxide (40 mg) induced impairment when given alone, paradoxically it antagonised some, but not all the effects of alcohol (0.54 g/kg).

When the low dose chlordiazepoxide (20 mg) was given with the high dose of alcohol (0.75 g/kg), synergism was observed only in the manual dexterity test.

(b) Diazepam

In general, psychomotor performance was slightly impaired by the effects of diazepam (10 mg).

The impairment induced by alcohol (0.75 g/kg) was enhanced by diazepam. This effect was also observed with the low dose of alcohol (0.54 g/kg).

The findings from our studies on the two minor tranquillizers chlordiazepoxide and diazepam showed that although they have similar pharmacological actions, and are prescribed for similar purposes, their interactive effects with alcohol are quite different. The low dose of chlordiazepoxide (20 mg) synergised with alcohol (0.75 g/kg) while the high dose (40 mg) antagonised some of the effects of alcohol (0.54 g/kg). Diazepam always synergised with alcohol.

Thus patients receiving diazepam should be warned of the dangers of operating complex machinery or driving a motor vehicle especially during the initial stages of therapy. They should also be made aware of the dangers of the drug and alcohol combination. With chlordiazepoxide, it is difficult to be precise. Certainly potential dangers exist, but they are not as great as with diazepam.

8. Conclusions and Recommendations

Impairment of human performance induced by alcohol was found to be dose-dependent.

The rate of alcohol metabolism and the detrimental effects induced by alcohol were not altered by either fructose or dextrose.

Decaffeinated coffee containing caffeine taken after alcohol did not have a "sobering-up" effect. However, caffeine did improve reaction time when this was impaired by alcohol.

Dexchlorpheniramine impaired psychomotor performance and when taken with alcohol delayed the recovery time of alcohol-induced impairment. Meclastine either taken alone or with alcohol did not modify psychomotor performance.

Diazepam when taken alone induced a slight decrement on psychomotor performance. A synergistic effect was found to occur when diazepam was taken with alcohol. Chlordiazepoxide had variable effects. A low dose (20 mg) did not modify the effects of alcohol to any great extent. A higher dose (40 mg), although induced some depressant effect on performance per se antagonised some but not all of the depressant effects of alcohol.

This series of studies reported to date have indicated some pertinent factors which might play a significant role in traffic crashes resulting from the combination of activities of drinking and driving and drink-driving under drug medication.

The study on fructose as a possible agent to increase the rate of alcohol metabolism suggests that the amount of fructose contained in commercial packets sold in many of Sydney's public bars is too small to have any significant effect on the blood alcohol concentration to beat the Breathalyzer. It has no significant effect in inhibiting the depressant effects of alcohol. A person with a blood concentration of 0.1% or more taking such a preparation who drives, places himself at risk in being involved in an accident. This is because impairment of human abilities induced by alcohol at these blood alcohol concentrations is explicit and the probability of being involved in an accident is at least five times greater than it is to a sober motorist.

There is no evidence to support the common belief that coffee will help to sober up an intoxicated person. Caffeine in coffee did not antagonise alcohol-induced impairment on psychomotor performance except reaction time where improved performance was found. This would suggest that there may be an inherent and unsuspected danger in the consumption of an allegedly sobering cup of coffee. A driver may be expected to feel more alert and perhaps notice an apparent reduction in his drunkenness. He would remain unaware of a continuing impairment of his other perceptual and motor functions which would affect his driving.

The difference in the effects of dexchlorpheniramine and meclastine either taken alone or with alcohol suggests that a "blanket warning" on antihistamines usage may not be warranted. Therefore it is suggested that the investigation of the effects of antihistamines on human performance should be carried out on the individual drugs.

It is clear from the studies on the tranquillizers that patients receiving diazepam should be warned of the dangers of operating complex machinery especially driving a motor vehicle. They should be made aware of the drug and alcohol combination which produces detrimental effects that are greater than the additive effects of the individual drugs. Although it is difficult to be precise with regard to chlordiazepoxide, certainly potential dangers exist but they are not as great as with diazepam.

The studies reported to date give some indications of the combined effects of alcohol and other drugs on human performance. However, the extent of the role played by a combination of alcohol and other drugs in traffic accidents in Australia has yet to be established. Because the use of drugs is a part of the life style of our urban society, continuing research into the effects of individual drugs and, most importantly, the combination of alcohol and other drugs on human performance is essential.

Laboratory investigation on alcohol should be extended to include the effects of different alcoholic beverages taken under various conditions so as to reflect the various patterns of social drinking in which the blood alcohol levels may vary with the type of beverage, situation and drinking pattern. In addition, further study is necessary to establish

(a) whether or not intellectual functions are more susceptible than motor functions to the depressant effects of alcohol; (b) the rate of recovery of various functions after alcohol.

Controlled field experiments should use actual driving performance which permits an analysis of change in driving behaviour resulting from the intake of alcohol and/or other drugs under various traffic situations. This is likely to yield a better insight of the findings from laboratory studies in real life situations.

For the assessment of the role which alcohol and drugs play in traffic accidents, a study of accident-involved and non-accident-involved drivers investigated under similar conditions is essential. Data concerning the involvement of alcohol and drugs from such a controlled study would almost certainly shed light on the risks involved of drinking drivers in the drug users population and the population at large.

A study on hospitalized drivers who have been involved in road accidents to determine the presence of alcohol and other drugs by blood tests should be given a high priority. Together with the data on the type, severity etc. of accidents, and sociological data of such drivers, the study will be invaluable to traffic safety management.

If traffic safety education is to succeed in its role to ameliorate the road toll, it must equip itself, amongst other things, with adequate research information.

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APPENDIX

Tests and Recording Apparatus Utilized in the Experiments.

Figure 3: Motor Performance Test (Vienna Determination Apparatus) - Stimulus/Response Panel.

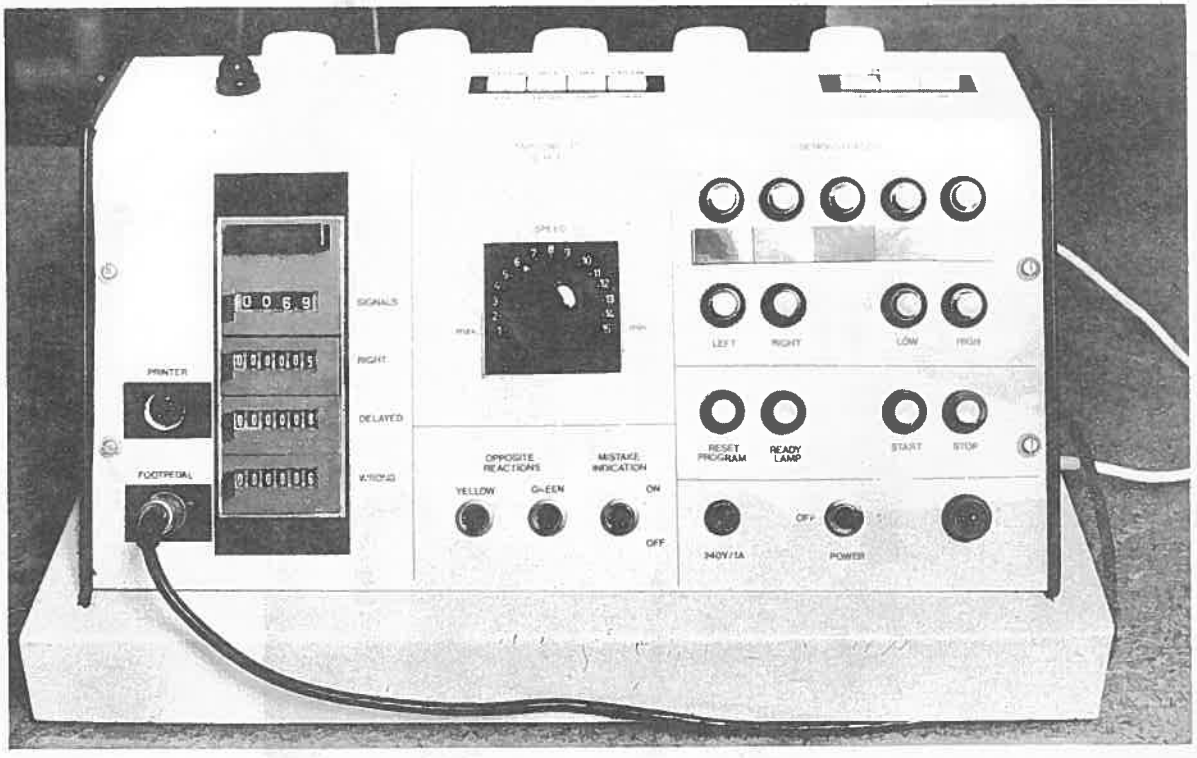

Figure 4: Motor Performance Test (Vienna Determination Apparatus) - Control and Recording Panel.

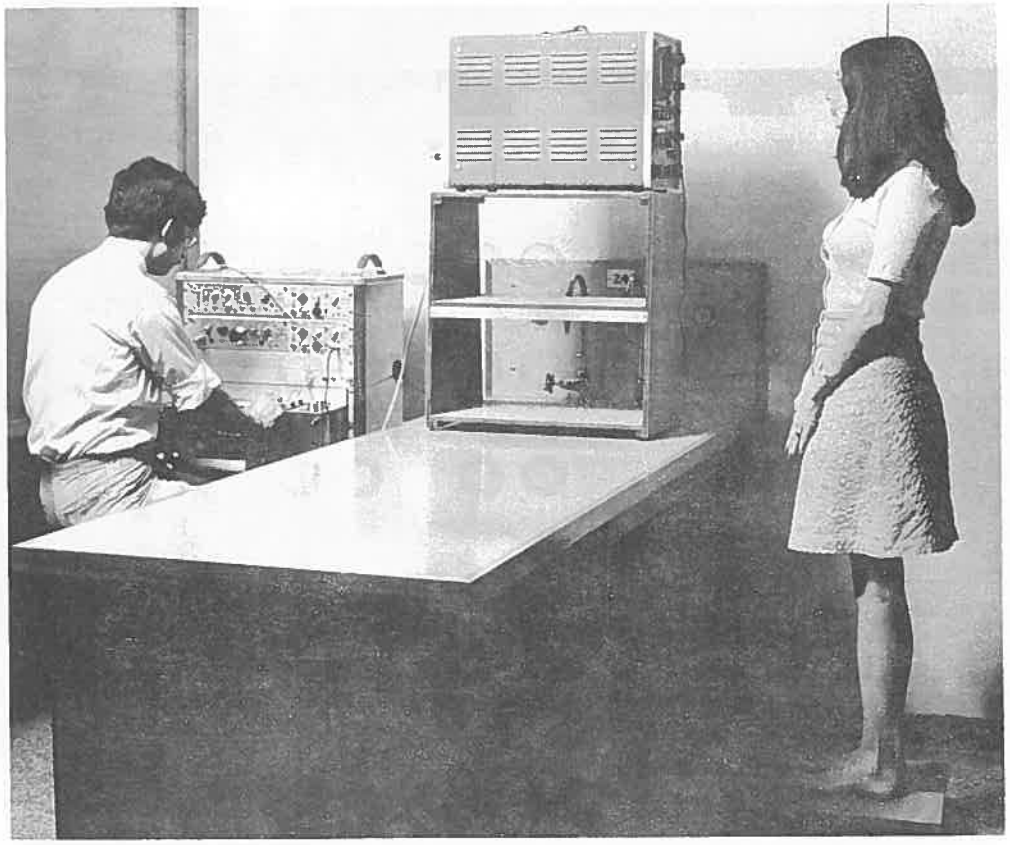

Figure 5: Standing Steadiness Test.

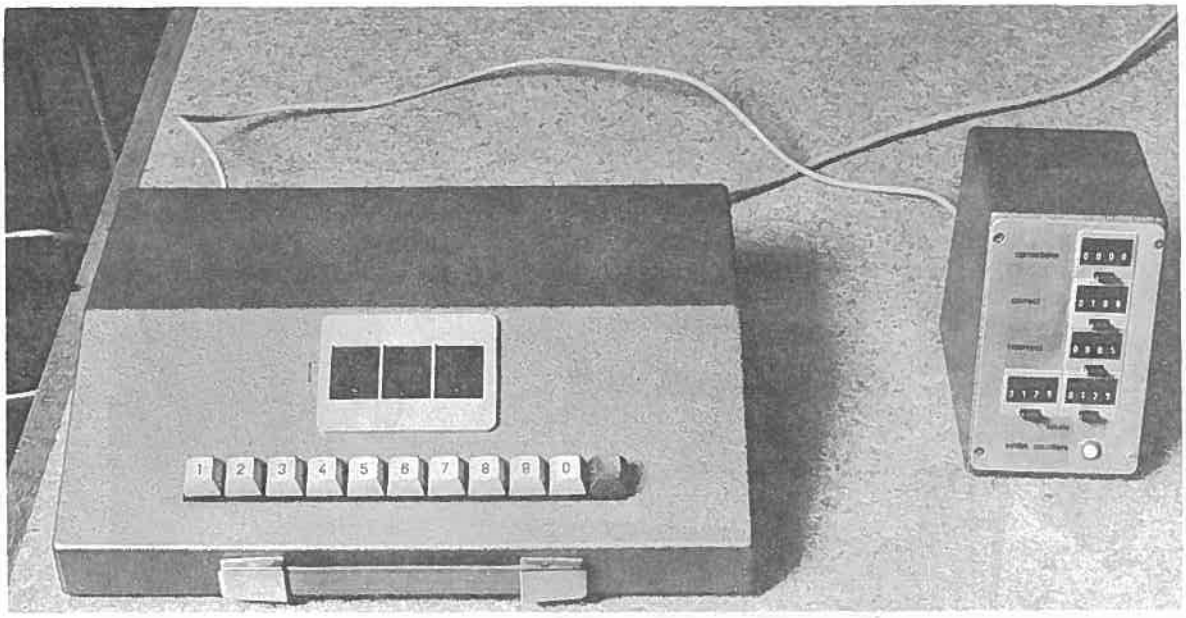

Figure 6: Arithmetic Test - Stimulus/Response and Recording Unit.

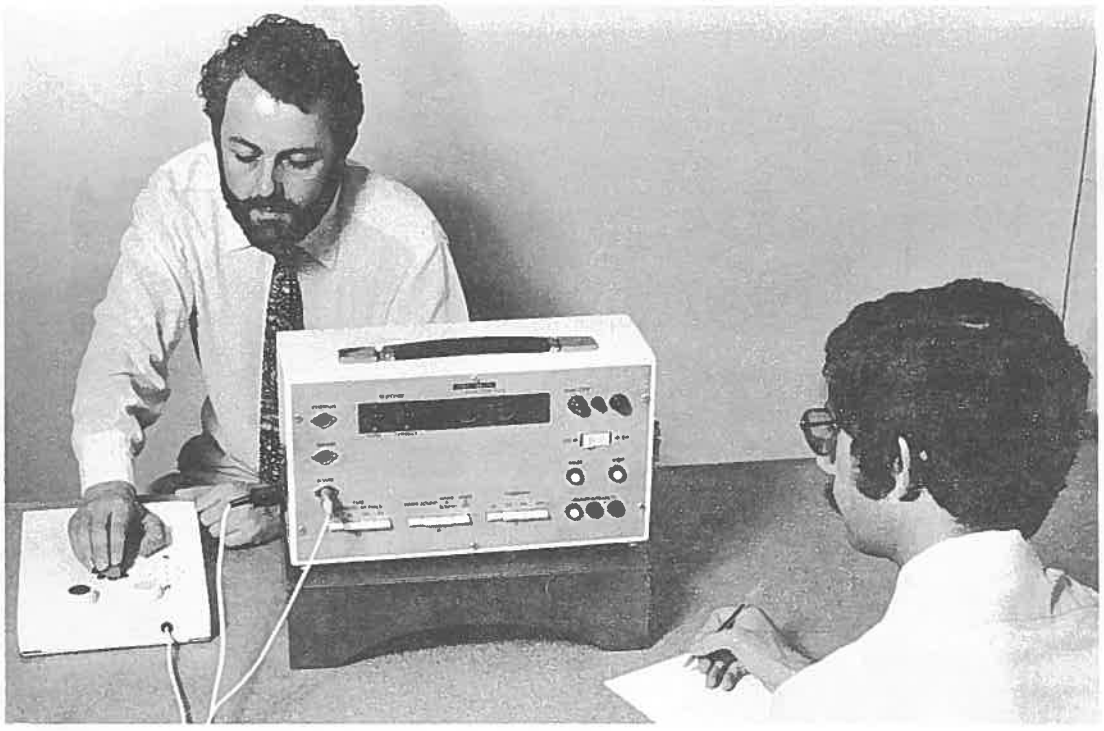

Figure 7: Reaction Time Test - Stimulus/Response and Recording Unit.

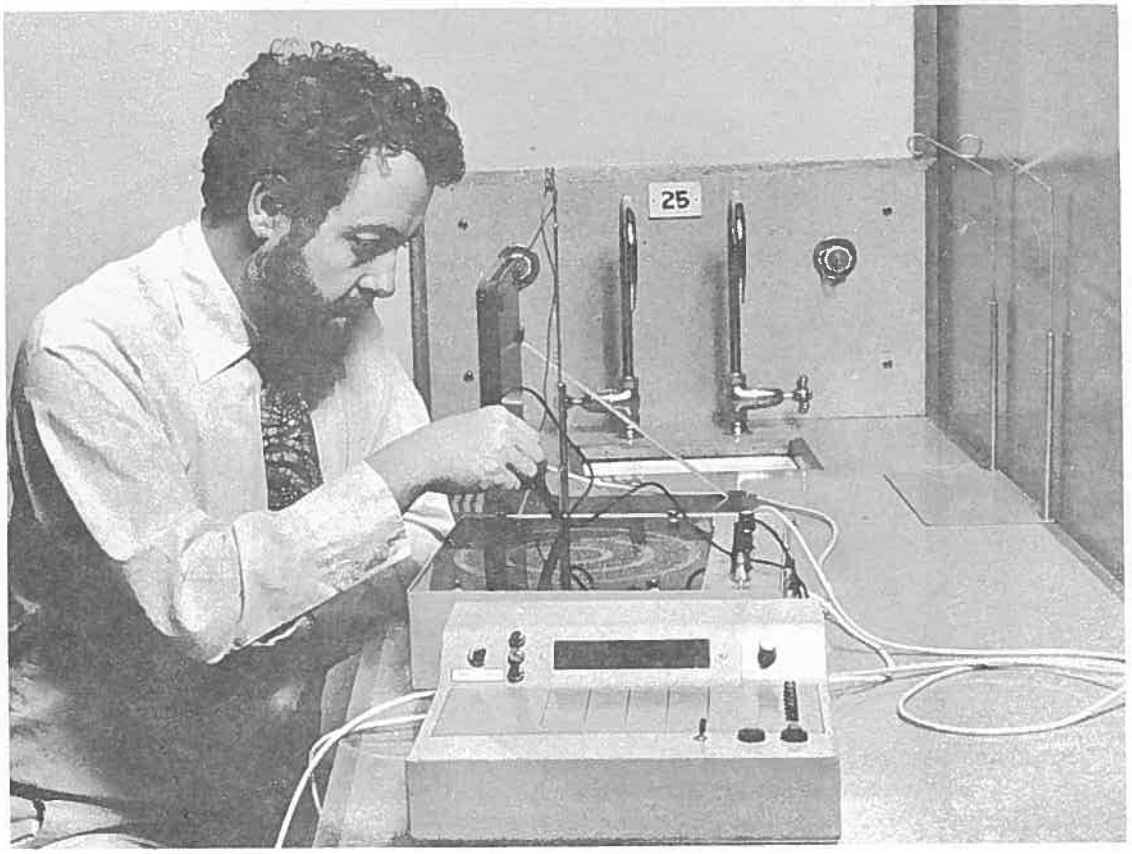

Figure 8: Motor Performance (Pursuit Rotor) - Stimulus/Response and Recording Unit.

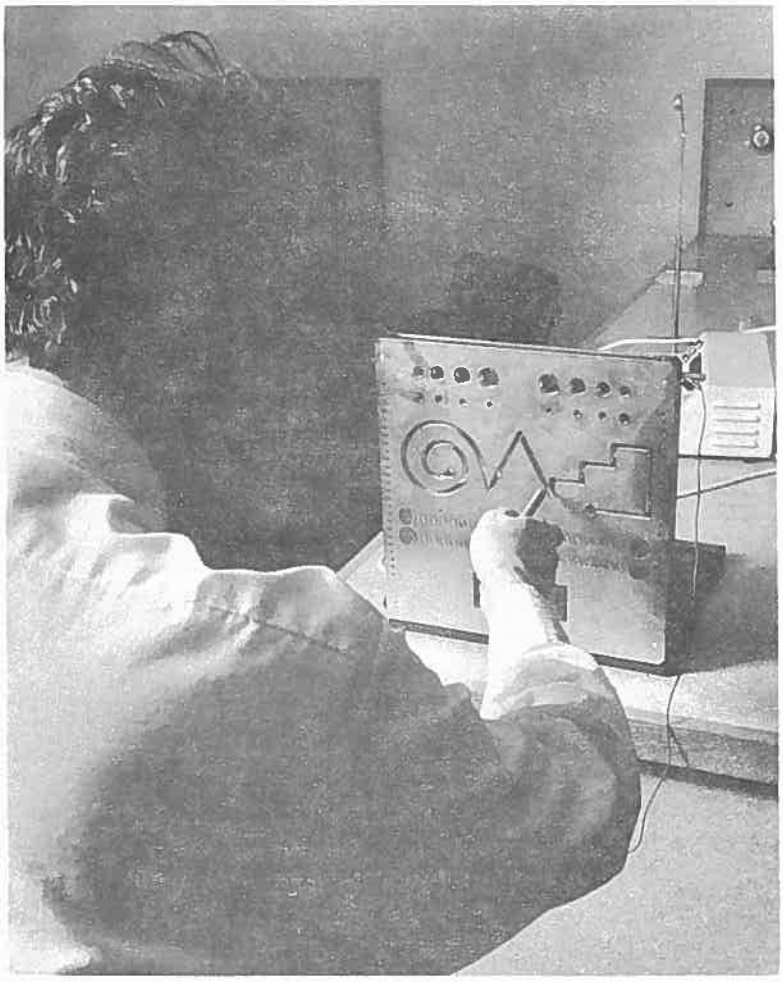

Figure 9: Motor Performance (Motorische Leistungsserie) - Work Panel.

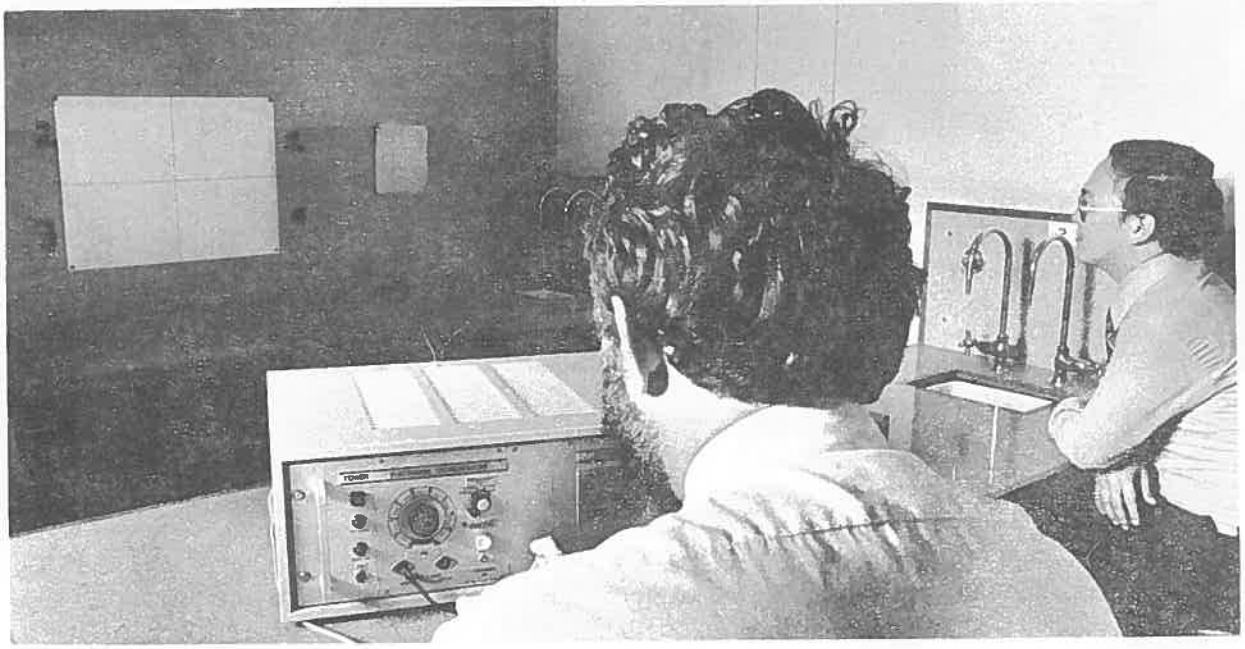

Figure 10: Perceptual Test (Tachistoscope) - Control Unit and Stimulus Display Board.

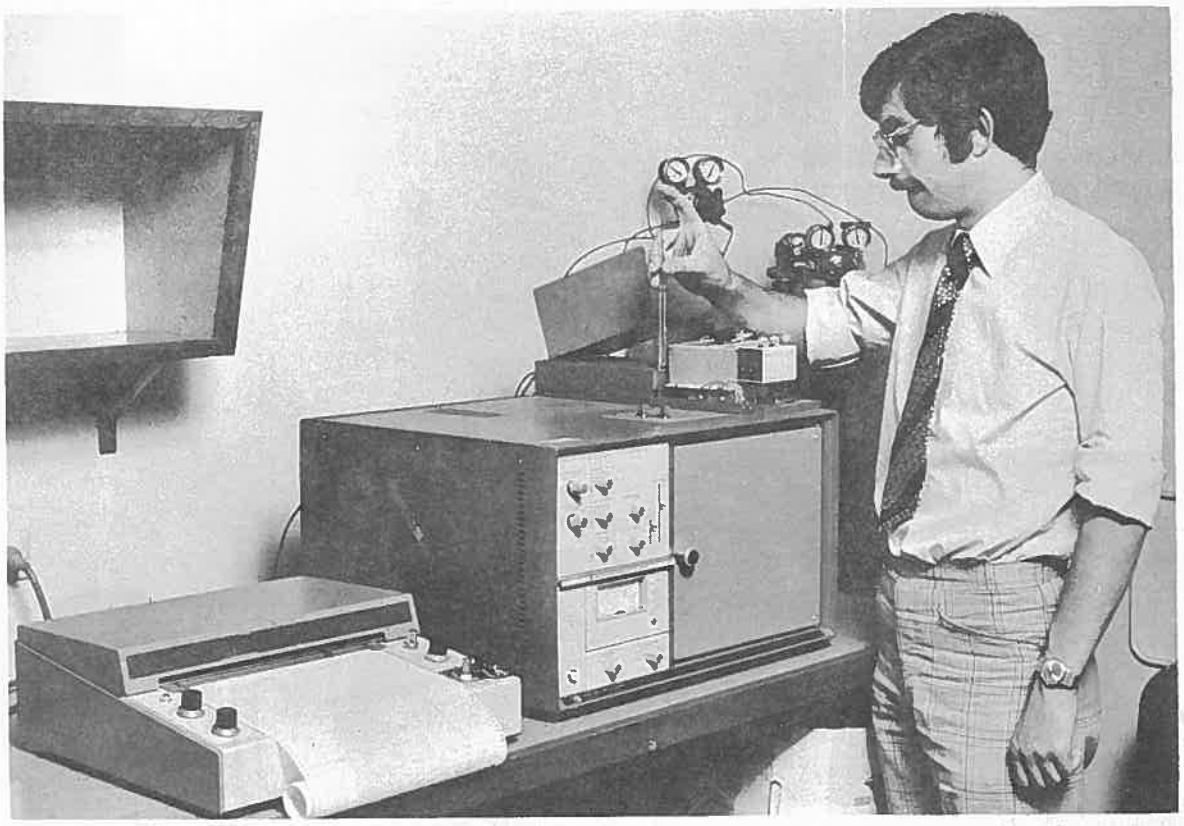

Figure 11: Gas-Liquid Chromatography - Analysis and Recording Unit.