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PHYSIOLOGICAL AND BEHAVIOURAL EFFECTS OF CIGARETTE SMOKING: IMPLICATIONS FOR CLASSIFICATION AND TREATMENT

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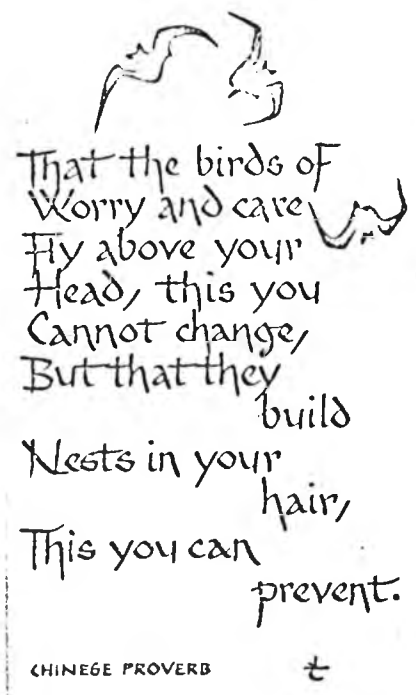
This thesis is gratefully dedicated

to my parents,

Sureyya and Fuat Yucesoy.



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PREFACE

This thesis is based upon a general interest in the understanding and modification of cigarette smoking.

A review of smoking modification methods is presented in chapter one. This review points out that although a diversity of treatment techniques have been applied to the modification of smoking behaviour, the general outcome has not been impressive. The common finding is that although almost any form of treatment induces short-term reductions or total abstinence in smoking frequency, relapse is a common phenomenon observed across treatments.

One possible reason for the failure of the treatment programs to extinguish smoking frequency successfully may be related to the major underlying assumptions prevalent in the area. Smoking modification researchers have generally viewed smokers as a homogeneous group, exhibiting a common problem behaviour, which is smoking. Thus, they have tried to develop a treatment strategy that will prove to be universally effective for all smokers. This approach does not consider the differences that might exist between individuals in their motives for smoking.

Research in smoking typology (chapter 2) shows that smokers give different reasons for why and when they smoke. Furthermore, the typology research points out the possibility of classifying smokers as pharmacologically addicted or non-addicted to smoking.

In chapter 3, the role of nicotine in the maintenance of smoking behaviour is discussed. Although, it is of crucial importance to

investigate the effects of nicotine manipulations on smoking parameters, this area of research is also dominated by the assumption that nicotine affects all smokers in the same way. Due to this view, findings on the role of nicotine for different types of smokers are very slender and at present inconclusive.

Experimental studies investigating the effects of cigarette smoking on physiological, behavioural and psychophysical measures are presented in chapter 4. Most of the measures discussed in this chapter are used in the experiments of this thesis. The aim of this chapter is to link the data from the verbal self-reports of smokers on why and when they smoke to objective evidence on the effects of smoking so as to formulate a model of smoking behaviour.

An analysis of these areas indicates that smokers may best be viewed as a heterogeneous group with different motives or a hierarchy of motives maintaining their smoking behaviour. This view necessitates the adoption of a differential treatment approach, by which appropriate treatment strategies can be devised to deal directly with the needs or motives of different types of smokers.

However, prior to the treatment phase, it is essential to develop a reliable method of identifying types of smokers. At present the classification of types of smoking is based on self-reports of smokers to questionnaires. So, firstly the objective validity of the typology scales needs to be established.

The experiments of the present thesis (chapters 5 and 6), were designed to investigate differences between pharmacologically addicted and non-addicted smokers as classified by Russell et al's (1974) "Smoking

Typology Scale". Differential effects of smoking and deprivation on these groups, in physiological, behavioural, and psychophysical measures and in smoking topography was investigated. The major aim of these studies was to provide an understanding of pharmacological addiction in terms of the effects of smoking and of cigarette deprivation.

On the basis of the findings from the two experiments of the present thesis and those reported in the literature, a modification of the concept of pharmacological addiction is proposed and its implications for a differential treatment approach to smoking is discussed in chapter 7.

CONTENTS

CHAPTER 1

THE MODIFICATION OF SMOKING BEHAVIOUR

1.1 INTRODUCTION

There have been numerous studies investigating the effectiveness of various intervention techniques in the modification of the smoking habit. Research interest in the treatment of smoking stems mainly from two sources.

Firstly, there has been growing evidence that cigarette smoking is causally linked or facilitative in the development of chronic bronchitis, cardiovascular disease, cancer of the lung and bladder, emphysema and other physical disorders (Royal College of Physicians Report on Smoking and Health, 1962; 1977; Report of the Surgeon Generals's Advisory Committee on Smoking and Health, 1964; 1973, American Heart Association, 1970). These findings have led to a general concern amongst smokers, and also an attempt by researchers to understand and modify smoking behaviour.

It has been reported that the majority of smokers (77%), want to stop smoking or have attempted to quit smoking unsuccessfully (McKennell & Thomas, 1967). However, only 15% of regular smokers were found to discontinue smoking and become permanent ex-smokers before the age of sixty, (Todd, 1972). These findings have pointed out the need for the development of effective treatment strategies to help smokers in their attempts to quit.

Secondly, smoking behaviour provides a useful field for more general hypothesis testing. Koenig and Masters (1965), stated that habitual smoking fits in perfectly with the theoretical, practical and logical requirements for investigations of treatment methods. Smoking behaviour is a maladaptive behaviour (i.e; health hazards imposed and the desire shared by 77% of smokers to quit), it occurs in discriminable units, thus lending itself

to objective measurement, and it occurs in high frequency in the population at large.

In this chapter experimental studies investigating the effectiveness of various smoking intervention techniques will be evaluated in the following sections:

A) General evaluation of treatment results,

B) Common methodological limitations,

C) Review of the "Smoking Modification" Literature,

D) Conclusions and directions for future research.

1.2 GENERAL EVALUATION OF TREATMENT RESULTS

A wide range of intervention techniques ranging from well controlled studies based on social-learning theory principles to pharmacological treatment methods, and to more ambiguous methods like hypnosis, have been used by investigators to modify cigarette smoking. However, the overall results, especially the long-term outcomes have not been very encouraging. The general findings can be summarized as follows:

i) There are no consistent differences between the outcomes of various treatment methods,

ii) Almost any form of treatment produces significant reductions in the smoking frequency or even total abstinence at the end of the treatment period,

iii) The impact of treatments are short lived and relapse is a common phenomenon. (Koenig & Masters, 1965; Keutzer, et al, 1968; Bernstein, 1969; Hunt & Bespalec, 1974; Bernstein & McAlister, 1976; Raw, 1977).

Large scale studies evaluating the outcomes of various smoking modification techniques have revealed high relapse rates following the termination of various treatment programs, (Hunt, et al, 1971; Marston & McFall,

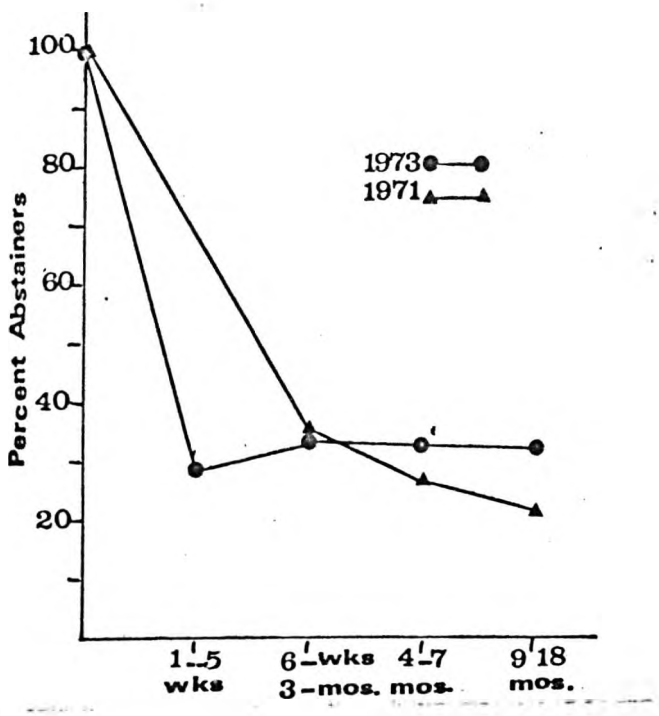
1971; Hunt & Bespalec, 1974).

Marston and McFall (1971), compared the outcomes of eight independent studies employing behaviour modification techniques in the modification of the smoking habit. They have noted that virtually all treated smokers had a 60 to 70 percent decrease in their consumption levels from the pre-treatment baseline smoking rate to the end of the therapy period. Including the drop-outs, total abstinence rates ranged between 7 percent to 40 percent with a mean of 26 percent at the end of treatment, and between 9 percent to 17 percent with a mean of 13 percent at long-term follow-up. Considering that most smokers participating in modification studies are well motivated to quit and receive treatment voluntarily, these findings are not very impressive.

In a more extensive review, Hunt and Bespalec (1974), evaluated the outcomes of 89 smoking treatment studies employing various techniques, published between 1968 and 1973. Such a composite evaluation provides a valuable criteria for assessing the success of individual treatment programs. The authors noted high relapse rates in all studies after the end of the treatment period which increased sharply during the three to six months follow-up period. Only 25% of smokers who completed treatment successfully and were abstemious at its termination were found to remain long-time abstainers. Figure 1.1 (overleaf) shows the mean relapse rates and time course of relapse reported by the studies included in this extensive review.

The authors have also compared individual treatment methods, in terms of the percentage of abstainers and percent reductions in pre-smoking rates at various follow-up periods. The variations in the follow-up periods and the differences in the criteria of success employed by various authors imposes some limitations on such a comparison. However, in terms of abstention

rates, "hypnosis", with a range of 15-88% abstention, was found to give the best results.



From Hunt and Bespalec (1974)

Fig. 1.1. Relapse rates after treatment for smoking.

Despite the wide range of techniques used by individual investigations and the improvement noted in research methodology, the prevention of relapse and the maintenance of treatment gains for the majority of smokers treated has not yet been satisfactorily dealt with. Bernstein (1969), in his evaluation of smoking modification studies has pointed out this failure, "the need for long-term maintenance of non smoking is largely ignored, with the result that much current research is following a directionless or, at best, circular course". An important area which has been neglected by the majority of researchers is the tailoring of treatment methods to subject variables. Although this approach has been emphasized in the application of behaviour modification techniques to other behavioural problems (e.g; obesity, Balch and Ross, 1975), there have been very few attempts of this amongst researchers

of smoking modification (e.g. Best & Steffy, 1971; Best, 1975). Research in smoking typology (see chapter 2), offers a valuable and promising adjunct to the tailoring of treatment strategies to the individual smokers' needs.

Hunt and Matarazzo (1973), have pointed out the need to tailor treatment to match individual needs, by stating ... "A last suggestion is that in designing our treatment programs we tailor them to the individual needs of the subject wherever possible and not rely on the blanket application of a common, mass attack", (pg. 112). Unfortunately, this suggestion has not been followed by the majority of researchers, and there are only a few studies reported in literature in this direction, (Harrington, 1978).

1.3 COMMON METHODOLOGICAL LIMITATIONS

i) SUBJECT SELECTION

In the evaluation of outcomes of smoking modification methods the selectivity of smokers participating in the smoking control studies needs to be considered. Mostly, subjects are voluntary participants, who are well motivated to quit smoking. The overall moderate impact of various treatment programs are even more discouraging considering the selectivity of subjects.

The second problem is the high-drop-out rates commonly observed in treatment programs. The prevention of drop-out has been tackled effectively by the collection of monetary deposits from the subjects returnable on the completion of treatment (e.g; Keutzer, 1968). The requirement of a deposit also serves the function of screening less motivated subjects and thus equates motivation levels amongst subjects to a certain degree. However, it can also be argued that this manipulation could well increase the subject selectivity, and produce a homogeneous group of well motivated smokers or alternatively smokers of a certain socio-economic class, who can afford to pay the

deposit without much inconvenience.

The major shortcoming imposed by the drop-outs is the lack of agreement between investigators on how to treat the data from such subjects. Although, some investigators include the data from drop-outs, the majority report outcomes only on subjects attending all the treatment meetings. It can be argued that the effectiveness of a particular treatment program can only be assessed by the behaviour of subjects completing the entire program. However, the exclusion of drop-outs leads to an artificial rise in the success rates reported, since by this method drop-outs are not considered as failures. Also, it is of interest to identify the characteristics of smokers stopping the treatment prematurely. There might well be some smokers who stop attending the treatment no matter what the type of treatment method is or, alternatively and more likely, some treatment programs might simply not be suitable for some types of smokers. The characterization of this group of smokers thus can aid in the understanding of appropriate treatment packages for smokers with different needs.

Control of relevant subject characteristics is another area of interest in experimental studies of treatment effectiveness. Although various treatment groups have been matched in terms of mean measures on some relevant characteristics, such as age, sex and number of cigarettes smoked, this can hardly be considered as a tight control. Between subject variations in the measures used have not generally been considered within experimental groups. Bernstein and McAlister (1976), in their review of smoking modification studies have also emphasized the need to match subjects on relevant smoking history and other variables and have cited two studies (Whitman, 1972; Levinson, et al, 1971), as exemplars of the practice of such a control of subject variables prior to random assignment to experimental conditions. However, an examination of the positive examples cited by these authors, reveals that the control

employed is far from being satisfactory. In Whitman's study the range in number of cigarettes smoked is between 10 - 60, the range in the years of smoking is between 2 - 43. Although the means of the treatment groups were not significantly different, the wide range in these measures represents what Hunt and Materazzo (1973), defined as .. "A blanket application of a common, mass attack". It is surprising that such studies are cited as employing control on relevant subject characteristics. Relevant subject characteristics should include "smoking typology" data, information on the parameters of smoking behaviour or physiological and behavioural responses during abstention from smoking. It seems more meaningful to form homogeneous groups of smokers, with a small range in the relevant characteristics chosen in a particular investigation, rather than attempting to establish groups of smokers, with a wide variation between subjects of each experimental group, but no significant difference between the means of different experimental groups.

A more promising and potentially fruitful approach would be to select subjects according to well defined and strict criteria and subsequently subject these groups to treatment techniques that seem appropriate for their characteristics. The problem of lack of strict control of subject variables stems partly from the notion of a universal and effective treatment program for a random sample (heterogeneous group) of smokers. Once a differential treatment approach is adopted the importance of forming homogeneous experimental groups on the basis of a relevant criterion becomes obvious.

ii) EXPERIMENTAL DESIGN

In investigating the effectiveness of different treatment strategies it is of great importance to include attention-placebo treatments with sufficient credibility for the subjects, and control groups of unaided smokers. A major design deficiency of earlier studies has been the omission of these control groups. However, the majority of recent studies have included

attention-placebo and unaided quitting groups to control for the non-specific factors (i.e; attention from therapist, commitment for treatment etc.) involved in treatment programs. Bernstein and McAlister (1976), recommended the inclusion of these experimental groups as a routine in every smoking modification study.

The second major problem is the variation in the duration of treatment, and follow-up periods amongst the published studies. Although the differences in the duration of the treatment periods are justifiable, in the case of follow-up periods it is highly undesirable. Since the main problem in the area of smoking modification is the maintenance of the treatment gains, reports of outcome at standard follow-up periods are crucial for comparing the effectiveness of different treatment methods. In some studies the follow-up periods are too short to provide a reliable measure of long-term success (e.g; Sachs, et al, 1970), and still in others no follow-up measures were taken (e.g; Upper and Meredith, 1970).

In addition to the above considerations, base-rate smoking data needs to be collected by careful recordings over a reasonable time period (e.g; one week), and whenever possible independent observers should be used to check the reliability of subjects' reports (e.g; Ober, 1968; Best, 1975). Number of cigarettes smoked generally represents the only dependent measure in the smoking modification studies. In the light of experimental evidence (Ashton and Watson, 1970), suggesting that smokers can also alter their smoking parameters (i.e; puff-rate, depth of inhalation, etc.), in response to nicotine manipulations, the validity of crude consumption levels needs to be investigated. Lando (1975), recommended the inclusion of breath CO levels, as an objective measure to supplement self-reported smoking data. A significant positive correlation was noted between consumption levels and breath CO levels. The author analyzed the breath samples of smokers who claimed abstinence for two months and those who reported continued smoking. The breath samples of

the former group was found to have five to 11 ppm of CO, whereas for the latter group CO ranged between 36 to 80 ppm. These findings tentatively supported the reliability of self-report data.

This section will present a review of the most widely used smoking intervention techniques. The impact of national anti-smoking campaigns, anti-smoking legislation and tobacco taxation have been excluded.

1.4 REVIEW OF LITERATURE

The general aim of all the treatment techniques that will be discussed in this section is to extinguish or reduce the frequency of smoking behaviour. However, different components of the smoking behaviour (i.e; cues leading to smoking, consequences, withdrawal symptoms, social factors or attitudes) have been dealt with depending on the view of what maintains the smoking behaviour.

1) Social-Learning Theory Approaches:

There is agreement amongst reviewers of smoking literature that social-learning approaches seem to be the most promising, mainly because of their emphasis on controlled hypothesis testing and operational definitions, which is more likely to generate valuable theoretical and practical knowledge about smoking behaviour, (Keutzer, et al., 1968, Bernstein, 1969., Lichtenstein & Keutzer, 1971., Bernstein & McAlister, 1976).

Social-learning approaches have aimed at either a) reducing the probability of smoking behaviour or b) increasing the probability of non-smoking behaviour or incompatible responses to smoking.

Despite the superiority of research methodology employed, social-learning approaches have not produced better treatment outcomes especially in

terms of long-term abstinence as compared with other methods of intervention, (e.g; hypnosis).

In the following section each of the major social-learning techniques will be discussed and evaluated.

Aversive Conditioning

Among the wide range of behavioural methods designed to inhibit and extinguish smoking, more attention has been directed to develop and evaluate aversive conditioning techniques (e.g; electric shock, rapid smoking, warm smoky air, covert sensitization, etc.) than to any other single approach. Aversive conditioning techniques attempt to modify smoking behaviour in two ways, viz.,

a) by making the aversive stimulus contingent upon actual or imagined smoking, or b) by pairing contiguously the total smoking response with a noxious stimulus, thus developing a conditioned aversion response in the subject and a consequent conditioned avoidance response.

The following factors need to be considered in developing an effective aversive technique for controlling smoking behaviour:

a) The "aversive" stimulus must indeed be aversive and its mode of administration should be reasonably convenient.

b) It should be possible to administer the procedure wherever and whenever the response naturally occurs.

c) The aversion should ideally be based upon intrinsic stimuli to the undesired response, (e.g; rapid smoking).

There is a mixed picture on the effectiveness of contingent versus non-contingent electric shock. Levine (1974), investigated the effects of contingent versus non-contingent electric shock in reducing cigarette consumption and has found that there were significant reductions in smoking frequency only in the contingent-shock group. However, Russell, Armstrong & Patel

1976), in a similar investigation, examining the effects of the temporal contiguity in electric aversion therapy, have reported that all treatments (i.e; contingent versus non-contingent electric shock, non-shock, simple support and attention from the therapist) were equally effective in reducing consumption, and that the effects of contiguous versus non-contiguous shocks did not differ. On the other hand, Pope & Mount (1975), using an electronic portable apparatus which emitted an auditory signal followed by a shock delivered to the subjects' preferred smoking arm in relation to his smoking frequency throughout his waking hours, reported a very high (80%) abstinence rate at a one year follow-up. This finding compares very favourably with the general outcomes of smoking treatment programs and its superiority could be due to the delivery of the shock continually in the natural setting whenever smoking occurs, as compared to laboratory aversive conditioning programs.

Administration of aversive taste substances contingent upon and concomitant with smoking was not found to have a permanent effect on the smoking behaviour (Marston & McFall, 1971., Whitman, 1972). However, Rosenberg (1977), found that the use of a chewing-gum, that produced an unpleasant taste in the mouth when tobacco smoke is inhaled, for two weeks was effective as a smoking deterrent. The reduction in cigarette consumption was still demonstrable one month after the end of the treatment. Although the results seem promising a replication with a longer follow-up period is warranged.

The studies discussed above employing electric shock and aversive taste substances mainly dealt with the overt (i.e; motoric) smoking responses but not with the covert (i.e; cognitive) responses (e.g; verbalizations) related to the smoking behaviour. Some investigators used aversive techniques to punish both the cognitive and also the motoric components of the smoking behaviour. Berecz (1972), investigated the therapeutic effectiveness of self-administered shocks contingent upon imagined smoking. It was predicted that if imagined smoking was punished, corresponding overt responses

would also be suppressed. It was found that for moderately smoking male subjects shock for imagined versus actual smoking was equally effective, whereas for heavy-smoking males, imagined smoking treatment was the most effective. In a later study, Berecz (1974), reported a procedure in which smokers self-administered shocks while having an urge to smoke. This procedure was directed at punishing the early components of the total smoking behaviour chain. The author reported that three subjects treated with this procedure were abstinent two years after the completion of the treatment program. Although the small number of subjects involved makes it difficult to draw reliable conclusions from this study, it seems to be a promising approach which can be used in natural settings and thus have a potential for generalizability.

The imaginary aversive conditioning technique of covert sensitization (Cautela, 1970), has also been used to modify smoking behaviour. In this technique the subject is required to imagine that he is about to engage in smoking. Then he is instructed to imagine that he is receiving a noxious stimulus (e.g; feeling of nausea and vomiting). This procedure is labeled as covert sensitization because both the behaviour to be modified and the aversive stimulus are presented in imagination, with the aim of producing avoidance behaviour. The outcomes of experimental studies employing covert sensitization procedures have not produced results to support its superiority to other techniques or placebo procedures in terms of both end of treatment and long term effectiveness (Wagner & Bragg, 1969; Wisocki & Rooney, 1974; Barrett & Sachs, 1974; Sipich., et al, 1974). Barrett and Sachs (1974), investigated the effectiveness of covert sensitization with a refined procedure. They have employed several variations of the covert sensitization procedure by arranging the order of the aversive scene (U.C.S.) and the smoking scene (C.S.) differently in each of their experimental groups, (i.e; smoking scene $\longrightarrow$ aversive scene), a backward conditioning group (i.e; aversive scene $\longrightarrow$ smoking scene), an interval group (i.e; same as backward conditioning group,

except an interval of 60 seconds was imposed between the two scenes), and finally a no-association group (i.e; presentation of the aversive scene only). The results showed that all treatments were equally effective in reducing cigarette consumption. Since both the interval and no association groups were as effective as the other two, the classical conditioning explanation was discarded in favour of cognitive, motivational, or non-specific factors involved.

In another approach of aversive conditioning of smoking, cigarettes themselves, have been used as the aversive component of the treatment program. Three main versions of this approach have been reported: Satiation (i.e; doubling or tripling smoking rate) before quitting and aversive conditioning of smoking by rapid smoking or by experimenter controlled warm-smoky air.

The satiation method is based upon the following assumptions: a) with continuous excessive presentation, the reinforcer loses its rewarding properties altogether and may actually become aversive, b) aversion based upon stimuli intrinsic to the response to be extinguished will have more likelihood of generalizability than that stemming from artificial sources (e.g; electric shock).

Resnich (1968), employing the satiation technique produced promising short-term results (60% abstinent after four months). Marrone, Merksamer and Salsberg (1970), replicated the above findings. They have also showed that outcome was related to the length of treatment. From the two experimental groups, the 20-hours satiation group had a higher percentage of abstainers (60%), as compared to the 10-hours satiation group (18%) at a 4 month follow-up period. However, recent research on the satiation technique failed to demonstrate its long-term effectiveness (Marston and McFall, 1971), or its differential efficacy to placebo (Claiborn, Lewis and Humble, 1972),

or other active treatment methods (Marston and McFall, 1971).

Similarly, aversive conditioning of smoking by warm smoky air was not found to produce long-term abstinence (Wilde, 1965; Franks, Fried & Ashem, 1966). However, Lichtenstein et al (1973), reported 60% abstinence rate after six months, when warm, smoky air was used in conjunction with rapid smoking.

Rapid smoking technique, used alone have not been found to produce strong treatment effects (15 - 20% abstinence after a year), (Lando, 1974). Social factors and positive expectations of the smokers treated were found to be related to treatment outcome (Harris and Lichtenstein, 1974).

In summary, although various aversive conditioning techniques have been shown to produce short-term reductions in cigarette consumption, their long-term effectiveness have not been demonstrated. Although techniques employing cigarettes themselves as the aversive component seem to be more promising in terms of generalizability of treatment effects than the use of electric shock, the results produced with all the aversive conditioning techniques seem to be confounded with non-specific treatment factors (i.e; social support, positive expectations, etc.).

Stimulus Control

Yates (1970), stated "Clearly the initiation of the smoking sequence becomes attached to so many environmental stimuli that laboratory control of smoking is unlikely (however successful), to maintain control over smoking behaviour outside the laboratory". Stimulus control approach is based on the assumption that smoking response is a learned habit that is initiated by a wide range of environmental stimuli. Thus, the treatment aims at restricting the distriminative cues initiating the smoking response, by restricting smoking to only

specified temporal periods or to specified environmental stimuli (e.g; buzzer of a pocket timer).

Stimulus control programs vary greatly in respect to how strictly they restrict the smoking response to specific environmental stimuli. Studies employing timed signalling devices to initiate smoking have revealed that the reduction in cigarette consumption finally stabilizes at a certain level (10 - 12 cig/day), after which further decreases in consumption become unlikely (Upper & Meredith, 1970; Bernard & Efran, 1972). This "floor" effect has been attributed to the appearance of pharmacological "withdrawal symptoms" by some authors (Levinson, et al,1971), or alternatively as an increase in the reinforcement value of the cigarettes as the level of consumption is reduced to fewer cigarettes, which makes the smoking response highly resistant to extinction, (Mausner, 1971). Both of these explanations seem to be plausible since, as will be seen in chapter 6, the reduction in cigarette consumption is likely to produce smoking behaviour that closely resembles post-deprivation smoking (i.e; with fewer cigarettes, there are longer time gaps between cigarettes). Smoking after a period of deprivation has been noted to produce marked changes in physiological activation levels. On the other hand, deprivation has been noted to produce lowered physiological activation levels as compared to normal smoking periods. Thus, with the gradual reduction method, it is likely that the smokers will experience some withdrawal symptoms. However, they will achieve more marked physiological effects with each cigarette, which may make each cigarette more reinforcing and thus further reductions in consumption may become difficult.

Self-Control Strategies

In this approach although stimulus-control techniques play a great part, attention has also been directed at teaching smokers various self-control techniques, like self-reward, self-punishment, self-monitoring, non-

smoking skills and environmental planning, (Harris & Rothberg, 1972).

Successful results have been reported in individual cases, where smokers were restricted to smoke only at a particular place, without engaging in any other activity (Nolan, 1968; Roberts, 1969). Although, self-control strategies in their flexibility, offer valuable possibilities for the tailoring of treatment methods with the needs and/or habits of individual smokers, more research is needed to delineate the role of various components of these procedures. More recent research has revealed that self-monitoring, usually employed to gather data on base-rate smoking frequency, reduces consumption levels and thus is a reactive measure, (McFall, 1970), and furthermore the timing of self-monitoring was also found to be important. Keeping a written record of consumption prior to lighting a cigarette was found to be more effective in reducing consumption than recording it after smoking (Rozensky, 1974).

In summary, although at present self-control strategies do not appear to produce long-term effects which are superior to other active or placebo treatments, more research is needed on its different components and also on the effects of tailoring self-control strategies to the needs of individual smokers.

Contractual Management

Behavioural contracts, providing social and/or monetary rewards contingent upon non-smoking have also been used as anti-smoking procedures. Contractual procedures have been found to produce short-term reductions in smoking frequency (Tooley & Pratt, 1967; Elliott & Tighe, 1968). Elliott & Tighe (1968), using a contractual system, in which subjects were required to deposit some money with the therapist which would be returned either to them or to other group members contingent upon the subjects smoking during

the specified periods, found a high success rate (84%), at the end of treatment. However, this rate was reduced to 38% at a fifteen month follow-up. It has been suggested that this procedure could be useful as a component of a more elaborate treatment program (Bernstein & McAlister, 1976).

Systematic Desensitization

Systematic desensitization (S.D.),[1] has also been used as a smoking intervention technique, either to relieve the tension caused by cigarette withdrawal or to reduce stress which leads to smoking. However, the rationale for employing S.D. does not seem to be justified for all types of smokers.

The occasions on which people usually smoke differ (McKennell & Thomas, 1967), and only some people report smoking to alleviate anxiety (Russell, Peto & Patel, 1974; Horn & Waingrow, 1966). So, although the relaxation training and an S.D. program might be an appropriate form of treatment for "Negative Affect" (tension reduction) smoking (Horn & Waingrow, 1966), or "Nervous Irritation" smoking (McKennell, 1970), or "Sedative" smoking (Russell, Peto & Patel, 1974), it does not seem to be a suitable choice of treatment for other types of smokers or smoking (e.g; "Indulgent Smoking - stimulation smoking" - See chapter 2).

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(1) Systematic desensitization - Method of treatment, based on learning theory principles. Subject is trained in muscular relaxation and subsequently he is required to think about anxiety or fear producing events while he is relaxed. The rationale is that relaxation is incompatible with fear or anxiety and that the subject can be reconditioned.

The results of controlled studies on the effectiveness of S.D. as an intervention method for smoking have failed to support its superiority to other active treatment methods or to non-specific factors involved in treatment programs (Koenig & Masters, 1965; Wagner & Bragg, 1970; Gerson & Lanyon, 1972). Wagner & Bragg (1970), used a combined covert sensitization and S.D. group, and noted that this group was more successful at follow-up (less increase in cigarette consumption) as compared to other treatment groups (S.D. alone, C.S. alone; relaxation alone, counselling.)

So, at present S.D. used on its own, does not appear to be more effective than any other treatment method. However, tailoring of S.D. and relaxation procedures with smokers who smoke mainly under anxiety provoking situations might provide more promising results.

In general, cigarette withdrawal, no matter what the nature of the dependence is (psychological or pharmacological) is likely to produce a need state, which in turn would produce tension. However, the alleviation of this tension by relaxation training or S.D. does not offer any alternative to the alleviation of the needs satisfied by smoking. Thus, although S.D. can be used as an adjunct to treatment with some smokers it needs to be supplemented with other treatment methods which aim directly at substituting alternative means of satisfying the particular motives for smoking in order to establish non-smoking behaviour.

MULTICOMPONENT TREATMENT PACKAGES

Some researchers have employed various social-learning strategies to form multicomponent intervention packages (Harris & Rothberg, 1972; Delahunt & Curran, 1976; Elliott & Denney, 1978). Aversive conditioning, training in self-control and non-smoking skills, environmental planning, contractual management and post-treatment contacts have been used in combination to modify the smoking behaviour. Although, at present there are few published reports on the effectiveness of such programs, Bernstein and McAllister (1976), have stated that "the multicomponent approach appears quite promising and would seem to warrant further and more rigorous evaluation" (pg; 97).

However, with this approach it is quite difficult to isolate the active components of the package and it might well be that the variety of strategies employed are differentially suitable for different smokers under treatment. In other words, with multicomponent packages, smokers are presented with a wide selection of treatment methods and "which" intervention method affects "who" is not explored.

Flaxman (1974), (from Bernstein & McAlister, 1976; pg. 97), reported a 63% abstinence rate at a six month follow-up, among smokers who quitted abruptly after being exposed to a multicomponent treatment program. Delahunt & Curran (1976), combined self-control strategies and negative practice procedures in a treatment package and noted a 56% abstinence rate for the nine subjects in the package condition. Lando (1977), reported a 76% abstinence rate 6 months after administering a treatment package that included aversion therapy, contractual management, booster sessions, group contact and support. Finally, Elliott and Denney (1978), compared the effectiveness of a package treatment which included eight different components (rapid smoking, relaxation, covert sensitization, systematic desensitization, self-reward

and punishment, behavioural rehearsal and emotional role playing) with a single treatment approach (rapid smoking) and control groups receiving no treatment or receiving only the non-specific factors involved. Although, this study was well controlled for therapist effects (multiple therapists) and checks were made on the reliability of reports on consumption levels by independent observers, the package program seems to cover almost the whole range of social-learning techniques and thus it seems to be quite obvious that due to the impact of such an intense program, be it specific or placebo, it will have more likelihood of producing superior results to those of a single treatment approach (i.e; rapid smoking). The results, not surprisingly, supported the superiority of the package program, which was found to produce a 45% abstinence rate six months after the termination of the treatment program.

Elliott & Denney (1978), advocated the use of multicomponent treatment packages by stating "from a strategic point of view, it would seem advisable to devise a complex treatment program that achieves the desired results in terms of bringing about persistent changes in these maladaptive behaviours and then subsequently to perform analytical studies to discover the effective components operating within the package" (pg; 1331). However, the problem is not only the alienation of the active components of the treatment program but also to explore "component and type of smoker" interactions.

In conclusion, although multicomponent approaches have produced more favourable long-term (6 months) results to those of single approaches, active components of the packages and "component-subject characteristic" interactions have not been explored. In terms of economy of treatment it would seem to be more appropriate to employ techniques that are based on a comprehensive and reliable understanding of the controlling variables maintaining the smoking act in homogeneous groups of smokers, rather than

employing a mass attack, by which higher abstinence rates are produced, but yet the active agent is unknown.

ii) PHARMACOLOGICAL TREATMENT APPROACHES:

Two main drug therapy approaches have been adopted by researchers in aiding cigarette withdrawal. The first one aims at alleviating the physical and psychological withdrawal symptoms (i.e; tension, irritability, hunger, craving, impairment of concentration, etc.), frequently reported by smokers with cessation of smoking, by prescribing tranquilizers (Whitehead & Davies, 1964), amphetamines or an amphetamine-barbiturate mixture (Ross, 1967). Research on the effects of these drugs have shown that they only produce temporary reductions in cigarette consumption, which is not superior to placebo drugs.

The second approach is based on the view that nicotine and its pharmacological actions play a predominant role in the maintenance of cigarette smoking. (See chapter 3). In line with this assumption either nicotine itself or drugs which mimic the pharmacological effects of nicotine have been prescribed for smokers. The most widely tested nicotinomimetic agent is lobeline, which produces pharmacological effects that closely resemble those of nicotine (Scott, et al, 1962; Davison & Rosen, 1972).

Lobeline sulphate was first tested as a smoking deterrant in 1936 by J. L. Dorsey. Although no data was reported, positive results were indicated. However, severe gastrointestinal side effects were noted with the ingestion of lobeline sulphate (Wright & Littaur, 1937). To overcome the unpleasant side effects some researchers used smaller doses of lobeline sulphate combined with fast-acting and slow-acting antacids (i.e; Bantron), and earlier studies with this compound yielded high short-term abstinence rates (60%), (Rapp & Olen, 1955). However, later a more controlled study by Bartlett & Whitehead (1957), did not confirm the above positive findings for the effectiveness of Bantron.

Research on the effects of other lobeline preparations (e.g: lobidan) as a smoking deterrant did not produce results to support their superiority over placebo tablets (Merry & Preston, 1963; Davison & Rosen, 1972). So, in conclusion although lobeline, in various compounds have been tested as a smoking deterrant by various researchers, it does not seem to be more effective than placebo tablets in the treatment of cigarette dependence. Bernstein & McAlister, (1976), in their review of drug therapy approaches to the modification of smoking, have stated that "Research on the effects of these agents has shown them to be relatively weak, temporary, and primarily a function of placebo and other non-specific effects associated with receiving medication rather than of specific drug characteristics". (pg; 91).

More recently nicotine itself has been administered to smokers by a nicotine containing chewing-gum, developed in Sweden (Fernö, Lictneckert & Lundgren, 1973), as a smoking deterrant. Although, nicotine-chewing-gum has been used to investigate the role of nicotine on short-term changes in smoking parameters, (see chapter 3), at present there are only a few studies evaluating its effectiveness as a long-term smoking deterrant. Brantmark et al (1973), in a one-week double-blind study compared the effectiveness of nicotine and placebo gums in aiding cigarette withdrawal. Their results supported the superiority of the nicotine-gum, especially among heavier smokers. Schneider et al (1977), in a case study, also found that nicotine-chewing gum was effective in helping a heavy smoker ($2\frac{1}{2}$ packs/day) to abstain from smoking. The subject was reported to be still abstemious at a 7 months follow-up. Both of the above studies indicate that heavy smokers are more likely to be dependent on nicotine and that a nicotine substitution technique might be a helpful adjunct to therapy for heavy smokers. At present due to the small number of subjects involved in the published reports it is not possible to draw a firm conclusion on the usefulness of nicotine chewing-gum as a smoking deterrant.

iii) Hypnosis

Hunt & Bespalec (1974), in their evaluation of various smoking treatment methods have commented on hypnosis as follows: "With a range from 15% to 88% this perhaps gives us our best results (for abstention)" (pg; 435).

Crasilneck and Hall (1968), reported an 82% abstinence rate at a one year follow-up period, using hypnosis. Similar favourable results were also obtained in some other studies using hypnosis (Watkins, 1976; Von Dedenroth, 1964, a, b; Nuland & Field, 1970). Although the results reported are impressive, in most of the studies the procedures are not clearly defined and other techniques such as contact with therapist via telephone calls (Nuland & Field, 1970), and various self-control strategies (Von Dedenroth, 1964, a, b) have also been used in conjunction with hypnosis.

When subjected to careful experimental control hypnosis was not found to produce better outcome than nonspecific placebo treatments (Perry & Mullen, 1975), or from a pharmacological treatment approach using lobeline (Edwards, 1964).

The main problem in the evaluation of the studies employing the so called "hypnosis" technique is the lack of an operational definition that clearly describes the procedures utilized. For this reason although most authors claim success with the technique the results can not be replicated. Mostly the treatment offered is complex, including unsystematically applied social-learning techniques, such as hypnotic suggestions to give cigarettes aversive taste or to associate smoking with aversive events and self-control strategies (i.e; smoking bans during certain periods, planned cut-downs, etc.), so that it is difficult to evaluate the specific role of hypnosis. In other words, at present it is not possible to delineate the effects of "hypnosis" per se, from other techniques embedded in the treatment packages, and thus conclusions on the effectiveness of this technique in the modification of

smoking awaits well controlled experimentation with matched control groups, clear definitions of the procedures employed and long-term follow-up data. Hypnosis might have therapeutic utility for some smokers, and for this reason it is important to investigate the characteristics of subjects who respond well to hypnosis.

iv) SMOKING CESSATION CLINICS AND GROUP THERAPY APPROACHES

Clinics mainly based on "group-therapy" approaches have been used widely to help smokers in their attempts to quit. They provide a setting of social support, in which smokers can come together and discuss either amongst themselves or with the therapist various aspects of their smoking habit , the difficulties encountered upon while quitting and how to resist temptation while abstaining. In conjunction with the support offered by the group setting, lectures on the health hazards of smoking, film shows, educative pamphlets, drug therapy and hypnotherapy have also been used in these clinics, (Cruickshank, 1963; Ross, 1967; Graff, et al, 1966).

Originally, clinics were formed purely as therapeutic settings and for this reason reports from the earlier clinics do not include data from control groups. Therefore, it is difficult to evaluate their effectiveness in producing behavioural changes, (e.g; Ejrup, 1964).

Smoking cessation clinics differ widely in terms of length, number and intensity of meetings, the duration of the treatment program, the specific treatments employed and the length of follow-up. (Keutzer, et al, 1968; Bernstein, 1969; Raw, 1977).

Success rates in terms of abstention or reductions in cigarette consumption at the end of the treatment period ranges from 34% (Ross, 1967), to 75% (Lawton, 1962). Although, the short term results seem promising,

especially considering the economy of treatment, the long-term results are very similar to those of other active treatments and placebo-treatments employing the non-specific factors (i.e; motivation, support from other group members and therapist, etc.) involved in group settings. The relatively low success rate reported by Ross, could be due to his inclusion of drop-out subjects as failures, which points out the need to adopt a standard reporting procedure by all researchers in this field. At one year follow-up periods, results are not very encouraging, success rates falling to 15 - 20%, (Lawton, 1962; Ejrup, 1964).

So, the clinics compare favourably with other treatment methods in producing temporary changes in smoking behaviour, however they produce similar long-term results to those of other treatment methods, active or placebo.

Studies employing control groups who were instructed to try quitting on their own have revealed that the group therapy approach is not superior to subjects' unaided efforts (Lawton, 1967; Mausner, 1966; Schwartz & Dubitzky, 1967). Also, the use of non-specific factors that are present in clinic settings (suggestion, motivation, participation, structure, etc.) in isolation was found to produce similar results to those of carefully planned clinic programs (Bernstein, 1969; McFall & Hammen, 1971; Lichtenstein, et al, 1973; Sipich et al, 1974).

Bernstein (1969), in his review of clinic approaches to the modification of smoking has commented "most clinic procedures reported to date represent a great deal of wasted time and effort" (pg; 431). In fact, considering the similarity of long-term success rates reported by clinic studies and other active treatment programs (around 20% abstinence rate at one year follow-up), two conclusions can be drawn. Firstly, the clinic results compare favourably with some other treatment methods (e.g; individual aversion therapy or hypnotherapy) in terms of economy of treatment (therapist

time and number of subjects treated). Secondly, group therapy approach, similar to most of the other therapy approaches is based on procedures which are assumed to be effective in controlling smoking behaviour. However, without a detailed and clear understanding of the hierarchies of controlling variables in individual smokers the application of haphazardly selected procedures, like the discussion of health hazards, hypnotherapy, social support etc., to heteregeneous groups of smokers seems unjustified.

So, at this stage it seems premature to conclude that clinics are a waste of time and with proper selection of subjects and supplementing techniques clinics might provide an economical and efficient method of modifying smoking behaviour.

v) SENSORY DEPRIVATION

Based on the evidence that a period of sensory deprivation leads to a generally increased persuasibility and responsiveness to external cues (Suedfeld, 1973), Suedfeld & Ikard, 1974 , employed sensory deprivation (S. Depr) (isolation in a dark chamber for 24 hours), and delivered antismoking messages (once in every 1½ hours), in an attempt to modify cigarette smoking. They have also used control groups of S. Depr. alone, message alone, and no messages-no S. Depr. (S's asked to seek treatment elsewhere). One year after the treatment the S. Depr.-message group was found to have reduced their rate of cigarette consumption by an average of 48%, compared with 16% for the control subjects. It was noted that a set of antismoking messages delivered without S. Depr. had no permanent effect. However, in terms of abstinence the outcome does not appear to be superior to other treatment methods. 27% of S. Depr.-message subjects and 6% of S. Depr. alone subjects were found to be abstinent at a 1 year follow-up.

More research is needed to delineate the active components of the S. Depr. technique, and the characteristics of smokers who respond well to this treatment method. For some smokers, a 24 hour deprivation period

might be too aversive and they might prefer to subject themselves to the hazards of smoking rather than to the long isolation period. In addition, with this method even if the smokers are persuaded to participate in the program and are then persuaded about the health hazards of smoking and the benefits of not smoking an attempt must still be made to offer them an alternative way of satisfying their needs (related to smoking). This may well be the reason why more favourable results are obtained in terms of reduction in consumption as opposed to total abstinence. Smokers treated with S. Depr. might be cutting down their consumption levels to such a level that they can still satisfy their needs (psychological, pharmacological, or both), with the remaining cigarettes. In this respect, it is important to use more refined dependent measures such as blood or butt-nicotine analysis rather than crude measures of consumption levels to evaluate the effectiveness of the treatment programs. The smokers might well be increasing their puff-rates or degree of inhalation and thus still be subjected to the hazards of smoking.

1.5 CONCLUSIONS AND DIRECTIONS FOR FUTURE RESEARCH

Kiesler's arguments against the "Uniformity Myths" prevalent in psychotherapy research are also applicable to the smoking modification area. Kiesler (1966), commented "Until our designs can incorporate relevant patient variables and crucial therapist dimensions so that one can assess which therapist behaviours are more effective with which type of patients we will continue to perpetuate confusion" (pg. 113). The importance of the role of different patient characteristics in determining the effectiveness of different therapy methods, has also been stated by Sanford (1953) ... "From the view point of science, the question 'Does psychotherapy do any good?' has little interest because it is virtually meaningless ... The question is which people, in what circumstances, responding to what psychotherapeutic stimuli." (Kiesler, 1966; pg. 113). The uniformity assumptions, to which

the above authors objected are also prevalent among smoking modification researchers.

Smoking intervention studies, with the exception of a few (Lichtenstein & Keutzer, 1967; Best & Steffy, 1971; Best, 1975; Harrington, 1978), have been conducted with the underlying assumption that smokers are a homogeneous population with a common maladaptive behaviour (i.e; smoking). Despite the research in smoking typologies (see chapter 2), investigators have been contented to take a sample of well motivated smokers and investigate the effectiveness of various treatment strategies. Although, attempts were made to match the experimental and control groups on some relevant characteristics in terms of overall mean levels, no attempts have been made to control the variance within the whole sample.

The researchers have been generally concerned with designing a treatment strategy that will be effective in modifying the smoking act and which will also produce superior results to those of other active and placebo treatments. This search for an effective treatment illuminates the second assumption, which is about the universal effectiveness of a single treatment program for all smokers. Various treatment methods have been compared and conclusions have been drawn either about the superiority of one active treatment method or the lack of any specific treatment effect, without considering the characteristics of smokers successful under a certain treatment method. As was noted in the previous section most of the treatment programs produce about 20% long-time abstainers. Although, the long-time abstainers could be a distinct group of smokers who might have abstained under any treatment method, or even with their own effort, still it is important to analyze the characteristics of the abstainers under different treatment conditions.

So, the limitations in the smoking modification area could well be due to the underlying assumptions (namely, the uniformity of smokers

and the effectiveness of a treatment method for all smokers) which governs the research designs. In the light of research in smoking typologies, it seems to be more appropriate to view smokers as a heterogeneous group, with different needs or a hierarchy of needs maintaining their smoking behaviour. Although various researchers (Horn & Tomkins, 1966; Waingrow, 1966; McKennell & Thomas, 1967; McKennell, 1973 - a; Russell, 1971 - b), have proposed models that differentiate types of smoking, very few investigators have used these typologies in a treatment context (Harrington, 1978).

At present, the typology models are limited to the subjective self-reports of smokers, so that firstly their objective validity needs to be established. However, even the differences in self-reports of smokers on when and why they smoke points out the need to abandon the uniformity assumption about smokers. There is certainly a need for large scale investigations examining behavioural, physiological and psychological changes prior to, during and after smoking and also during cigarette deprivation. Such an investigation could provide a reliable and fundamental understanding of the smoking act and will subsequently facilitate the formation of a valid, comprehensive and operationally useful model of smoking behaviour.

Smoking intervention programs should be built upon such comprehensive models of the smoking behaviour. However, research in intervention techniques has outnumbered the research aimed at acquiring a fundamental understanding of the controlling variables of smoking, motives for smoking and needs satisfied by smoking in different individuals, so that no meaningful conclusion, except that mass attacks on heterogeneous groups of smokers fail to produce permanent changes in the smoking behaviour, can be drawn from the outcomes of the vast number of studies published in the literature.

Some researchers investigated the association of personality

variables with treatment outcome and the effects of tailoring suitable treatment procedures with personality variables. Keutzer (1968), compared three behaviour modification techniques and also investigated the role of demographic and personality variables in predicting treatment outcome. Only one personality variable "Effective Cognitive Dissonance" (E.C.D.) about smoking, was found to be significantly associated with treatment outcome. The higher the E.C.D. score, the more likely the subject was found to achieve success in abstinence or significant reduction in smoking. Best & Steffy (1971), also stressed the need to tailor appropriate treatment methods with subject characteristics, "which procedure of two competing alternatives should one use with what clients?" (pg; 178). Best & Steffy (1971), in a study designed to investigate the tailoring of smoking modification procedures to subject characteristics, contrasted the utility of an internalized "will-power" oriented approach to smoking control with an externalized environment-based approach. They have analysed the relative effectiveness of these procedures for clients falling at the internal and external end of Rotter (1966), "Locus of Control" (L.O.C.) scale. The interaction of a dissonance inducing procedure with pretreatment levels of dissonance as measured by Keutzer's "E.C.D." scale, was also investigated. Their results failed to show any interaction between the L.O.C. score and treatment manipulations. However, they found that subjects initially having little cognitive dissonance about smoking had benefited more at treatment follow-up, from procedures designed to induce dissonance. Best (1975), investigated the differential efficacy of three treatment procedures, designed to enhance treatment generalizability and durability as a function of subject characteristics. The first procedure, treatment focus, compared procedures considered to be suitable for internal versus external locus of control subjects. The second, punishment, was designed to enhance treatment durability, by requiring Ss to double or triple (i.e; satiation) their smoking rate if they relapse after treatment. The third, timing of attitude change, was given either before or after the change in smoking behaviour

during treatment. Treatment focus and timing of attitude change was found to interact significantly with subject characteristics (L.O.C.-score) in determining treatment outcome. Finally, Yucesoy (1976), investigated the differential effectiveness of two therapy programs, hypothesized to be differentially suitable for "Internal" and "External" locus of control (Rotter, 1966), subjects. The treatments were "self-control" (suitable for I Ss), and group-pressure (suitable for E Ss) and an attention placebo group. The results did not reveal any L.O.C. score and treatment type interaction as was predicted. However, it was found that internally oriented individuals had a higher success rate (percent reduction) than externally oriented subjects under all treatment conditions, both at the end of the therapy and at a 2 months follow-up period, which might imply that some smokers are more likely to reduce their consumption levels, no matter what the type of treatment is.

Harrington (1978), supported the possibility that some smokers no matter what the treatment procedures are, benefit more from treatment and find it easier to abstain or reduce their consumption levels than others. He compared the effectiveness of two different treatment programs among smokers classified as "cravers" and "non-cravers" according to Ikard et al, (1969), smoking typology test. The results revealed that "non-cravers" had a better outcome than "cravers" under all treatment conditions. Although, Harrington's study represents a valuable attempt to investigate differential treatment effects on smoking types, the treatment procedures employed do not appear to be appropriate. One of the treatment conditions was a "self-control" program in which smokers were given detailed instructions on environmental planning, stimulus control tactics, etc., whereas the other group, labelled as "positive self-control" was only provided with social reinforcement contingencies and positive rewards (e.g; saving money). It is not very clear why the second treatment was labelled as a "positive self-control" group, since social factors, outside the individual are also a component of the program. A more

suitable treatment approach could have been a "pharmacological treatment approach". Since both of the treatment conditions in this study dealt with the habit components of smoking, it does not seem unexpected that "non-cravers" had a higher success rate under all treatment conditions. So, this study needs to be replicated with a more appropriate form of therapy for "cravers", before a conclusion can be drawn on type of smoking and treatment interaction.

The studies discussed above point out that some personality variables (e.g; L.O.C.), cognitive factors (e.g; Dissonance about smoking) and smoking types (e.g; cravers and non-cravers) are associated with outcome in therapy. However, more research is warranted before they can be used to design more effective treatment procedures for different types of smokers.

Also, procedures effective in maintaining the treatment gains need to be investigated further. Best (1975), pointed out "smoking modification may be conceptualized as a two-stage process. Achieving abstinence may be relatively unrelated to maintaining abstinence" (pg; 1). At present, short-term success (abstinence or significant reductions in smoking frequency) can be achieved with almost any type of treatment, or even with non-specific factors (i.e; motivation, attention from therapist, etc.) present in modification studies. However, with the tailoring of appropriate treatment strategies to the needs of individual smokers a more consistent and higher success rate may be achieved, since with this method the motives for smoking in individual cases can be taken into consideration.

For the durability of treatment results, techniques that facilitate generalizability to natural settings need to be incorporated into the intervention programs. It can also be argued that since treatment strategies directly built upon a comprehensive model of smoking behaviour are likely to include methods to deal with withdrawal symptoms, this in itself, might increase the durability of treatment effects.

Russell et al (1974), included " withdrawal relief" as a type of positive reinforcement maintaining " addictive smoking". The components of the withdrawal stage need to be analyzed comprehensively, in order to devise intervention techniques that can deal with it directly. At present, techniques to enhance treatment durability (e.g; therapist contact, booster sessions, etc.), have mainly concentrated on the smoking behaviour and its extinction, but not with the withdrawal state that accompanies abstention. Although, some of the self-control techniques have aimed at teaching smokers the so called "non-smoking" skills (e.g; telling others that you have stopped smoking, throwing the smoking related articles away, etc.), these techniques only impose limits on smoking but do not offer any alternative to relieve the withdrawal state, so that they can only be effective with smokers who do not experience strong withdrawal symptoms.

1.6 CHAPTER SUMMARY

Various intervention techniques, ranging from methods based on social-learning theory, to pharmacological treatment, hypnosis, group-therapy approaches and sensory deprivation have been used to modify the smoking behaviour. However, the general outcome has not been very encouraging. Almost any form of treatment produces short-term success (i.e; abstinence or significant reductions in smoking frequency). However, the impact of treatments are short lived, and treatment gains are only maintained in a small proportion (13% - 25%) of treated smokers.

Although, methodological shortcomings, like the selectivity of subjects, lack of adequate control groups, standard follow-up periods and reliable dependent measures poses serious problems in the smoking modification research, a more serious problem arises from the assumptions of "uniformity of smokers" and "universal effectiveness of a single treatment approach".

The appropriateness of viewing smokers as a "heterogeneous" group and tailoring appropriate treatment strategies to the needs of homogeneous groups of smokers, classified according to a reliable criteria has been emphasized. The need for further research to gain a fundamental understanding of the smoking behaviour and its implications for intervention methods has also been discussed.

CHAPTER 2

MODELS OF SMOKING BEHAVIOUR AND SMOKING TYPOLOGIES

2.1 INTRODUCTION

In the previous chapter the need to abandon the "uniformity assumptions" about smokers and treatment effects has been emphasized. In order to adopt a differential treatment approach first it is necessary to develop reliable and valid measures that can be used to identify different types of smokers or smoking. In this chapter various conceptualizations of smoking behaviour and the typology scales derived from them will be reviewed and their implications for smoking intervention programs will be discussed.

"Why do people smoke cigarettes?", is the basic question addressed by the smoking models. A distinction is made between different types of smokers or smoking in terms of the variables (i.e; motives, reinforcements, arousal levels and occasions) controlling the smoking act. Models of smoking behaviour are mainly based on theoretical conceptualizations of the smoking behaviour and at present empirical data to support the validity of the typologies proposed by various models are very scarce.

The typology scales to differentiate types of smokers are mainly limited to the self-reports of smokers to paper-pencil tests. Thus, the typologies might purely be reflecting the subjective self-images of the smokers rather than any objectively identifiable differences between them. This is the major shortcoming of the typology research and more research is needed to establish the objective validity of the types delineated by the typology scales. Despite this shortcoming the classification schemas proposed by various authors are very valuable in terms of: a) pointing out the need to view smokers as a non-uniform group, and b) offering possibilities for tailoring appropriate treatment methods with different types of smokers.

2.2 MODELS OF SMOKING BEHAVIOUR AND SMOKING TYPOLOGY SCALES

Although, in the past smokers have been classified as light or heavy, regular or intermittent, inhaler or non-inhaler, recently there have been attempts to use more comprehensive models to type smokers. In this section these models and the typology scales derived from them will be reviewed.

i) HORN-TOMKINS TYPOLOGY (Smoking as management of affect)

Tomkins (1966), proposed a model of smoking behaviour based on his theory of affect (i.e; feelings and emotions). He delineated eight primary and innate affects which he further classified into two broader groups, namely, positive (excitement, enjoyment, surprise) and negative (distress, anger, fear, shame, contempt) affects. Making an anology between sucking response in childhood and smoking behaviour, he stated, "Sucking or smoking, therefore, is innately capable of reducing negative affect of distress and of evoking the positive affect of enjoyment" (pg; 18). Although, his argument about the role of sucking in interfering with crying in childhood, both behaviourally and also neurally (relaxation of mouth muscles) seems to be plausible, this view appears to be too simplistic and inadequate in dealing with smoking behaviour in adulthood.

On the basis of his theory of affect, Tomkins distinguished four general types of smoking:

a) Smoking to increase Positive Affect: Positive affect smoking was further divided into two subtypes, smoking as a stimulant and smoking as a relaxant. According to Tomkins, "stimulant type of smoking occurs whenever smoking is used to give the person a lift from the positive affect of excitement" (e.g; when an adult smokes for the excitement of something to do) and "relaxant" type of smoking occurs in those individuals who characteristically smoke under pleasant circumstances which are relaxing" (e.g; at the end of a meal, or during a pleasant conversation). Another type of positive affect smoking,

which was suggested by D. Horn is associated with the sensorimotor aspects of smoking (e.g; lighting and handling cigarettes, watching the smoke, etc.) (Tomkins, 1966; pg. 19).

b) Negative Affect Smoking (Sedative smoking): (Smoking to reduce negative affect)

According to Tomkins in this type of smoking the individual smokes primarily to reduce his negative feelings. Two subtypes of sedative smoking were delineated:

i) Partial sedative smoking, in which smoking is used to reduce negative feelings so that the smoker can face and solve his problems, and ii) Complete sedative smoking, in which smoking is used as an opiate, in order to reduce negative feelings and to avoid any confrontation with the source of distress.

Tomkins stated that the sedative smoker may not smoke at all when he is feeling good and in contrast positive affect smoker may never smoke when he feels bad.

c) Habitual Smoking : (Smoking with no affect)

Tomkins viewed habitual smoking as a highly developed skill which involves a minimal degree of awareness. In his words, "the individual originally may have smoked to reduce his negative affect or to experience positive affect but he has long since ceased to do so". In this type of smoking, the act becomes so automatic that the smoker may frequently be unaware that he has lit a cigarette.

d) Addictive Smoking :

Both positive and negative affect are operative in addictive smoking. Tomkins viewed it as follows, i) the smoker is always aware of the fact that he is not smoking whenever this occurs, ii) such awareness of not

smoking invokes negative affect, which the addicted smoker believes to be only reduced by smoking, iii) the negative affect increases in intensity until it is intolerable as the duration of deprivation increases, and iv) when a cigarette is finally smoked, the smoker experiences a sudden decrease in negative affect and at the same time starts to experience positive affect.

The above chain of feelings and the evocation of positive affect with smoking confirms the addicted smoker's view that only a cigarette will reduce his distress and bring relief.

The four general types of smoking described above are formulated purely on the basis of Tomkins' theory of affect and the validity of the types proposed needs to be empirically established. Before discussing such attempts, the suggestions made by Tomkins on the specific techniques of cessation appropriate for different types of smokers will be mentioned.

Tomkins stated, "If there are these varieties of smoking behaviours then clearly attempts to control them must be designed in the light of these differences". In dealing with habitual smoking, "the major effort must be directed at increasing the degree of awareness of the act so that it again becomes possible for the individual to choose whether and when to smoke". So, for habitual smoking "self-control" strategies, by which the smoker can be instructed to keep records of when and how many he smokes, seems to be appropriate. By this procedure the smoker can become aware of his smoking and will also gain an understanding of when he smokes, so that he can apply stimulus control strategies to restrict his smoking frequency.

For positive affect (stimulant and relaxant) smoking Tomkins suggested, "the individual must be directed to alternative substitute sources of positive excitement and enjoyment". Tomkins' model does not include pharmacological rewards that might be involved in the maintenance of the smoking

habit. Although, he proposed the stimulant type of smoking, it only covers the excitement evoked by having something to do, rather than the physiological and behavioural arousal produced by the inhalation of nicotine. In this respect although his model covers a stimulant type of smoker, his conception of the mechanisms of stimulation seems to be inadequate. In connection with this limitation his suggestions for dealing with this kind of smoking seems to be too ambiguous. However, although not explicitly stated, his suggestion of "substitute sources of positive excitement" can also accommodate a pharmacological treatment approach.

In the case of negative affect (sedative) smoking Tomkins suggested, "either an attack, must be made on the sources of negative affect, to reduce their frequency and severity, or the individual must be taught alternative ways of making himself feel better on such occasions, or to more directly confront and solve his problems rather than to sedate himself". Although, behaviour therapy techniques, such as systematic desensitization and relaxation training can be suitable for this type of smoking, the recommendations made by Tomkins are again ambiguous and seem to require an intensive psychotherapy program.

Finally, in dealing with addictive smoking Tomkins proposed two strategies, "One is to interfere with the first link in the long chain, i.e; to so arrange his life that he ceases to become aware of the fact that he is not smoking when he is not smoking. The other major strategy is to intensify the cold turkey method so that the crisis of deprivation affect is reached more quickly and with more intensity, to enable the individual to learn that the apparently intolerable negative affect is in fact tolerable". This suggestion does not deal adequately with the needs satisfied by smoking and even if the smoker can be made to realise that non-smoking is tolerable, still no alternative means of satisfying his needs is offered.

The cessation methods proposed by Tomkins are in no way novel, and have also been used by other researchers (see chapter 1). However, Tomkins' suggestions for tailoring appropriate treatment methods with different types of smoking represents a novel and valuable approach to the modification of smoking behaviour. The majority of studies reviewed in chapter 1 share the notion of 'the universal effectiveness' of a particular treatment approach, whereas Tomkins' suggestions point out the need to adopt a differential treatment approach, by which smokers are viewed as a heterogeneous group, and treatment effectiveness depends on who is being treated by which particular technique.

Several investigators have attempted to develop a scale to differentiate the types of smoking proposed by Tomkins. Horn and Waingrow (1966), devised a 23-item scale based on Tomkins' model, and gathered data from a national survey of adults conducted for the U.S. Public Health Service. The factor analysis of this data yielded six rotated factors, representing the following types of smoking: habitual, addictive, negative affect reduction, pleasurable relaxation, stimulation, and sensorimotor manipulation. With the exception of addictive items, which were also loaded to a moderate degree on the negative affect reduction factor, the individual items were not found to be loaded on more than one factor. Subsequently, six sub-scales corresponding to the six factors listed above were formed.

Significant sex differences were noted for the "habitual", "addictive", and "negative "affect" smoking sub-scales. Males were found to score higher than females on the "habitual" and "addictive" sub-scales, whereas, females scored higher on the "negative affect reduction" sub-scale. This finding is consistent with the data on differences in dosage levels between sexes. Males on the average smoke more cigarettes, smoke cigarettes with a higher tar content and inhale deeper and more frequently than females (Waingrow, et al, 1968).

Correlations ranging from 0.38 to 0.58 were obtained among the habitual, addictive and negative affect reduction sub-scales. On the other hand, the three positive affect sub-scales indicated only a slight interrelationship (r+0.09-0.30). Moderate positive correlations were reported between the mean number of cigarettes smoked per day and habitual, addictive and negative affect reduction sub-scales, (r=0.27-0.41 for males and r=0.40-0.53 for females).

Later, Ikard, et al, (1969), utilizing both the factor loadings and item intercorrelations developed an 18-item questionnaire, consisting of six sub-scales (3 items per sub-scale), corresponding to the six rotated factors listed above.

Ikard and Tomkins (1973), conducted a series of studies in an attempt to validate Tomkins' model. One of these studies, was designed to measure the degree of craving experienced by different types of smokers (i.e; addictive, preaddictive, negative and positive affect), during a three hours deprivation period. It was found that addictives experience the most craving during the deprivation period and the positive and negative affect smokers, the least. On the basis of these results the authors stated, "types of smoking can clearly provide a guide in terms of expected difficulty in a cessation attempt. Seperate and different techniques may be required for the different types of smokers in order to deal with the level of suffering expected". (pg. 177). Although, the results of this study show that deprivation has a differential effect on different types of smokers in terms of the subjective craving ratings, it does not provide any physiological or behavioural measures to demonstrate an objective difference between the groups. Thus, although the classification based on the subjective self-reports of smokers to a questionnaire seem to predict a difference in yet another subjective measure (degree of craving), the correspondance between the two measures can simply reflect the self-images of the smokers rather than any difference in the degree of objective

withdrawal symptoms experienced. Thus, the findings of this study do not offer any practical guide for how to treat particular groups of smokers, apart from pointing out that addictive smokers, who were also found to have a high consumption rate will experience the greatest difficulty (i.e; craving) in cessation. In another study Ikard and Tomkins (1973), instructed smokers to smoke on a one-hour schedule, as a part of a method of smoking cessation. The ability to adhere to the schedule was found to be related to the type of smoker, addictive smokers were found to have the greatest difficulty in following a one-hour schedule.

The studies mentioned above demonstrate the relationship between types of smoking derived from a theoretical model and some external variables (i.e; degree of craving during deprivation and ability to restrict smoking). However, the data provided is only descriptive and adds little practical information about why these smokers are different. Both Tomkins' model and the typology scales based on it emphasize the role of smoking in controlling affect. Although this view may be plausible, more intensive research investigating which aspects of smoking (i.e; psychological, pharmacological or behavioural) influence affect and the mechanisms of this effect in different individuals is needed.

ii) McKENNELL AND THOMAS TYPOLOGY:

McKennell and Thomas (1967), attempted to type smokers according to the kinds of occasions on which they most characteristically smoke. They developed a list of "smoking occasion" items derived from a content analysis of unstructured interviews with smokers and ex-smokers on the occasions on which they said they smoked. Finally, a 42-item questionnaire was prepared and administered to representative national samples of adolescent (N=490), and adult (N=603) smokers and both to adult and adolescent ex-smokers (N=146).

The factor analysis of the replies to the check-list questionnaire yielded seven types of smoking factors, which were further categorized under two broader groups of factors, which were:

a) Inner Need (Personal) Smoking :

1 - Nervous irritation smoking (e.g; smokes when irritable, anxious, worried, etc.)

2 - Relaxation smoking, (e.g; smokes when happy, resting, etc.)

3 - Smoking alone, (e.g; smokes when by self and feeling alone, gets more pleasure when smoking alone, etc.)

4 - Activity accompaniment, (e.g; smokes when working hard, smoking helps concentration)

5 - Food substitution, (e.g; smokes when feeling hungry, to keep slim, etc.)

(r = 0.27-0.56 between the above five inner-need factors)

b) Social Factor :

6 - Social smoking, (e.g; smokes in company, when drinking alcohol, tea or coffee, at a party, etc.)

7 - Social confidence smoking, (e.g; feels more relaxed in a group when smoking, more self-confident with smokers, etc.)

(r = 0.22-0.37 between the above two social factors)

The five "inner-need" factors were found to be moderately correlated with consumption rate, (r = 0.29-0.53). However, when amount smoked was held constant by partialling it out in the factor analysis, the same seven factors were again obtained, which demonstrated that these factors characterized smokers by the type of occasion on which they are most likely to smoke, rather than on the amount they smoked.

In order to demonstrate the link between the occasions for smoking and the motives for smoking, the seven smoking occasion factors were correlated with several external variables indicative of need and motivation for smoking. Substantial correlations, (r = 0.39-054) were obtained between the "inner-need" factors and a measure of craving (i.e; craving was operationally

defined as those smokers who say that "when they haven't got a cigarette on them they feel a craving for one"). "Nervous irritation" smoking had the highest correlation ($r = 0.54$, before amount smoked was held constant, and $r = 0.40$ when amount smoked was partialled out) with this measure of craving.

The correlations between the seven factors and another external variable, namely, the extent to which smoking serves a useful function, measured by a scale consisting of the items, "smoking is pleasurable","smoking can help people relax", "smoking can help people when they feel nervous and embarrassed", were also investigated. "Occasions for smoking" factors were found to be related to the "perceived helpfullness" of smoking, ($r = 0.43$ for smoking alone; $r = 0.39$ for nervous irritation smoking). Finally, differences were reported between the factor profiles of smokers and ex-smokers, the former scoring higher on the five "inner-need" factors, on amount smoked and on the measure of craving, than do the ex-smokers. So, smokers who score low on the inner-need factors, who do not experience craving if cigarettes are not available seem to have less difficulty in abstaining from smoking, than the ones scoring high on the inner-need factors and craving.

McKennell (1973-a), conducted a further study, in which he investigated the overlap between the "Horn" and "McKennell" factor structures. In this study, he confirmed the stability of the factor structures reported by both of the models. He added an additional factor, namely reluctant smoker to the McKennell factor structure, which was similar to Horn-Tomkins' "habitual smoking" factor. Horn's "pleasurable relaxation" and McKennell's "relaxation" factors were found to be measuring different variables ($r = 0.15$ between these two). The former being concerned with the smokers general attitude to smoking, while the latter being related to the desire to smoke in relaxed situations. On the other hand, Horn's "tension reduction" and McKennell's "nervous irritation", ($r = 0.61$); Horn's "habitual smoking" and McKennell's "reluctant smoking", ($r = 0.46$); and Horn's "addictive smoking" and McKennell's measure of

addiction, ($r = 0.56$), were found to be alternative measures of the same trait.

Although, Horn and McKennell classification schemes overlap to some extent, an important difference between them lies in the status given to the variable of addiction. In Horn's typology, the addicted smoker is seen as a different kind of smoker whose motives are the satisfaction of craving, whereas in McKennell's (1970), study, addiction (craving) is included as a type of motive underlying the individual's smoking behaviour. McKennell (1973-b), investigated the nature of addictive smoking further and found that the variance in addiction as measured by Horn and Ikard's addiction factor could be accounted for by a combination of other smoking traits, (i.e; Addiction = Tension Reduction + Habitual Smoking - in Horn; or Addiction = Nervous Irritation + Reluctant Smoking - in McKennell). So, McKennell (1973-b), showed that addiction is not an independent trait as it appears in the Horn typology, but is in large part a compound of other traits. These traits emerged as the same on both models.

McKennell & Thomas (1967), also proposed a further distinction between smokers, namely dissonant and consonant smokers. Dissonant smokers were found to agree with a wide variety of negative statements about smoking and they either want to stop smoking or have tried unsuccessfully to do so in the past. These smokers were found to be ambivalent towards their habit and compared to ex-smokers they scored higher on measures of "addiction" and the "inner-need" factors. Whereas, the consonant smokers express no wish to give up smoking and tend to reject anti-smoking arguments. McKennell suggested that the latter group would be more prone to attitude change manipulations, (i.e; anti-smoking campaigns), whereas the dissonant smokers do not seem to be a good target for anti-smoking appeals, because their problem is not to decide whether to stop, but how to stop smoking.

McKennell (1970), recommended the application of learning theory and behaviour therapy techniques in breaking the links associated with the smoking habit. However, this recommendation indicates that smoking is purely maintained by learning to smoke in the presence of certain cues (i.e; occasions), and does not consider the needs or motives which may be playing a role in initiating smoking on different occasions. For this reason, this classification schema does not appear to be adequate in explaining how and why smokers develop different smoking patterns and the specific needs involved in each pattern. The two motivational factors delineated, namely, "craving for a cigarette" and "perceiving smoking as helpful" are based on very crude operational definitions. In this respect these independent measures contribute very little to the identification of the aspects of smoking behaviour that are of some value for the smoker, (i.e; sensorimotor, pharmacological, social etc.). For example, "smoking is pleasurable", one of the items of the "belief smoking is helpful scale" gives little information on which specific effect of smoking is conceived of as being pleasurable. So, the external variables used by McKennell to demonstrate the link between occasions for smoking and motivation for smoking seem to be too general to yield any information on the specific motives or needs for smoking. The smoker might have a craving for a cigarette when none is available, however, it does not seem useful to conclude that the need for smoking arises from the craving for cigarettes. It is more important to delineate the mechanisms and underlying causes of craving and reinforcers of smoking behaviour rather than simply concluding that craving is the motive maintaining the smoking behaviour.

Classification of smoking according to the predominant patterns of reinforcement, as proposed by Russell (1971-b), which will be discussed next, appears to be more adequate in delineating the motives for smoking.

iii) RUSSELL - TYPOLOGY (Based on a composite reinforcement model of smoking)

Russell (1971-b), proposed a scheme to classify smokers according to the pattern of reinforcers, or motives maintaining their smoking behaviour. He distinguished the following three main groups of motives:

a) Psychosocial Rewards; According to Russell these include smoking to conform and gain acceptance and to increase social confidence.

b) Sensory Rewards; These include the sensory satisfactions (i.e: smell, taste, handling, watching the smoke, etc.), and oral gratifications.

c) Pharmacological Rewards: These include the euphoriant, sedative and stimulant effects of nicotine and the drive of withdrawal relief in physiologically dependent smokers. Russell's main contribution was the inclusion of pharmacological rewards into a smoking model.

Russell (1971-b), delineated five types of smoking based on these three main motives. Fig. 2.1 illustrates the scheme proposed by Russell for classifying types of smoking according to the predominant pattern of reinforcement.

Russell, Peto & Patel (1974), criticised the two smoking models discussed earlier by stating, "The shortcomings of these previous attempts at classification appears to stem from two sources. First, the items or behaviours sampled have been incomplete and much of relevance may have been left out. Second, and more importantly, the classification schemas have not been woven into a comprehensive account of smoking; one which incorporates psychophysiological and psychopharmacological as well as behavioural data". (pg: 314). The authors also stated that both Horn and McKennell typologies are merely descriptions of events occurring within relatively restricted domains and they fail to explain how smoking alters affect or why it occurs in certain situations.

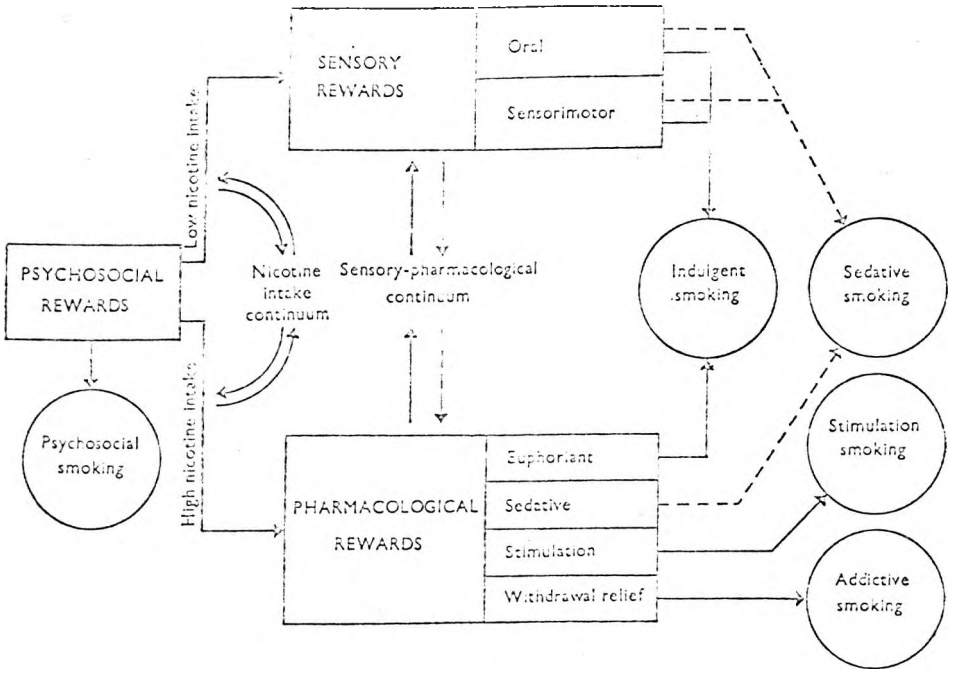

Fig. 2.1. Schema for classifying smoking according to the predominant pattern of reinforcement. (from: Russell (1971-b))

The five types of smoking delineated by Russell (1971-b) were:

a) Psychosocial Smoking - (derived from psychosocial rewards).

This type of smoking is mostly observed during the early stages of smoking. Smoking is intermittent and occurs only in social situations. According to Russell there may be virtually no nicotine intake at this stage and except for a few smokers who do not inhale, the absorption of nicotine ensures the evolution of smoking for pharmacological rewards. However, even non-inhalers do absorb some nicotine through the buccal mucosa (Kersbaum, et al, 1967). Therefore, it would be more appropriate to view social-smoking as being predominantly reinforced by social rewards, although some pharmacological action may also be involved.

This type of smoking appears to be equivalent to McKennell's "social" and "social confidence" smoking.

b) Indulgent Smoking - (derived from euphoriant (pharmacological) rewards and/or oral (sensory) rewards).

According to Russell this is smoking purely for pleasure. There may be gaps between smoking, however, on indulgent occasions (i.e; watching television, resting, after meals) smoking may be frequent. The pleasure of smoking may be derived from oral rewards (oral - indulgent), sensorimotor rewards (sensorimotor - indulgent) or a mixture of these rewards (mixed-indulgent). The oral indulgent smoker may resort to oral compensation when giving up smoking, and seems to be equivalent to the "food substitution smoking" of McKennell.

Although, indulgent smoking, as defined by Russell, seems to be maintained by non-pharmacological rewards, the concept of smoking for pleasure seems to be misleading. Since, other types of smoking (i.e; stimulation, tranquilization, etc.) also may produce pleasurable effects for smokers, it seems more appropriate to describe this type of smoking as being maintained by oral and/or sensorimotor rewards.

c) Tranquilization Smoking - (derived from sedative (pharmacological) rewards and/or oral and sensorimotor (sensory) rewards.

In this type of smoking the sedative effect of nicotine and the calming effect of oral gratification and the occupation of hands predominate in the maintenance of the smoking behaviour. Smoking frequency largely depends on the emotional state of the smoker. This type of smoking appears to be similar to Tomkins' "negative affect" and McKennell's "nervous irritation" smoking.

d) Stimulation Smoking - (derived from stimulation (pharmacological) rewards).

In this type of smoking the stimulant properties of nicotine are used and the frequency of smoking increases in monotonous situations or in situations requiring alertness. This implies smoking in low-arousal situations to raise the level of arousal (physiological and/or behavioural) and is similar to Frith's typology which will be discussed later. The sensorimotor factors involved in smoking may also influence the level of arousal and it would be appropriate to include the effects of these alongside with the stimulant properties of nicotine as determinants of stimulation smoking.

e) Addictive Smoking - (derived from withdrawal relief (pharmacological) rewards.

In this type of smoking, the frequency of smoking is mainly dependent upon internal rather than external cues, and the motive is to maintain a consistent blood nicotine level. According to Russell, "the smoker experiences withdrawal symptoms whenever he has gone 20 - 30 minutes without smoking, and he smokes to avoid or relieve this distressing state".

Russell's "addictive smoking" appears to be quite similar to Tomkins' addictive smoking, in which the smoker also experiences negative affect when not smoking and believes that only smoking will alleviate his negative affect.

Russell et al (1974), attempted to link Horn and McKennell typologies with the pharmacological evidence and with other psychological findings. The psychosocial aspects suggested by McKennell and five of Horn's factors were included in this study. The aim of the study was to expand Horn's scale to include the factors omitted, (i.e; social and pharmacological), to compare the similarity and overlap between Horn and McKennell typologies and finally to relate the new scheme to Russell's composite reinforcement model of smoking.

A 34-item self-completion questionnaire, including most of the original Horn and McKennell (psychosocial and activity accompaniment) was administered to 175 smokers from the general smoking population, and to 103 smokers attending the Maudsley Hospital Smokers' Clinic. The latter group was used as a criterion group of addicted heavy smokers, (50% of whom smoked more than 30 cigs/day, compared with 11% of the main sample).

The factor analysis of the replies from the two samples yielded six factors, which were:

Factor I: Stimulation smoking - (comprised of 6 items). Horn's stimulation factor and McKennell's activity accompaniment items were included together.

Factor II: Indulgent smoking - (contains 4 items). This factor combined Horn's and McKennell's concept of smoking for pleasure in relaxed situations. However, the items included in this factor were more closely linked with McKennell's relaxation items.

Factor III: Psychosocial smoking - (comprised of 6 items). This factor included McKennell's social and social-confidence smoking items.

Factor IV: Addictive smoking - (contains 4 items). Three of Horn's addictive smoking items were included in this factor.

Factor V: Sensorimotor smoking - (contains 5 items). This factor included Horn's sensorimotor manipulation items.

Factor VI: Automatic smoking - (contains 3 items). This factor was essentially the same as Horn's habitual smoking.

A "sedative smoking" factor did not emerge from the factor analysis. Russell et al (1974), suggested that this is probably due to the inclusion of McKennell type activity accompaniment items, and when these were excluded from the analysis a sedative factor re-emerged. McKennell (1973-a), also found Horn's "Tension Reduction" factor to be the least stable with a tendency to merge with the addictive factor.

A very major and valuable finding of Russell et al's (1974) study is the finding of two orthogonal unrotated factors. The major one represented the degree of dependence or addiction while the second bipolar factor represented a non-pharmacological versus pharmacological dimension. The six factors listed above and their items fell into two distinct clusters when plotted according to their loadings on the two second-order unrotated factors. All the items covered by Indulgent, Psychosocial and Sensorimotor factors loaded at the non-pharmacological end, whereas with the exception of one of the addiction items, Stimulation, Addictive and Automatic factors were found to be loaded on the pharmacological end. On the other hand, it was noted that both the pharmacological and non-pharmacological factors spread along the dependence dimension. However, non-pharmacological items were not found to appear as far along as the pharmacological items on the dependence dimension. This suggested that smokers have a likelihood of getting dependent on cigarettes either pharmacologically or non-pharmacologically. The loading of one of the addictive items on the non-pharmacological end could also be explained by this characteristic of dependence.

Moderate positive correlations (r=0.27-0.63), were noted between number of cigarettes smoked per day and the factors loading at the pharmacological end. The relationship of addictive, automatic and stimulation factors with each other (r=0.21-0.39), and with the daily cigarette consumption, their clustering along the pharmacological and dependence dimensions, and also the reported marked difference between the Smokers' Clinic sample and the main sample (i.e; the former scoring much higher) on these factors suggested and supported a main pharmacological dimension. Russell et al (1974), suggested, "it may prove more useful to classify smokers according to their position on the single dimension of pharmacological addiction to nicotine rather than in terms of their profiles on the six types of smoking" (pg: 332).

The study discussed above substantiated the composite reinforcement model (see Fig. 2.1), proposed by Russell. Apart from psychosocial

smoking, which was negatively correlated with age (-0.23), no strong relationship was noted between age and the other factors. This suggested that although there is a progression from psychosocial smoking to other types of smoking, addictive smoking is not inevitable and occurs only in a proportion of smokers. It is of crucial importance to investigate the characteristics of those smokers who score high on the pharmacological dimension, and to examine the role of nicotine in their smoking habit.

Although, Russell et al (1974), by developing a simple questionnaire which included psychological, social and pharmacological aspects of the smoking behaviour have progressed beyond the findings of the previous typologies, still due to the nature of the investigation (i.e; subjective self-reports to preselected questions), the classification derived from this model needs to be validated by objective measures. The authors themselves have pointed out the shortcomings of this method, "the sceptic is still entitled to ask whether we have discovered anything more profound than high correlations between answers to similar questions and moderate correlations between related, if less repetitive items". "There is a fundamental difference between questionnaire response and physical measurement. A questionnaire reflects the subject's conceptual scheme and there must be a tendency, whether self-conscious or subconscious, to produce a set of answers which are consistent with self-image".

So, in conclusion, although Russell, et al's typology test provides a practical guide to identify the predominant motives maintaining the smoking behaviour in different individuals, it is necessary to investigate, a) whether the types delineated by this model can also be identified by objective measures, and b) why some smokers, but not others become pharmacologically addicted to nicotine.

Finally, the typology questionnaire developed by Frith (1971-a), which links smoking with arousal levels will be discussed.

IV) FRITH-TYPOLOGY - (Based on the effects of smoking on arousal levels)

Frith (1971-a), proposed a model based on the view that the main role of smoking is to regulate behavioural and physiological arousal levels.

Although, the psychopharmacological effects of smoking (nicotine) are discussed in chapter four, here a brief account will be helpful in discussing Frith's model. At present, there is a great deal of evidence to suggest that the main effect of smoking doses of nicotine is to increase the level of arousal of both the peripheral autonomic nervous system and the central nervous system, (Lucchesi et al., 1967; Domino, 1967). However, nicotine in larger doses have been found to have a sedative action, (Armitage, et al., 1969).

Although, nicotine in larger doses have been observed to produce a decrease in the level of arousal in animals, for humans, it is necessary to demonstrate that smokers inhale deeper and thus achieve higher blood nicotine levels when they are in a state of high arousal. However, it is quite difficult to determine this experimentally. There have been some attempts to investigate the role of arousal levels on the smoking behaviour, however, the measures used (e.g; puff rate, number of cigarettes, latency to the next cigarette, etc.) fail to provide a direct answer to the dosage of nicotine inhaled under experimental conditions designed to induce low and high arousal levels. It is crucial to employ an operational definition of arousal and to check the reliability of experimental conditions designed to induce low or high arousal levels with physiological and behavioural measures.

Frith (1971-a), on the basis of these physiological findings argued, "It should be possible to isolate two extremes of smoking behaviour. There should be one group of people who smoke in situations inducing low levels of arousal in order to increase their arousal level, and there should

be another group of people who smoke in situations which induce high levels of arousal in order to reduce their arousal level". Frith also included the characteristic level of arousal of the smoker into his model, which implied that smoking can either be used to manipulate this characteristic level of arousal or arousal induced by external situations. So, this model implies that smoking is mainly maintained by its pharmacological actions and that even a light smoker, if he smokes consistently in specific situations, can be regarded as dependent on the pharmacological actions of nicotine.

Frith (1971-a), constructed a 22-item questionnaire, in which situations inducive of high and low arousal were described. The respondents had to imagine themselves in these situations and rate what their desire for a cigarette would be in these situations. Twelve high arousal situations, mainly related to emotional stress, anxiety, and stress induced by mental activity, and ten low arousal situations, mainly related to relaxation, boredom, repetitive work and bodily tiredness were used. The questionnaire was administered to a sample of 89 (50 male and 39 female), cigarette smokers. The main findings were, a) For the whole sample low-arousal situations tended to induce more desire for a cigarette than high-arousal situations. This finding is in line with nicotine acting as a stimulant to increase the level of arousal, b) Heavy smokers were found to have a desire for a cigarette in any situation. This finding seems to be in agreement with Russell's view of addicted smokers, who have a wish to smoke every 20 - 30 minutes. Although, it can also be explained purely by habit mechanisms and the high frequency of smoking among the heavy smokers, Russell's notion of some individuals smoking in order to maintain a consistent blood-nicotine level also seems plausible.

Russell's view implies that the smoking behaviour of addicted smokers is regulated by internal cues rather than the changing external situations. Frith's finding that heavy smokers have a desire for a cigarette in

any situation can also be explained by the possibility of the dominance of internal cues (i.e; blood nicotine levels) for heavy smokers. Herman (1974), investigated the role of external (i.e; cigarette cue prominence) and internal (pharmacological-nicotine manipulations) smoking cues as determinants of smoking behaviour in heavy (i.e; more than 20 cig/day) and light (i.e; less than 15 cig/day) smokers. It was predicted that light smokers would be more affected by the manipulation of external cues, whereas heavy smokers would be more affected by the manipulation of internal cues. However, the results showed that both light and heavy smokers were affected by the manipulation of internal cues (i.e; high or low nicotine cigarettes and no cigarettes), whereas the effects of external cues (i.e; saliency manipulation) was found to be confined to light smokers (i.e; smoking more and sooner in the high saliency situation). One of the major shortcomings of this study is that as a manipulation of internal cues, low, high nicotine cigarettes and no cigarettes were used. Nicotine-free cigarettes would have been more appropriate to assess the role of internal cues (i.e; nicotine deprivation), than the " no-cigarette" condition. Due to this shortcoming, the results of this study do not appear to be conclusive.

c) Men were reported to have the highest desire for a cigarette in situations inducing boredom and tiredness, whereas women were found to have their highest desire to smoke in stress inducing situations. This finding is in line with the results of Horn's survey, in which women agreed significantly more often than men to smoke in situations of stress and high arousal (e.g; feeling upset, worried, angry, etc.).

From these findings Frith(1971-a), attempted to define the characteristic level of arousal of different smokers and have stated, " on this basis the results suggest that women are usually more highly aroused than men". It seems to be a gross over-generalization to infer from the finding that women have a higher desire to smoke under high-arousal situations, that they also characteristically have a higher arousal level the data

presented by Frith seems to be inadequate to make any statements on the characteristic level of physiological and/or behavioural functioning.

The physiological and behavioural evidence on the effects of smoking (see chapter 4), does suggest that smoking can be used as a means to control behavioural and physiological arousal levels. However, Frith's study, apart from pointing out that smokers fall into two groups according to their desire to smoke in high or low-arousal situations does not provide a novel typology. Russell, McKennell, and Horn Typologies have also made this distinction. Frith's only departure from the previous schemas is his exclusive emphasis on the pharmacological aspects of smoking. However, even he himself as a conclusion stated that some psychological or behavioural aspects, like the ritual of lighting and holding a cigarette, might be the important motives for smoking to relieve tension. So, although this model offers a simple classification schema, it puts too much emphasis on pharmacological aspects and thus does not offer a comprehensive and adequate model to account for different motivations involved in different smokers.

Finally, two studies investigating the relationship between types of smokers and the behavioural and subjective effects of smoking will be reviewed.

Fuller and Forest (1973), investigated the relationship between smoking and arousal levels in heavy (15 or more cig/day) and light (5 or fewer cig/day) smokers. Although, the classification of smokers into light and heavy in terms of levels of cigarette consumption is quite arbitrary, this approach merits acknowledgement for viewing smokers as a heterogeneous population. One of the experimental conditions was designed to induce high arousal levels (i.e; watching a stressful film about industrial accidents), and the second was designed to induce low-levels of arousal (i.e; relaxing alone on a couch). The reliability of the experimental manipulations were

confirmed by physiological (i.e: higher heart rate and skin-conductance level in the high-arousal condition, etc.) measures and subjective self-reports. The results of the experiment showed that although heavy smokers took significantly less puffs during the high-arousal condition as compared to the low-arousal condition, their nicotine intake was the same on both conditions. It was also found from butt-nicotine analysis that under the low-arousal condition heavy smokers took more nicotine (1.41 mg/cig) than the light smokers (1.27 mg/cig). Finally, there was an overall increase in the rate of smoking during the relaxation condition for both light and heavy smokers, which indicated that smokers may have a faster rate of smoking when there is nothing else to do. Considering the relationship between the effects of nicotine and the dosage levels, Fuller and Forest's results do not support the notion that smokers will seek higher dosages of nicotine in high arousal situations in order to sedate themselves. However, in this study subjects were restricted to only one cigarette and it is plausible that they might have smoked more cigarettes to regulate the dosage of nicotine. On the other hand, the finding that heavy smokers obtained similar doses of nicotine in both of the experimental conditions is in line with the view that internal cues might be the determinants of smoking for this group.

Myrsten, et al (1975), also investigated the interaction between arousal levels induced by specific experimental conditions and smoking habits (i.e; low-arousal smokers versus high-arousal smokers). They constructed a questionnaire for selecting smokers whose desire to smoke was greatest in either low-arousal (e.g; situations inducing boredom and monotony) or high-arousal (e.g; situations inducing anxiety and excitement) situations. Subjects were classified as either low-arousal or high-arousal smokers on the basis of this questionnaire. Two experimental conditions designed to induce either a low-level of arousal (i.e; performing a visual vigilance-test) or a high-arousal level (i.e; performing a complex sensorimotor task) were used. The only data to support the high-arousal inducing characteristics of the

"complex sensorimotor task" condition was that both groups of subjects had a higher heart rate during the 'high-arousal - non-smoking condition' than the 'low-arousal - non-smoking' condition. The differential effects of smoking two cigarettes in these conditions were examined for the low and high arousal smokers in terms of their performance efficiency and subjective reports of alertness. The results showed that in the low-arousal situation, smoking produced an improvement in performance for the low-arousal smokers, whereas a slight impairment was noted for high-arousal smokers. On the other hand, a reversed pattern was obtained in the high-arousal situation, where the high-arousal smokers seemed to benefit from smoking, while the low-arousal smokers did not. Subjective self-reports were also in line with the above findings, low-arousal smokers reported to be less bored and more alert when they smoked in the low-arousal condition than in low-arousal non-smoking condition, whereas high-arousal smokers stated that they felt less alert and more bored when they were allowed to smoke in this condition.

So, the results of the above study supported the assumption that different smoking habits are related to specific differences in the immediate effects produced by smoking. However, the results failed to provide any information on why smoking affects these groups differently. No consistent differences in psychological or physiological measures were noted between the low and high-arousal smokers in pre-experimental baseline recordings and under non-smoking conditions. This finding makes it even more difficult to interpret the noted interaction between type of habit, experimental condition and the dependent variables measured. Although, the authors proposed the hypothesis that pharmacological motives may be predominant in low-arousal smokers and psychological motives in high-arousal smokers, the results of the study do not rule out the possibility of pharmacological motives for high-arousal smokers. The indicator of arousal (i.e; heart rate) does not seem to be an adequare measure on its own and more extensive physiological and behavioural measures are necessary to establish the objective validity of the experimental conditions.

2.3 DISCUSSION AND CONCLUSIONS:

Four models of smoking behaviour and the typology scales based on them have been reviewed in the previous section. These models have been based upon theoretical formulations of smoking behaviour and empirical data to support their validity is limited to the analysis of responses to paper-pencil, self-report questionnaires.

Table 2.1 (overleaf), gives a summary of these classification schemas and illustrates the overlap between the types of smoking delineated by different models. As can be seen, smoking to stimulate onsself, in low-arousal situations or a low-arousal state have been included in all of the models. Likewise, smoking to sedate and calm oneself down, in high-arousal situations or in a state of high-arousal is also common to all models. Russell and Frith have emphasized the role of pharmacological effects of nicotine in the maintenance of these two kinds of smoking, whereas the other models failed to explain adequately how smoking accounts for stimulation or sedation.

As has been pointed out earlier, the value of typology scales lies in their potential for developing differential treatment programs that will be appropriate for the needs of different types of smokers. However, except for Russell's model, the rest have not attempted to explain the needs or motives involved in different types of smoking. Tomkins failed to provide an adequate explanation of how smoking alters affect.

Russell (1971)	*Stimulation*	*Addictive*	*Automatic*	Sedative	*Indulgent*	Psycho-Social	Sensori-motor	
McKennell & Thomas (1967)	Activity Acc.	———	Reluctant	Nervous Irritation	Relaxation & Food Subs.	Social & Social Confid.	———	
Horn & Waingrow (1966)	Stimulation	Addictive (craving)	Habitual	Tension Reduction	Pleasurable Relaxation	———	Sensorimotor manip.	
Tomkins (1966)	Stimulant (Positive Aff.)	Addictive (Positive & negative Aff.)	Habitual (No Aff.)	Sedative	Relaxant (Positive Aff.)	———	———	
Frith (1971)	Low-Arousal	———	———	High-Arousal	———	———	———	

Table 2.1. A comparison of the smoking types proposed by Russell, et al (1974), Horn and Waingrow (1966), McKennell & Thomas (1967), Tomkins (1966) and Frith (1971-a).

Tomkins' model can be illustrated as follows:

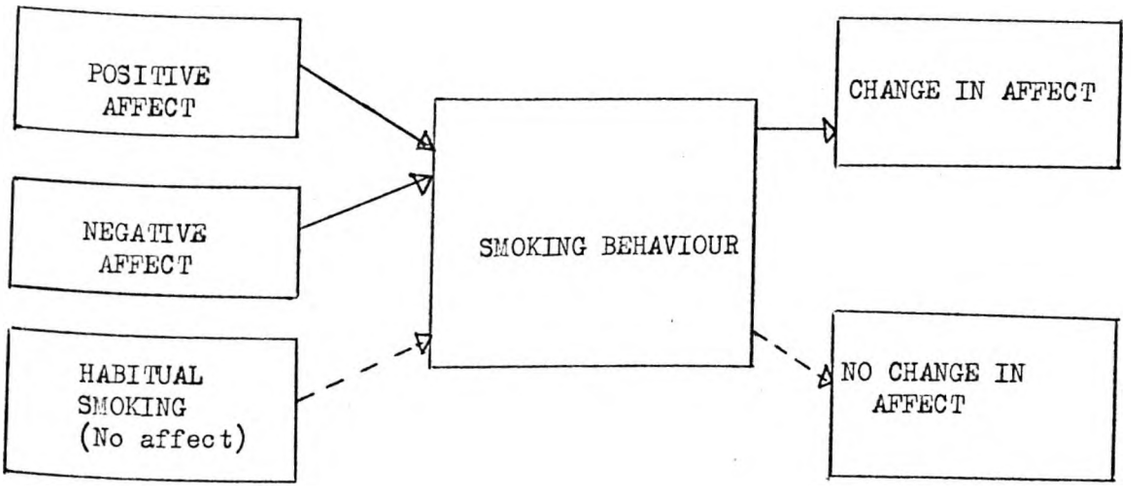

Fig. 2.2. Tomkins' model of smoking behaviour: Smoking as management of affect.

Although, this model points out that smoking is related to how a smoker feels (affect) and that smoking alters the way he feels (change in affect), it gives no information on the mechanisms of this change. So, in terms of understanding the smoking behaviour this model only contributes the information that people smoke when they experience a certain affect and smoking helps to modify or alter this affect. However, in terms of smoking intervention it is important to investigate why certain people smoke under certain affective states, the physiological and behavioural functioning at these states and what aspects (i.e; psychological, behavioural or pharmacological), of smoking alter the state of the individual.

McKennell's model also shares the same shortcoming of Tomkins' model.

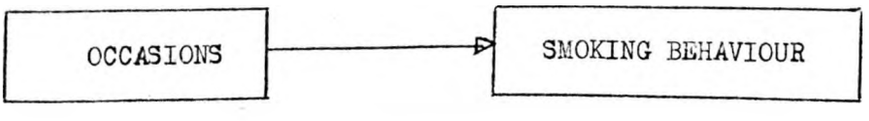

Fig. 2.3 McKennell's model of smoking behaviour: Smoking linked to certain occasions.

This model points out that people smoke on certain occasions and that smokers can be typed according to the occasions on which they most frequently engage in smoking. This model only gives a description of when a smoker is likely to smoke but fails to offer any information on why different individuals smoke in particular occasions. Although, McKennell's model can be explained in terms of learning theory principles, still it is necessary to investigate the motives for smoking in particular situations, the characteristics (i.e; in terms of physiological or behavioural activation levels) of these occasions, their effects on the smoker and the effects of smoking and not-smoking (i.e; deprivation), on these occasions.

Although very simplistic, Frith's model emphasizes the manipulation of arousal levels as a predominant motive in the maintenance of smoking.

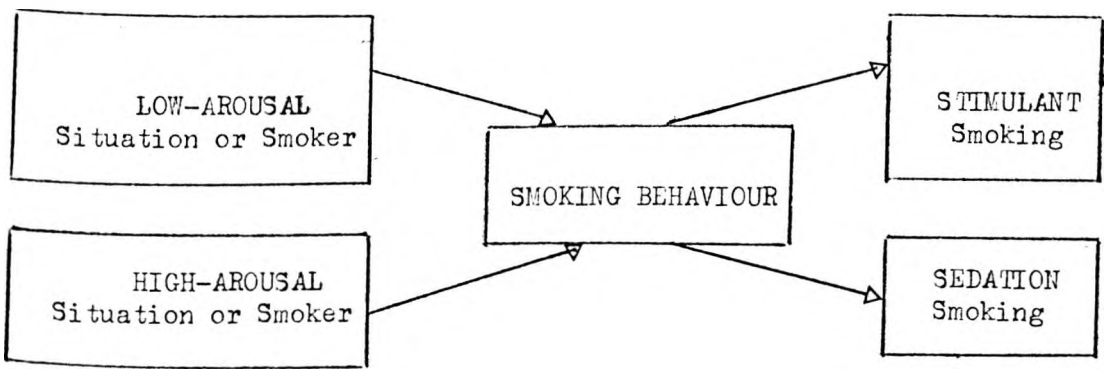

Fig. 2.4 Frith's model of smoking: Smoking to alter arousal levels.

Pharmacological motives for smoking (nicotine) have been exclusively emphasized by Frith. However, the model does not explain why some smokers have the need to smoke under high-arousal situations whereas, others smoke in low-arousal situations. It is also crucial to define arousal operationally and to investigate the measures of arousal prior to, during and after smoking in order to establish the objective validity of this model.

Russell's model departs from the others in providing a

schema based on the predominant patterns of reinforcement (see Fig. 2.1). His model provides the motives for smoking in all the types of smoking delineated by his own and other models. Russell's model combines the present evidence on physiological, behavioural and subjective effects of smoking and provides a comprehensive account of the smoking behaviour. However, the typology scale based on his model is still limited to the self-reports of smokers to pre-selected questions. In this sense, although it offers a valuable guide for designing therapy programs (e.g; pharmacological versus other forms of treatment), more research is needed to a) establish the objective validity of the typology proposed by this model with psychophysiological and behavioural measures, and b) understand the smoking behaviour in greater detail rather than prematurely accepting the present evidence on its effects as the main motives for its maintenance.

An intensive investigation of physiological, micro-behavioural, environmental and subjective (attitudinal) variables prior to, during and after smoking in different smokers, preferably in natural settings, is required in order to gain a more comprehensive, accurate and reliable understanding of the smoking behaviour.

It also seems to be very important to investigate the effects of nicotine deprivation under conditions similar to the ones initiating the smoking behaviour,on physiological, subjective and behavioural measures.

In conclusion, the present models of smoking behaviour and smoking types derived from them point out that social, sensory and pharmacological motives might be playing a role in the maintenance of the smoking behaviour and that smoking can be classified according to the predominance of these motives.

Types of smoking, (stimulation and sedative smoking) indicative

of the involvement of pharmacological motives in the maintenance of the smoking habit have been included by all of the models. For this reason, it is important to investigate the role of nicotine in the maintenance of smoking. There have been numerous studies investigating the role of nicotine in the smoking habit. However, as it is pointed out by the typology scales not all smokers smoke to stimulate or sedate themselves or to manipulate their arousal levels. Therefore, it can not be expected that nicotine is the predominant reinforcer for all smokers. As will be seen in the next chapter, surprisingly very few researchers have made distinctions between types of smoking when investigating the role of nicotine. Even the ones viewing smokers as a non-uniform group, differentiated them purely on the basis of consumption levels.

So, although the research described in this chapter provides very useful and practical research questions, generally the possibilities offered by this valuable body of research have been overlooked, both by clinicians in designing their treatment programs and also by experimentalists who attempted to identify the role of nicotine in the maintenance of the smoking habit. As will be seen in the next chapter and as has already been pointed out in the smoking treatment review, this has led to research described by Bernstein (1969), as, "directionless, or circular".

CHAPTER SUMMARY

Various researchers have viewed smokers as a non-uniform group and have attempted to identify types of smoking on the basis of theoretical models of smoking behaviour or self-report data from questionnaires.

Tomkins (1966), viewed smoking as management of affect, and typed smoking according to his theory of affect. McKennell (1967), differentiated types of smoking according to the characteristics of occasions smokers said they most frequently smoked. Frith (1971), viewed smoking as a means to manipulate arousal levels and, finally Russell (1971), proposed a typology based on a composite reinforcement model of smoking.

The models proposed by the above authors and the typology scales based on these models have been reviewed and their implications for a differential treatment approach have been discussed. The need to investigate the objective validity of the typology scales has been emphasized.

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CHAPTER 3

THE ROLE OF NICOTINE IN THE SMOKING BEHAVIOUR

3.1 INTRODUCTION

A number of investigators have opposed the overemphasis of psychosocial determinants of smoking behaviour and have proposed that smoking is mainly maintained by pharmacological reinforcement (Russell, 1971-b; Jarvik, 1970; Armitage, 1973). In this approach smokers are viewed as smoking to obtain a consistent and individually characteristic level of nicotine. Based on this assumption a "nicotine-titration" hypothesis was proposed, which suggested that some smokers regulate nicotine intake in order to achieve an optimum dose.

Russell (1976-b), stated that "nicotine is the one alkaloid present in tobacco and tobacco smoke that is rapidly absorbed and distributed throughout the body, including the brain, in sufficient amounts to produce a striking array of pharmacological effects both centrally and peripherally". The authors supporting the "nicotine-titration" hypothesis attributed to nicotine the central role in the maintenance of smoking behaviour and only a few of them made a distinction between different types of smokers and the differential role of various reinforcers for each group.

Although, nicotine is a powerful drug, the level of intake and the particular pharmacological effects produced in individual smokers or groups of smokers needs to be considered. There have been very few studies in which smokers were classified according to their initial consumption levels (i.e; heavy versus light smokers), (Ague, 1972; Schachter, 1977). Although, some investigators did observe different and individually consistent patterns of change in plasma nicotine levels in response to nicotine manipulations (Russell, et al, 1975), amongst the subjects, the characteristics of smokers exhibiting similar response patterns have not been examined.

To support the role of nicotine, Russell (1976-b), pointed out that there is historical evidence in favour of the role of nicotine, ... "throughout its history, tobacco use has fluctuated between chewing, snuffing and smoking, but no population has dispensed with one form of tobacco use without replacing it with another". However, although it is clear that one common factor in all the variety of usages of tobacco is nicotine, which is absorbed through the lungs in cigarette smoking, the buccal mucosa in chewing tobacco and nasal mucosa in snuffing, oral gratification, social influences, and the satisfaction of manipulative tendencies which have been proposed to play a role in the maintenance of cigarette smoking can also be applied to other usages of tobacco.

The role of nicotine may be examined by investigating whether or not smokers accept nicotine-free or low-nicotine cigarettes which are identical to standard brands of cigarettes in tar yield, draw resistance, burning characteristics and flavour. Unfortunately, commercially available cigarettes render this method inapplicable since at present the tar and nicotine yields of these cigarettes are highly correlated ($r = 0.96$; Goldfarb, et al, 1976). However, some investigators have overcome this problem by using experimental cigarettes which are naturally low in nicotine and then adding nicotine to them. This issue, could also be examined by investigating whether nicotine administered alone, can substitute for tobacco. However, since cigarette smoking is the most efficient way of delivering nicotine to the body and brain, (Russell, 1976-b,; Armitage et al (1968), this approach awaits further improvements in the technology of administering nicotine by alternative routes.

The above limitations have led the investigators to attempt to demonstrate a "nicotine-titration" effect amongst smokers, which suggests a consistent and individually characteristic level of nicotine intake, which is sufficient for the production of a particular pattern of pharmacological effects. In seeking evidence to support the "nicotine-titration" effect

investigators have analysed the changes in the parameters of smoking behaviour (i.e; number of cigarettes smoked, puff analysis, nicotine-bu.t analysis, plasma nicotine analysis, etc.) in response to:

a) manipulations of the nicotine yields of cigarettes smoked (i.e; low, middle and high nicotine yield cigarettes or nicotine-free cigarettes);

b) administration of nicotine by routes other than tobacco smoke (i.e; nicotine chewing-gum and nicotine aerosol); and administration of nicotine antagonists (i.e; drugs that are known to block the pharmacological effects of nicotine, e.g; mecamylamine);

c) manipulation of the acidity level of urine.

3.2 GENERAL EVALUATION OF STUDIES ASSESSING THE ROLE OF NICOTINE

Before discussing the methods and outcomes of studies using the experimental manipulations mentioned above, various measures of change in smoking behaviour will be defined and their limitations will be pointed out.

i) Measures of change in the smoking behaviour:

a) Number of cigarettes smoked:- This is simply the frequency of smoking over a certain time period. Although consumption level is a useful measure and can be fairly easily and accurately assessed, it needs to be supplemented with more direct measures of nicotine intake. It has been shown that smokers can modify their puff-rate, the depth of inhalation, weight of tobacco burned, etc., on switching from high to low nicotine cigarettes (Ashton & Watson, 1970; Turner, et al, 1974). So, a crude measure of number of cigarettes smoked might conceal important information on the amount of nicotine delivered to the smoker. This measure is also prone to recording errors when the consumption is only recorded by the smoker himself without any independent observer. However, it is a practical and economical measure and can be used in a wide range of experimental studies. Investigators using this measure as the only dependent variable must exercise caution in interpreting their findings. The limitations of this measure are more likely to mask the nicotine regulation amongst the smokers, since even if the frequency of smoking remains constant following nicotine manipulations, the smoker might alter other

parameters of his smoking behaviour and increase or decrease his nicotine intake.

b) Topographical Components of smoking behaviour:- These measures involve analysis of puffing for rate of puffing, volume of puffing and inter-puff-interval (time elapsed between two consecutive puffs); cigarette duration (time elapsed between lighting up and putting the cigarette out); weight of tobacco burned (the weight of the cigarette stub subtracted from the weight of an unlit cigarette). As stated above (see (a)), it has been observed that smokers do change the topography of their smoking responses following the manipulation of the nicotine yields of cigarettes, without necessarily altering their consumption levels. So, it is important to assess the changes in the topographical components of smoking following nicotine manipulations. However, since these measures are not directly indicative of levels of nicotine intake, it is of crucial importance to investigate the changes produced in blood nicotine levels by alterations of smoking topography. Data from blood nicotine analysis which requires gas chromotography, will enable researchers to concentrate on the analysis of response topography when appropriate facilities are not available to perform blood nicotine analysis, or when there are practical limitations imposed by the nature of the investigation, to obtain blood samples at appropriate time periods (i.e; investigations taking place in natural environments).

c) Cigarette butt analysis for nicotine:-

This measure is used as an index of the amount of nicotine drawn into the mouth and it does not provide a direct measure of nicotine inhaled into the lungs. It is calculated on the basis of the filter retention efficiency(1)

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(1) The filter retention efficiency is calculated from measurements of the mainstream smoke nicotine and filter nicotine for any brand of cigarette. Filter retention efficiency (F) = $\frac{N r}{Ns + Nr}$; where Nr is the filter nicotine and Ns is the mainstream smoke nicotine (Rawbone, et al, 1978).

of the filter and the amount of nicotine retained in the filter after smoking. The formula for the calculation of the nicotine presented to the smoker was given by Ashton and Watson (1970), and Rawbone et al.(1978), as:

$$Ns = \frac{Nr \quad (1-F)}{F}$$

where Ns= nicotine presented to the smoker (i.e; mainstream smoke nicotine); Nr=nicotine retained in tip; F= filter retention efficiency.

This is certainly a more reliable and direct measure of nicotine intake in comparison to the number of cigarettes smoked. However, this method reflects the dose of nicotine presented to the smoker and not the dose absorbed. Therefore, it is important to investigate the relationship between estimated nicotine intake from butt analysis and other measures reflecting the amount of nicotine absorbed. Rawbone et al(1978), investigated the relationship between alveolar carbon monoxide (calculated from expired air, and related to the dose of nicotine absorbed), and nicotine presented to the smoker (from butt-nicotine analysis). No significant relationship was noted between these measures (i.e; $r= 0.28$, $p>0.05$). Thus, on the basis of this result the dose of nicotine presented to the smoker as estimated from butt-nicotine analysis do not seem to reflect the dose inhaled from the mouth to the lungs. However, Kumar et al (1977), have reported a significant positive correlation between puff volume and nicotine butt analysis.

d) Blood and Urinary Nicotine Levels:- The rate of excretion of nicotine in the urine is highly dependent on its acidity level, so that unless the acidity of urine samples is kept constant, the excretion rate of nicotine does not offer reliable and meaningful data. On the other hand, blood nicotine analysis is the most direct way of measuring the dose of nicotine intake from a cigarette. It permits the investigation of the time course of elimination of nicotine from blood after smoking one cigarette and also the cumulative effects of several cigarettes. It also reveals the differences between smokers in terms of the peak blood nicotine levels after smoking a particular

cigarette.

However, this method also presents some problems. Firstly, the timing of the post-smoking blood sample is of crucial importance, since nicotine is rapidly eliminated from the blood in the period following administration. Moreover, the peak nicotine levels may depend on the puff frequency and depth of inhalation, so that blood samples taken after the termination of smoking may not provide the peak measures (Ashton, Stepney & Thompson, 1978). An alternative to the post-smoking blood sampling has been recommended by Russell (1976-b), "more refined continuously repeated sampling of arterial blood would enable the estimation of nicotine concentrations during and between boli".

In addition to these difficulties which necessitate caution in interpreting the results of blood nicotine analysis, the method also poses some practical limitations. Blood sampling may interfere with the other experimental measures (i.e; topographical components of smoking behaviour or psychophysiological measurement) and may induce changes in these measures (i.e; due to the stress). Also, the analysis of blood samples requires specialized technology and equipment (i.e; gas-liquid chromatography; Isaac & Rand, 1972) which may not be available for the experimenter.

e) Blood Carboxyhaemoglobin (COHb) levels:- Carbon monoxide (CO) is not absorbed buccally, so any increase in blood COHb is proportional to the degree of inhalation (Russell, 1976-b). Thus, blood COHb levels have been used as an index of degree of inhalation. The analysis of blood with a CO-Oximeter reveals the COHb levels (Russell, et al, 1973). Reduced COHb levels were used to support a lowered nicotine intake either by a decrease in the number of cigarettes smoked or lesser degree of inhalation. It is a useful measure for comparing the degree of inhalation between smokers and associated changes in blood nicotine levels, however the practical limitations (i.e; interference with other experimental measures and the technological facilities required for analysis), of the blood nicotine analysis are also

applicable for this measure.

ii) Methodological Shortcomings:

a) Subject Selection:-

The majority of researchers seeking evidence to support the "nicotine-regulation" hypothesis have conceived smokers as a homogeneous group and attempted to demonstrate a nicotine regulation attempt in all smokers. In this approach the inherent assumption is that pharmacological reinforcement is universal for all smokers alike. Although, it was emphasized that smokers seek an individually characteristic and consistent level of nicotine intake, no attempt was made to differentiate smokers in terms of their characteristic levels of nicotine requirements. It needs to be investigated whether individual smokers require different levels of nicotine intake due to their different physiological and chemical constitutions, in order to obtain similar pharmacologic effects or whether various smokers seek different levels of pharmacologic effects. In order to examine this issue it is necessary to have strict control on the sample of smokers participating in experimental studies or alternatively, to analyse the characteristics of smokers in a random sample showing similar trends of change.

For example, Russell, et al (1976-a), reported that "some individuals increase their nicotine levels on switching to the high nicotine brand, but others do not". It would be valuable to analyse the characteristics of those individuals showing similar trends. However, with a few exceptions (Ague, 1972), the majority of studies have used experimental groups matched on mean age, initial consumption levels and sex but failed to use homogeneous groups of smokers, classified by a typology test or according to consumption levels. Ague, (1972) & Schachter (1977), have investigated the effects of nicotine manipulations amongst heavy and light smokers. This type of an approach is more likely to reveal useful information on the role of a particular reinforcer. Surprisingly, some studies have used subjects with a wide range of initial consumption. For example, Kozlowski, et al (1975),

reported that their subjects smoked between 1 to 60 cigarettes per day. Although, their experimental groups were matched in terms of mean consumption levels, the results of such a study will be highly masked, due to the variance between subjects and not very informative on the individual predominant reinforcers for dissimilar smokers.

b) The use of standard brands of cigarettes, and knowledge of experimental manipulations:-

Although some researchers used specially manufactured cigarettes with different nicotine yields (Turner, et al, 1974; Ague, 1972) or experimental cigarettes with different nicotine yields but equivalent tar content (Goldfarb, et al, 1976), some others have used standard brands of cigarettes without any attempt to conceal their nicotine yields from the subjects (Russell, et al, 1975), which limits the reliability of the findings. The use of lettuce-leaf nicotine free cigarettes also poses such a problem, since these cigarettes have an unusual taste and smell, which makes them easily identifiable. In administering nicotine by routes other than smoking, smokers have been usually aware of the nature of the experimental manipulations. In investigations employing nicotine-chewing gum, the subjects were usually informed that they would be taking nicotine, however they were not informed of the non-nicotine placebo gum. The findings of studies in which nicotine manipulations were not completely blind needs to be interpreted with caution.

In summary, studies seeking evidence to support the "nicotine-titration" effect have used various measures of change in smoking behaviour in response to nicotine manipulations, some of which have limitations when used on their own. The choice of a particular dependant variable is mainly determined by the nature of the investigation (i.e; laboratory versus experiments in natural settings), and also by the availability of technological facilities. The limitations of the measures taken needs to be considered in the interpretation of the results.

3.3 REVIEW OF EXPERIMENTAL STUDIES:

The experimental studies investigating changes in smoking parameters in response to nicotine manipulations will be surveyed in this section.

i) Manipulations of the nicotine yields of cigarettes smoked:-

The findings of the majority of studies using this manipulation have indicated that "blind" substitution of low-nicotine cigarettes has led to an increase in consumption and puff-frequency and also to a shorter latency between cigarettes smoked, which supported a nicotine regulation view. However, some others reported contradictory results and did not find evidence of compensation for the nicotine decrement on switching to half-cut cigarettes (Goldfarb & Jarvik, 1972) or to low nicotine yield cigarettes (Forbes, et al, 1976). Considering the diversity of measures used as an index of change in the smoking behaviour, the lack of specification of smokers according to a reliable and relevant criteria and the small sample size in most of the studies, the emergence of these conflicting results does not seem surprising.

Ashton and Watson (1970), showed that smokers smoking low-nicotine cigarettes took significantly more puffs than smokers smoking high-nicotine cigarettes. The butt nicotine analysis revealed that there was no significant difference in the amount of nicotine delivered to the smoker from the two types of cigarettes. This study confirms a titration effect by demonstrating that smokers do compensate for the low-nicotine delivery of a cigarette by increasing their puff-rates and obtaining similar amounts of nicotine from low and high nicotine cigarettes. Although there was a wide range in initial consumption levels of subjects (3 to 30 cig/day), which could have masked the results, since a smoker smoking 3 cig/day is not likely to smoke in order to achieve a certain dose of nicotine, the modification of a parameter of smoking behaviour (i.e; puff-rate), to obtain a particular dose of nicotine was demonstrated. However, the increase in puff-rates could also be due to the differences in the draw resistances of the two experimental cigarettes and in

this connection it is highly desirable to equate cigarettes on characteristics other than nicotine yields.

The effects of varying the nicotine contents of cigarettes on consumption levels was also investigated by Frith (1971-b). It was found that the greater the nicotine yield of the cigarette, the less was the number smoked during the eight hour experimental period, which indicated a nicotine regulation effect. However, number of cigarettes smoked is a rather crude measure, when employed on its own and a replication of this study with a more direct measure of nicotine intake would be useful.

Russell, et al (1973), have also noted a significant increase in the number and weight of cigarettes smoked on switching to low nicotine cigarettes, and a decrease in consumption on changing to high nicotine cigarettes.

Turner, et al (1974), confirmed the nicotine regulation hypothesis. Changes, indicating compensation for nicotine decrement, or increment were observed in some smoking parameters in response to smoking three kinds of cigarettes, ranging from medium to very low nicotine yields, over a 3 week period. They noted a significant increase in consumption on switching from medium to low nicotine brands. Butt-nicotine analysis showed that there was a higher nicotine intake than would be expected (with a standard smoking machine), from the very low nicotine cigarettes. All subjects rated the very-low nicotine cigarettes as too weak and unsatisfying. These findings showed that smokers try to compensate for low nicotine yields by increasing their consumption and extract more than expected amounts of nicotine from very low nicotine cigarettes. However, a major shortcoming of this study was the use of standard brands of cigarettes, which casts some doubt on the changes observed.

Kozlowski, et al (1975), reported that the latency to the next cigarette was shorter after smoking a low nicotine cigarette, which was interpreted

as indicating a nicotine regulation effect. However, total puff-time in a free smoking period showed that differential nicotine levels in cigarettes did not influence the puff-times. Although the shorter latency of smoking can be interpreted as a compensation, unless all the other properties of low and high nicotine cigarettes are equated (e.g; burning rate, draw resistance, etc.) in addition to nicotine yields, it is not safe to draw a conclusion to support the role of nicotine. It could be possible that some other property of low nicotine cigarettes apart from their lower nicotine yields are unsatisfying for the smokers and might lead them to smoke more frequently. Although this study was conducted with a large sample (N = 56), there were wide differences between initial consumption levels amongst the subjects (1 to 60 cigs/day). It would have been useful to include butt nicotine, blood nicotine or COHb analysis to support the relation between latency and nicotine intake. The inherent assumption of shorter latency to the next cigarette is a lowered nicotine intake and this assumption needs to be confirmed by measures of actual nicotine intake, (since other investigators have shown that parameters of smoking behaviour are altered to obtain higher doses of nicotine from low nicotine cigarettes than would be expected) (Ashton and Watson, 1970).

Russell, et al (1973), examined the effects of high and low nicotine cigarettes on blood nicotine and carboxyhaemoglobin levels as well as number of cigarettes smoked during a 5 hour experimental period. They have confirmed a nicotine regulation effect by showing that 1) midmorning plasma nicotine levels obtained on four different days after subjects had smoked their usual cigarettes were fairly consistent within subjects although there were great variations between subjects, 2) there was a significant decrease in consumption on switching to high nicotine cigarettes, and 3) the average blood nicotine levels obtained after smoking medium and high nicotine cigarettes were not significantly different. However, an examination of individual nicotine levels revealed that the subjects showed different patterns of change, half of them had an increase in blood nicotine levels,

whereas the rest had lower levels of nicotine on switching to high nicotine cigarettes. The mean measures conceal this individual variation and it seems to be more appropriate to analyse individual results and the characteristics of smokers showing similar trends of change (i.e; increase or decrease). In spite of the significant decrease in consumption on switching to high nicotine cigarettes, half of the subjects did in fact display higher nicotine levels. This would imply, contrary to what the authors stated, that for at least some smokers the plasma nicotine levels, measured after the completion of a particular cigarette, does depend on the nicotine yield of that cigarette and that some smokers might have consistent topographical responses which are not altered by changes in nicotine yields. A decrease in COHb levels was also noted on switching to high nicotine cigarettes, which suggests a lesser degree of inhalation. It is again desirable to examine individual levels of change, before arriving at a general conclusion. This study is valuable in demonstrating different requirements of nicotine intake amongst smokers and dissimilar trends of change, which supports the necessity of a differential investigation of effects of various reinforcers.

Although, it is crucial to equate cigarettes of different nicotine yields on other properties, there have been very few attempts of this reported in the literature. It has been mentioned earlier that tar and nicotine levels in commercially available cigarettes are highly correlated ($r = 0.96$). For this reason we can not evaluate the results of the studies on nicotine-titration effect without having reservations about the conclusions. Smokers might well be regulating their tar intake. Three studies have been reported, in which attempts were made to exclude tar as a confounding factor in titration. The most recent and well controlled one was conducted by Goldfarb, et al (1976). They have used experimental cigarettes with low correlations ($r = .14$), between tar levels and nicotine yields as compared to commercial cigarettes. By this they were able to investigate the effects of varying the nicotine content of cigarettes independent of tar levels. It was

noted that the mean number of the experimental cigarettes smoked tended to fall significantly as their rated delivery of nicotine increased. There was no effect of tar on this measure, and the effect of nicotine yield did not differ as a function of the tar level. Ratings of the "strength" of the experimental cigarettes were found to be consistently and directly related to the nicotine yields, and not to their tar levels. So, the findings of this study excluded tar as a confounding factor and have confirmed earlier reports, by showing that nicotine is a factor that can influence both the number of cigarettes smoked and their perceived "strength". The use of additional and more direct measures of nicotine intake to support the noted change in consumption levels would have been commendable.

In an earlier study reported by Goldfarb, et al (1970), other characteristics of cigarettes with different nicotine yields were equated by adding nicotine to lettuce leaf cigarettes. Subjects' baseline smoking rates with their own brands of cigarettes were compared to their rates when smoking the experimental cigarettes (0, 1.26, and 2.25 mg of nicotine/cig). The authors noted a general decrease in smoking rate for all types of experimental cigarettes, as compared to the base-line smoking rate. This finding did not support a regulation of nicotine intake view since different nicotine delivery cigarettes produced similar decreases in smoking frequency. Russell (1976-b), in his evaluation of this study stated that .. "the nasty taste of the lettuce smoke seems to have swamped all other effects, causing the subjects to smoke fewer experimental cigarettes than their usual brand", what Russell did not mention is the fact that the decrease in consumption was still observed when the subjects switched back to their own brands at the last experimental period. An important procedure in Goldfarb et al's study was the requirement to keep careful records of self-smoking frequency. Several investigators have shown that self-monitoring (i.e; keeping a record of smoking frequency), is a reactive measure and produces decreases in consumption levels (McFall, 1970; McFall & Hammen, 1971; Rozensky, 1974), probably by making smokers more aware of their smoking. So, the decrease in consumption, which was still observed after switching to usual brands,

could well be due to the effects of self-monitoring rather than the taste of lettuce leaf cigarettes or other experimental manipulations. The general findings of this study does not support a nicotine-titration hypothesis, however the reactivity of self-monitoring and the unusual taste of lettuce leaf cigarettes makes it difficult to draw any firm conclusions.

In an earlier study Finnegan, et al (1945), also reported findings based on the changes in smoking behaviour induced by varying the nicotine yields of cigarettes, which were similar in other respects. They used tobacco naturally low in nicotine to produce low-nicotine cigarettes and then added nicotine to this tobacco to produce a cigarette with a higher nicotine yield. By this manipulation, the subjects' responses could be attributed solely to differences in nicotine yields. No statistical analysis was reported. However, it was noted that the subjects who increased their consumption on switching to low-nicotine cigarettes did not experience withdrawal symptoms. On the other hand, subjects who did not increase their consumption experienced severe withdrawal symptoms. Heightened irritability, decreased ability to concentrate on mental tasks, feelings of inner hunger and emptiness were the common symptoms reported by this group. The authors evaluated their findings as supporting the role of nicotine for only some smokers, stating that ... "with many individuals nicotine is not a factor in their smoking habit". This conclusion lends support to an approach of differential role of nicotine for various smokers. Although the sample size was too small (N = 25), to arrive at a reliable conclusion on the roles of different reinforcers for smokers, the exclusion of other confounding factors in cigarettes with different nicotine yields and the conclusions of the authors are very valuable. Considering that this is a fairly early publication in this field it seems surprising that other investigators have not followed the direction of research pointed out by these authors.

Two independent studies, investigating the role of nicotine for heavy and light smokers, have been reported. The first one was the study by

Ague (1972), who showed that heavy smokers (i.e; > 10 cig/day) preferred the experimental cigarettes with varying nicotine yields to non-nicotine lettuce leaf cigarettes, whereas light (i.e; < 10 cig/day) smokers rated the lettuce leaf cigarettes higher in preference and showed a dislike for the high-nicotine cigarettes. The author concluded that these findings supported the importance of nicotine for the heavy smokers. The second investigation was conducted by Schachter (1977), who used two groups of subjects, classified according to initial consumption levels and some other smoking history criteria as heavy (> 20 cig/day) and light (< 15 cig/day) smokers. It was noted that heavy smokers reduced their consumption on switching to high nicotine cigarettes, whereas no consistent regulation was observed in light smokers. The poor regulators among the heavy smokers were the ones to report withdrawal symptoms. Schachter, on the basis of these findings stated that "there appears to be no question but that long term heavy smokers regulate nicotine intake". This is an invaluable investigation in its approach to the differential effects of nicotine manipulations on groups of smokers classified according to an objective criteria. Only the consumption levels of heavy smokers were effected consistently in a trend that suggests a regulation of a consistent level of nicotine intake. Since such a change was not observed amongst the light smokers, it seems likely that some other reinforcer which needs to be identified is operating in the maintenance of their smoking behaviour. These two studies are in agreement with each other in suggesting the operation of different reinforcers for light and heavy smokers.

Two studies using similar manipulations of experimental cigarettes, but different measures of change by independent authors have been published, one favouring the nicotine titration effect (Gritz, et al, 1976) and the other (Goldfarb & Jarvik, 1972) disfavouring it. These studies demonstrate well the limitations imposed by the lack of a standard and reliable measure of change in smoking behaviour and point out the need

Forbes et al (1976), have discredited a nicotine regulation effect by showing that for the majority of subjects changing to cigarettes with lower nicotine yields than their usual brands has led to a reduction in the total daily mouth-level exposure to nicotine (by butt-nicotine analysis). The extent of the reduction was related to the nicotine decrement (i.e; the difference between the nicotine yields of the ordinary and low-nicotine cigarettes). The findings of this study disfavoured a nicotine regulation effect, by showing that there was no tendency to increase mouth-level nicotine intake while smoking cigarettes with nicotine yields lower than accustomed brands. An interesting finding was that there was a substantial variation in the mouth-level exposure to nicotine even among smokers smoking cigarettes with similar nicotine yields, which indicates that smokers have different smoking parameters (i.e; puff rate; depth of inhalation, etc.) which influence their nicotine intakes. It would be interesting to investigate whether various smokers need different levels of nicotine intake to obtain comparable pharmacological effects or whether some smokers have consistent smoking patterns that are unrelated to the nicotine delivery of the cigarettes they smoke.

Lastly, a study by Freedman and Fletcher (1976), disfavouring the nicotine regulation hypothesis will be discussed. This study was conducted over a much longer experimental period (20 months) as compared to the other studies discussed, thus it shows the long-term changes in the smoking behaviour on switching to lower nicotine yield cigarettes, allowing subjects to adjust to the low nicotine levels. The authors have noted that cigarette consumption remained constant on changing to "New Smoking Material" cigarettes (NSM). This finding indicated that subjects accepted the decrement in the nicotine intake without any change in their consumption levels. The results of butt-nicotine analysis also supported the above interpretation, in that nicotine presented to the smoker from the commercial and NSM cigarettes were found to be similar to the expected nicotine

to be cautious in the interpretation of results based only on one and crude measure of change (i.e; number of cigarettes smoked). In both of the studies, subjects were provided with full length cigarettes of their own brands, a full-length cigarette marked at half-length (distal condition), and a cigarette cut at the one half-length (proximal condition). Goldfarb & Jarvik (1972), did not report a significant increase in consumption levels on switching to the experimental cigarettes. However, 12 out of 18 subjects increased consumption by 5 cigarettes per day, which can be interpreted as an attempt to compensate for the decrease in nicotine delivery of half cigarettes. Subjects could also have compensated by altering their puff-rates, depth of inhalation, etc., for the reduction in nicotine delivery. However, these measures were not taken. The authors have stated that the results showed a functional autonomy of smoking behaviour from manipulations of cigarette nicotine delivery. On the other hand, Gritz, et al (1976), by using urine nicotine analysis and keeping consumption levels constant have shown that nicotine manipulations did in fact alter the smoking parameters. This finding casts doubt on Goldfarb & Jarvik's results. Gritz, et al by keeping consumption constant expected that if subjects attempted to titrate nicotine intake, then they had to adjust parameters of smoking behaviour other than the number of cigarettes smoked. Urine nicotine analysis supported a nicotine regulation effect. There was no significant difference between nicotine excreted in the full-length and proximal conditions, despite the greater length of full-length cigarettes. Although, the authorsdid not measure any topographical component of smoking to support their findings and also to identify the responses that were altered while smoking the half-cut cigarettes, they have stated that topographical components must have been altered by the subjects in order to maximize nicotine intake. Gritz, et al's study can be regarded as a replication of the former study disfavouring a regulation effect and the use of different measures of change seems to have led to disparate conclusions.

deliveries of the two cigarettes as measured by a standard smoking machine. So, in conclusion the findings of this study were not in line with a nicotine regulation view, especially considering that consumption remained unchanged despite the free supply of cigarettes. However, butt-nicotine analysis is only an index of mouth-level exposure to nicotine and the subjects could have increased their depth of inhalation and obtained higher doses of nicotine than expected from the NSM cigarettes which would not be detected by butt-nicotine analysis.

In summary, the evidence from this class of experiments, manipulating the nicotine yields of cigarettes smoked are conflicting, some confirming a nicotine-regulation effect and some failing to note any attempt to regulate nicotine intake and obtain consistent doses of nicotine. Although it is crucial to investigate the role of nicotine which is indeed a potent substance delivered to smokers by cigarette smoking, it seems more appropriate to investigate its role in homogeneous groups of smokers or to analyse the characteristics of smokers showing similar trends of change. It seems likely that the disagreement in results are mainly due to attributing to nicotine a universal and central role in the maintenance of the smoking habit, without allowing for a non-uniformity of needs amongst smokers. A second reason for the lack of agreement in the reported findings could be due to the shortcomings of some of the measures used and experimental methodology.

It seems clear that for some smokers nicotine does play a central role and the main issue is to identify these smokers and also to assess the degree of pharmacological effects produced by different levels of nicotine intake in different smokers. It is also highly desirable to exclude factors other than nicotine as confounding factors, in order to attribute the changes in smoking behaviour solely to nicotine manipulations.

ii) <u>Effects of administering nicotine by alternative routes and nicotine antagonists on the smoking behaviour</u>:

On the basis of the "nicotine-regulation" view it can be

argued that if smokers can obtain sufficient amounts of nicotine to satisfy their needs, from sources other than cigarettes then they will reduce their consumption levels. On the other hand, if they are given nicotine antagonists, which will block the pharmacological effects of nicotine obtained from cigarettes, then they are likely to increase their consumption levels in order to compensate. In this section studies investigating the changes in the smoking behaviour following the administration of "nicotine-chewing-gum", "intraveneous nicotine injections" and "nicotine antagonists" will be surveyed.

In order to provide evidence for the role of nicotine in the maintenance of the smoking habit it is necessary to demonstrate that simultaneous administrations of nicotine via an alternative route leads to a decrease in the frequency of smoking. However, cigarette smoking is the most efficient method of nicotine administration, in terms of the puff by puff boli and higher peak levels of plasma nicotine. It is difficult to establish other methods of administration with comparable efficiency, (Russell, 1976-b).

Stemming from the findings demonstrating the role of nicotine and the importance of pharmacological reinforcement, a chewing-gum containing nicotine bound to an ion exchanger was devised in Sweden, (Fernö, et al, 1973). The rationale was to avoid the risks imposed by carbon monoxide, tar and other harmful constituents of tobacco smoke, and to satisfy the smokers' craving for nicotine. The gum is prepared with varying amounts of nicotine (0, 1, 2, 4 mg of nicotine). Intensive chewing causes about 60% of the nicotine to be released after ten minutes and most of the remainder after a further ten minutes. Buffer substances were added to increase the rate of buccal absorption. Fernö, (1975), reported that after chewing the 4 mg nicotine-gum every hour or smoking one cigarette every hour the blood nicotine levels just before chewing or smoking were virtually identical. Russell, et al (1976-a), confirmed the above finding, however they also reported that the absorption from the gum was much slower and did not produce the same peak levels or puff-by-puff boli obtained by cigarette smoking. Another disadvantage

of the gum is its pungent taste and side effects (e.g; local irritation of mouth, hiccups, increase in heartburn).

Although, the nicotine chewing-gum offers a practical method of nicotine administration and permits the investigation of effects of alternative nicotine administrations, the act of chewing provides an oral substitute for smoking behaviour and thus needs to be controlled for by double-blind studies with placebo and active gums.

Since the chewing-gum has been produced fairly recently, there are only a few experimental studies on its effects. Brantmark, et al (1973), assessed the effects of the 4-mg nicotine-chewing-gum and a non-nicotine placebo-gum in reducing cigarette consumption. In this double-blind one week trial subjects were given either the active or the placebo gums and were required to substitute as much of the tobacco as possible with the chewing-gum. The results showed that tobacco consumption was significantly lower in the active treatment group than in the placebo group. Subjects were found to consume less nicotine-gums than placebo-gums. Subsequently, the authors divided the sample into light and heavy smokers and noted that a higher proportion of heavy smokers were successful in the active-gum group than in the placebo group. This finding supports the likelihood of pharmacological addiction to nicotine in heavy smokers, since administration of nicotine-chewing-gum was found to lead to a higher proportion of successes amongst the heavy smokers. On the other hand, a higher proportion of successes were observed amongst the light smokers in the placebo group than amongst the heavy smokers. These findings suggest a differential treatment effect for light and heavy smokers and fit in well with the view that heavy smokers, who are more likely to be dependent on nicotine, will benefit more from a pharmacological treatment approach. The authors concluded that the nicotine-chewing-gum is a valuable adjunct in the first phase of tobacco withdrawal, firstly by its nicotine content, which was important for heavy smokers and secondly by providing a form of oral

satisfaction. This study illustrates how the effects of nicotine manipulations might be masked by using heterogeneous samples and points out the appropriateness of a differential assessment approach.

Russell and his co-workers conducted a series of well-controlled investigations on the effects of nicotine-chewing-gum on smoking frequency and plasma nicotine levels. In general, their findings did not provide a strong support for the therapeutic value of the chewing-gum as a substitute for cigarettes, mainly because of the slower rate of nicotine absorption.

Russell, et al (1976-a), compared the plasma nicotine levels obtained after smoking cigarettes (1.2 mg of nic) and nicotine-chewing-gum (2 and 4 mg of nic), in a single subject over seven hours. It was found that the peak nicotine levels after cigarette smoking was higher compared with the levels obtained with the 2 and 4 mg chewing gums. Nicotine was absorbed much more slowly from the gums (peak levels at 15 - 30 minutes for 4 mg gum), than from cigarettes (peak levels at 2 minutes). The authors have also examined the plasma nicotine levels after smoking and after taking the 2 mg nicotine gum in 15 smokers attending a "smokers" clinic. The average plasma nicotine levels at the end of a day, when subjects only took the 2 mg gum was significantly lower as compared with a day, when they smoked as usual. This finding suggested that the 2 mg gum is too small a dose for adequate nicotine substitution. On the other hand, Russell, et al (1977), found that on a fixed schedule of one piece of 4 mg gum per hour, the plasma nicotine levels produced by the gum in 21 subjects were similar to the levels produced by ad libitum smoking. In this study the authors have also reported that the subjective ratings of degree of missing cigarettes were not related to the plasma nicotine or to the blood COHb levels, both after smoking and chewing a 4 mg-gum. So, although the 4 mg-gum can probably provide an adequate nicotine substitution, it does not seem to satisfy the subjective need for cigarettes. The majority of subjects in this study experienced side effects and found the gum unpleasant.

Considering these findings it is difficult to attribute any change in the smoking behaviour following the administration of the nicotine chewing-gum solely to its nicotine yield. Since the gum offers oral satisfaction and also distraction for twenty minutes while it is being chewed, these factors need further investigation.

Yet in another study Russell, et al (1976-b), found that when smokers took 2-mg nicotine-gum, or placebo gums in a double-blind crossover trial, both gums led to a decrease in consumption when subjects were permitted to smoke as inclined. The reduction in consumption was more for the active gum than for the placebo gum, which indicated an inhibitory effect of nicotine introduced from another source in smoking frequency. However, the major decline in consumption was observed when subjects tried not to smoke. At this experimental period, the difference between the active and the placebo gum was not found to be significant. The interesting finding of this study was that initial cigarette consumption, plasma nicotine and COHb levels and sex of the subjects were not found to be related to success in giving up smoking, which does not support the view that heavy smokers would benefit more from a pharmacological treatment method. However, the 2 mg gum was shown to produce much lower blood nicotine levels, so it can be argued that the pharmacological treatment offered was not an adequate substitute for the pharmacological reinforcement derived from cigarettes. The plasma nicotine levels were lower while taking the placebo-gum, which does not support a self-regulation of nicotine intake view, however in this study, being in a clinic and taking chewing-gums might in itself have motivated the subjects to change their smoking behaviour and thus decrease consumption.

Finally, a study by Schneider, et al (1977) who investigated the effects of nicotine chewing-gum on reductions in consumption level will be discussed because of its value in demonstrating the appropriateness of a single-case approach. They have demonstrated that a heavy smoker (50 cigs/day),

stopped smoking and was abstinent after seven months, with the aid of nocotine chewing-gums. He was given gums of different nicotine yields and also a placebo gum. It was noted that the subject could make distinctions between the active gums in terms of the duration of their effects and strength. He could also identify the placebo gum by its lack of effects. The subject gradually reduced the nicotine intake from the gums by switching to lower nicotine yield gums and finally discontinued the use of gums altogether. The demonstration of the efficacy of the nicotine-gum in helping a heavy smoker to abstain from cigarettes indicates the importance of the selection of an appropriate form of therapy for the individual smoker. However, a single case study might have involved more encouragement from the experimenter due to the one-to-one contact and it is necessary to replicate this study with a larger homogeneous sample.

In conclusion, the studies discussed above, suggest that the nicotine-chewing-gum is not an adequate substitute for cigarette smoking, mainly because of its slower rate of absorption. Thus, it does not offer a suitable method to investigate the nicotine-regulation hypothesis. However, some of the findings do suggest a differential role of the gum for heavy and light smokers and indicate that it might have therapeutic ubility for heavy smokers.

A novel approach was adopted by Stolerman, et al (1973), who used a centrally acting pharmacological antagonist of nicotine (mecamylamine), a peripherally acting antagonist of nicotine (pentolinium) and placebo capsules in order to examine the consequences of reducing the nicotine potency of cigarettes smoked. Mecamylamine, in comparison with the placebo capsules, led to a 30% increase in the number of cigarettes smoked during the experimental period. Puff-rate was also noted to be higher, which suggested that more nicotine was needed to counteract the effects of the antagonist. Although these findings are in line with the nicotine-regulation hypothesis, the compensation did not seem to be complete. The authors reported that hand

steadiness, which has been shown to deteriorate after cigarette smoking, was improved after the administration of nicotine antagonists. If smokers did have a fine interoceptive regulation mechanism, and achieved full compensation then the improvement in hand steadiness would not be expected. However, it is difficult to argue about the degree of compensation and nicotine regulation in a study in which the subjects had to perform various tasks (e.g; digit substitution and hand steadiness) which will restrict the number of cigarettes that could be smoked during the experimental period and thus the degree of compensation. So, it is desirable to replicate this study with an experimental period during which subjects are free or preferably in natural settings over a longer period of time.

Effects of Intravenous nicotine injections on the smoking behaviour

Intravenous nicotine injections overcome the problem of a confounding oral-substitute factor introduced by the "nicotine-chewing-gum". However, very few studies have used this manipulation and comparative measures of blood nicotine levels after intravenous injections and smoking have not been taken. However, Russell (1976-b), reported that some pilot work they have undertaken suggested that blood nicotine levels were lower after intravenous injections than after smoking.

The first study on nicotine injections was conducted by Johnston (1942). Nicotine was administered hypodermically and intravenously in doses ranging from gr. 1/50 to gr. 1/10 to 35 volunteers, some smokers and others non-smokers. It was reported that "smokers invariably thought the sensation pleasant and given an adequate dose, were disinclined to smoke for some time thereafter, whereas the non-smokers usually termed it "queer" ". Johnston also demonstrated that smokers were able to tolerate considerably higher doses of nicotine than non-smokers. Intravenous injections of 1/500 to 1/700 gr. induced sensations simulating those induced by inhalations of cigarette smoke. The author administered 80 doses of nicotine to himself,

and preferred the injections to cigarettes and experienced withdrawal symptoms when the drug was discontinued. This study is based only on subjective reports without any objective measurement and statistical analysis. The nicotine administered was neither blind nor controlled with placebo solutions. Nevertheless, it was the first attempt to examine the role of nicotine in smoking behaviour by using intravenous nicotine injections and there were no further reports on effects of intravenous nicotine injections till 1967. The second study was reported by Lucchesi, et al (1967). They measured number of cigarettes smoked and puff-frequency as compared to the subjective reports of Johnston, in a six hours experimental period. The number of cigarettes smoked, puff frequency and also the amount of each cigarette smoked decreased significantly when five smokers received a blind intravenous infusion of 2 to 4 mg of nicotine bitartrate per hour. Although the findings of this study showed that intravenous nicotine suppressed smoking behaviour and thus lent support to the nicotine-regulation hypothesis, it also indicated that the amount of decrease was small in relation to the dose of nicotine injected, so that nicotine regulation was not complete.

Russell (1976-b), stated that slow infusion of nicotine is not an efficient substitute for inhaled smoke. Kumar, et al (1977), in a well controlled investigation examined and compared the effects of various doses of inhaled tobacco smoke and comparable doses of rapid intravenous injections of nicotine on the puff-rate, puff-volume and some physiological variables (i.e; heart-rate; electroencephalogram; skin-conductance). They found that although inhaled tobacco smoke reduced subsequent puff-rate and volume in a dose related way, comparable intravenous injections failed to affect the smoking parameters. It was also reported that both intravenous and inhaled doses of nicotine produced very similar physiological effects, which suggested that rapid injections produced comparable nicotine levels both in the blood and in the brain. Thus, the findings of this study are not in agreement with the nicotine-regulation hypothesis and point out the need to investigate the role of nicotine further.

There has been very few studies with intravenous nicotine injections and the findings are not yet conclusive. However, in summary the studies demonstrating the inhibitory effect of nicotine injections do not support a full compensation, and the last study discussed above does not favour a nicotine-regulation view.

iii) Effects of manipulating urinary Ph levels on the smoking behaviour:

Goodman and Gilman (1958), stated that "when the urine is alkaline, only one fourth as much nicotine is excreted as when the urine is acid". This is explained by the fact that nicotine base is reabsorbed from an alkaline urine.

So, it appears reasonable that increasing the acidity of the urine can have substantial effects on plasma-nicotine levels. If the nicotine-regulation view holds, then this will in turn lead to an increase in consumption or a change in other parameters of smoking behaviour to compensate for the high excretion rate of nicotine.

Schachter, et al (1977), adopted the above approach to demonstrate regulation of nicotine levels in smokers. They have noted that acidification of urine, by administering vitamin C , increased cigarette consumption, and thus confirmed the nicotine regulation view. They have also noted a significant negative correlation between number of cigarettes smoked daily and urine alkalinity, which suggested that smokers with high consumption levels are compensating for the high nicotine excretion rate. Although, this finding needs to be further investigated, it implicates the role of nicotine and pharmacological reinforcement for all smokers and does not support the conception of dissimilar groups of smokers, with different motives for smoking.

This approach is potentially very useful in investigating the possible reasons for different consumption levels amongst smokers. It

could be possible that light smokers due to their physiological and chemical constitution do not require as much nicotine as heavy smokers to obtain similar pharmacological effects. However, the higher acidity observed in the urine samples of heavy smokers could well be a consequence of heavy smoking and this issue needs further investigation.

3.4 CONCLUSIONS

Although, cigarette smoking has been regarded as a form of nicotine dependence, the evidence for this is as yet slender and inconclusive. Even the studies favouring a "nicotine-titration effect" suggest that there is only partial regulation of nicotine intake amongst smokers. The assessment of the role of nicotine, including the dose of nicotine and its physiological and behavioural effects in homogeneous groups of smokers, classified according to reliable criteria seems to be more appropriate to clarify the conflicting findings from the various studies discussed in this chapter. Although nicotine is a powerful drug, its universal role as the predominant reinforcer maintaining the smoking behaviour of all smokers seems to be disputable.

3.5 CHAPTER SUMMARY

Experimental studies investigating the role of nicotine as the predominant reinforcer of smoking behaviour have attempted to provide evidence favouring a "nicotine-titration effect". This suggests that smokers will alter their smoking parameters in order to obtain a fairly consistent and individually characteristic level of nicotine intake.

Evidence for this has been sought by measuring changes in the smoking parameters in response to manipulations of nicotine yields of cigarettes, administration of nicotine by alternative routes and nicotine antagonists and finally altering nicotine excretion by manipulating urinary Ph levels.

The findings of the studies on the role of nicotine are conflicting and the evidence favouring the "nicotine-titration effect" is as yet inconclusive. Differential assessment of the role of nicotine for homogeneous groups of smokers seems to be more appropriate.

CHAPTER 4

PSYCHOPHYSIOLOGICAL AND BEHAVIOURAL EFFECTS OF CIGARETTE SMOKING

4.1 INTRODUCTION

The various conceptualizations of smoking motives and types indicates that smoking and/or nicotine may serve the function of manipulating subjective and/or physiological activation levels (e.g; stimulation, sedation, low-arousal and high-arousal smoking; see ch. 2). Research also indicates that nicotine is an important factor, at least for heavy smokers in determining the frequency and the parameters of smoking behaviour (see ch. 3). In this chapter the effects of smoking and/or nicotine on physiological, subjective and behavioural activation levels will be examined, in order to link the pharmacological effects of smoking with the reasons given by smokers for why they smoke. In other words, this chapter will aim at providing data on the pharmacological and behavioural bases of the changes in arousal levels (i.e; increasing or decreasing activation levels), reported by smokers.

Arousal, as used in this discussion refers to increases in physiological activation levels (i.e; cortical activation as manifested by the desynchronization of the electroencephologram, and increased sympathetic activity in the autonomic and peripheral nervous systems), subjective feeling of increased alertness as reported by smokers, and the behavioural manifestations of these physiological and subjective changes. Arousal can best be conceptualized as a continuum. Therefore, the effects of cigarette smoking and/or nicotine on activation levels and behavioural functioning may

be elucidated by examining changes in relevant activities from pre-to post-smoking and normal smoking day to deprivation day.

The pharmacological effects of nicotine depend on the dose per unit time, mode and route of administration and the physiological state of the organism at the time of administration. Larson, et al (1961), in their extensive review of the actions of nicotine have stated, "... so far as tobacco smoking is concerned much of what we already know of the neuropharmacological and other actions of nicotine is clearly irrelevant. The reasons behind this irrelevancy are, first, the quantitative matter of dosage, and secondly, the qualitative fact that nicotine is not the only, though it would seem to be indispensable factor, pharmacological or otherwise, involved in tobacco use", (pg.137). So, since the aim of the present chapter is to relate the pharmacological effects of cigarette smoking to motives for smoking the review will be limited to the effects of nicotine in man, in the doses inhaled in tobacco smoke or in equivalent doses of nicotine injections.

Before discussing the effects of smoking on specific physiological and behavioural measures, a brief account of the pharmacological effects of nicotine will be presented. Several authors have provided detailed reviews of the area (Comroe, 1960; Ginzel,1967; Murphree,1967; Jarvik,1970; Russell, 1976-b; Stephens,1977).

The predominant effect of nicotine in the whole intact animal or human has been reported to be sympathomimetic (Jarvik,1970). However, the actions of nicotine both centrally and peripherally are multitudinous and somewhat paradoxical. The pharmacological effects of nicotine are a complex function of dose, route and mode of administration. In addition to this, nicotine has a biphasic action at cholinoceptive sites. It first acts as a

cholinergic agonist and then as a blocker of acethycholine (Russell, 1976-b).

In low concentrations nicotine can stimulate the sympathetic ganglion cells, whether they are in the paravertebral chain or not and also the parasympathetic ganglion cells. Since, it can stimulate both of these opposing systems, it is important to know which of them is stimulated by the doses of nicotine involved in cigarette smoking.

By its stimulant actions on the sympathetic ganglion cells it can produce all the effects of sympathetic stimulation on the heart, blood vessels, eye, gastrointestinal tract, bronchioles and bladder. It produces an increase in metabolic rate and blood unesterified fatty acids (Comroe,1960).

Adrenergic effects are also produced by the liberation of adrenaline from the adrenal medulla and noradrenaline from sympathetic nerve endings, which in turn play an important role in the peripheral cardiovascular, respiratory and renal effects observed with nicotine and also during smoking (Jarvik,1970). All these effects have been noted to be abolished by the administration of large doses of hexamethonium or nicotine itself (Comroe,1960).

Nicotine also stimulates the chemoreceptors of the carotid and aortic bodies, which detect the increases in O_2 and CO_2 and pH levels in arterial blood and signals the respiratory and vasomotor centers to take appropriate action. Typical effects of nicotine on chemoreceptors are an increase in rate and depth of breathing, and circulatory actions typical of sympathetic stimulation such as cardiac acceleration, increase in blood pressure and

vasoconstriction in skin (Comroe, 1960; Ginzel, 1967). It has been suggested that in its smallest effective doses, nicotine does not stimulate the sympathetic ganglion cells directly but carries its action via chemoreceptors. Comroe (1960), pointed out that after the denervation of the carotid and aortic bodies, the increase in respiration rate and blood pressure following one mg/kg nicotine administration in dogs or cats could no longer be obtained.

Nicotine has been noted to produce central excitation, evidenced by the activation of electroencephalogram. In high doses it produces convulsions (Jarvik,1970). The central stimulant actions of nicotine can be antagonized by nicotinic ganglionic blocking drugs, like mecamylamine. However, actions of nicotine on the brain are complex. It causes a release of ACh from the cortex. Since the ACh is not released when nicotine is directly applied to the cortex, this effect has been suggested to be secondary to the actions of nicotine on other areas. Compared to the EEG arousal, ACh release has been noted to be more prolonged, suggesting that the effects may be mediated by different processes (Russell, 1976-b).

Domino (1973), reported that nicotine injections (20 µg/kg) produces a marked increase in spontaneous Renshaw cell (i.e; inhibitory interneurons in the spinal cord) activity. The author examined the effects of cigarette smoking on the patellar reflex and found that smoking depresses the patellar reflex. The depression was noted to be dose related, and was also produced by inhaling nicotine from an aerosol. No significant change was noted after smoking lettuce-leaf nicotine free cigarettes. The reflex response was noted to return toward the control levels within 25 minutes of the end of smoking. An increase in heart rate was also noted after the inhalation of nicotine aerosol, however HR values returned to control levels

earlier than the patellar reflex. This result indicated that the time-course of effects of nicotine are not the same in different systems.

It can be seen from this brief account of the neuropharmacological effects of nicotine that dose is very important in determining the response. Although, the general effect of smoking doses of nicotine seems to be sympathomimetic, it can also produce a relaxing effect on skeletal muscle tone.

As will be seen from the following review, the findings of the majority of studies indicate that cigarette smoking and nicotine administered *via* alternative routes increases physiological activation levels, as indicated by the desynchronization of the EEG, increase in pulse rate and blood pressure, increase in urinary catecholamine levels, etc. Conversely, cigarette deprivation produces physiological withdrawal symptoms, mainly indicative of lowered physiological activation levels (i.e; drop in pulse rate and blood pressure, synchronization of EEG, etc)(Ulett & Itil,1969; Myrsten *et al*, 1977). Other smoking related factors, such as deep breathing and sham smoking were not noted to produce significant changes in the majority of the measures examined (Ashton *et al* ,1973; Agué,1974), which indicates that nicotine is the factor responsible for the physiological and behavioural effects.

The effects of smoking and deprivation briefly outlined above, indicate that smoking increases physiological activation levels, and thus are in line with the reports of smokers who state that they smoke in low-arousal situations in order to stimulate themselves. On the other hand, some studies (i.e; on contingent negative variation and patellar reflex), have shown that smoking may also produce sedative effects. However, at present the evidence for the sedative effects of smoking is meagre, and it is difficult to explain why some smokers find smoking calming (i.e; sedative smoking).

4.2 LITERATURE REVIEW

Table 4.1 (overleaf), provides a summary of the effects of smoking, nicotine administrations, cigarette deprivation and other smoking related factors on the measures reviewed in this chapter.

i) CORTICAL ACTIVITY

Silvette et al (1962), have stated that small doses of nicotine are used in tobacco smoking for the primary purpose of central nervous system (CNS), stimulation. In order, to assess the central effects of smoking, EEG recordings have been taken by various investigators before, during and after smoking.

It has been found that smoking a cigarette after a 2 hours deprivation period produces a significant decrease in the abundance of alpha wave activity (1), which lasts for approximately 20 minutes after smoking (Phillips, 1971). An increase in Beta activity (2), during and after smoking has also been reported (Kumar et al ,1978). Kumar et al (1978), noted a significant increase in Beta activity of the EEG, following intravenous nicotine injections. In line with this, deprivation from cigarettes for 24 hours

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(1) Alpha Activity: Smooth electrical activity of 8-13 Hz, recorded from the brain. Alpha activity is generally associated with a state of relaxation (Carlson,1977).

(2) Beta Activity: Irregular electrical activity of 13-30 Hz, recorded from the brain. Beta activity is generally associated with a state of arousal (Carlson,1977).

MEASURES	SMOKING	NIC-ADMIN.	DEPRIVATION	OTHER SMOKING RELATED FACT·
Cortical Activity				
a-EEG	Desynchronization Increase in Beta activ. (1;2)	Same as smoking (2)	Synchronization Inc. in Alpha activ. (3)	——
b-CNV	Inc. or Decr. in CNV magnitude (4)	Same as smoking	——	No significant change with sham smoking (4)

(continued)

Table 4.1 (Continued)

MEASURES	*SMOKING*	*NIC-ADMIN*	*DEPRIVATION*	OTHER SMOKING RELATED FACT
Autonomic (Peripheral)				
a-Heart Rate	Increase, (dose related more after a period of deprivation) (5;6;7;8)	Same as smoking (2;10;11)	Decrease related to length of depr. (8;9)	No change in HR in natural life cond. (12) No significant change with sham smoking & nic-free cigs. (6;7;10;20)
b-Blood Press.	Increase (13;14)	———	Decrease (8)	No change with sham smoking (10)
c-Skin Cond.	Increase, not dose related. (2;7;15)	Nic. aerosol leads to inc. (15)	———	———
d-Skin Temp.	Decrease (5;7;14;16;18)	Same as smoking (17)	Increase (19)	Sham smoking, deep breathing inspiration, etc. also produces changes (17)
e-Catecholamine excretion	Increase (14;19)	———	Decrease (19)	Stress of exp. task also effects adrenaline excretion (9 & 20)

(Continued)

Table 4.1 (Continued)

MEASURES	*SMOKING*	*NIC-ADMIN.*	*DEPRIVATION*	*OTHER SMOKING RELATED FACTORS*
Behavioural & Psychophysical				
a-CFFT	Increase or decrease (22;23)	Increase (nic. tablets) (23)	Decrease or Increase (23)	———
b-PVF	Decrease (24)	———	Increase (9;24;25)	No change with denicotinized cigs. (25)
c-Hand Stead.	Deterioration (14;26)	———	Improvement (19;26)	Effect noted only for S's who inhaled (26)
d-Vigilance	Prevents perf. decrement/time. (27;28;29;30)	Nic. tablets have same effect as smoking (27)	Perf. decrement/ time (27;28;29)	Sham smoking & placebo tablets do not prevent decrement in perf. (27)

Table 4.1 Effects of smoking and/or nicotine on cortical activation, autonomic (peripheral) functioning and behavioural efficiency.

(1-Phillips, 1971; 2-Kumar et al (1978); 3-Ulett & Itil (1974); 4-Ashton et al (1973; 1978); 5-Larson et al (1961); 6-Elliott & Thysell (1968; 7-Ague (1974); 8-Knapp et al (1963); 9- Myrsten et al (1972); 10-Irwing & Yamamoto (1963); 11-Herxheimer et al (1967); 12-Erwin (1971); 13-Thomas et al (1956); 14-Frankenhauser et al (1968); 15-Ague & Frith (1969); 16-Stephens (1977); 17-Burch & DePasquale (1961); 18-Gershon-Cohen et al (1969); 19-Myrsten et al (1977); 20-Andersson & Post (1974); 21-Fabricant & Rose (1951); 22-Garner et al (1954); 23-Warwich & Eysenck (1963); 24-Johnston (1965); 25-Krippner (1970); 26-Edwards (1948); 27-Wesnes & Warburton (1978); 28-Tong et al (1971); 29-Tarriere & Hartemann (1964); 30-Heimstra et al (1967)

was found to increase the abundance of alpha wave activity (Ulett & Itil,1969). Ulett and Itil (1969), reported that the change in the EEG was accompanied by behavioural symptoms such as drowsiness, restlessness and dysphoria. Although, some of the behavioural symptoms seem to be contradictory with each other (e.g; drowsiness and restlessness), a significant decrease in HR, which supports the EEG findings, was noted. The EEG pattern and the behavioural symptoms were reversed (i.e; return to pre-deprivation measures) by smoking a cigarette. Knott and Venables (1977), compared the EEG recordings of deprived (DS), and non-deprived (NDS) smokers, before and after smoking, and non-smokers (NS). They noted that in the pre-smoking period DS had significantly slower dominant alpha frequencies as compared to NDS and NS. No significant difference was noted between the NDS and NS. Post-smoking measures showed that there was no longer a difference in dominant alpha frequency between the three groups. The authors concluded that smoking has a normalizing effect on cortical activity.

The findings of the EEG studies discussed so far reveal a clear picture on the effects of smoking. It has been shown that smoking and intraveneous nicotine injections produce an increase in beta activity, whereas deprivation increases the alpha activity. So, these studies consistently indicate that smoking and nicotine has an activating effect on CNS functioning and support the stimulating effect of smoking and/or nicotine.

However, investigations using contingent negative variation (CNV)[3], to assess the state of the CNS, have revealed a more complicated picture for the effects of smoking on cortical activity.

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(3) Contingent Negative Variation (CNV) : is recorded between electrodes on the vertex and mastoid or ear lobe and consists of a small negative potential which builds up between a warning signal and an imperative signal

..... Cont.

Ashton et al (1978), found that a centrally stimulant drug (i.e; caffeine citrate, 300 mg), produced a significant increase in CNV magnitude whereas, a centrally depressant drug (i.e; nitrazepam, 2.5 mg), led to a significant decrease in CNV magnitude. Based on these findings it was hypothesized that if nicotine has a stimulant effect on the CNS then an increase in CNV magnitude, similar to that found with caffeine citrate would be expected. The authors found that in 22 smokers tested, smoking either increased or decreased the CNV magnitude.

Intraveneous nicotine injections were also found to either increase or to decrease the magnitude of the CNV. However, no significant change in CNV magnitude was noted following sham smoking (Ashton et al, 1973). The authors interpreted the dual changes in CNV magnitude as reflecting the stimulant and depressant effects of nicotine. They have shown an interaction between personality (Introversion/Extraversion- Eysenck Personality Inventory), nicotine intake and change in the direction of CNV. This interaction suggested that the rate of nicotine intake in extraverted smokers was slower and it was associated with a stimulant effect, while in introverts the rate of nicotine intake was faster and it was associated with a depressant effect, in terms of changes in CNV magnitude. Ashton et al (1973), also noted that when intraveneous injections, in doses similar to what was obtained by smoking was injected to the same subjects, the changes in CNV magnitude was in the

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(3)-Cont.

requiring the subject to carry out some response, usually a motor response. The CNV thus occurs in an expectancy situation and is sometimes referred to as an expectancy wave. The magnitude of CNV is thought to reflect the degree of activity in the ascending reticular activating system and limbic system as well as in the cortex, and is related to the degree of alertness of the subject (Ashton et al ,1978).

same direction as occurred after smoking.

In summary, the findings of the above authors suggest that the effects of nicotine on the CNS is dependent on the dose of nicotine and/or the personality (or the state of the individual at the time of smoking) of the smoker. The findings seem to suggest that individuals adjust the dose in order to obtain either a stimulant or a sedative effect. Further support for the sedative and stimulant effects of nicotine came from a thesis put forward by Nelson (1978), derived from animal studies. It was hypothesized that arousal is mediated by the limbic system(4), and the reticular formation(5) (RF), and that these two centers are mutually inhibitory. Nelson et al (1975), investigated the effects of nicotine as an antagonist of the behavioural disruptions resulting from stimulation of the RF of the rat by chronically implanted electrodes. They found that the electrical stimulation of the RF resulted in a marked disruption on a learned visual retention task. Systemically injected nicotine partially blocked the disruptive influence of the RF stimulation upon the task performance. The authors interpreted this finding as an increased level of limbic system activation which counteracts the increased RF activation. The authors suggested that these findings may be extended to human smoking behaviour and could be used to explain the calming effects of smoking reported by smokers. Nelson et al (1975), stated, " It is

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(4) A group of brain regions including the anterior thalamus, amygdala, hypothalamus, as well as their interconnecting fiber bundles (Carlson,1977). Dunn (1978), suggested that the limbic system allows for more selectively motivated or goal-directed behaviours.

(5) A large network of neural tissue located in central regions of the brain stem, from the medulla to the diencephalon, generally taken to be the center for generalized activation or arousal (Dunn,1978).

not inconceivable that one of the motivations underlying smoking behaviour is the desire to reduce an RF activation level which is manifested in an hyperstimulated or anxious state inappropriate for effective behaviour and to engender what might be considered a state of useful behavioural arousal".

Support for these authors' view is provided by studies in frustrating long tasks, where it is generally observed that although subjective reports of anger and frustration are increased in all experimental groups, smokers allowed to smoke sustain their performance at a constant level, whereas deprived smokers and non-smokers show a decrement in performance over time (Heimstra, 1973; Jones and Lieser, 1976, cf. Dunn,1978). These studies indicate that smoking may have an advantageous effect in situations inducing non-specific affective arousal (e.g; anger, fear, frustration).

Although, more research is needed to elucidate the sedative qualities of smoking, the thesis advanced by Nelson seems very attractive in suggesting a dual effect of smoking as a result of an interaction between nicotine intake and the state of the smoker.

So, in summary although the EEG studies support the view that smoking in laborotory conditions (usually under resting conditions), has stimulating effects on cortical activity, research utilizing CNV and the work of Nelson et al points out that the actions of nicotine and/or smoking are determined by an interaction of factors (i.e; dose of nicotine and state of the individual and the characteristics of the task). The latter findings throw some light on the reasons why smokers state that smoking helps them concentrate and that they feel calmer after smoking. A distinction must be made between everyday smoking, and smoking after a period of deprivation in a laborotory resting condition. Although, smoking may have a stimulating effect on cortical

activation levels under resting conditions, in everyday smoking, as suggested by the self-reports of smokers it may be inhibiting activity in certain systems of the brain and thus produce what is conceived of as a sedative effect which helps to overcome the distracting effects of overexcitement and thus help concentration under high arousal situations. However, more research is needed to explain the dual (i.e; stimulative and sedative) effects of cigarette smoking.

ii) AUTONOMIC (PERIPHERAL) ACTIVITY

a) PULSE RATE

Russell (1976-b), has summarized the cardiovascular effects of smoking doses of nicotine as predominantly stimulant, whether administered by injections, aerosol or by cigarette smoking. He commented, " It is not clear how much the cardiovascular effects of smoking are due to the stimulation of the sympathetic nervous system, to the stimulation of chemoreceptors of carotid and aortic bodies, or to the output of adrenaline from the adrenal medulla ". As has been pointed out earlier, the dose of nicotine inhaled is an important factor in determining which system is effected directly. However, it seems that the interaction of the above listed systems is producing a stimulant effect on the cardiovascular system.

Larson et al (1961), in their extensive review of smoking literature have reported 95 publications from 1863 to 1959, in which pulse rate increase was noted following smoking. This effect was observed consistently, after smoking cigarettes, cigars and pipes upon non-smokers as well as habitual smokers, and ranged between a few to over 50 bpm. However, the same authors also listed 12 publications reporting no change in pulse rate, and 11 publications noting a decrease in heart rate (HR) following smoking. As will be seen from the following review, the effects of smoking on HR seems to depend

on the conditions prior to testing (i.e; after abstinence or normal smoking), and thus the basal HR levels.

HR after smoking has been noted to be higher than pre-smoking values. Furthermore, the increase in HR has been reported to be more pronounced after a period of deprivation than after regular smoking. This may be related to the lower base-line levels during deprivation.

Elliott and Thysell (1968), compared the effects of smoking on HR, in two groups of smokers, one of which abstained from smoking for three hours, and the other smoked as usual before the experiment. They reported that smoking after abstinence produced a more marked increase in HR (20 bpm). The time course for the dissipation of effects of smoking was noted to be slower after regular smoking before the experiment than following smoking after a few hours deprivation. This was interpreted by the authors as a cumulative effect of smoking.

Irwing and Yamamoto (1963), reported an increase in the cardiac output (i.e; quantity of blood ejected per minute by the heart), ranging up to 200 per cent over control values during the inhalation of tobacco smoke, whether from cigarettes or a pipe and also following intraveneous nicotine injections. The cardiac output was noted to return to control values in 10-15 minutes after the termination of smoking. Other investigators have reported values ranging from 20 minutes to one-hour for the pulse rate to return to pre-smoking values (Armitage, 1978; Agué, 1974). Herxheimer et al (1967), showed that the inhalation of nicotine aerosol also leads to an increase in HR.

Knapp et al (1963), reported a lenear relationship between HR in-

creases and the nicotine yields of cigarettes smoked. Armitage (1978), provided evidence to support the relationship between plasma nicotine concentrations and increase in HR. Peak levels of arterial blood nicotine concentrations (40 ng/ml), were found to coincide with maximum increases in HR. After 20 minutes of smoking the arterial blood concentration was found to be 10 ng/ml.

Cigarette deprivation has been noted to produce a drop in pulse rate. Knapp et al (1963), found that deprivation produced a decrease in pulse rate. Pulse rates for a deprived group were significantly lower than a non-deprived group of smokers. Similarly, Myrsten et al (1972), found that HR was significantly higher in the smoking condition, at all points during the experimental period as compared to a non-smoking condition.

So, the findings discussed above suggest that smoking and/or nicotine injections increase and deprivation produces a decrease in HR. Although, the studies using nicotine injections and nicotine aerosol suggest that nicotine is the factor in cigarette smoking responsible for the changes in HR, some authors have investigated the effects of other smoking related factors (i.e; deep breathing, nicotine-free cigarettes, etc.) on HR.

Nicotine-free cigarettes (Andersson & Post, 1974; Agué ,1974), sham smoking, deep breathing and smoking without inhalation (Elliott & Thysell, 1968; Irwing & Yamamoto, 1963) were not found to produce significant changes in HR.

Although, the experimental findings strongly suggest that smoking has a stimulant effect on HR, and nicotine delivered *via* smoking is the major factor in producing this effect, a further point needs to be considered. The common experimental procedure across studies investigating the effects of smoking on HR is that pre-smoking values, taken under laborotory resting conditions, some after a period of abstinence, have been compared with the post-smoking values. Thus, the consistent higher HR noted by investigators is based on comparisons of post-smoking values with very low, resting HR levels.

A valuable departure from the above methodology has been made by Erwin (1971), who examined HR alterations during spontaneous cigarette smoking with an EKG radiotelemeter. Subjects were ambulant and both the initiation and rate of smoking were voluntary. No significant changes were noted in HR before, during or after cigarette smoking. Although, this study needs to be replicated in other samples, it is very valuable in its approach (i.e; analyzing HR changes during spontaneous smoking in ambulant subjects). It points out the unsufficiency of laboratory research in elucidating the conditions prior to and following voluntary smoking in natural settings.

b) BLOOD PRESSURE (BP)

Changes in BP following smoking have usually been reported to be less marked than the changes in HR. Thomas *et al* (1956), reported a mean systolic blood pressure (SBP), increase of 2.9 mg Hg and mean diastolic blood pressure (DPB), increase of 4.6 mg Hg, in 113 smokers who smoked one standard cigarette. No further changes were observed after smoking a second

cigarette. Similarly, Frankenhaeuser et al (1968), noted increases in SBP following smoking. Sham smoking and smoking without inhaling was not found to produce significant changes in BP (Irwing & Yamamoto, 1963).

On the other hand, Knapp et al (1963), found that DPB was lower in 15 heavy smokers during abstinence as compared to values during normal smoking.

Although, considerable variability between subjects has been noted with blood pressure changes, the consistent effect seems to be an increase (Stephens ,1977).

c) SKIN CONDUCTANCE (SC)

Activity of the eccrine sweat glands, which are found to be densest (6) on the palm of the hand and sole of the foot, have been found to be of major importance in the production of electrodermal phenomena. Innervation of eccrine glands is solely via the sympathetic branch of the autonomic nervous system, however, the postganglionic synapse is cholinergic, having acethycholine, rather than the usual sympathetic neurotransmitter nor-adrenaline (Venables & Christie, 1979). Thomas and Korr (1957), obtained a linear relationship between the number of active sweat glands per unit area and skin conductance levels (SCL). The use of SC, rather than its reciprocal skin resistance, has been recommended by Lykken and Venables (1971), who maintained, " SC bears a simpler and more linear relationship to the underlying processes of psychological interest than

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(6) - 2000/cm^2 on palm and sole of foot (Weiner and Hellman (1960)).

does its reciprocal skin resistance " [7](pg. 659).

Although, from the stimulant actions of nicotine on the sympathetic ganglia, an increase in SCL following smoking may be predicted, it has been suggested that SC is not a good indicator of drug action. However, the background sweat-gland activity can provide useful information on "general activation " levels (Lader & Wing, 1966; cf. Agué, 1974).

The effects of smoking on SCL has not been widely investigated. Frith and Agué (1969), noted increases in SCL lasting for about 30 minutes after smoking a standard tobacco cigarette and also after the administration of nicotine aerosol. Agué (1974), reported a marked increase (0.9 log micromhos) in SCL above base-line values in 24 habitual smokers immediately after smoking (cigarettes in 3 different doses). The increase in SCL persisted throughout the 60 minutes experimental period with a slight tendency to diminish at the end of the period. No significant difference was

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(7)

It has been suggested that the skin consists of multiple parallel resistances which can individually change in value (Tregear, 1966). " Resistance in a parallel circuit is a complex function of the individual resistances, and the change produced in one branch depends upon the resistances of all the other branches. In contrast, the conductance of a parallel circuit is a simple sum of the conductances in parallel and a change in one of these produces simply an equivalent change in the total, independently of the values of others. In addition with conductance, the problem of the dependence of phasic response upon the tonic level is simplified " (Lykken & Venables, 1971; pg.658).

noted between different doses. However, it was observed that the high nicotine cigarettes(1.02 and 2.11 mg nic), had opposed effects during the morning and afternoon sessions. In the afternoon they caused a sharp increase in conductance, whereas, in the morning they produced SC values close to pre-smoking levels. The difference between morning and afternoon SC levels were found to be significant. Rate of smoking (slow or fast frequency of puffing), were also found to be related to the changes in mean conductance levels throughout the experimental period, with fast rate of smoking producing a significantly higher SCL as compared to the slow rate of smoking. The results of this experiment suggest that the effects of smoking on SCL depends on the rate of administration, time of day and initial levels of " alertness " of the subject. However, the relationship between the rate of puffing and change in SCL, may also be related to different respiratory patterns.

Kumar et al (1978), also failed to find any dose-related effects of cigarette smoking (0 - 2.6 mg nic) and intravenous nicotine injections (0 - 0.07 mg/kg) on the SCL of 12 smokers.

In conclusion, changes in skin conductance levels following smoking do not seem to be related to different doses of nicotine. SCL might prove to be a useful measure of the effects of smoking on general activation levels, rather than reflecting the specific effects of nicotine dosage delivered to the smoker. Further research, investigating the factors in smoking behaviour contributing to the changes observed in SCL needs to be undertaken.

d) SKIN TEMPERATURE (ST)

Vasoconstriction in fingers and toes, producing a decrease in skin temperature, occurring both in healthy smokers and subjects with peripheral vascular disease after smoking one or more cigarettes have been mentioned in the literature (Frankenhaeuser et al ,1968; Agué ,1974; Stephens, 1977). However, there is considerable disagreement regarding its mechanisms. It has been attributed to the effects of nicotine on the chemoreceptors of aortic and carotid bodies, to the stimulant actions of nicotine on the sympathetic ganglia or to the release of adrenaline from the adrenal medulla (Comroe, 1960).

Several investigators have shown that decreases in ST following smoking were only partially related to the nicotine content of cigarettes. Burch and DePasquale (1961), have noted that offering a subject a cigarette, simply lighting a cigarette and sham smoking also produced decreases in digital blood flow in the finger. Rapid intravenous infusion of nicotine bitartrate was also found to produce a decrease in digital blood flow in fingers. Similarly, Agué (1973), reported mean ST decreases of 3.5° C below base levels with 2.11 and 1.02 mg nic. cigarettes. On the other hand, lettuce-leaf cigarettes produced a decrease of 2.0° C, which returned to base-line levels within 10 minutes, whereas for the nicotine cigarettes the ST was noted to be still 1° C below the base-line levels one hour after smoking.

Larson et al (1961), reported that a large number of investigators have observed a decrease in skin temperature following smoking ranging from 0° C to 4° C for finger and 0° C to 2.8° C for the toe.

Gershon-Cohen et al (1969), took thermographic scans of hands, forearms and feet after smoking and noted decreases in ST ranging from 0.5°C to 3.0°C. In line with this, abstinence from smoking (5 days), was found to produce an increase in the finger temperature (Myrsten et al,1977).

These results point out that smoking causes decreases in the peripheral blood flow, and thus lowers the temperature of the extremities (i.e; fingers and toes). Although, these changes were found to be related to the nicotine content of the cigarettes, sham smoking, deep inspiration etc, were also noted to produce changes in ST in the same direction. So, the total act of smoking (i.e; nicotine intake, motor responses involved) seems to be contributing to the observed changes.

e) CATECHOLAMINE EXCRETION

Nicotine can cause the adrenal medulla (8) to liberate catecholamines (i.e; adrenaline and noradrenaline)(Comroe, 1960). Russell (1976-b), pointed out that in situations of stress adrenaline is released from the adrenal medulla into the blood stream, and that its stimulant effects help to overcome the stress. Thus, smokers may smoke in stressful situations for this effect.

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(8)- "The adrenal medulla, closely resembles a sympathetic ganglion. It is innervated by preganglionic fibers and its secretory cells are analoguous of post-ganglionic sympathetic neurons. These cells secrete adrenaline and a little noradrenaline when they are stimulated. Secretions of the adrenal medulla function chiefly as an adjunct to the direct neural effects of sympathetic activity; for example, adrenaline increases heart rate and constricts peripheral blood vessels. This gland also stimulates a function that cannot be mediated neurally- an increase in the conversion of glycogen into glucose within the skeletal muscle cells" (Carlson,1977)

The excretion rates of adrenaline and noradrenaline in the urine were found to be higher after smoking, and lower after a period of cigarette deprivation. Although, in the same direction the changes in noradrenaline levels are usually smaller than for adrenaline levels (Elgerot,1975).

Myrsten et al (1977), reported that after 5 days of abstention adrenaline excretion was significantly lower than base-line measures, and there was an increase in urine adrenaline levels after smoking. Noradrenaline values were found to exhibit similar but less regular trends. Frankenhauser et al (1968), noted a positive relationship between adrenaline excretion and number of cigarettes smoked.

However, some investigators found that the increase in adrenaline excretion is not totally dependent upon smoking, especially in experiments in which subjects are confronted with demanding performance tasks. Andersson and Post (1974), reported that there were no differences in the urinary adrenaline levels of subjects smoking nicotine or nicotine-free cigarettes while performing a verbal rote learning task. An increase in adrenaline levels was noted for both groups. Similarly, Myrsten et al (1972), noted that both the smoking (4 cigarettes) and the no-smoking conditions produced an increase in urinary adrenaline levels, in a two hr experiment in which subjects were required to perform simple- and choice-reaction time tasks. The authors have concluded that the experimental situation was in itself sufficiently stressful to bring about an increase in adrenaline output and under these circumstances smoking did not produce a further increase in adrenaline excretion. In this study, it was noted that smoking increased the performance efficiency in the simple-reaction time task and it also increased HR and SBP. So, based on these results the excretion rate of

adrenaline seems to depend on the nature of the task. If the experimental task is too demanding, then smoking does not seem to produce a further increase in adrenaline levels over those produced by the task itself.

In conclusion, although the rate of adrenaline excretion is related to smoking, the demands imposed by the experimental situation also seem to play an important role in determining the levels of adrenaline in urine.

iii) BEHAVIOURAL AND PSYCHOPHYSICAL MEASURES

There have been numereous studies investigating the relationship between smoking and behavioural efficiency (e.g; vigilance, learning and retention, sensory thresholds : critical flicker fusion, peripheral visual field, taste, etc, motor tasks: hand steadiness, etc.). This section will be limited to the effects of smoking on some sensory threshold measures, vigilance and hand steadiness tasks, which have been used in the experiments of this thesis. These measures have been chosen because of their relationship to the physiological changes associated with cigarette smoking (i.e; changes in activation levels).

a) CRITICAL FLICKER FUSION THRESHOLD (CFFT)

The fusion frequency of flicker [9] has been defined as the highest number of impulses the retino-cortical system can perceive in a unit time. Flicker fusion thresholds have been used as an index of the functional state of the retino-cortical system (i.e; sensitivity), and by inference, of the functional state of the CNS as a whole (Larson et al ,1950).

Knott (1978), suggested that, " the important factor relating to efficient flicker fusion performance is the 'rate or speed' at which the 'noise' generated by the first stimulus is dissipated by the CNS, i.e; the initial stimulus input creates noise which carries over to the second stimulus and makes the detection of the second input more difficult "(pg.121).

It has been reported that decrease in fusion frequency of flicker parallels development of subjective fatique (Simonson et al, 1941), and that fusion frequency of flicker varies directly with the oxygen supply to the visual pathway (i.e; the more O_2 supply, the higher the fusion frequency) (Simonson et al, 1943, cf. Larson et al, 1950).

Fabricant and Rose (1951), explained the increases in CFFT commonly observed after smoking and the relationship between CFFT and oxygen supply, by the increased velocity of blood [10] flowing through the cerebral vessels, which brings more oxygen to the neural tissues.

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(9)- " Frequency is measured in cycles per second. At low frequency the '0' perceives a series of flashes of light; as the frequency is gradually increased the impression changes successively to coarse flicker and fine flicker, and perfectly steady light. The frequency at which all flicker disappears is called the fusion frequency. The higher the fusion frequency, the more efficient is the operation of the light-registrating mechanism, the better its resolving power in time" (Woodworth & Scholosberg, 1966)

(10)- " Smoking causes a peripheral vasoconstriction with a rise in blood pressure. Thus, the total amount of blood is increased in terms of speed rather than in terms of enlargement of the total capacity of the vascular bed" (Fabricant & Rose, 1951).

The results of experiments investigating the changes in CFFT following smoking are at present inconclusive. Although, some investigators noted an increase in CFFT following smoking (Larson et al ,1950; Fabricant and Rose, 1951), some others have failed to note any consistent change in CFFT related to smoking (Garner et al, 1954).

Garner et al (1954), reported that in smokers who abstained from smoking for ten hours, and non-smokers, smoking did not produce a consistent change in CFFT, some subjects having an increase, some a decrease and some no change in CFFT.

Warwick and Eysenck (1963), investigated the effects of smoking one cigarette and nicotine tablets (0.1 mg nic. or placebo tablets), on the CFF thresholds of smokers and non-smokers. They found that CFF thresholds were raised both after smoking and after the administration of nicotine tablets. However, no change was noted for smokers who had not abstained before the experiment and for non-smokers after smoking (11). The increase in CFF was noted to last for 15 minutes after smoking.

So, although there is evidence to suggest that nicotine delivered by smoking and nicotine tablets raises the CFF thresholds, the evidence as yet is inconclusive, and further research investigating the effects of other

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(11)- "..presumably because the non-smokers did not inhale" (Warwick and Eysenck, 1963, pg.225). However, the validity of this statement needs to be confirmed by blood or butt-nicotine analysis.

smoking related factors (i.e; sham smoking, deep breathing, nicotine-free cigarettes), on CFF thresholds needs to be undertaken. It also needs to be investigated whether smoking, depending on the dose of nicotine inhaled or individual characteristics, have a dual effect (i.e; increase or decrease) on CFFT, similar to the one noted with contingent negative variation.

b) PERIPHERAL VISUAL FIELD (PVF)

Although, the mechanisms are not clear, smoking has been found to decrease the size of the peripheral visual field (Johnston,1965; Krippner, 1970).

Johnston (1965), reported a significant increase in the PVF after a two-weeks abstinence period in eight smokers. However, there was a considerable variation between subjects (increases ranging from 16 to 85 %). Krippner (1970), with a larger sample (N=40), also found an increase in the PVF, especially for the temporal (i.e;0°)meridian, after eight hours of abstinence. He also noted that there was no difference in the PVF measurements of subjects who abstained during the experiment and those who smoked denicotinized cigarettes, which indicated that the effects of smoking on the peripheral vision may be attributed to the nicotine content of cigarettes.

Research on the effects of smoking on the peripheral visual field is slender, and although the findings of the present studies indicate that smoking has a deleterious effect on the PVF, the mechanisms of this effect are not deliniated.

c) VIGILANCE TASKS

The maintenance of attention over relatively long periods of time has been conceptualized as vigilance, defined as the readiness to detect and respond to certain specific changes occurring at random time intervals in the environment (Mackworth, 1957). In vigilance tasks it is generally observed that as the time spent performing the task increases, the probability that the observer will correctly detect the experimental signal decreases (referred to as performance decrement).

Although, performance decrement over time has been a common finding, the rate of decrement in performance efficiency has been found to be related to the type of task used, stimulus conditions and some organismic states. Although, measures of physiological activity taken under resting conditions were generally not found to be correlated with vigilance performance, the latency of the galvanic skin response (GSR), to a novel stimulus was found to be positively correlated with performance decrement (i.e; the shorter the latency of GSR the less the decrement in performance over time) (Coles & Gale, 1971).

On the other hand, changes in physiological measures in the direction of a lowered level of activation have been noted with time on task in vigilance tests. Decline in EEG alpha output (cf. Coles & Gale, 1971), skin conductance levels (Stern, 1966), and the concentration of adrenaline in blood (O'Hanlon, 1965), have been reported. O'Hanlon (1965), noted a significant positive correlation between performance decrement in a 3-hours visual vigilance task and decrement in blood adrenaline levels. Bakan (1963), reported that at the end of a 48 minutes auditory vigilance test period the retrospective reports given by the subjects indicated that

the task had a soporific effect for most of the subjects. The subjective reports of drowsiness and lapses in attention were found to be related to the number of signal detections on the vigilance task.

The findings cited above indicate a relationship between physiological and subjective arousal levels and vigilance test performance. Warburton (1978), (cf. Wesnes & Warburton,1978), proposed that lower levels of cortical arousal resulted in impaired information processing, so that as a person's cortical activity becomes more synchronized (i.e; higher amplitude, lower frequency electroencephalographic activity), he attends less to external stimuli.

It has been noted earlier that smoking gererally leads to the desynchronization (i.e; irregular electrical activity, which represents the random spontaneous activity of the cells at the cortex and is generally associated with a state of arousal; Carlson,1977), of the EEG, although the effects of smoking on cortical activation depends on the dose of nicotine delivered and the initial level of arousal (Armitage et al, 1968). So, it can be hypothesized that smoking may enhance performance efficiency in vigilance tasks by preventing performance decrement over time.

The effects of smoking and/or nicotine administered by alternative routes on vigilance performance have been investigated both in the framework of a general arousal theory of vigilance (i.e; based on the central stimulant actions of nicotine, which increases cortical activation and thus would be expected to enhance vigilance performance), and in relation to the activity of specific arousal systems (i.e; nicotine

being a cholinergic drug may be expected to influence cholinergic mechanisms involved in attention tasks) [12].

Studies investigating the effects of cigarette smoking and nicotine administered via alternative routes on the performance of various vigilance tasks (i.e; auditory, visual and rapid information processing), have generally shown that smokers who are allowed to smoke maintain a stable performance level while non-smokers and deprived smokers show a performance decrement over time. So, the latter two groups exhibit a performance pattern similar to what would be generally expected in vigilance tasks (i.e; lowered arousal and performance decrement with time on task), whereas smoking prevents this commonly observed decline in performance efficiency.

Table 4.2 (overleaf), gives a summary of various studies investigating the effects of cigarette smoking and nicotine tablets upon performance in various vigilance tasks (the type of subjects, tests and mode of nicotine administration and results have been briefly outlined). As can be seen from Table 4.2, most studies discussed in this review have been conducted by Wesnes & Warburton. Thus, unless otherwise stated, the findings discussed below are reported by these authors.

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(12) - Wesnes & Warburton (1978), have hypothesized that an increase in the activity of cholinergic pathways ascending to the cortex will result in improved selection of information both from the internal and external world. They maintained that the cortical desynchronization produced by nicotine can be used as an evidence that these pathways are being activated by this drug. Based on these points it has been suggested that in order to investigate the possibility that human attention is controlled by cholinergic mechanisms, attentional effect of drugs (i.e; nicotine) which influences cholinergic activity needs to be investigated.

	AUTHORS	SUBJECTS	TEST	MODE OF NIC. ADMIN.	RESULTS
EFFECTS OF CIGARETTE SMOKING AND DEPRIVATION	1-Wesnes & Warburton (1978)	Smokers, Depr. Smks. & Non-Smks. (12 hrs. depr. for all Smks. prior to the exp.)	Visual Vig. (80 mins)	Cigs or no-Cigs	Smks. allowed to smoke in the exp. showed a constant superior perf. Depr-Smks & Non-Smks had a perf. decrement / time.
	2-Tong et al (1977)	Smks-smoking (SS) Smks-Depr (DS) Non-Smks (NS)	Auditory Vig. (60 mins)	Cigs or no-Cigs	SS showed an improvement, while DS had a relatively stable perf and NS had a deterioration/ time.
	3-Tarriere & Hartemenn (1964)	SS; DS; NS (N=24/grp)	Visual Vig. (150 mins)	Cigs or no-Cigs	SS had a stable level of perf. while NS & DS had a decr/time. Perf. efficiency corresponded to cardiac rate.
	4-Heimstra et al (1967)	SS:DS:NS	Simulated driving (6 hrs)	Cigs or no-Cigs	SS consistently tended to perform better on reaction time & vigil tasks than DS & NS. DS markedly inferior to the SS and NS.
EFFECTS OF CIGARETTES WITH DIFFERENT NICOTINE YIELDS	1-Wesnes & Warburton (1978)	Heavy Smks (N=12)	Rapid inf. processing (20 mins)	0.9;1.8 mg nic. or nic-free cigs & no-smk.	Perf. with nic, cigs, showed an immediate improvement & then a decline. Perf. with nic-free cigs & no-smk. showed a decrement/time.
	2-Wesnes & Warburton (1978)	Heavy Smks (N=23)	Rapid Inf. processing (20 mins)	0.28; 0.7 & 1.65 mg. nic/cig	Only 1.65 mg. nic/cig. produced an improvement in perf.
	3-Wesnes & Warburton (1978)	Smks	Auditory Vig. (80 mins)	Nic & non-nic cigs.	Perf. with nic. cigs. was better than non-nic. cigs.
	4-Wesnes & Warburton (1978)	Heavy Smks (N=12)	Visual Vig. (80 mins)	0.28; 0.7 & 1.65 mg. nic. cigs.	The only signif. diff. in perf. was between 0.28 & 0.7 mg. nic. cigs. (0.7 producing better results)

Continued...

Table 4.2 Continued.

	AUTHORS	SUBJECTS	TEST	MODE OF NIC. ADMIN.	RESULTS
EFFECTS OF NICOTINE TABLETS	1-Wesnes & Warburton (1978)	Light & Heavy Smks. (n=12/grp)	Visual Vig. (80 mins)	Oral admin. 0; 1; 2 mg. nic tablets	Perf. was superior with nic. tablets than with the placebo tablet. Light & Heavy smks. responsivity to the drug could not be differentiated.
	2-Wesnes & Warburton (1978)	Smks & NS N=6/grp	Stroop Effect	0; 1; 2 mg. nic tablets	Nic. tablets reduced the magnitude of stroop eff. as compared to placebo tabl. No diff. between nic. doses.
	3-Wesnes & Warburton (1978)	Non-Smks	Visual Vig. (80 mins)	0; 1; 2 mg. nic tablets	Nic. tablets did not have a consistent effect. However, a dose related increase in HR was noted.

Table 4.2 Effects of smoking, cigarette deprivation and nicotine tablets upon vigilance test performance.

Wesnes and Warburton (1978), tested smokers allowed to smoke, deprived-smokers (DS) and non-smokers (NS), in an 80 minutes visual vigilance test. The results showed that the smokers allowed to smoke maintained a constant superior level of performance as compared to the NS and DS groups. There was no significant difference between the NS and DS groups, both of which exhibited a performance decrement over time. Tong et al (1977), also reported results confirming the above findings in an auditory vigilance task.

Tarriere and Hartemenn (1964), also noted that smokers who were allowed to smoke had a stable level of performance, whereas non-smokers had a significant decline in performance efficiency over time in a visual vigilance test. The authors also measured the cardiac rate throughout the experimental period and observed that the different levels in the cardiac rate curves of the experimental groups throughout the task corresponded to the differences in the level of performance curves. This result is very valuable in terms of relating a physiological measure indicative of activation to performance efficiency.

Wesnes and Warburton (1978), have shown that the beneficial effects of smoking on vigilance performance is related to the nicotine content of the cigarettes. They have found that in a rapid information processing task, subjects smoking nicotine cigarettes showed an immediate improvement in performance efficiency (i.e; 10 minutes after smoking) followed by a decline in efficiency. Performance with nicotine cigarettes was found to be superior to the performance in no-smoking and nicotine-free cigarette conditions. The latter two conditions produced a general decrement in vigilance over time, and were not significantly different from each other. Thus, the findings of this study implicate nicotine as the factor in cigarette smoking influencing performance, since the pure act of

smoking (i.e; nicotine-free cigarettes), was not found to produce a higher level of performance as compared to no-smoking condition. Higher performance efficiency with nicotine cigarettes as compared to non-nicotine cigarettes has also been noted in auditory and visual vigilance tasks by these authors.

Further support for the role of nicotine in enhancing performance in vigilance tasks comes from studies with orally administered nicotine (nicotine tablets of 1 & 2 mg. nic.). Wesnes and Warburton (1978), reported that nicotine tablets enhanced performance in a visual vigilance task and in a stroop effect [13] test, as compared to placebo tablets. However, for non-smokers, although a dose dependant increase was noted in HR. no consistent effect was found on their performance efficiency.

The responsivity of light ($\leqslant 5$ cigs/day) and heavy ($\geqslant 15$ cigs/day), smokers to nicotine tablets, as manifested in their performance efficiency has also been investigated. No statistical difference was found between the groups in terms of the differences in the performance scores under nicotine-and placebo-tablet conditions. The authors also investigated the relationship between consumption levels and the pharmacological addiction scores derived from Russell et al (1974), typology test (see chapter 2). They found a significant positive correlation between daily cigarette consumption and pharmacological addiction score. However, no significant correlation was noted between the addiction score and the difference in performance with nicotine and placebo tablets, which indicates that there is no evidence in terms of

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(13) - Stroop Effect: is the distraction caused by incongruent colour words. The test measures the time taken to name colour inks in which a series of incongruent colour names are printed. Nicotine is predicted to reduce the stroop effect (Wesnes & Warburton, 1978).

the performance efficiency in a visual vigilance task that smokers who score high on pharmacological addiction have a specific need for nicotine to enhance their performance efficiency.

So, in conclusion these studies indicate that smoking and/or nicotine tablets prevents the performance decrement commonly observed in vigilance tasks. This effect can mainly be attributed to the nicotine content of the cigarettes.

d) HAND STEADINESS

Cigarette smoking has been found to induce finger tremor (Edwards, 1948), and a deterioration in fine motor movements (i.e; hand steadiness).

Edwards (1948), measured finger movements with a tromometer (i.e; an apparatus that permits a tridimensional measurement of finger movements) and found that after smoking half a cigarette there was a marked increase in finger tremor (39%), compared to base-line values. The increase in tremor was found to be higher for women than for men. The author also compared the effects of inhaling and not inhaling from a standard tobacco cigarette on finger tremor and noted that there was only an increase in finger tremor for the subjects who inhaled. Smoking dried corn silk did not produce any change in finger tremor. It was also found that for habitual smokers a period of 2 hours abstinence from cigarettes produced a significant decrease in finger tremor. These results seem to indicate that the increase in finger tremor is related to the nicotine content of cigarettes rather than to other factors involved in smoking.

Frankenhauser et al (1968), noted a more marked deterioration

in hand steadiness[14] with the first cigarette after a period of abstinence. On the other hand, Myrsten et al (1972), noted an improvement in hand steadiness after 100 minutes of no-smoking period. Myrsten et al (1977), also reported an improvement in hand steadiness with abstention from cigarettes.

These results seem to indicate that smoking has a deleterious effect on tasks demanding fine motor coordination.

4.3 CONCLUSIONS

The majority of experimental findings reviewed in the previous section, indicate that cigarette smoking and nicotine administrations via alternative routes have a stimulant effect, as manifested by cortical and autonomic (peripheral) activation. Concurrent changes in behavioural efficiency (i.e; vigilance test performance and flicker fusion thresholds), have also been observed. All these changes, support the smokers' reports of a 'stimulant' effect of smoking.

Some studies point out that smoking can have a dual effect (i.e; stimulant and depressant), depending on the level of arousal of the smoker at the time of smoking and the rate and dose of nicotine inhaled. However, at present the findings demonstrating the sedative action of smoking are slender and it is difficult to explain the basis of sedative smoking. Although, smoking has been observed to prevent performance decrement in vigilance tasks, and increase the sensitivity to detect light flashes (i.e; CFF), it has been shown to impair fine motor movements and the

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(14) - Hand Steadiness - The task requires the subject to hold a metal stylus (1 mm in diameter) in the aperture of a metal plate (2.5 mm. in diameter)

size of the peripheral visual field.

So, these findings point out that the effects of smoking and/or nicotine are complex, and thus no simple psychological motive can be delineated to account for a pharmacological addiction to smoking. It is only possible, at present to conclude that smokers who state either or all of the following motives may be dependant on the pharmacological actions of smoking, and thus be pharmacologically addicted; a) stimulation, b) sedation, c) withdrawal relief. Russell (1976-b), made a distinction between pure psychological addiction not involving the pharmacological effects of smoking and psychological addiction maintained by a dependance on the pharmacological effects of nicotine. However, since even smokers who do not inhale obtain some nicotine through buccal absorption, it seems difficult to differentiate types of addiction. For this reason, at present one way of identifying the type of psychological addiction is to ask smokers why they smoke. If they report smoking for any reason that is in line with the pharmacological effects of smoking, then it seems likely that they are dependant on the pharmacological actions of smoking.

Although, 'sedative' smoking has not emerged as a smoking type in Russell et al's factor analysis, the subjective reports of smokers, and the experimental work of Nelson et al (1975) in animals, Heimstra (1973) and others in humans, point out that smoking may counteract the distractive effects of non-specific arousal. Furthermore, smoking may have a tranquilizing effect by its relaxing effect on skeletal muscle tone (i.e; depression of patellar reflex). At present it seems premature to discard sedative smoking as a possible smoking type. Further research investigating the sedative actions of smoking and/or nicotine is needed.

As stated earlier, the dual effects of smoking depend on dose and rate of administration as well as the state of the individual. In view of this, it seems more likely that smokers would adjust their nicotine intake according to their level of arousal and the characteristics (or demands) of the situation, rather than titrate their nicotine intake to achieve a consistent level of nicotine intake, as has been suggested by some researchers (see chapter 3).

A re-evaluation of the smoking typology research in the light of the studies on the pharmacological actions of smoking seems to suggest that a combination of Russell et al (1974), pharmacological addiction dimension and Frith (1971), typology model would yield a more comprehensive account of pharmacological addiction, than the models provided by each typology separately. Although, the 'pharmacological addiction' dimension proposed by Russell et al provides a general account of the pharmacological actions of nicotine that might serve as reinforcers in the maintenance of the smoking behaviour, it does not elucidate or allow for the analysis of individual predominant reinforcers.

The model summarized in table 4.3 (overleaf), indicates that it is important to know whether a smoker smokes in order to avoid the withdrawal symptoms associated with deprivation or the stimulant or sedative actions of smoking and/or nicotine.

Stimulation and sedation smoking is likely to lead to an adjustment of nicotine intake in order to achieve an optimum level for a particular situation, whereas withdrawal relief and automatic smoking is likely to lead to a high frequency and regular smoking in order to maintain a consistent blood nicotine level. Although, this model provides a basic understanding of pharmacological addiction, pure types (i.e; smoking only

to stimulate or sedate oneself) might not be very common, and further research is needed in this area. This model indicates that light smokers can also be pharmacologically addicted to smoking if they show a consistent smoking pattern in certain situations or states.

MOTIVES	TYPE	NICOTINE REGULATION
Withdrawal Relief & Automatic smoking (Addictive smoking) (Russell et al)	General Pharmacological Addiction	Consistent blood nicotine levels
STIMULATION: Smoking in low-arousal situations or in low-arousal state (Frith, 71; Russell et al, 74)	Stimulant Smoking	Adjustment of nicotine intake, more in low-arousal state or situation.
SEDATION: Smoking in high-arousal situations or in high-arousal state (Frith, 71; Russell et al, 74)	Sedation Smoking	Adjustment of nicotine intake, more in high-arousal state or situation.

Table 4.3 Model for pharmacological addiction based on the effects of smoking and typology research.

The model explains psychological addiction on the basis of the pharmacological effects of smoking and/or nicotine. However, it is not unlikely that for some smokers non-pharmacological motives may (i.e; sensorimotor and oral aspects, social rewards, etc.) predominate.

4.4 CHAPTER SUMMARY

The role attributed to nicotine as the major reinforcer in the maintenance of smoking behaviour and the pharmacological motives delineated by the typology research necessitates the investigation of the psychophysiological effects of cigarette smoking.

The effect of nicotine delivered by smoking is predominantly stimulant. It leads to the desynchronization of electroencephalogram, an increase in heart rate, blood pressure, skin conductance level and adrenaline secretion, produces vasoconstriction in the peripheral vasculature and thus decreases skin temperature. All these physiological changes are accompanied by changes in behavioural and psychophysical measures (i.e; vigilance task performance, sensory thresholds, hand steadiness).

The relationship between the effects of smoking and the smokers' motives for smoking (stimulation and sedation-pharmacological) has been discussed and a model to account for pharmacological addiction has been proposed.

CHAPTER 5

EXPERIMENT 1

PHYSIOLOGICAL AND BEHAVIOURAL EFFECTS OF CIGARETTE DEPRIVATION: AN ATTEMPT TO VALIDATE A SMOKING TYPOLOGY SCALE

5.1 INTRODUCTION

The present experiment aimed at providing a preliminary assessment of the relationship between pharmacological addiction to smoking and/or nicotine and the response (physiological, psychophysical and behavioural), to cigarette deprivation.

The main focus of this experiment was to investigate the differences between smokers scoring high (i.e; addicted) and low (i.e; non-addicted) on the pharmacological addiction dimension (i.e; stimulation, addictive and automatic factors), as proposed by Russell et al (1974) [1], in their physiological, psychophysical and behavioural responses to cigarette deprivation. The effects of deprivation were assessed by comparing measures taken under normal smoking conditions with those taken under deprivation conditions.

On the basis of the types that have been clustered together to form the pharmacological addiction dimension it was hypothesized that the effects of deprivation would be more pronounced for the addicted smokers

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(1) In order to simplify the terminology, the following labels have been used in this and following chapters.

Addicted Smokers: Smokers who score high on the pharmacological addiction dimension proposed by Russell et al (1974).

Non-Addicted Smokers: Smokers who score low on the pharmacological addiction dimension proposed by Russell et al (1974). Addiction as used in this chapter refers to pharmacological dependance and does not have any implication for psychological addiction.

as compared with the non-addicted smokers (i.e; the difference between the measures taken in the smoking and deprivation conditions would be greater for the addicted smokers).

This hypothesis was based upon the "addictive smoking" type, which suggests that smokers experience withdrawal symptoms whenever they have gone 20 - 30 minutes without smoking and that they smoke in order to alleviate this distressing state. So, if the withdrawal symptoms are related to physiological changes (i.e; lowered physiological activation levels), and to concurrent changes in behavioural efficiency and perceptual sensitivity then this may be evidenced by a comparison of measures taken under deprivation and smoking conditions, from addicted and non-addicted smokers.

It was also hypothesized that a lowered arousal level (as manifested by the physiological and behavioural_perceptual measures) would be observed for the whole sample in the deprivation condition as compared to the normal smoking condition. This hypothesis was based upon the findings discussed in chapter 4.

5.2 METHOD

i) Subjects:

The subjects were 23 voluntary adult smokers (15 females and 8 males), who responded to an article published in the local newspaper (see appendix A-i), requiring volunteers to help with research in "The effects and methods of giving up smoking".

Table 5.1 (overleaf), shows the characteristics of the sample.

Table 5.1 Distribution of subjects over demographic and smoking history variables. (N = 23)

Variable		Number of Ss	% of Ss	
AGE	20 - 29	3	13.05	
	30 - 39	7	30.05	(X = 41.35)
	40 - 49	6	26.08	(S.D. = 9.68)
	50 - 59	7	30.44	
X. NO. OF CIGS/DAY	0 - 9	1	4.30	
	10 - 19	8	34.78	(X = 21.91)
	20 - 29	8	34.78	(S.D. = 7.66)
	30 - 39	4	17.39	
	40 - 49	2	8.69	
NO. OF YEARS SMOKING	10 - 19	10	43.48	
	20 - 29	5	21.74	
	30 - 39	8	34.78	
MARITAL STATUS	Single	3	13.04	
	Married	17	73.90	
	Divorced	1	4.3	
	Widowed	1	4.3	
	Seperated	1	4.3	
WHETHER THE MOST IMP. PERSON THEY SPEND TIME WITH IS A SMK?	Yes	8	34.47	
	No	14	60.88	
	Can not say	1	4.3	
NO. OF ATTEMPTS TO QUIT	0	5	21.73	
	1	7	30.43	
	2	2	8.69	
	3 - 7	6	26.08	
	>8	3	13.04	
PERIOD OF COMPLETE ABSTINENCE	Several days	4	22.20	
	Several weeks	5	27.80	
	Several months	6	33.30	
	Several years	3	17.70	
DO THEY WANT TO QUIT?	Yes	23	100.00	
	No	0	0	

Table 5.1 (Continued)

REASONS FOR WANTING TO QUIT	Health	19	82.60
	Cost	14	60.86
	Aesthetic	2	8.69
	Social	2	8.69
TYPE OF CIG. SMOKED	Manufactured with filter	19	82.60
	Manufactured plain	2	8.69
	Hand rolled	2	8.69
BRANDS OF CIGS. SMOKED (based on tables issued by the Health Dept., U,K., 1975)	Low Tar - Nic. (nic. yield; 0.3 - 0.7 mg)	9	39.00
	Low-Middle Tar - nic. (1.3 mg nic)	4	17.40
	Middle Tar-nic	5	21.70
	(Hand rolled tob. users and Ss not having a consistently regular brand were excluded)		
ANTICIPATED WITHDRAWAL SYMPTOMS	Do not know	2	8.69
	None	1	4.34
	Lack of concentration	3	13.04
	Fuzziness in head	2	8.69
	Nervous tension & irritability	10	43.48
	Craving	2	8.69
	Increased Appetite	3	13.04
	Depression	3	13.04
MEAN FACTOR SCORES ON THE PHARMACOLOGICAL ADDICTION DIM. (RUSSELL ET AL, 1974)	1 - 5	1	4.34
	6 - 10	1	4.34
	11 - 15	1	4.34
	16 - 20	3	13.04
	21 - 25	5	21.74
	26 - 30	5	21.74
	31 - 35	3	13.04
	36 - 40	4	17.39

ii) General Procedures and Design

A letter was posted to each respondent explaining the procedures of the experiment (see appendix A-ii), together with two questionnaires. One of these questionnaires was the "Smoking Typology Scale" developed by Russell et al (1974), (see appendix A-iii), and the second one was a questionnaire developed by the present author, aimed at obtaining factual information on smoking habits. An "Addiction Index Score" was also derived from this scale (see appendix A-iv).

Subjects were asked to complete the questionnaires and send them back. On receipt of the filled-in tests, the subjects were invited to the University for an introductory meeting during which the procedures of the experiment were discussed in detail, and they were assigned to an experimental group and were provided with a form to record their smoking frequency for four consecutive days (see appendix A-v), and instructions appropriate for the experimental group they were assigned to (see appendix A-vi).

There were two experimental conditions:

a) Smoking Condition:

Subjects were asked to smoke as usual till their arrival for testing, but to abstain from alcohol and drugs. They were then required to smoke one cigarette of their usual brand, in their usual way. Testing was initiated two minutes after the termination of smoking. This procedure was employed in order to control for the recency of the last cigarette smoked.

b) Deprivation Condition:

In this condition subjects were asked to refrain from smoking (also alcohol and drugs), from 12 p.m. onwards, the evening before the testing day.

Subjects served as their own control and they were tested in both of the above conditions. Order of the test conditions was counterbalanced across subjects. So, half of the subjects (N = 11), were assigned to "experimental group 1" where smoking condition was followed by the deprivation condition, and the other half (N = 12), to "Exp. group 2", where the order of the conditions were reversed. For each experimental group testing took place on two consecutive days, at the same time of the day for each subject. Hours of deprivation ranged from 10 to 18 hours between subjects, according to the time of testing.

Following the completion of the experiment, a set of "Self-control" techniques were posted to the subjects to aid them in giving up smoking (see appendix A-vii).

iii) Procedures of testing, measures taken and apparatus utilized:

The testing procedure is outlined in Fig. 5.1, below.

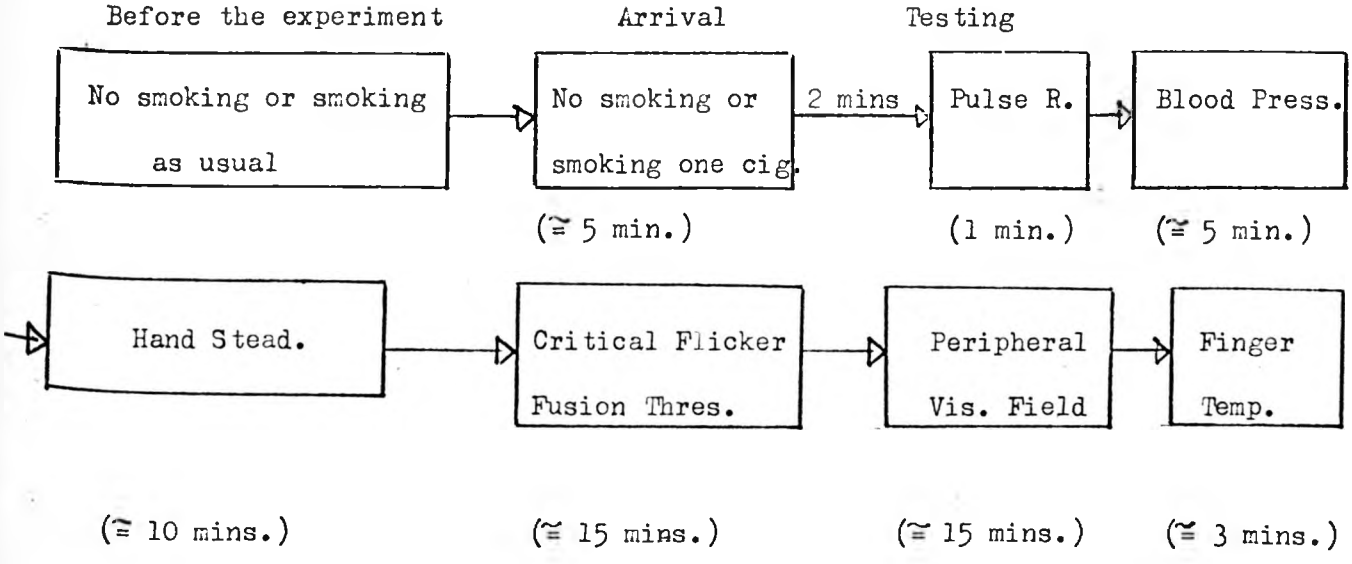

Fig. 5.1 Summary of the experimental procedure.

The experiment took place in a centrally heated room and the room temperature ranged between 19° - 21° C.

Measures taken and Apparatus (in the order of testing):

a) Pulse Rate:

Pulse rate was measured by the palpation of the radial artery of the dominant arm for a 60 seconds period.

b) Blood Pressure:

Systolic and diastolic blood pressures were recorded by the standard clinical method employing a sphygmomanometer and stethoscope (Model 104, Clayton Industries).

c) Hand Steadiness:

Apparatus: 1 - A metal stylus (1 mm in diameter), and a metal plate with an aperture of 2.5 mm diameter. 2 - timer (set to 20 seconds). 3 - automatic counter to record the number of contacts between the stylus and the aperture.

Experimental Task: To hold the metal stylus in the aperture of the metal plate steadily without touching the sides of the aperture for two "20 secs" periods.

Procedure:

1 - The subject was asked to read the instructions for the task. (see appendix B-i).

2 - He was asked to hold the metal stylus in the aperture of the metal plate, with his dominant hand, without supporting his arm against his body or any object.

3 - When the subject had placed the stylus in the aperture the experimenter counted up to five and then gave a "ready, now" signal, simultaneously operating the timer (set to 20 secs). The number of contacts with the sides of the aperture were recorded by the counter.

4 - Two "20 seconds" practice trials (with one min. rest period in between) were given. Following the practice trials, two experimental trials of 20 sec.

duration were given. The mean number of contacts recorded from the two experimental trials yielded the hand steadiness score.

Figure 5.2 outlines the procedure for the hand steadiness measurement.

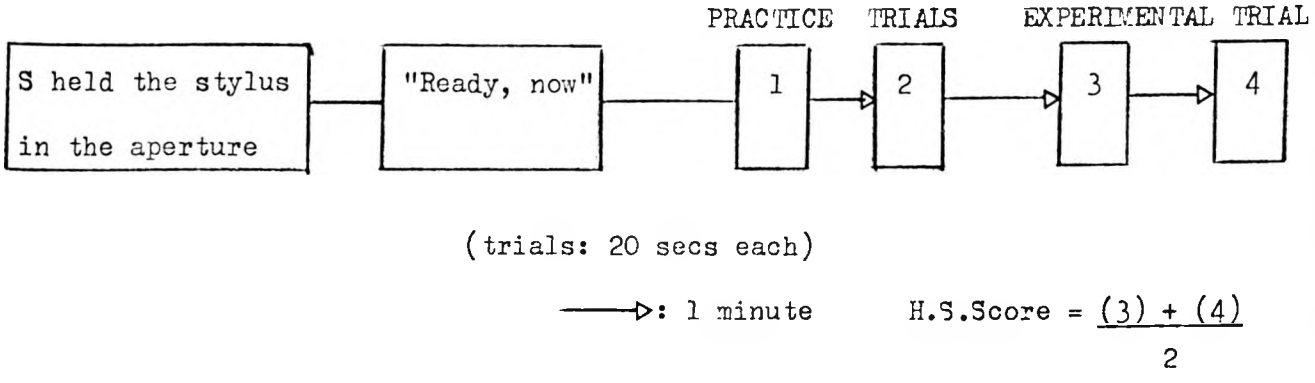

Fig. 5.2 Summary of the procedure for the hand steadiness task.

d) Critical Flicker Fusion Threshold (CFFT)

Apparatus: A red-light emitting diode (RS type 576-327), mounted on a black background. An adjustable black rubber tube mounted on a metal holder, enclosing the light-emitting diode. An electronic circuit enabled the frequency with which the light flashed to be set to any value in the range of 20 - 50 Hz.

Procedure:

1 - The subject was asked to read the instructions for the task and procedures to be followed (see appendix B-ii).

2 - The S's left eye was occluded with a black eye-patch, and the experimental room was darkened.

3 - The subject was seated on an adjustable stool in front of the table, on which the apparatus was placed. Testing started after 5 minutes of dark adaptation.

4 - The experimenter presented the stimulus by pressing a button which operated the light for 1 sec. duration. The frequency of the light flashes

was altered by a knob, with scale values ranging from 6 to 9, corresponding to 20 to 50 cycles per second. These were values at which the light flash was reliably rated as 'flickering' or 'steady' by independent judges (N = 6).

5 - A 'Ready, now' signal was given before each presentation.

6 - The CFFT was determined by the method of limits. The stimuli (different frequencies of light flashes), were presented consecutively in small increments or decrements. Three ascending (i.e; progressively increasing frequencies, starting from flicker and increasing in steps to fusion), and three descending (i.e; progressively decreasing frequencies, starting from fusion and decreasing in steps to flicker), series were given. The frequencies were incremented or decremented by 0.2 scale points (i.e; 2 cycles/sec.).

7 - The subject had two practice trials, followed by six experimental trials. A 30 seconds pause was given after the presentation of each series.

8 - The Ss reports were recorded on a form ('+' for fusion, '-' for flicker). The criterion for the CFFT was a change in the reports from flicker to fusion or vice versa, which was maintained on two consecutive responses. The mean of the six experimental trials (3 ascending and 3 descending series) yielded the CFFT score, (the higher the value, the more able the subject is to perceive flicker in light flashes of higher frequencies).

Figure 5.3 outlines the procedure for the measurement of the CFFT.

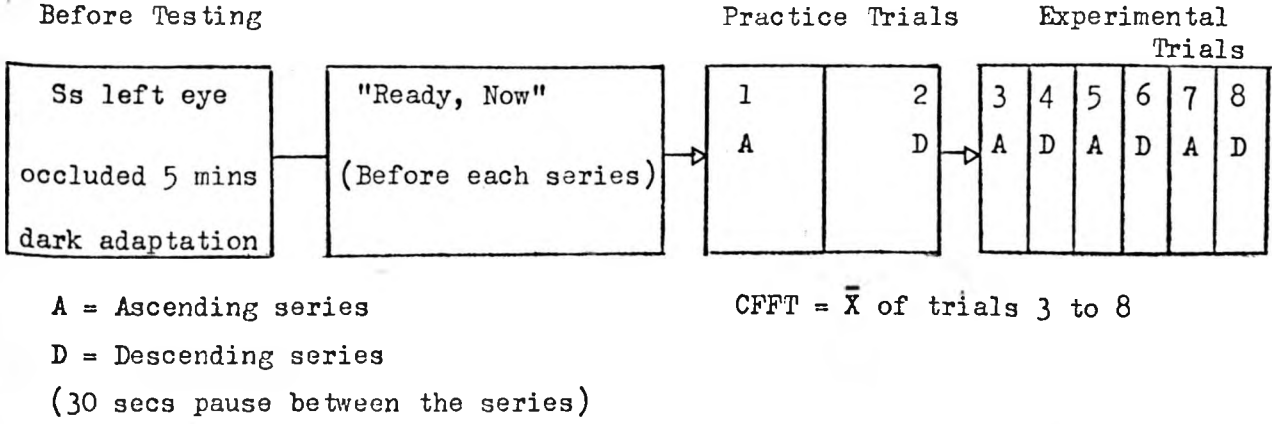

Fig. 5.3 Summary of the procedures for the critical flicker fusion test.

c) Peripheral Visual Field (PVF)

Apparatus: Aimark Projection Perimeter

Test Stimulus: Size: 3.0 mm; Colour: White; Brightness: 0.6 (density filter) (3.00 Lamp-foot).

Procedure:

1 - The subject read the instructions explaining the experimental task and procedures to be followed. (see appendix B-iii).

2 - His left eye was occluded and he was seated on an adjustable stool, with his arms resting on the table, on either side of the main base plate of the projection perimeter.

3 - He was asked to place his chin in the left cup of the double chin rest, so that his right eye was opposite to the fixation cross. The Ss head was in a vertical position with his eyes at level with the fixation cross, which was at the center of the arc of the perimeter. The S was instructed to fixate his right eye on the cross throughout the testing.

4 - Testing was conducted in a darkened room and the subject was dark adapted for 3 minutes before the testing was started.

5 - The test stimulus (i.e; light-dot) was presented at different angles on the arc. The S was asked to report whether he could see the light-dot or not.

6 - Testing was conducted in four meridians (0°, 90°, 180°, 270°) and the method of limits was used to determine the visual field.

7 - One practice trial was given in each meridian, followed by the experimental trials.

8 - The experimental trials consisted of 2 ascending and 2 descending series in each meridian.

9 - A "Ready, Now" signal was given before each stimulus presentation. A 30 seconds pause was given between the presentation of each series.

10 - The Ss responses were recorded on a form ('-' for no; '+' for yes). The criteria for the PVF was two consecutive 'Yes' responses after 'No' responses or vice versa.

11 - The mean of the four test trials in each meridian yielded the peripheral visual field score for that meridian. A total peripheral visual field score was derived from the following formulae:

$$\text{Total P.V.F.} = \frac{(0^\circ + 180^\circ) + (90^\circ + 270^\circ)}{2} \times 0.36 \quad \text{(Johnston, 1965)}$$

Figure 5.4 outlines the procedure for the PVF measurement.

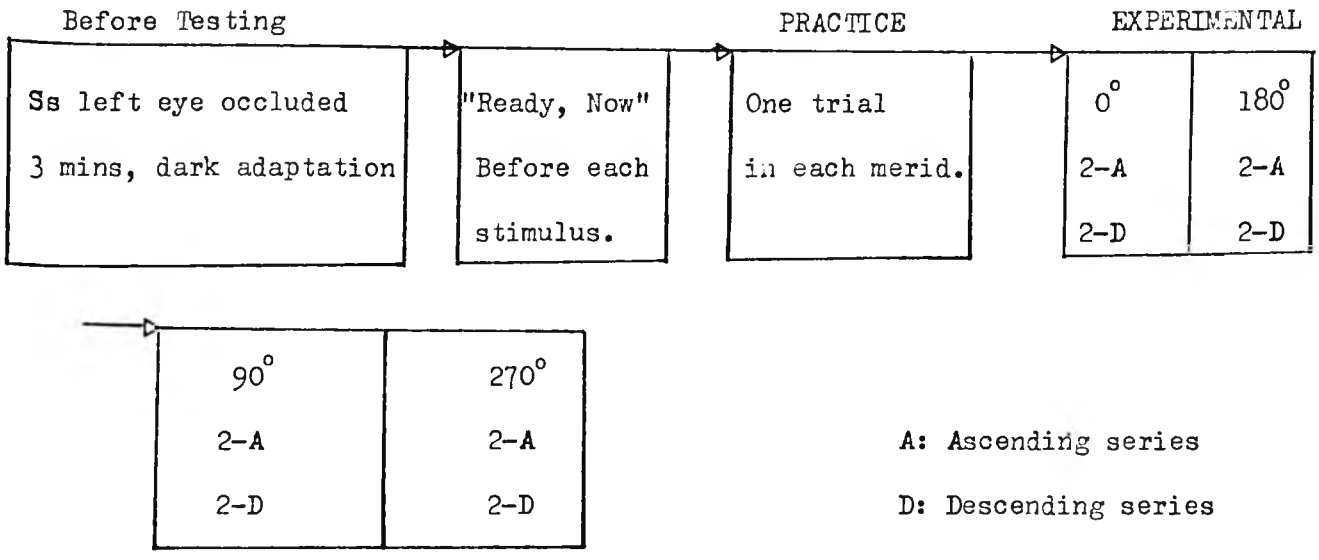

Fig. 5.4 Summary of the procedure for the measurement of peripheral visual field.

d) Finger Temperature:

Apparatus: A thermo-electric probe.

Procedure: The subject held the thermo-electric probe between his thumb and index finger of the dominant hand for 3 minutes.

III) RESULTS

The results of this study will be presented in four sections, each section addressing a specific hypothesis. Each result section will be followed by a discussion of findings and their implications.

Statistical analysis: "Elliott 903" computer (Algol programming language) was used to analyse the results.

A: THE EFFECTS OF CIGARETTE DEPRIVATION ON PHYSIOLOGICAL AND BEHAVIOURAL MEASURES

The difference between the "smoking" and "deprivation" condition values was analysed with "t" tests (correlated samples, N = 23).

Figure 5.5, (overleaf) illustrates the mean values in the smoking and deprivation conditions for the measures utilized.

The "t" values are shown in Table 5.2, below.

MEASURES	t
Pulse Rate	7.45^{xx}
Systolic B.P.	5.02^{xx}
Diastolic B.P.	1.70 NS
Hand Steadiness	1.28 NS
C.F.F.T.	1.40 NS
Tot. P.V.F.	1.15 NS
0 Merid.	0.44 NS
180 Merid	0.24 NS
90 Merid	2.81^{x}
270 Merid	0.72 NS
Finger Temp.	1.62 NS

df = 21
(two-tailed test)
x $p < 0.02$
xx $p < 0.001$
NS $p > 0.05$

Table 5.2 "t" values for the difference between 'smoking' and 'deprivation' conditions.

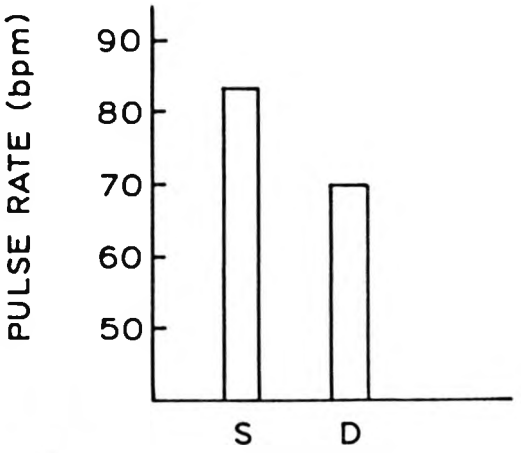

Fig. 5.5(a) Mean pulse rate

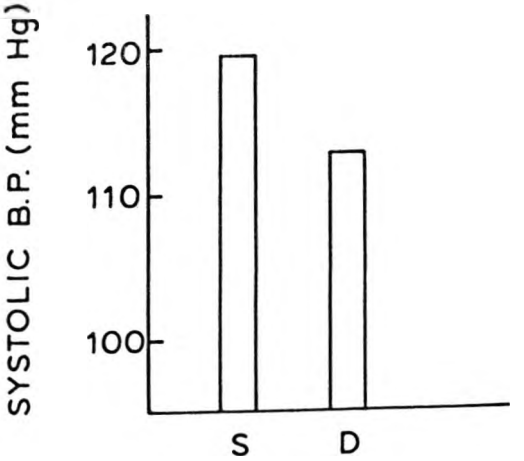

Fig. 5.5(b) Mean SBP

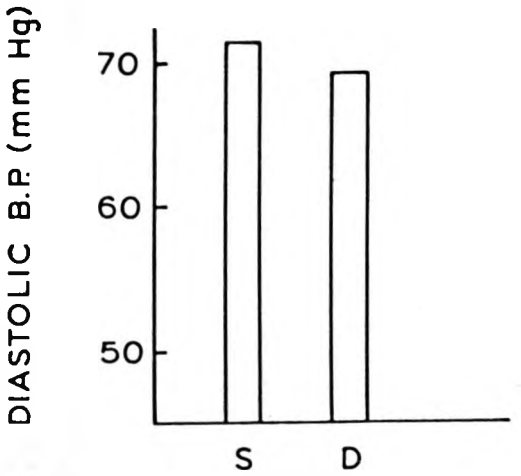

Fig. 5.5(c) Mean DBP

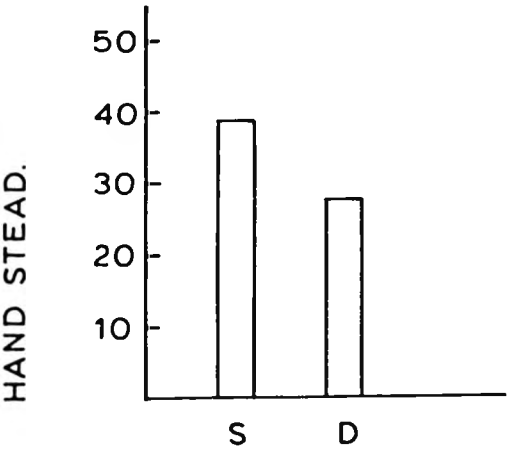

Fig. 5.5(d) Mean HS score

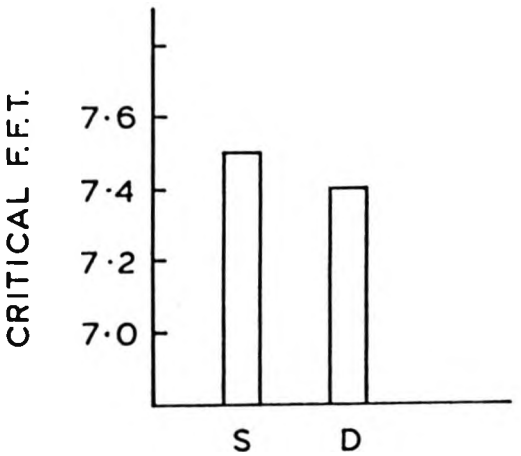

Fig. 5.5(e) Mean CFFT

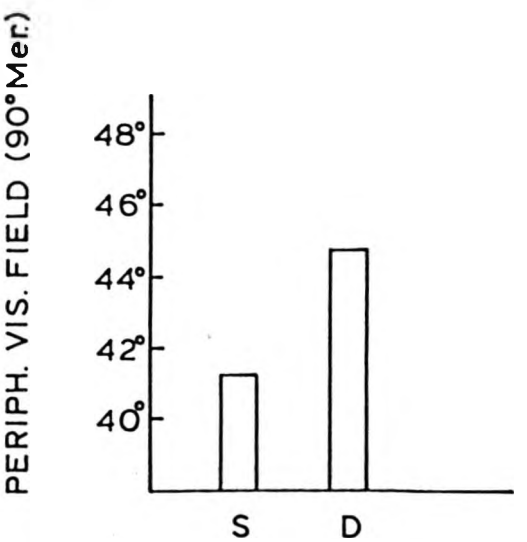

Fig. 5.5(f) Mean PVF in 90° Merid.

Table 5.2 shows that there was a significant difference between the smoking and deprivation conditions in pulse rate, systolic blood pressure and peripheral visual field recorded from 90° meridian.

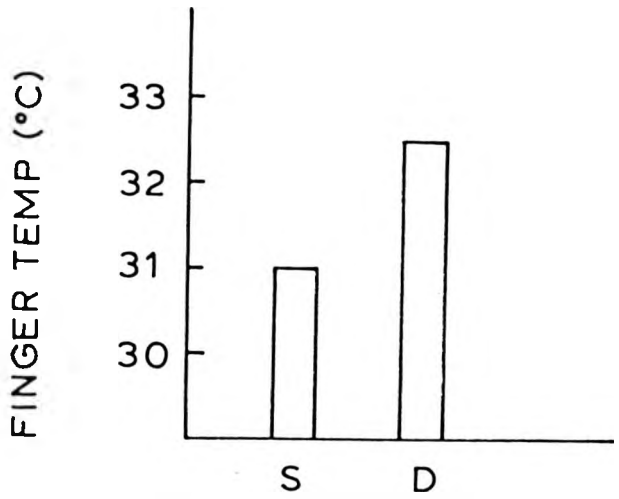

Fig. 5.5(g) Mean finger temp.

Fig. 5.5 (a - g) Measures taken in the Smoking (S) and Deprivation (D) conditions.

As will be seen from these results, cigarette deprivation produced a lower pulse rate, systolic blood pressure and increased the PVF in the 90° meridian.

DISCUSSION

Cigarette deprivation has been reported to produce a lowered level of autonomic activation as indicated by lower pulse rate and blood pressure levels as compared to normal smoking values. On the other hand, higher sensitivity to perceive flicker in light flashes of higher frequencies, and a deterioration in the performance of fine motor movements (e.g; hand steadiness), were also noted folowing smoking (see chapter 4).

The results of this experiment suggest a lowered level of autonomic activation after a period of cigarette deprivation as compared to normal smoking periods. The increase in peripheral visual field (90°meridian) noted in the present study is in agreement with the findings reported in literature. Although, it is interesting to note that deprivation has a beneficial effect on the PVF, at present the mechanisms of this change are not clear. Thus, further research is needed to elucidate the mechanisms of this effect.

Although, the change in the other physiological and behavioural measures showed a trend which was in line with the reports of other investigators, the difference between the smoking and the deprivation conditions were not found to be statistically significant. An improvement in hand steadiness, increase in finger temperature and decrease in the CFF thresholds (i.e; less sensitivity), were noted with deprivation.

The difference between the results of this experiment and the ones reviewed in chapter 4 could be due to two factors. Firstly, it has been noted by researchers that the first cigarette smoked after a period of deprivation produces a more marked change. Thus, the majority of investigators have imposed a period of abstinence before allowing the subjects to smoke. Since, in the present study there was no limitation on the number of cigarettes smoked before the experiment in the smoking condition, (i.e; no imposed period of abstinence before smoking) the results although exhibiting a similar trend to the ones observed in literature, may have failed to reach significance. Secondly, since only one cigarette was smoked before the initiation of testing, some of the measures might have been taken when the effects of smoking had started to dissipate. Isaac and Rand (1972), reported the plasma half life of nicotine to be less than 30 minutes, which is followed by an initial rapid decay, followed by a

slower phase of excretion and metabolism. So, after smoking a cigarette, decay in blood nicotine levels were observed starting 30 minutes and continuing up to 60 minutes of the completion of smoking.

In the present study testing was initiated 2 minutes after the completion of the experimental cigarette, so that the blood nicotine levels during the course of measurement would have declined according to the plasma half life of nicotine. Thus, the first measures immediately following smoking might be reflecting the effects of smoking more than the latter measurements. Investigators, who reported significant changes in hand steadiness, CFFT, PVF and finger temperature have usually tested the smokers immediately after smoking, when the blood nicotine levels would be expected to be quite high.

These considerations suggest that it would be more appropriate to take continuous records of physiological measures before, during and after smoking. Also, the effects of smoking on sensory sensitivity and behavioural efficiency might be reflected more clearly if measures are taken within a 20 - 30 minutes post-smoking period. Furthermore, it would be more appropriate to employ a standard period of deprivation for all subjects. Although, it would have been interesting to examine the effects of smoking in subjects who had different periods of deprivation, in the present study subjects were deprived for at least ten hours. It would be more useful to examine the effects of progressively increasing hours of deprivation (e.g; starting from one hour). The effect of duration of deprivation is particularly of interest when we examine light smokers, who leave long time gaps between their cigarettes. Thus, it would be useful to investigate how much time is necessary to observe a significant reduction in activation levels, as compared to smoking values.

Although, it is of interest to investigate the effects of a single cigarette after a period of deprivation, the choice of this procedure purely to observe a more pronounced change in the measures employed does not seem to be advisable. Since in normal daily smoking, smokers (especially heavy ones), do not have long periods of deprivation, such a procedure is not likely to generate information relevant to the effects of smoking in natural environments. However, the marked effects of post-deprivation smoking may indicate that for light smokers a single cigarette after a period of no-smoking may produce a greater effect in physiological activation levels and also behavioural efficiency.

So, in conclusion the findings of this experiment indicate that smoking deprivation produces a lowered level of autonomic activation as manifested by lowered pulse rate and blood pressure. Although, the trend in the psychophysical (i.e; Peripheral visual field), and behavioural measures (i.e; hand steadiness) indicates that deprivation has beneficial effects in some tasks, the trend for a decreased sensitivity to detect flicker in light flashes during deprivation suggests that the effects of deprivation on behavioural efficiency depends on the nature of the task.

B: THE RELATIONSHIP BETWEEN PHARMACOLOGICAL ADDICTION SCORES AND RESPONSE TO SMOKING AND DEPRIVATION AS INDICATED BY PHYSIOLOGICAL, PSYCHOPHYSICAL AND BEHAVIOURAL MEASURES: CORRELATIONAL ANALYSES

Correlational analyses (Pearson r- correlation matrix), to examine the relationship between questionnaire scores and differential effects of smoking and deprivation on physiological, psychophysical and behavioural measures were undertaken.

The following variables were tested by the correlational analyses:

1 - Hours of deprivation, 2 - Age, 3 - Daily cigarette consumption, 4 - Pharmacological Addiction score (Russell et al,1974), 5 - Addiction Indes score (from the 'Smoking Habits Questionnaire'), and the difference between the 'smoking' and 'deprivation' condition scores (i.e; for each subject; Smoking-Deprivation)in; 6 - Pulse rate; 7 - Systolic B.P; 8 - Diastolic B.P; 9 - CFFT; 10 - Hand Steadiness; and deprivation-smoking scores in; 11 - Peripheral V.F. in 90° meridian; 12 - Finger temperature.

The correlations matrix describing the relationship between these variables is presented in Table 5.3 (overleaf).

The following measures were found to be significantly correlated:

1 - Mean daily cigarette consumption correlated positively with the "Addiction Index score" (AIS) ($p < 0.02$), and the "Pharmacological Addiction score" (PAS) ($p < 0.05$).

2 - "Pharmacological addiction score" and the "Addiction index score" were found to be positively correlated. ($p < 0.01$).

3 - "AIS" was found to be negatively correlated with the change in diastolic blood pressure from the smoking to the deprivation condition ($p < 0.02$).

4 - Hours of deprivation was negatively correlated with the change in DBP ($p < 0.02$).

5 - The change in systolic and diastolic blood pressures were found to be positively correlated ($p < 0.02$).

	2	3	4	5	6	7	8	9	10	11	Change in F. Temp. 12
Hours of deprivation (1)	0.34	-0.01	0.07	-0.03	0.15	-0.28	-0.45**	0.24	0.35	0.15	0.01
Age (2)		-0.04	-0.01	-0.14	0.32	-0.14	-0.34	-0.02	0.33	0.23	-0.15
Daily cigarette consumption (3)			0.42*	0.47**	-0.15	0.21	-0.10	0.11	0.15	0.18	0.26
Pharm. Add. score (4)				0.62***	0.03	-0.20	-0.31	-0.17	0.31	0.11	-0.03
Addiction index score (5)					-0.09	0.06	-0.46**	0.03	0.09	0.33	0.21
Change in Pulse rate (6)						-0.08	-0.14	0.20	0.11	-0.10	0.18
Change in systolic BP (7)							0.51**	-0.32	-0.15	-0.15	0.27
Change in diastolic BP. (8)								-0.32	-0.24	-0.34	-0.05
Change in hand steadiness (9)									-0.01	-0.13	0.10
Change in critical flicker F (10)										0.04	0.28
Change in peripheral Vis. Field (90° Meridian) (11)											0.02

Table 5.3 Correlation matrix (Pearson r) for questionnaire scores and differences between smoking and deprivation conditions.

df = 21

* $p < 0.05$

** $p < 0.02$

*** $p < 0.01$

(two-tailed test)

DISCUSSION:

On the basis of the smoking motives contributing to the pharmacological addiction dimension (i.e; smoking to avoid withdrawal symptoms), it was hypothesized that smokers scoring high on this dimension are likely to exhibit more pronounced physiological and behavioural withdrawal symptoms (i.e; the difference between smoking and deprivation condition scores will be larger) after a period of cigarette deprivation.

The results of the correlational analysis did not support this hypothesis. No significant relation was noted between the pharmacological addiction scores and the 'difference scores' in the measures employed. In other words, the results of the correlational analysis indicate that the score on pharmacological addiction dimension is not a good predictor of physiological and behavioural consequences of cigarette deprivation.

Contrary to the expectations the addiction index score (AIS) and the change in diastolic blood pressure from the smoking to the deprivation condition was found to be negatively correlated. Since the items contributing to the 'AIS' suggest a high and consistent level of nicotine intake (i.e; deep inhalation and regular smoking throughout the day), blood pressure values in a smoking day would be expected to be higher in proportion with the dose of nicotine intake. Thus, smokers scoring high on the addiction index, could be expected to have a larger difference in blood pressure (i.e; Smoking-Deprivation values). However, tolerance to the effects of smoking and/or nicotine needs to be considered. If the addicted smokers, due to their high frequency of smoking and thus nicotine intake, develop a tolerance to the effects of smoking, then the experimental

cigarette smoked prior to testing might not have led to a large increase in their blood pressure recordings on this day. Thus, as the results indicate, the difference between smoking and deprivation conditions might be comparatively smaller for the addicted smokers than it is for the non-addicted or light smokers. However, this interpretation is speculative and needs to be experimentally verified.

Addiction scores derived from the two questionnaires were found to be correlated with each other and also with the daily cigarette consumption rate. It is interesting to note that the two scales utilized in this experiment yielded significantly correlated addiction scores. The 'Addiction Index score' (see appendix A-iv), is derived from direct factual information on smoking habits (i.e; mainly : How and when), like the degree of inhalation, whether the smoker chain smokes to compensate for the effects of temporary abstinence, and whether smoking frequency is distributed regularly throughout the day. On the other hand, the items contributing to the 'Pharmacological addiction dimension' are mainly related to the motives (i.e; Why) of smoking. The correspondance between the scores from the two scales seems to indicate that smokers do have a fairly consistent image of why (from Russell et al typology scale) and how intensely (i.e; behavioural, from the Smoking Habits scale) they smoke.

The correlation between both of the addiction scores and daily cigarette consumption rate indicates that the subjective reports of smokers (i.e; smoking to avoid withdrawal symptoms as indicated by the typology scale) are in line with the consumption rate obtained from a four days base-line period.

A negative correlation was noted between the hours of deprivation and change in diastolic B.P. This finding points out that it

is important to equate the hours of deprivation across subjects.

In conclusion, the findings of the correlational analyses did not suggest a relationship between a general measure of pharmacological addiction and response to cigarette deprivation.

C: PHYSIOLOGICAL, PSYCHOPHYSICAL AND BEHAVIOURAL EFFECTS OF CIGARETTE DEPRIVATION ON SMOKERS CLASSIFIED AS PHARMACOLOGICALLY ADDICTED AND NON-ADDICTED ON THE BASIS OF RUSSELL ET AL (1974), PHARMACOLOGICAL ADDICTION DIMENSION:

Subjects who scored at the highest and lowest 25% of the pharmacological addiction score distribution (of the present sample) were identified to form 2 experimental groups, namely, 'Addicted' and 'Non-Addicted' smokers. There were 4 females and 2 males in each group (N = 6). Table 5.4, outlines the characteristics of the 'Non-Addicted' and 'Addicted' groups.

	Mean Age	Mean No. of cigs/day (2)	Mean Pharm. Add. score	Mean Addic. Index score
Non-Addicted	46	17.83 (s.d. = 7.24)	14.99	8.0
Addicted	43	22.66 (s.d. = 4.67)	36.33	13.83

Table 5.4 Age, daily cigarette consumption, pharmacological addiction score and addiction index score for the 'non-addicted' and 'addicted' groups.

(2) - There was no significant difference between the two groups in the cigarette consumption rates ($t = 1.056$, $df = 10$; $p > 0.05$)

2 x 2 analyses of variance with repeated measures on one factor (B; Condition), were computed on pulse rate, blood pressure, hand steadiness, critical flicker fusion, peripheral visual field and finger temperature data.

The factors were:

A = Groups (A_1 = Non-Addicted; A_2 = Addicted)

B = Conditions (repeated measure, B_1 = Smoking condition; B_2 = Deprivation condition)

The "F" ratios yielded by the analyses of variance are presented in Table 5.6. (see appendix C for the ANOVA tables).

As can be seen from Table 5.5 (overleaf) the groups by conditions interaction (i.e; AB) was not significant for any of the measures utilized. The following main effects were found to be significant.

1) Condition (B): (i.e; smoking versus deprivation values)
   i) Pulse Rate ($F = 20.32$; $df = 1/10$; $p < 0.01$). There was a decrease in pulse rate from the smoking to the deprivation condition for both groups.
   ii) Systolic Blood Pressure ($F = 15.16$; $df = 1/10$; $p < 0.01$). There was a decrease in SBP from the smoking to the deprivation condition in both groups.

Figure 5.6 ((a) and (b)), (overleaf) shows the pulse rate and systolic blood pressure values in the two experimental conditions for 'addicted' and 'non-addicted' groups.

	Pulse R	Systolic B.P.	Diastolic B.P.	Hand Stead	C.F.F.T.	I. Finger Temp.
A	.288 N.S.	7.590 **	.621 N.S.	.044 N.S.	8.403 **	1.369 N.S.
B	20.322 ***	15.158 ***	1.949 N.S.	.107 N.S.	.009 N.S.	1.189 N.S.
AB	.037 N.S.	.105 N.S.	.866 N.S.	1.991 N.S.	.908 N.S.	.003 N.S.
A at B_1	.303 N.S.	7.159 **	.005 N.S.	1.134 N.S.	7.413 **	.875 N.S.
A at B_2	.109 N.S.	5.917 **	1.470 N.S.	.551 N.S.	1.895 N.S.	1.000 N.S.
B at A_1	11.044 **	8.895 **	2.707 N.S.	1.511 N.S.	1.205 N.S.	.534 N.S.
B at A_2	9.315 **	6.368 *	.108 N.S.	.587 N.S.	1.837 N.S.	.659 N.S.

	Total P.V.F.	0° Merid.	180° Merid.	90° Merid.	270° Merid.
A	.241 N.S.	.457 N.S.	.457 N.S.	.012 N.S.	2.173 N.S.
B	.283 N.S.	.089 N.S.	.164 N.S.	4.215 N.S.	.701 N.S.
AB	.022 N.S.	.412 N.S.	.048 N.S.	.441 N.S.	2.608 N.S.
A at B_1	.259 N.S.	.110 N.S.	.504 N.S.	.109 N.S.	3.868 N.S.
A at B_2	.151 N.S.	.787 N.S.	.343 N.S.	.015 N.S.	.598 N.S.
B at A_1	.073 N.S.	.443 N.S.	.194 N.S.	3.691 N.S.	3.006 N.S.
B at A_2	.232 N.S.	.058 N.S.	.017 N.S.	.965 N.S.	.302 N.S.

Table 5.5 "F" ratios derived from a 2 x 2 analysis of variance with repeated measures on factor "B", (N = 12)

Note: A_1 = Non-Addicted group, A_2 = Addicted group. B_1 = Smoking Condition, B_2 = Deprivation Condition.

* ($p < .05$)
** ($p < .025$)
***($p < .01$)

df = 1/10 for A; B; B at A_1 and A_2
1/20 for A at B_1 and B_2

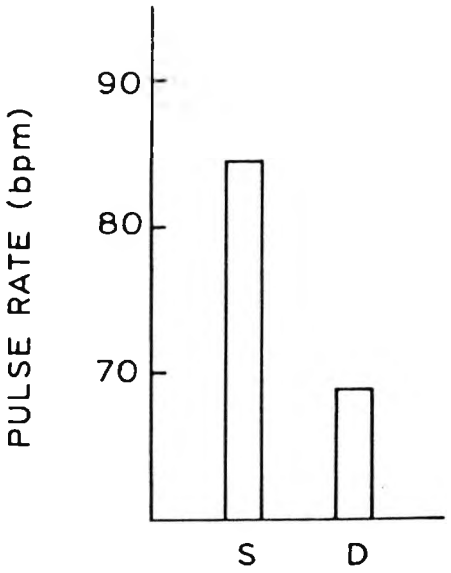

Fig. 5.6(a) Mean pulse rate: Main conditions (B) effect

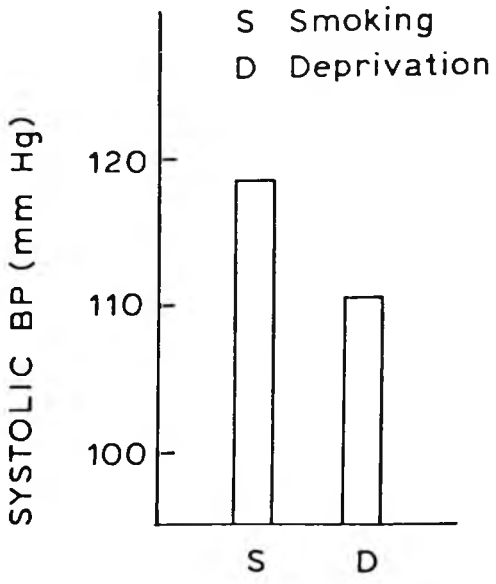

Fig. 5.6(b) Mean SBP: Main conditions (B) effect

No significant condition effect was noted for the other measures utilized.

2) Groups (A): (i.e; addicted versus non-addicted)

i) Systolic Blood Pressure ($F = 7.59$; $df = 1/10$; $p < 0.025$). Addicted and non-addicted groups were found to have significantly different SBP values in the smoking ($F = 7.16$; $df = 1/20$, $p < 0.025$) and also in the deprivation conditions ($F = 5.92$; $df = 1/20$; $p < 0.025$), the non-addicted group having a higher SBP on both occasions. The SBP values are presented in Figure 5.7 (overleaf)

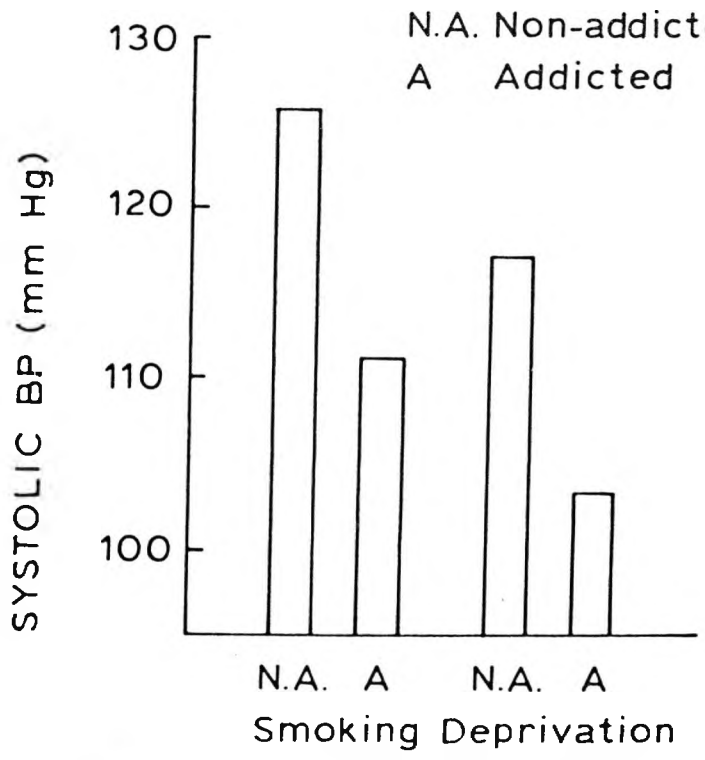

Fig. 5.7 Mean SBP values for the addicted and non-addicted groups in the smoking and deprivation conditions

ii) Critical Flicker Fusion Threshold ($F = 8.403$; $df = 1/10$; $p < 0.025$). Further analysis revealed that the groups were only significantly different in the smoking condition ($F = 7.413$; $df = 1/20$; $p < 0.025$). An inspection of figure 5.8, (overleaf) shows that the non-addicted group had a significantly higher CFFT in this condition.

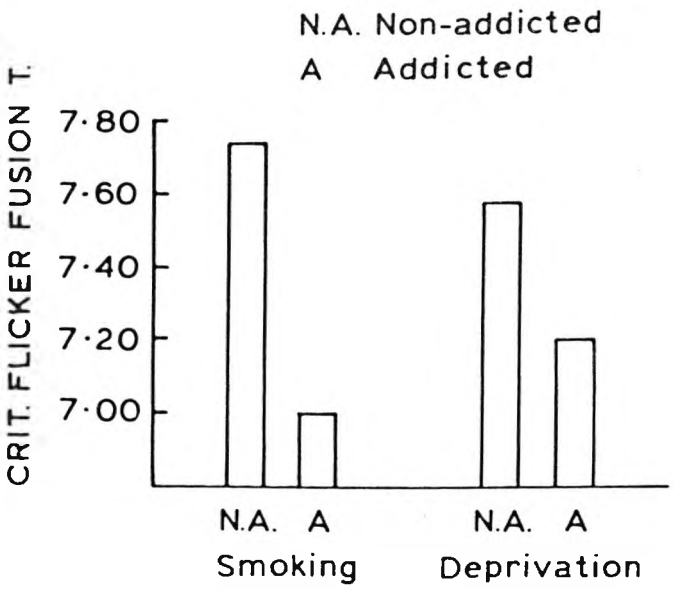

Fig. 5.8 Mean CFF thresholds for the addicted and non-addicted groups in the smoking and deprivation conditions

There was no significant groups (A) effect in the other measures.

DISCUSSION

The results of the analysis of variance presented in the preceding section provide two main findings. Firstly, it has been noted that deprivation produces a lowered level of autonomic activation as manifested by a decrease in pulse rate and SBP from the smoking to the deprivation conditions for both groups. This finding is in agreement with the results of the 't' test presented in section (A).

Secondly, although the groups by conditions interaction was not found to be significant for any of the measures, it was noted that the

addicted and non-addicted groups differed significantly in SBP and CFFT. The non-addicted smokers had a significantly higher SBP in both of the experimental conditions as compared to the addicted group. Although, the higher SBP for the non-addicted group in the smoking condition could be indicative of a lower tolerance level to nicotine, without appropriate measures of nicotine intake it does not seem advisable to speculate on this finding. Since the groups differ significantly in both conditions, the differences may well be due to constitutional differences between the subjects in this measure.

On the other hand, for CFFT, the difference between the groups was only noted in the smoking condition. The non-addicted group had a higher CFFT (indicative of greater sensitivity) in the smoking condition as compared to the addicted smokers. No significant difference was noted between the groups in the deprivation condition. Although, without any measure of nicotine intake it is difficult to derive a firm conclusion from this finding, it is interesting to note that smoking raises the CFFT (indicative of cortical arousal, see chapter 4) of the non-addicted smokers significantly above that of addicted smokers. This finding indicates that the non-addicted smokers obtain a more pronounced effect from smoking.

So, in conclusion, apart from the effects noted for CFFT, the results of the analyses of variance did not provide evidence to support the hypothesis that 'addicted' and 'non-addicted' smokers are differentially effected by smoking deprivation. On the other hand, a differential effect of normal smoking was noted for CFFT, which showed that smoking produced a higher CFFT in the 'non-addicted' group as compared to the 'addicted' group. This was the only finding in line with the expectation that smoking and/or deprivation will have different effects on the two experimental groups.

D): SMOKING TYPOLOGY SCALE SCORES: COMPARISON OF TL SCORES OF THE PRESENT SAMPLE (N = 23) WITH RUSSEL ET AL'S 'MAIN SMOKERS' (N = 174) AND 'SMOKING CLINIC' (N = 103), SAMPLE:

i) Figure 5.9 (overleaf), illustrates the factor scores (i.e; each factor score divided by the number of items contributing to that factor) of the present sample in relation to the two samples (i.e; main smokers and smokers clinic) employed by Russell et al (1974).

A marked difference was observed between the factor scores of the present sample and that of Russell et al's in the smoking types contributing to the pharmacological addiction dimension (i.e; Stimulation, Addictive, and Automatic smoking factors). The scores of the present sample were similar to the " smoking clinic sample" of Russell et al , with the exception of male smokers scoring lower than the clinic sample on the Automatic factor.

Figure 5.10 shows the factor scores of the 'Addicted' and 'Non-Addicted' groups identified in the present study and Russell et al's samples, on the smoking types included in the pharmacological addiction dimension.

As can be seen from Fig. 5.10 (overleaf), the " Addicted" group scored higher than the "smoking clinic" sample on the "stimulation" "addictive" and "automatic " factors. On the other hand the " Non- Addicted" group scored close to the smoking clinic sample on the stimulation factor.

ii) The factor scores on the seven smoking factors derived from Russell et al 'Typology Test' (i.e; Stimulation, Addictive, Automatic, Indulgent, Psychosocial, Sensorimotor and Sedative factors), for the present sample were subjected to a ranked order correlation matrix analysis. The correlations thus obtained are presented in Table 5.7 (overleaf).

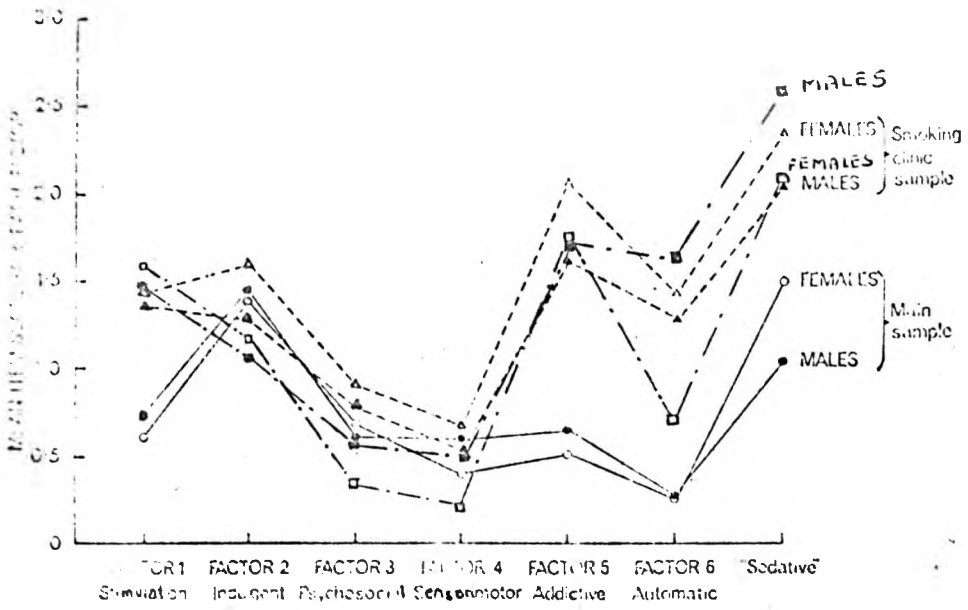

Fig. 5.9 Mean item scores for each factor by sex for the present study and Russell et al (1974) "smoking clinic" and "main sample"

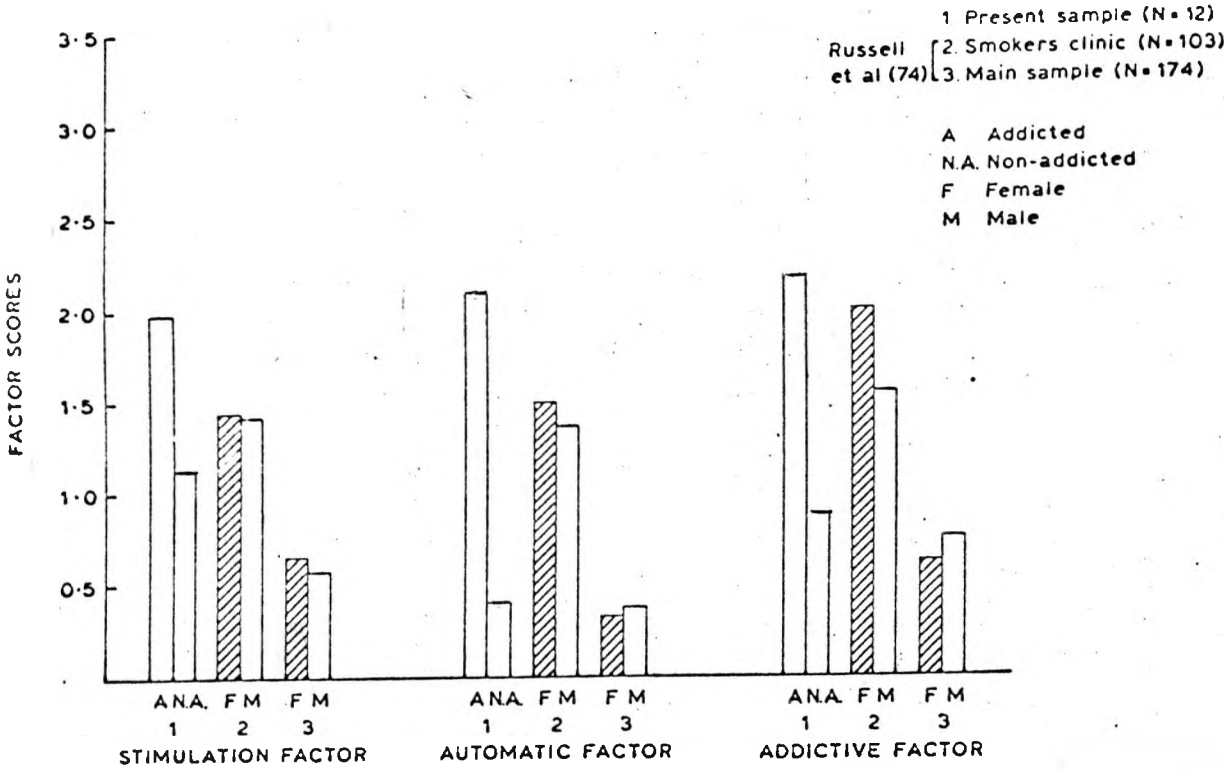

Fig. 5.10 Mean factor scores for the "addicted" and "non-addicted" groups of the present study and Russell et al (1974) "smoking clinic" and "main sample".

	Sensorimot.	Indulgent	Stimul.	Automat.	Addict.	Sedative
Psychosoc.	0.433*	0.191	-0.071	0.229	0.134	0.111
Sensorimot.		0.015	0.255	0.202	0.506**	0.200
Indulgent			0.032	0.024	-0.219	0.024
Stimulation				0.325	0.184	0.400
Automatic					0.393	0.229
Addictive						0.616***

Table 5.7 Inter-Correlations of the factor scores on the seven sub-scales of Russell et al (1974) Typology Scale.

* $p < 0.05$

** $p < 0.02$ df = 23

*** $p < 0.01$ (two-tailed test)

As can be seen from Table 5.7, the following significant correlations were noted:

1 - Psychosocial and sensorimotor factors were found to be positively related ($r = 0.43$, $p < 0.05$)

2 - A positive correlation was noted between the sensorimotor and addictive factors ($r = 0.506$, $p < 0.02$).

3 - Addictive factor was found to be positively related to the sedative factor ($r = 0.616$, $p < 0.01$).

DISCUSSION

Mean factor scores of the present sample in comparison with the main smoking sample and smokers clinic sample of Russell et al (1974), showed a marked difference in the pharmacological addiction dimension

(i.e: stimulation, addictive and automatic factors) (see fig.5.9). The scores of the present sample were similar to the "smoking clinic " sample of Russell et al, with the exception of male smokers scoring lower than the clinic sample on the "Automatic" factor. Considering that there were only eight males in the present sample, even for the automatic factor 65 % of the present sample scored slightly higher than the clinic sample on this factor. Russell et al(1974), used the higher scores obtained by the clinic sample to support their postulated pharmacological addiction dimension. A further figure was produced to investigate the factor scores of the "addicted" and "non-addicted" groups of the present sample, in comparison with the Russell et al's samples (see fig 5.10). The addicted group was noted to score higher than the smoking clinic sample on the stimulation, automatic and addictive smoking factors. The non-addicted smokers were noted to score higher than the main smokers sample of Russell et al, on the stimulation and addictive factors.

So, it was seen that the present sample, who were all voluntary respondents to the newspaper article, resembled the smokers clinic sample in their scores on the pharmacological addiction dimension.

In order to investigate the differences between groups formed on the basis of the score distribution on the pharmacological addiction dimension it is necessary to obtain representative samples (i.e; subjects scoring close to or less than the main sample, and those who score close to or higher than the clinic sample). This has been the major drawback of the present investigation. The failure to substantiate the hypothesis that deprivation will affect the addicted and non-addicted groups differentially

could well be due to this sampling error. It was seen that smokers who express a wish to discontinue smoking and who were willing to commit themselves for experimentation on two consecutive days, in majority score high on the pharmacological dimension. It is interesting to note that the present sample scored even higher than the smoking clinic sample, which might again reflect differences in the degree of motivation. The smokers clinic sample might have included smokers who were referred to the clinic by their physicians and they might not have been aware of the degree of their addiction, whereas the present sample (100%), reported that they want to give up smoking and expected treatment at the end of experimentation. They might have biased their responses to the typology test, by trying to present themselves as addicted smokers. So, it seems advisable to use alternative methods of subject recruitment in order to obtain representative samples of addicted and non-addicted smokers.

Correlational analyses between the factor scores for the present sample on the seven sub-scales of Russell et al's typology scale revealed that the scores on the sensorimotor (i.e; non-pharmacological) and addictive (i.e; pharmacological) and sedative and addictive factors were positively related. It is of value to note that smokers scoring high on addiction also tend to score high on sedative smoking. This finding points out that sedative smoking may also be related to pharmacological addiction. Although, presently, evidence to support the sedative effect of smoking is slender (see chapter 4), it seems premature to exclude sedative smoking as a smoking type.

On the other hand the relationship between scores on the sensorimotor and addictive factors does not lend support to an independent general pharmacological addiction dimension since scores on the sensorimotor

factor which loads on the non-pharmacological dimension were found to be significantly related to addictive smoking, which belongs to the pharmacological dimension.

iv) GENERAL DISCUSSION AND CONCLUSIONS

The present study was intended to provide a preliminary assessment of the effects of cigarette deprivation as manifested by physiological, psychophysical and behavioural measures in a voluntary adult sample. The specific interest was to investigate the differential effects of deprivation (i.e; Differences between normal and deprivation condition values) on smokers classified as pharmacologically 'addicted' and 'non-addicted' according to the scores on Russell et al (1974), Smoking Typology scale.

The findings of this study, did not provide evidence to support the hypothesis that 'addicted' smokers will exhibit more pronounced withdrawal symptoms after a period of cigarette deprivation.

The effects of deprivation noted in the present study were in line with the findings of previous researchers. A lowered level of autonomic activation as manifested by a drop in pulse rate and systolic blood pressure from the smoking to the deprivation condition was noted for the whole sample. Although, this finding in itself was not the major focus of the present study, it indicates that the subjects did comply with the instructions and abstained from smoking in the deprivation condition.

The two indices of pharmacological addiction (i.e; Addiction Index score, and Pharmacological addiction score), employed were not found to be related to the change in the physiological and behavioural measures brought about by the experimental conditions (i.e; smoking and deprivation).

Thus, the correlational analyses did not provide evidence to support the view that degree of addiction may be related to the degree of withdrawal effects. However, it was noted that addiction and the change in diastolic blood pressure from the smoking to the deprivation condition was negatively related. Although, this finding is contrary to the view that addicted smokers will exhibit more pronounced withdrawal symptoms, it may be explained by a possibly higher level of tolerance to the effects of smoking. If the addicted smokers have developed a higher level of tolerance to smoking and/or nicotine then a single cigarette may not produce a large change in their DBP (i.e; in the smoking condition). Thus, since the effects of deprivation are assessed in relation to the values in the smoking condition the change for the addicted smokers may seem small. The differential effects of smoking on the addicted and non-addicted groups in critical flicker fusion test, lends support to this view. It was noted that CFFT following smoking was significantly higher (i.e; more sensitivity) for the non-addicted smokers, which indicates that the single cigarette smoked prior to testing had a more pronounced effect for these subjects. Although, these results supported the view that the non-addicted group had a lower tolerance for nicotine (i.e; the time course for the dissipation of nicotine in their blood may be slower) it can not be explained by differences in consumption levels. No significant difference was noted between the daily cigarette consumption rates of the addicted and non-addicted smokers. (See section C).

Except for the differential effects of smoking on CFFT, as mentioned above, the results of the present study did not support the expectation that the two experimental groups will be differentially affected by deprivation. However, there were two major shortcomings in the present study: the timing of the measurements and the sample bias.

It has been pointed out earlier that the plasma half life of nicotine have been reported to be less than 30 minutes (Isaac & Rand, 1972), so that since the present experiment took about 50 minutes some of the measures were taken when the effects of the single cigarette smoked prior to testing would have dissipated. So, it seems more appropriate to take continuous recordings of physiological measures before, during and after smoking.

The pharmacological addiction scores (i.e; Sum of factor scores on the stimulation, addictive and automatic factors) of the present sample were noted to be markedly higher than the smokers clinic sample of Russell et al. Even the subjects scoring at the lower 25% of the present score distribution were found to score close to the smokers clinic sample which was used as a criterion group of heavy addicted smokers. This finding indicated that the present sample were biased towards the pharmacological dimension. The failure to support the experimental hypothesis could well be due to this sampling bias.

CONCLUSIONS

Classifying smokers according to their position on the single dimension of pharmacological addiction to nicotine, as suggested by Russell et al (1974), seems very promising for a treatment approach emphasizing the tailoring of treatment of subject characteristics. However, first of all it is necessary to provide a satisfactory definition of pharmacological addiction. We may define pharmacological addiction to smoking as any type of smoking that is predominantly maintained by the pharmacological effects of smoking and/or nicotine. It seems more appropriate to regard the total pharmacological addiction score proposed by Russell et al as an indicator of degree of pharmacological addiction, rather than as a unitary type or class. For

example, the intensity and the frequency of pharmacological effects may depend on the frequency of smoking. Some smokers report that they only smoke in certain situations (i.e; high or low arousal - Frith). Thus, even light smokers, if they consistently smoke at certain time periods or occasions may be dependent on the pharmacological effects of smoking. Since, the main goal of the typology research is to understand the mechanisms and motives of smoking for individual smokers or groups of similar smokers, a gross measure of pharmacological addiction seems to be defeating the original aim.

The findings of the present study did not substantiate the hypothesis that the position of scoring on the pharmacological addiction dimension is related to the effects of deprivation. However, there was a failure in formulating representative samples and a procedural flow in the timing of the measurements. A second study, which will be presented in the next chapter, was designed in order to investigate the same hypothesis as formulated in this chapter, but employing continuous recording procedures, a selection criteria for the subjects, and equal hours of deprivation for all subjects.

5.5)

CHAPTER SUMMARY

In an attempt to validate Russell et al (1974) Typology Scale and to investigate the effects of cigarette deprivation, 23 adult smokers (15 female and 8 male) were tested under a normal smoking and a deprivation condition, each subject serving as his/her own control. Pulse rate, systolic and diastolic blood pressures, hand steadiness, critical flicker fusion threshold, peripheral visual field and index finger temperature were measured under the two experimental conditions, on two consecutive days, at the same time of the day. Subsequently, two experimental groups, namely 'Pharmacologically Addicted' and 'Non-Addicted' were formed according to scores on the pharmacological addiction to nicotine dimension. Significant condition effects were noted for pulse rate, systolic blood pressure and peripheral visual field for the whole sample. Also, significant group effects were noted for systolic blood pressure and critical flicker fusion threshold, the non-addicted smokers scoring higher on both measures in the smoking condition.

The findings of the present study failed to demonstrate differential effects of smoking and deprivation on the two experimental groups, however, this could be due to an experimental error in obtaining representative samples and some procedural (i.e; timing) errors. Suggestions for future work and criticisms of a general pharmacological dimension have been made.

CHAPTER 6

EXPERIMENT II

PHARMACOLOGICALLY ADDICTED AND NON-ADDICTED MALE SMOKERS: DIFFERENTIAL RESPONSIVITY TO CIGARETTE SMOKING AND DEPRIVATION IN PHYSIOLOGICAL MEASURES, NICOTINE INTAKE, SMOKING PARAMETERS AND ATTENTIONAL PERFORMANCE.

6.1 INTRODUCTION

Although, the results of the first experiment presented in the preceding chapter did not bear out the expectation that "addicted" and "non-addicted" smokers are affected differentially by cigarette deprivation, it pointed out that non-addicted smokers may have a lower tolerance level to the effects of nicotine. However, the scoring distribution of the subjects and error in the timing of measurements limited the conclusions that can be drawn from the first experiment.

The present study was designed in order to investigate the differential effects of cigarette smoking and deprivation on pre-selected groups of "addicted" and "non-addicted" smokers. Considering the shortcomings of the previous study continuous recording procedures and a criterion typology score (i.e; for pharmacological addiction) for the allocation of subjects into the experimental groups were employed. A control group of non-smokers was also used to control for the time effect (i.e; changes associated with experimental manipulations other than smoking).

In addition to the physiological measures, topographical smoking measures (i.e; cigarette and puff duration, inter puff intervals, etc.) nicotine intake (from butt nicotine analysis), the latency to a second cigarette smoked voluntarily and attentional performance in an auditory

vigilance test was also taken. The sensory threshold tests used in the previous experiment were not used due to the restrictions imposed by electrode application for the physiological measures.

The present study was a further attempt to investigate the correspondence between classifications derived from verbal reports and physiological and behavioural concomitants of cigarette smoking and deprivation. The main assumption underlying both of the experiments of this thesis is that the smokers who report (i.e; typology scale scores), that they will experience withdrawal symptoms on cessation of smoking and who give reasons for their smoking, which are in line with the pharmacological effects of smoking and/or nicotine are likely to exhibit more pronounced physiological and behavioural withdrawal symptoms with deprivation. The problem one encounters here is whether the verbal reports of smokers reflect the physiological and behavioural changes brought about by deprivation or whether they are shaped by cognitive (i.e; attitudinal) or other factors. In other words, 'are smokers aware (i.e; are able to describe) of the physiological and behavioural effects of smoking and deprivation?'

Thus, the present experiment was designed to address the above issue, investigating the differential effects of smoking and deprivation on some physiological and behavioural-attentional measures, in two groups of smokers (i.e; pharmacologically addicted and non-addicted) identified on the basis of their scores on Russell et al (1974), smoking typology scale.

6.2 METHOD

i) Subjects:

Subjects were paid male students (£1/hour), recruited by a notice advertised in the union and departmental notice boards of Hull University,

requiring male smokers and non-smokers to participate in an experiment on the effects of giving up smoking.

The respondents (smokers only) were required to complete Russell et al (1974), Smoking Typology scale. According to their scores on factors contributing to the pharmacological addiction dimension they were assigned either to the 'addicted' or the 'non-addicted' experimental groups, or were not accepted if they scored outside the criterion bands.

Subjects scoring 25 or more on the pharmacological addiction dimension were assigned to the 'addicted' group (N = 9), and Ss scoring 15 or less were assigned to the 'non-addicted' group (N = 9). The respondents scoring between 15 and 25 were not accepted, (N = 14). Non-smokers (N = 9), were refused only if they were ex-smokers and had stopped smoking within the last five years.

Of the 9 male smokers assigned to each experimental group, 2 from the addicted and 3 from the non-addicted group failed to attend their second testing session, and new subjects were assigned in their place.

Table 6.1 (overleaf) shows the characteristics of the sample.

ii) General Procedures and Design:

After completing the Russell et al (1974), Typology scale, smokers were assigned to the 'addicted' or 'non-addicted' groups, if their pharmacological addiction scores met the criteria. Following this they were taken to the laboratory, where the testing took place and were given an introduction to the procedures and requirements of the experiment.

		Addicted	Non-Addicted
Mean Age (1)	Non-Smokers 25.11 (s.d = 4.457)	24.56 (s.d = 4.035)	21.67 (s.d = 4.416)
Mean numb. of cigs/day (2)		26.11 (s.d = 3.333)	11.00 (s.d = 7.67)
Mean numb. of years smoking (3)		7.44 (s.d = 3.206)	3.44 (s.d = 1.509)
Do they want to quit?	Yes	66.67 %	11.11 %
	No	33.33 %	88.89 %
Previous attempts to quit	None	-	44.44 %
	1	22.22 %	22.22 %
	2	11.11 %	11.11 %
	3 - 7	66.67 %	22.22 %
Types of cigs. smoked:	Manufac. filter	66.64 %	77.78 %
	Manufac. plain	-	-
	Hand rolled	33.33 %	22.22 %
Brands of cigs. smoked rated on tar-nic yield (H.D. U.K., 1975)	Low Tar-Nic	11.11 %	11.11 %
	Low-Middle Tar -Nic	-	-
	Middle Tar-Nic	55.55 %	66.67 % Hand rolled tob. users excluded.
Anticipated withdrawal symptoms (No. of Ss)	None	0	5
Lack of concentration		2	0
Tension & Irritability		7	2
Depression		2	0
Increased appetite		3	0
Craving for cigarettes		3	0
Sensorimotor deprivation (e.g; fiddling with hands, etc.)		2	2
Whether they smoke at regular time intervals	Yes	66.67 %	-
	No	33.33 %	100 %
Pharmacological Addiction Score (Russell et al, 1974, Typology Scale)		5.49	1.33
Addiction Index score		16.55	7.44

Table 6.1 Smoking history and age: 'Addicted' and 'Non-Addicted' groups.

(footnotes overleaf)

For smokers there were two experimental conditions:

a) Smoking Condition -

Subjects were allowed to smoke as usual before they arrived for testing, but were asked to refrain from alcohol and drugs.

b) Deprivation Condition -

Subjects were asked to refrain from smoking from 12 p.m. onwards the evening before the testing day (also alcohol and drugs).

Subjects (i.e; smokers) served as their own controls and were tested in both of the above conditions. Order of testing was counterbalanced in each group, four Ss in each group were first tested in the deprivation condition followed by smoking condition and five subjects per group were first tested in the smoking condition followed by the deprivation condition. For each subject the two testing sessions were completed within a week (i.e; time between the testing days ranged from 1 - 4 days). For all subjects (smokers and non-smokers) the experimental sessions were held at 2 O'clock in the afternoon.

Non-smokers had only one experimental session, and were matched randomly with smokers for the timing of experimental manipulations.

After the introductory meeting each subject (i.e; smokers) was provided with three experimental cigarettes (U.K. King size filter cigarettes). They were asked to smoke these cigarettes the day before their first experimental session, one as their first cigarette in the morning,

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(1) - Difference between the two groups was not significant ($t = 1.45$, $df = 8$, $p > 0.05$)

(2) - Significant difference between the two groups ($t = 5.42$, $df = 8$, $p < 0.001$)

(3) - Significant difference between the groups ($t = 3.39$, $df = 8$, $p < 0.02$)

(two-tailed test)

second as their first cigarette after lunch and the third as their first cigarette after dinner. They were asked to place the butts of these cigarettes in the plastic bags provided and to return them to the experimenter (see appendix D-i for the instructions). Subsequently, these butts were sent to the British American Tobacco Company and were analyzed for nicotine.

The date for the first experimental session was arranged and the Ss were given written instructions for their first session (See appendix D-ii).

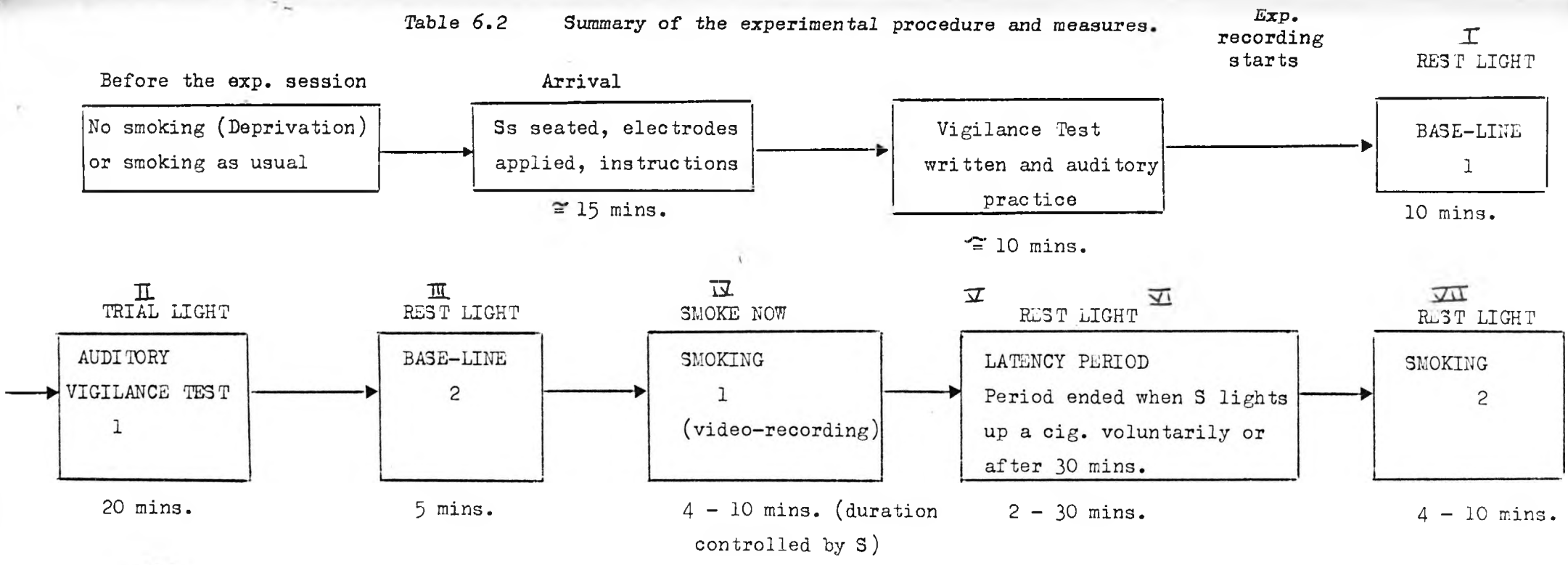
iii) Experimental Sessions: Procedures, measures taken and apparatus:

The procedures for the two experimental sessions and the measures taken are outlined in Fig. 6.2 (overleaf).

The experiment took place in a laboratory (Human Performance Lab.), equipped with psychophysiological recording apparatus. On arrival the subject was asked to wash his hands with soap and water (for the skin conductance measurement, as recommended by Venables & Christie, 1979), and was then taken to the experimental cubicle which was sound and light attenuated. He was seated on a reclining garden chair. Electrodes for the measurement of heart rate, respiration and skin conductance were applied. Following this the subject was asked to read the general procedures and instructions for the experiment (See appendix D-iii). He was then given written (see appendix D-iv) and auditory practice in the vigilance task.

After the completion of this preparatory period the experimental recording was started. Experimental periods were signalled to the subject by a panel located on the wall facing the subject. Three signals were used throughout the experiment, namely: 'Trial', 'Rest', and 'Smoke, Now'.

Table 6.2 Summary of the experimental procedure and measures.



MEASURES

Physiological (Contin. recording)

1 - Heart rate; 2 - Respiration; 3 - Skin conductance.

Smoking:

1 - Videotape recording of smoking analysed for:
Number and duration of puffs, cigarette duration, etc.

2 - Butt-nicotine analysis.

Vigilance test performance

These signals were illuminated from a control switch outside the experimental cubicle. At the end of the first smoking period the experimenter communicated with the subject through an intercom (Eagle International transistor intercom), to inform him that there is no restriction on his smoking ("In this rest period you are free to smoke. You can smoke a cigarette whenever you want to while the rest light is on"). Timing of the rest periods were made by a stopwatch.

All the physiological measures were continuously monitored using a Grass 7 D polygraph. The signals were also stored on a magnetic tape recorder (Racal, Thermionic, Store 4) for subsequent off-line computer analysis. Subjects were filmed during the two smoking periods by a videocorder (Sony - AV-3620CE) for subsequent analysis of the smoking topography measures, and were monitored throughout the latency period in order to mark the time elapsed between the first and second cigarettes. Figures 6.1 and 6.2 (overleaf), show the apparatus utilized. The experimental chamber, and electrode positions on a subject can be seen from Fig. 6.3 ((a) and (b)).

Physiological measures:

(Recorded by Grass 7D Polygraph and stored on magnetic tape (see Fig. 6.1 and 6.2) for off-line analysis).

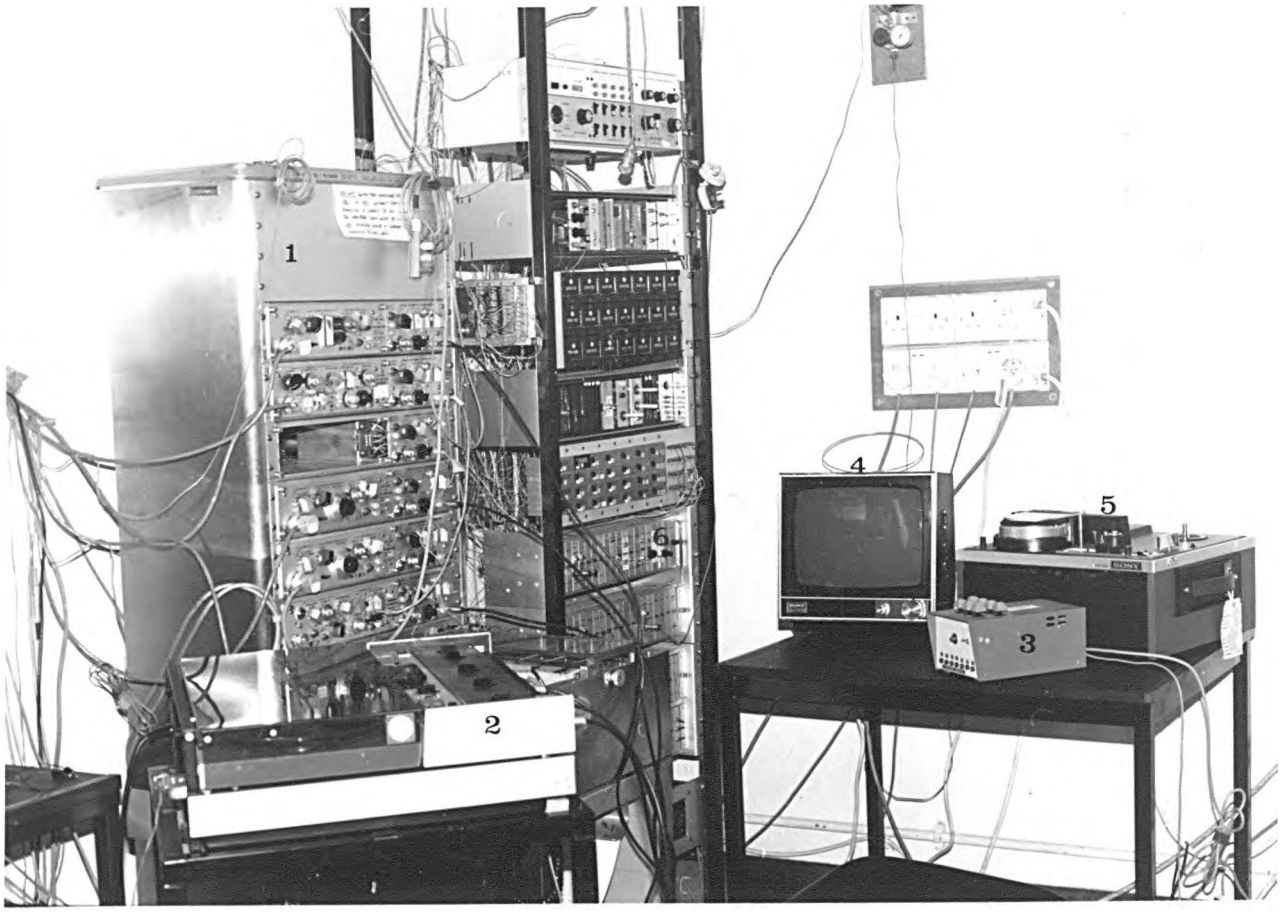


Fig. 6.1 Side view of the apparatus.

1 - Grass 7D Polygraph; 2 - Magnetic Tape Recorder (Racal-Thermionic, Store 4)

3 - Event Marker (connected to the tape recorder); 4 - Video-television;

5 - Videocorder (Sony).

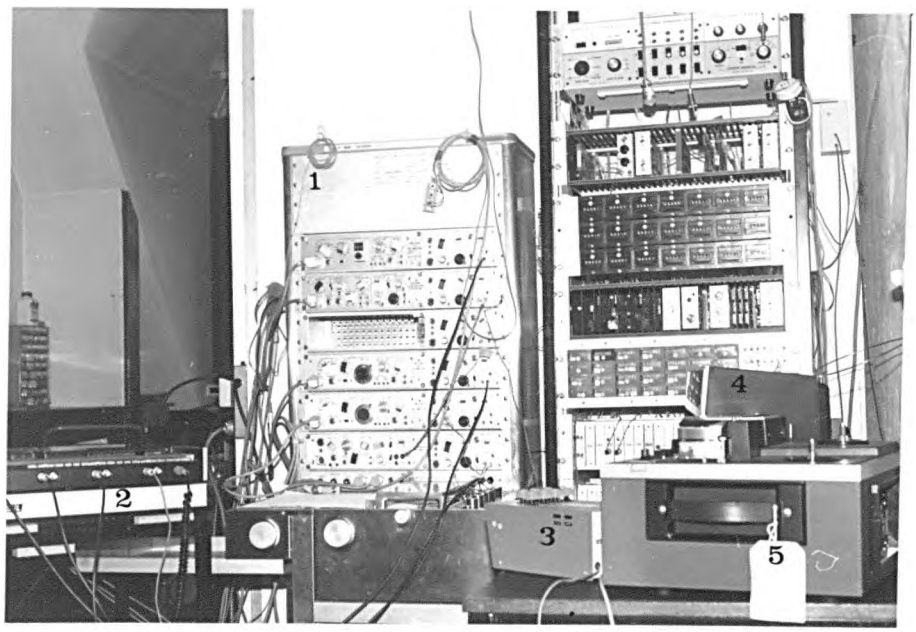
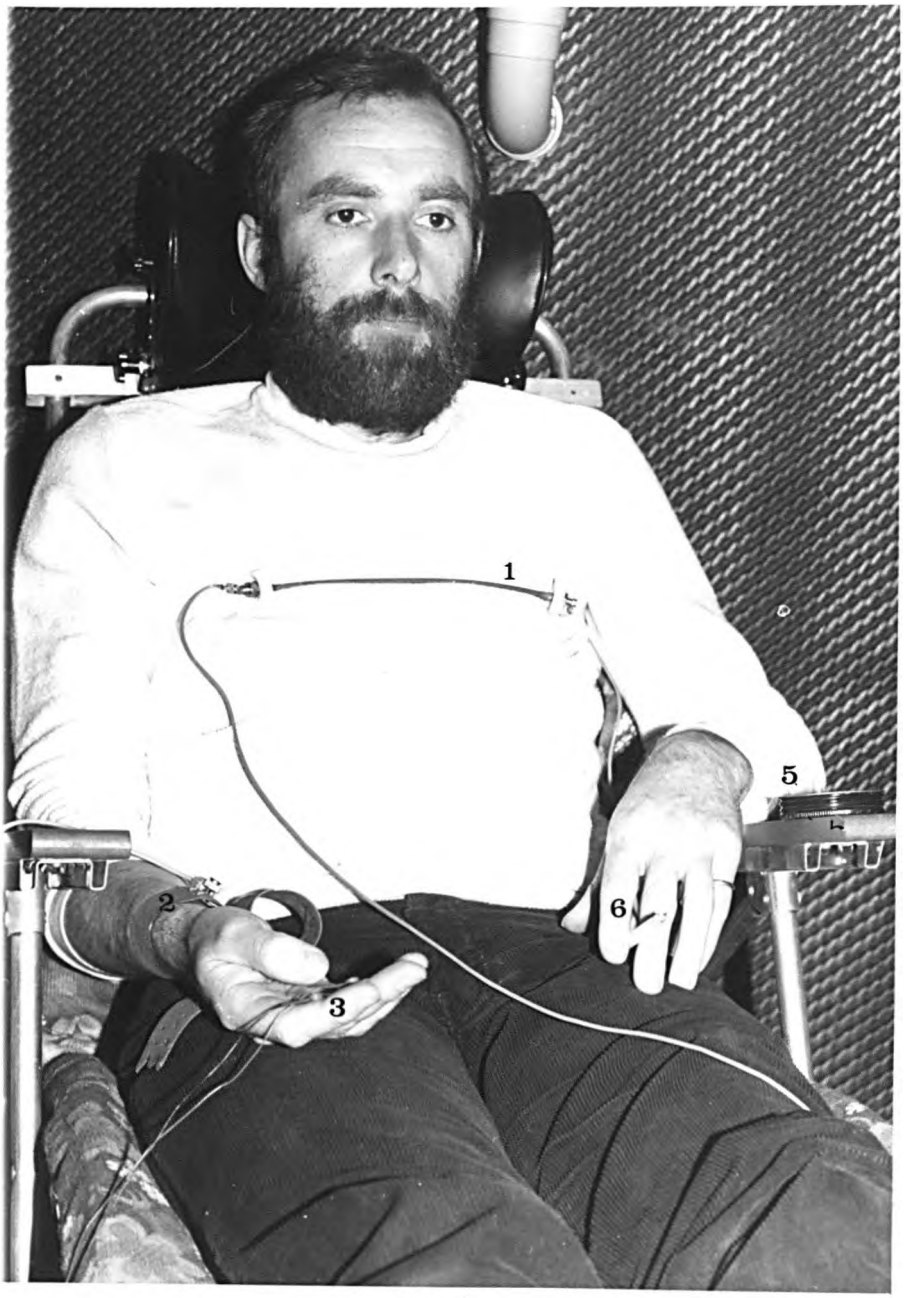


Fig. 6.2 Front view of the apparatus.

(a)

Front view



1; Strain gauge

2:& 4: Lead II ECG electrodes.

3: Skin Cond. electrod.

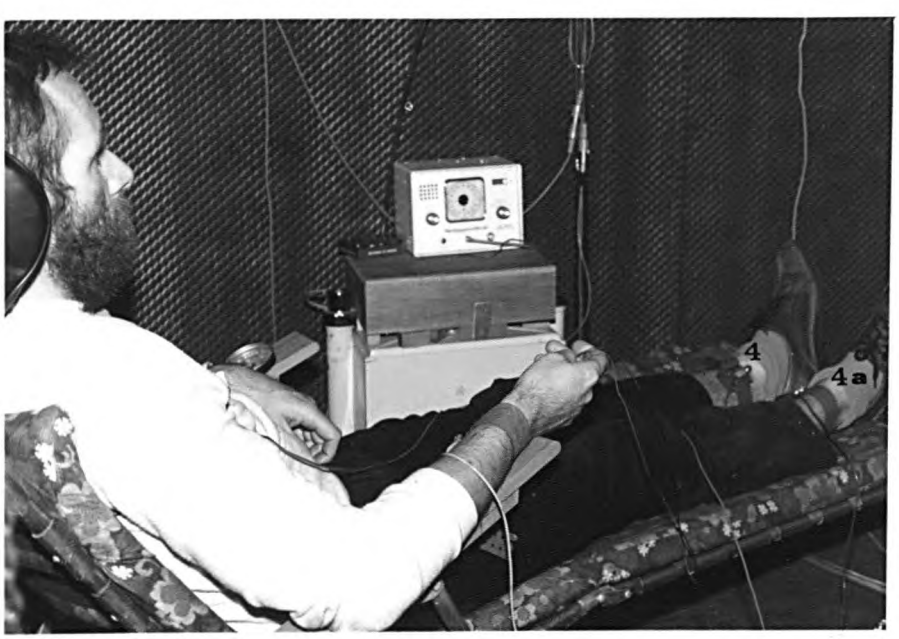
5:Ash-tray.

6: Smoking with left hand.

Fig.6.3 (a) & (b) View of the experimental cubicle and electrode locations.

(b)

Side view



a) Heart Rate: (Electrocardiogram [4] - ECG and Cardiotachometer [5])

Standard bipolar limb lead II (i.e; between the right wrist and left ankle, right ankle earth) was used for the ECG recording (see Fig. 6.3 (a) and (b). Before the electrodes were applied the electrode sites were wiped with alcohol. The ECG electrode cream (Cam Creme, Cambridge Medical Instruments, Ltd.), was spread over the electrodes, which were then applied to the prepared sites. The electrodes were secured in place by adjustable rubber straps.

Polygraph channel 2 (Wide Band A.C pre-amplifier, time constant: 0.2 secs* and D.C driver amplifier) was used for ECG recording and channel 6 (Tachograph pre-amplifier and driver amplifier model 7 DA C, Range = 40 - 120 bpm) for the cardiotachometer. The signal from the driver amplifier output J6, was fed into the magnetic tape recorder and stored for off-line computer analysis.

Sections of polygraph records for the ECG and cardiotachometer can be seen from Figures 6.4 and 6.5.

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(4) - By placing electrodes at strategic loci on the body surface the electrical activity of the heart (i.e; sequence of depolarization and repolarization), during successive cardiac cycles are recorded as ECG. The external action potential detected is referred to as the QRS complex (i.e; deflections corresponding to ventricular excitation) (Thompson & Patterson, 1974).

(5) - Cardiotachometer functions as a time to amplitude convertor. It measures the time between successive R waves and provides a voltage output proportional in amplitude to the HR equivalent of the interval between two R waves.

* - This is a short time constant for ECG recordings and was employed to minimize amplifier blocking which otherwise followed movement-induced artifacts.

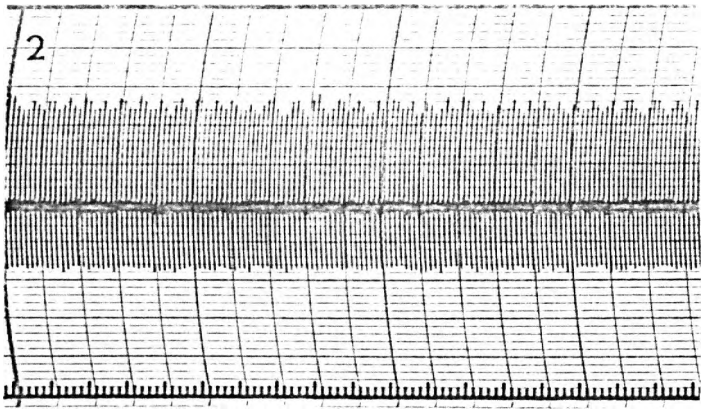

Fig. 6.4 Section of polygraph "ECG" record, (paper speed = 1.0 mm/sec.)

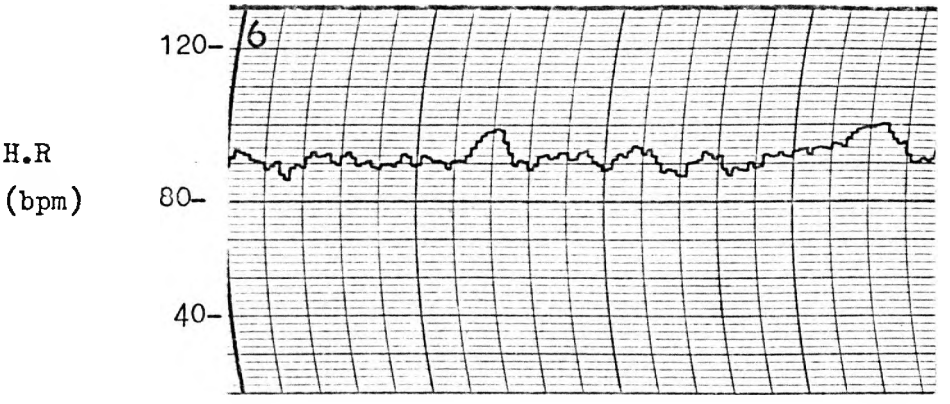

Fig. 6.5 Section of polygraph "cardiotachometer" record (paper speed = 1.0 mm/sec.)

b) Respiration:

A strain gauge[6] (18 cm. long, 7 mm.diameter rubber tube filled with copper sulphate solution, attached to leather straps), was used to record the respiratory activity. The strain gauge was strapped around the chest and the leads were connected to polygraph channel 1 (low level DC pre-amplifier, 10 mv/cm, driver amplifier model 7DA D) (See Fig. 6.3 - 1).

---

(6) - The changes in the size of the chest circumference during each respiratory cycle (i.e; inspiration and expiration) can be detected by the strain gauge. The electrical resistance between the two ends of the rubber tube changes (i.e; during inspiration it increases whereas during expiration it decreases), due to the changes in tension produced by chest movements. This resistance change is detected by the polygraph.

Fig. 6.6 shows a section of polygraph recording of respiratory activity.

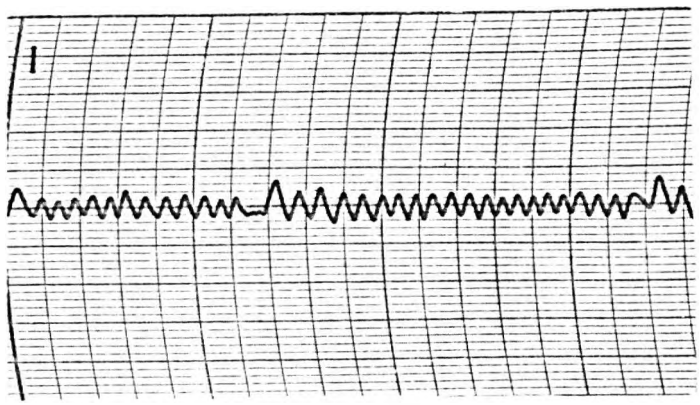

Fig. 6.6 Section of polygraph "respiration" record, (paper speeds = 1.0 mm/sec.)

The signal from the driver amplifier (out J6) was fed into the magnetic tape and stored for off-line analysis.

Respiration rate (i.e; number of peaks per minute), and respiration amplitude (i.e; Difference between the highest and lowest points in each respiratory cycle) were subsequently analyzed on the computer.

c) Skin Conductance:

Skin conductance was measured by the constant voltage method, using bipolar electrodes (silver/silver chloride disc electrodes, 1.0 cm. diameter, 0.3 cm. diameter center) placed on the medial phalanges of the index and middle fingers of the right hand (see Fig. 6.3 - 3).

The subject was asked to wash his hands with soap and water. The center of the electrodes were filled with Beckman (electrodeelectrolyte) cream. The electrodes were held in place by double sided adhesive colors (2.0 cm. diameter, with a 0.4 cm. diametered hole in the center).

Polygraph channel 4 (low-level DC pre-amplifier, 1 mv/cm, with model 7 DA C Driver amplifier) was used for recording the skin conductance level (SCL) (1 micromho = 1 cm. deflection on the polygraph record). An offset adjustment on channel 4 was used to set the initial SCL within the recording range and during the course of the experiment whenever SCL exceeded 2 cms. above or below the baseline (i.e; center line).

Every time a new setting was made a signal was recorded on the event channel of the tape recorder (manipulated by the experimenter by pressing a button on an event marker). The new offset values were fed into the computer in the course of off-line analysis.

Fig.6.7, shows a section of the polygraph record for the skin conductance.

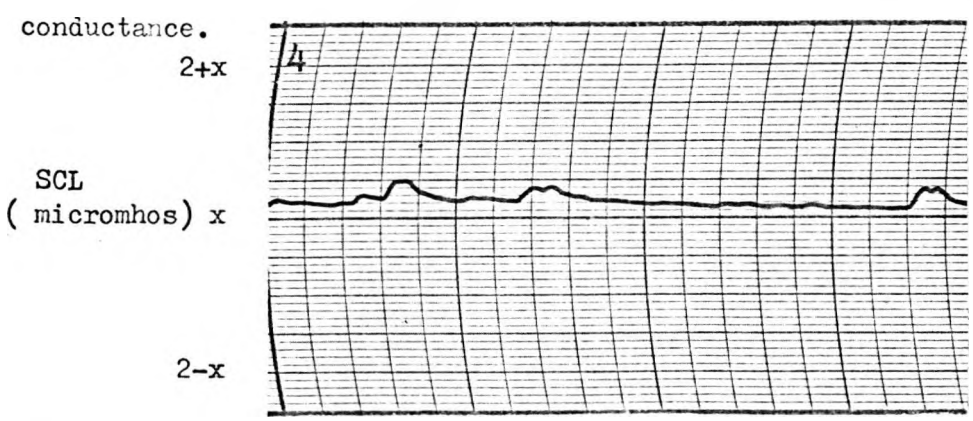

Fig. 6.7 Section of polygraph "Skin Conductance" record (x = the SCL offset value)

In the course of off-line analysis two measures were derived from the skin conductance data.

1 - Skin Conductance Level (SCL): tonic level of activity in micromhos.

2 - Non-Specific Skin Conductance responses (NS.SCR) or Lability (7) :

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(7) - Lability or NS.SCR's refers to fluctuations in electrodermal activity, which have the appearance of responses but which nevertheless cannot be associated with a particular, identifiable, external stimulus (Venables & Christie, 1979). NS.SCR's were found to increase in response to the administration of amphetamines and to decrease in response to barbiturates (Burch and Greiner, 1960). Elevated rate of spontaneous electrodermal responses are generally related to biologically or psychologically induced arousal states (Katkin, 1966; Burch & Greiner, 1960).

Response criteria; an increase followed by a decrease in S. Conductance of 0.03 micromhos magnitude, that occurs within 3 seconds.

DATA ANALYSIS

Signals from the polygraph driver amplifier (out J6) were fed and stored on the magnetic tape recorder (4 channels) for off-line computer analysis. The three channels of the tape recorder were used for storing heart rate, respiration and skin conductance data. The fourth channel was used to signal events related to the beginning and end of experimental periods and for marking the change in the offset value of the SCL recording. The experimenter operated the event market (see Fig. 6.2 - 3), by which two different voltages were fed into the fourth channel of the tape recorder. One for marking the periods and the second for a new SCL value.

Subsequently, the tape recorder was connected to a microprocessor based computer (Cromemco, Z-2D-Computer system). Several computer programs were developed (in Basic computer language) to process the data stored in the tape and to transfer them on to mini-disks. The tape was run automatically by the computer. One sample per second was taken, the analogue signals were converted into digital values, H.R. artifacts were eliminated and data was averaged over 10 second periods. The computer programs and sample data are presented in appendix E.

Smoking Measures:

1 - Videotape Records:

The subjects were filmed during the two smoking periods. These films were analysed for:

a) Cigarette duration (i.e; time between lighting the cigarette and putting it out (by stopwatch)).

b) Puff frequency (i.e; number of puffs from the cigarette).

c) Puff duration (i.e; glowing time, mean puff duration was calculated by dividing the total puff duration in seconds to the number of puffs minus one).

d) Inter puff interval (i.e; total puff duration subtracted from the cigarette duration).

2 - Butt-Nicotine Analysis:

The amount of nicotine taken into the mouth was estimated from the amount of nicotine retained in the butt. Butts of all the cigarettes smoked in the deprivation condition (36), the butts of the three experimental cigarettes smoked by the subject in daily life conditions (46, some Ss refused to smoke the 3 cigarettes given, especially the light smokers who stated that they never smoke before the afternoon), and some butts from the smoking condition (22) were analyzed by the British-American Tobacco Company for the amount of nicotine trapped in the filter.

The method for the measurement of nicotine by autoanalysis is given by Rothwell & Grant (1974).

The calculation of nicotine in smoke delivered to the smoker is based on the filtration efficiency (FE) of the filter and the amount of nicotine retained in the butt.

$$\text{Nic. in smoke from filter (mg)} = \text{Nic. in filter tip} \times \frac{1 - \frac{FE}{100}}{\frac{FE}{100}}$$

The experimental cigarettes used in this experiment were U.K. King size filter tip cigarettes with a filtration efficiency of 39% and nicotine yield of 1.5 mg/cig.

3 - Latency: time taken to light a second cigarette voluntarily after finishing the first cigarette. If the subject did not smoke within 30 mins. he was asked to smoke another cigarette.

Attentional Performance:

Vigilance task: Two 20 minutes vigilance tests, one before smoking and the second after smoking, were given during the experiment.

Subjects listened to a tape recording of digits, spoken at the rate of 1 per second. The task of the subject (8) was to detect signals, defined as the occurrence of three successive odd digits in one and even digits in the other test (e.g; 739 or 286) (Bakan, 1963). Each time the subject detected a signal he was asked to press a button (placed under his left hand). There were 20 signals in each test. Written and auditory practice trials of 8 mins. duration, were given before the experiment proper started (see appendix D-v).

The digits were recorded on one channel of a stereo tape recorder (Teac A-3340, 4 channel simul-sync). On the second channel corresponding to each correct sequence of stimuli a signal was recorded. Whenever the subject pressed the button at the same time or 2 seconds after this signal the response was automatically recorded as a correct detection. Any other button press was counted up as an incorrect response (i.e; false alarm). Number of correct detections and false alarms were recorded automatically by an encoder/decoder device connected to the output of the second channel of the tape recorder.

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(8) - Instruction: "Now you will hear digits from 1 to 9 spoken at a random order. Please, listen carefully, and press the button whenever you hear a sequence of three consecutive odd (or even) but unequal digits. For example, 739 (or 286) Ready?"

An index of attentional performance was calculated by the following formula:

$$\text{Performance score} = \frac{CD - FA}{20} \times 100$$

CD: Number of correct detections

FA: Number of false alarms

The order of the two tests (i.e; detection of odd or even digits) was counterbalanced across subjects of each experimental group (i.e; half taking the odd digits first followed by even digits test and the other half in the reverse order).

6.3 RESULTS

The mean values for the physiological measures in each experimental period were retrieved from the mini-disks and subjected to analyses of variance and post-hoc tests (Duncan Multiple Range test). The analyses were conducted on "Elliott 903" computer (Algol programming language).

For clarity of presentation, results (ANOVA on physiological and smoking measures) will be presented in separate sections, each part addressing a specific issue. Results for each section will be followed by an evaluation and discussion of findings. At the end of this chapter a general overview, with an interpretation of the results will be presented. However, before presenting the separate sections a summary of the statistical analysis will be provided.

Each experimental session was divided into eight experimental periods. Mean values for these periods were used in the analyses.

Experimental periods (as used in the analysis of variance tests and figures):

I: Base-line 1- (BL 1: mean of the last 5 minutes of the first base-line period),

II: Vigilance Test 1- (Vig 1: mean of the 20 minutes recording during the first vigilance test),

III: Base-line 2- (BL 2: 5 minutes mean)

IV: Smoking 1- (SM 1: mean of the first record (five minutes or less) of the "smoking 1" period) (9).

V: Latency 1 (Lat 1) - Mean of the first record (5 minutes or less) preceding the first smoking period. (10)

VI: Latency 2 (Lat 2) - Mean of the last record (5 minutes or less) prior to the second smoking period.

VII: Smoking 2 (SM 2) - Mean of the first record for the second smoking period (5 minutes or less).

VIII: Vigilance Test 2 (Vig 2) - Mean of the 20 minutes recording period for the second vigilance test.

The computer program "New.Graph" (see appendix E-vi), was used to plot more detailed histograms on the polygraph, showing the mean values of each record for each subject (smokers and non-smokers). These histograms are presented in appendix F.

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(9) - Since the duration of smoking was determined by the subjects, the records of the smoking periods were not equal in time across Ss. In the analysis, data from the first smoking record which was either the mean of 5 minutes or less if smoking terminated sooner was used.

(10) - The duration of the latency period (i.e; time taken to light a second cigarette voluntarily) was determined by the Ss. The first record after the "SM 1" period was used as latency 1 and the record before the "SM 2" was used as latency 2. However, since some Ss had a latency of less than 5 minutes, for some Ss the same data was used for the two latency periods.

Summary of the statistical analysis:

1 - Data (i.e; for heart rate, skin conductance level and lability, respiration rate and amplitude and smoking topography measures) for the addicted and non-addicted groups in the normal smoking and deprivation conditions were subjected to a three-factor analysis of variance (Factors: A = Groups; B = Conditions; C = Periods, with repeated measures on factors B and C). The ANOVA tables are presented in appendix G. A summary of the main and interaction effects is presented in Tables 6.3 and 6.4. (overleaf)

	HR	SCL	Lability	RESP. R	RESP. AMP
A	0.159 NS	0.462 NS	0.168 NS	1.163 NS	1.269 NS
B	12.613***	3.811 NS	1.055 NS	3.620 NS	0.815 NS
AB	0.001 NS	4.610*	0.400 NS	1.139 NS	2.070 NS
C	17.53****	19.632****	6.193****	29.028****	27.370****
AC	0.990 NS	2.789*	0.822 NS	2.644**	0.777 NS
BC	9.049****	1.512 NS	2.076 NS	1.486 NS	0.241 NS
ABC	0.560 NS	0.732 NS	0.609 NS	1.924 NS	1.393 NS

Table 6.3 Summary of "ANOVA" F ratios for the main and interaction effects for the physiological measures.

* $p < 0.05$

** $p < 0.025$

*** $p < 0.01$

**** $p < 0.001$

	I.P.I.	Cig. Duration	Number of puffs
A	1.310 NS	1.442 NS	0.024 NS
B	0.090 NS	0.128 NS	2.305 NS
AB	0.065 NS	2.648 NS	2.101 NS
C	3.274 NS	0.024 NS	14.391 ***
AC	0.236 NS	0.858 NS	7.418 **
BC	10.242 ***	9.758 ***	1.302 NS
ABC	0.007 NS	0.917 NS	0.443 NS

Table 6.4 Summary of "ANOVA" F ratios for the smoking topography measures.

2 - The duration of the latency period to the second cigarette in the two experimental conditions were subjected to a 2 factor ANOVA (A = Groups; B = Conditions; Repeated measures on B). The ANOVA table is presented in appendix G-ix.

3 - Correlations between questionnaire scores and pre-to-post smoking changes in physiological measures, and smoking topography measures were computed by 'Pearson r'. The correlations matrix is presented in section F.

4 - The factor scores from the 7 factors of Russell et al (1974), Typology scale and the "Addiction Index score" from the smoking habits questionnaire were also subjected to correlational analysis. This correlation matrix is presented in section F.

5 - Data for the non-smoker group were subjected to a one factor ANOVA, with repeated measures. ANOVA tables for the physiological measures are presented in appendix K. Table 6.5 (overleaf) gives an outline of the "F" values.

	H.R.	S.C.L.	Lability	RESP. R	RESP. AMP
B	8.18 ****	0.78 NS	0.54 NS	13.36 ****	2.47 *

Table 6.5 "F" Values: One factor ANOVA (repeated measures on periods, df = 7/56; * $p < 0.05$ **** $p < 0.001$)

6 - Estimated nicotine intake (i.e; from the butt-nicotine analysis) in the two experimental conditions and daily life were analyzed by ANOVA tests (see appendix H).

The subjects were divided into two groups, namely high and low-nicotine intake groups, and data for H.R. for these groups was analysed by a 3 factors ANOVA. (See appendix I).

7 - The vigilance test scores of smokers (i.e; addicted and non-addicted) and non-smokers were analysed by two separate ANOVA tests (see appendix J).

In the following results sections main and interaction effects that were found to be significant in the ANOVA tests listed above will be discussed in relevant sections.

A: DIFFERENTIAL EFFECTS OF CIGARETTE DEPRIVATION ON THE ADDICTED AND NON-ADDICTED GROUPS

The verbal reports of smokers classified as pharmacologically addicted to nicotine indicate that they experience craving and profound withdrawal symptoms when deprived of cigarettes. Cigarette deprivation has been shown to produce lowered physiological activation levels (see chapter 4). If the withdrawal symptoms and craving reported by the addicted smokers are related to the changes in activation levels, then it seems likely that the deprivation condition will produce significantly lower physiological activation

levels as compared to a normal smoking day condition in this group. Skin conductance level was the only variable to show a differential effect of deprivation on the addicted and non-addicted groups.

RESULTS AND DISCUSSION:

Fig. 6.8 shows the skin conductance levels for the addicted and non-addicted groups in the normal smoking (N) and deprivation (D) conditions.

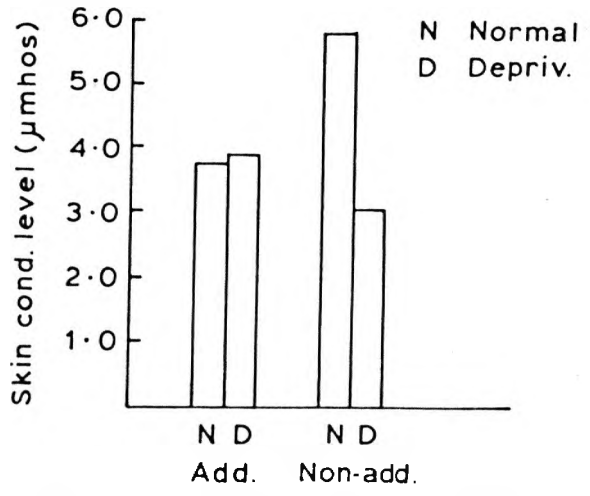

Fig. 6.8 Mean SCL's : Groups x conditions interaction.

The analysis of the skin conductance data showed that addicted and non-addicted groups were effected differentially by deprivation (ANOVA, Groups x conditions interaction, $F = 6.103$; $df = 1/16$, $p < 0.05$, see appendix G-ii). Deprivation did not alter the SCL's of the addicted group, whereas it produced a significant decrease in the SCl's of the non-addicted group (Duncan M.R.T. $df = 16$; $MSe = 32.77$, $p < 0.05$; see appendix G-ii).

The results of the SCL data were not consistent with the verbal self-reports of the addicted smokers. Cigarette deprivation does not seem to produce changes in physiological activation levels as indicated by SCL's, in addicted smokers. It was interesting to note that the non-addicted smokers were affected markedly by deprivation. Since the effects of deprivation were examined in relation to physiological activation levels in a normal smoking condition, it is plausible that the non-addicted smokers have a lower tolerance level to the effects of smoking and smoking therefore increases their SCL markedly.

Although, cigarette smoking has been noted to increase SCL's, this increase was not found to be related to the dose of nicotine inhaled (Kumar et al, 1978). Since SCL was the only measure that showed a differential effect of deprivation on the two experimental groups, these results need to be interpreted with caution.

B: DIFFERENTIAL EFFECTS OF CIGARETTE SMOKING ON THE ADDICTED AND NON-ADDICTED GROUPS

In the previous section it was suggested that the non-addicted smokers may have a lower tolerance level to the effects of smoking than the addicted smokers. The difference in tolerance levels between the two experimental groups could be due to their consumption levels. Addicted smokers were noted to have a significantly higher consumption level than the non-addicted smokers (see section 6.2). Since the addicted smokers are likely to smoke at a high frequency throughout the day one additional cigarette during a normal smoking day is not likely to produce marked changes in their physiological activation levels over the base-line values. Several physiological (i.e; SCL and RR) and smoking topography (i.e; puff-rate and butt-length) showed that smoking affected the addicted and non-addicted smokers differently. These results are presented below.

Smoking was noted to have a differential effect on the SCL's of the two experimental groups (ANOVA, Groups (A) x Periods (C) interaction, F = 2.789; df = 7/112, $p < 0.025$, see appendix G-ii).

Figure 6.9 shows the SCL values for the addicted and non-addicted groups over the 8 experimental periods.

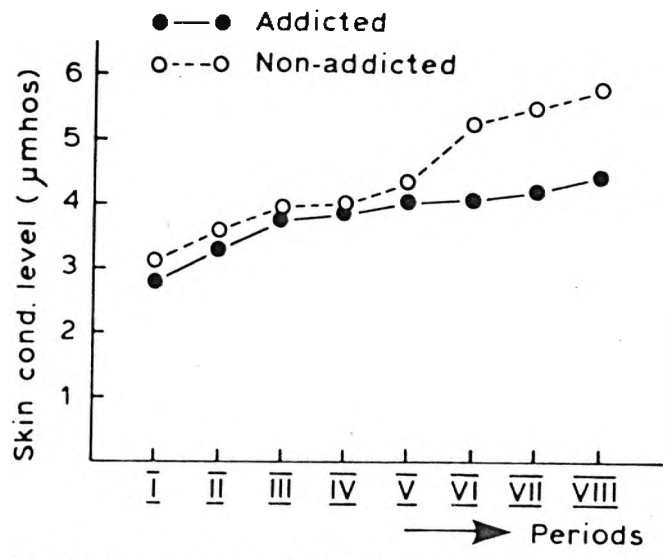

Fig. 6.9 Mean SCL : Groups x periods (AC) interaction.

Significant post-smoking changes (i.e; increase), in SCL compared to pre-smoking levels were noted for the non-addicted group, whereas for the addicted group there was no significant change in SCL following smoking (Duncan M.R.T.: df = 112; MSe = 0.998, $p < 0.05$, See appendix 6-ii).

The two experimental groups had similar SCL values till the Lat 2 period. However, from then on the non-addicted group had a significantly higher SCL as compared to the addicted group.

These results indicate that the non-addicted smokers are affected markedly by smoking as manifested by significant post-smoking increases in SCL. This finding also sheds some light on the marked difference between the normal smoking and deprivation condition values noted in the previous section for the non-addicted group. Since, smoking was noted to increase the SCL of only the non-addicted smokers, the higher SCL observed during a normal smoking day condition for this group may be explained by a more marked effect of smoking on the N.A smokers. If the major motive behind pharmacological addiction is the manipulation of arousal levels (i.e; stimulation), then judging from the SCL data the non-addicted smokers seem to be having more success in increasing their general activation levels than the addicted smokers.

Although, the differential effects of smoking on the two groups may be explained by differences in consumption levels and thus tolerance, it is useful to investigate whether the two groups differ in the way they smoke their cigarettes. It may be possible that the non-addicted group achieve marked effects not because they are more sensitive to the effects of smoking and/or nicotine, but because they smoke more intensely.

Inspection of the polygraph records showed that the typical respiratory pattern during smoking was of high amplitude and slow rate. A negative correlation was noted between the respiration rate and amplitude (see section F). The decrease in RR and the increase in R.A may be used as indices of degree of inhalation. Thus, differences in respiratory activity between the two experimental groups may yield information on their intensity of smoking.

Fig. 6.10 (overleaf) shows the mean respiration rates (per minute) over the eight experimental periods for the two groups.

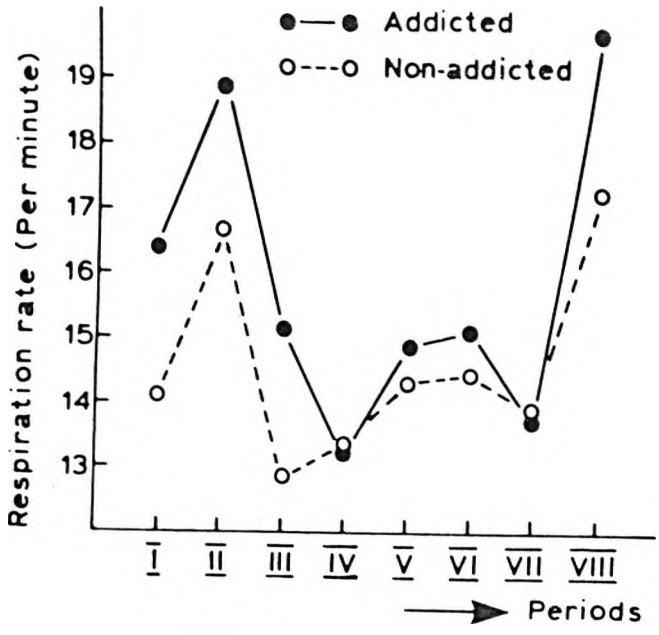

Fig. 6.10 Mean R R's : Groups x Periods (AxC) interaction.

Analysis of the respiration rate data indicated that the addicted and the non-addicted groups were effected differentially by the experimental periods (ANOVA, Groups (A) x Periods (C) interaction, F = 2.644; df = 7/112; $p < 0.025$, see appendix G-iv). A significant decrease in RR from BL 2 to the smoking periods (i.e; SM 1 & 2) was noted for the addicted group, whereas for the non-addicted group there was, in fact, an increase in RR from BL 2 to the SM 2 period, and no significant change in the SM 1 period (Duncan M.R.T, df = 112; MSe = 4.41; $p < 0.05$, see appendix G-iv). Although, it was noted that the groups did not differ in RR during the two smoking periods, it was found that the addicted smokers had a higher RR as compared to the non-addicted group in all the other experimental periods (11). So, here the

(11) - Another point is raised by these results. Higher RR's have been associated with higher activation levels (Duffy, 1962). The addicted smokers were noted to have a higher RR in all experimental periods except the two smoking periods as compared with the non-addicted group. So, that the reduction in RR induced by smoking may be perceived by these smokers as a sedative effect of smoking. Although, this interpretation suggests a possible tranquilizing action of smoking, at present it is merely speculative and needs to be experimentally verified.

direction of change from BL 2 to smoking periods needs to be examined. Although, the two groups did not differ in the smoking periods, one had a decrease (i.e; addicted) and the other (i.e; non-addicted) had an increase in RR, from BL 2 to the smoking periods. These results may imply that the addicted group displayed more intense smoking behaviour or that they inhaled more deeply than the non-addicted group. In view of this finding, the more marked effect of smoking on the SCL levels of the non-addicted smokers seems to be related to higher sensitivity to the effects of nicotine rather than to smoking behaviour.

Some of the smoking topography measures also revealed differences between the addicted and the non-addicted groups, which were in line with the above findings.

Figure 6.11 illustrates the puff-rates from the first and second experimental cigarettes for the two groups.

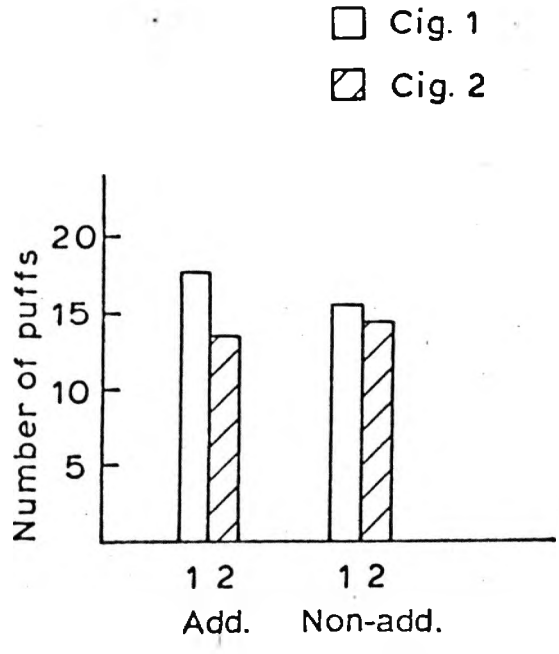

Fig. 6.11 Total number of puffs: Groups x periods (AxC) interaction.

Analysis of the puff-rates indicated that the two experimental groups differed in respect to puff-rates from the first and second cigarettes (ANOVA, Groups (A) x Periods (C) interaction, $F = 7.418$, $df = 1/16$, $p < 0.025$, See appendix G-vii). The non-addicted group took significantly fewer puffs from both of their experimental cigarettes than did the addicted smokers from their first cigarettes (Duncan M.R.T, $df = 16$; $MSe = 5.871$; $p < 0.05$; see Appendix G-vii). This result supported the RR analysis and confirms that the addicted group smoked their first experimental cigarette more intensely than the non-addicted group. It was also noted that the addicted group altered their puff-rate in the second smoking period, and took less puffs from their second cigarettes as compared to their first cigarettes. No significant difference was noted in the puff-rates of the non-addicted smokers from their first and second cigarettes. This finding indicated that only the addicted smokers were altering their smoking parameters and were smoking their first experimental cigarettes more intensely than their second cigarettes. This alteration may represent an attempt to adjust nicotine intake.

The analysis of the butt-lengths of the two experimental cigarettes collected in the deprivation condition showed that only the addicted smokers altered this smoking parameter between their first and second cigarettes. They left longer butts from their second post-deprivation cigarettes than their first cigarettes (ANOVA, Groups (A) x Periods (B) effect, $F = 8.976$; $df = 1/16$; $p < 0.01$, see appendix G-x). No significant difference was noted between the butt-lengths of the first and second cigarettes for the non-addicted group.

Figure 6.12 (overleaf) shows the butt-lengths of the first and second cigarettes for the addicted and non-addicted groups.

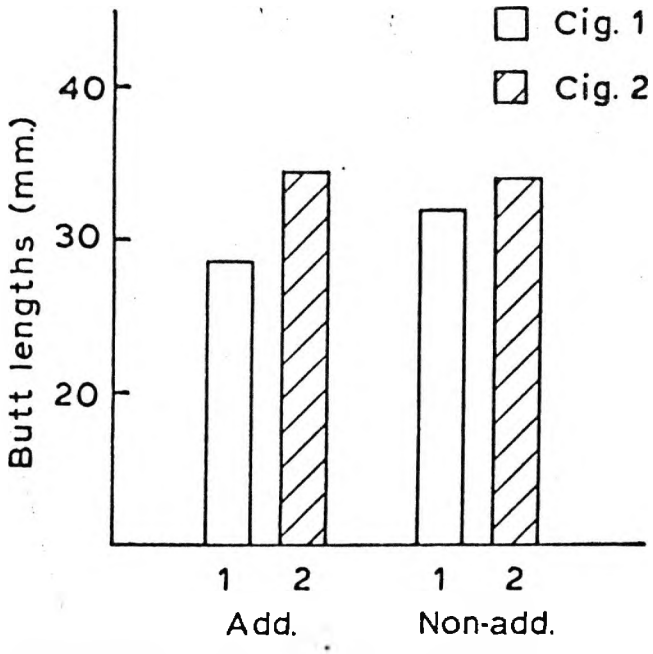

Fig. 6.12 Mean butt lengths: Groups x cigarettes interaction.

Thus, the results of puff-rate and butt-length analysis are in agreement, and they suggest that addicted smokers alter their smoking parameters with each cigarette, whereas the non-addicted group show a consistent smoking habit (i.e; no significant change in smoking parameters from first to second cigarette).

This alteration in the smoking parameters could be related to the duration of time elapsed (i.e; latency) between the first and second experimental cigarettes. A significant difference was noted in latency between the two experimental groups (ANOVA, Groups (A) effect, $F = 13.863$; $df = 1/16$; $p < 0.01$, see appendix G-ix). The addicted smokers were observed to have a shorter latency to their second cigarette in both the normal smoking

(ANOVA, Groups (A) at normal smoking cond. (B 1), F = 10.948, df = 1/32, $p < 0.01$, see appendix G-ix), and the deprivation conditions (ANOVA, Groups (A) at deprivation cond. (B 2), F = 9.151, df = 1/32, $p < 0.01$, see appendix G-ix).

Mean values for the addicted and non-addicted smokers are illustrated in Fig. 6.13.

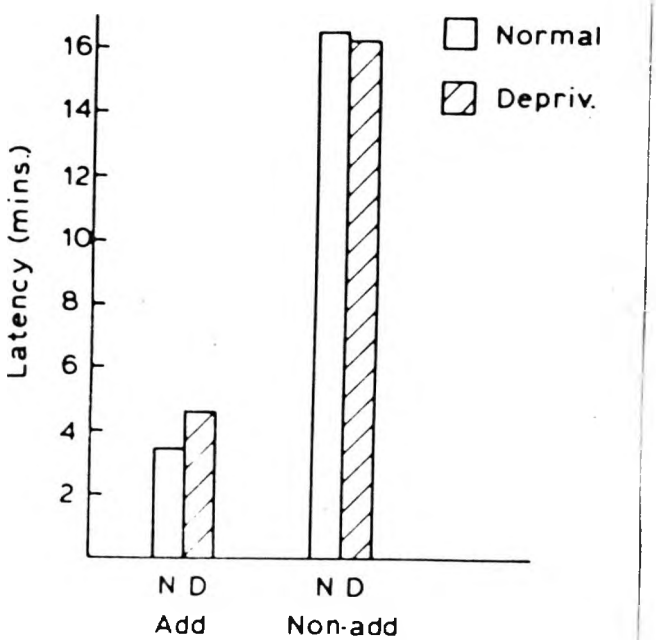

Fig 6.13 Latency to the second cigarette.

These results indicate that the addicted smokers, when free to smoke, light up a second cigarette much sooner than the non-addicted smokers. Four out of nine non-addicted smokers were asked to smoke a second cigarette 30 minutes after their first cigarette. Thus, the difference in latency would have been greater between the two experimental groups, if the subjects were completely free to smoke their second cigarettes whenever they wanted. However, due to practical limitations a maximum latency of 30 minutes was imposed. This may well be a quite unrepresentative smoking frequency for some non-addicted smokers, and is a shortcoming of the present study.

The alterations in some of the smoking parameters (i.e; butt-length and puff-rate) from the first to the second cigarettes noted for the addicted group may be related to the shorter latency noted for this group. They might have smoked their second cigarette for reasons other than obtaining nicotine (i.e; having nothing else to do in the experimental chamber), and might have smoked it less intensely.

In conclusion, the addicted group displayed more intense smoking behaviour than the non-addicted group. They smoked their second cigarette sooner and took more puffs from their first experimental cigarettes than the non-addicted group. On the basis of these findings the addicted group may be expected to display more marked changes in physiological activation levels following smoking than the non-addicted group. Contrary to this expectation, it was found that there was a significant elevation of SCL following smoking only for the non-addicted group. For the addicted group the difference between the pre- and post- smoking SCL's was not significant. This result suggested that either due to lower tolerance levels for nicotine or due to constitutional differences, the non-addicted group, even though they seem to show a less intense smoking behaviour (i.e; less number of puffs, longer latency to the second cigarette, less marked changes in respiration rate during smoking) achieved a marked change in activation levels as manifested by a significant elevation of SCL following smoking.

C: EFFECTS OF CIGARETTE DEPRIVATION ON THE WHOLE SAMPLE:

Although, the effects of deprivation on the whole sample is not the major focus of this thesis, the results that will be presented in this section are presented firstly, to provide an objective assessment of whether smokers followed the instructions for the deprivation condition and abstained from smoking, and secondly, to see how sensitive the measures utilized were

to the effects of deprivation. Heart rate alone reliably reflected the effect of deprivation across the two smoking groups.

RESULTS AND DISCUSSION:

HR was lower in the deprivation condition as compared to the normal smoking condition (ANOVA, Conditions (B) main effect, $F = 12.61$, $df = 1/16$; $p < 0.01$, see appendix G-i).

Figure 6.14 illustrates the HR values observed in the deprivation and normal smoking conditions.

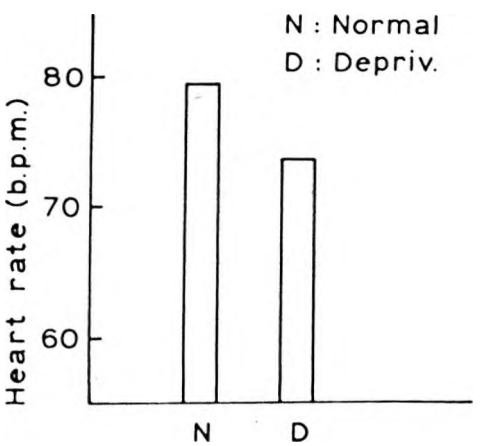

Fig. 6.14 Mean HR's : Condition (B) effect.

The lower heart rate values observed in the deprivation condition are in agreement with the previous reports (see chapter 4). This finding suggests that the subjects did comply with the instructions and abstained from smoking prior to the testing session. It is of interest to note that, even though the Ss smoked two cigarettes in the deprivation condition testing session the mean HR for the whole session was still

significantly lower than that of the normal smoking day, which indicates a cumulative effect of smoking on HR in the normal smoking condition. However, the effects of smoking in the normal and deprivation conditions will become more clear in the section dealing with the differential effects of smoking in the two experimental conditions.

D: EFFECTS OF CIGARETTE SMOKING ON THE WHOLE SAMPLE (REGARDLESS OF THE EXPERIMENTAL CONDITIONS)

In the previous section it was noted that heart rate was significantly lower in the deprivation condition as compared to the values in the normal smoking condition. Cigarette smoking has been reported to increase physiological activation levels (see chapter 4). Thus, it is important to examine the changes in physiological activation levels throughout the experimental session for smokers and non-smokers, in order to assess the effects of cigarette smoking and other experimental manipulations (i.e; vigilance task). In the following section changes in physiological measures over the eight experimental periods for smokers and non-smokers will be presented.

RESULTS AND DISCUSSION

The analysis of HR data throughout the eight experimental periods revealed that there were significant changes in HR values for both the smokers (ANOVA, Periods (C) main effect, $F = 17.52$, $df = 7/112$, $p < 0.001$, see appendix G-i), and the non-smokers (ANOVA, Periods effect, $F = 8.18$, $df = 7/56$, $p < 0.01$, see appendix K-i).

Figure 6.15 (overleaf) shows the mean HR values for the smoker and non-smoker groups throughout the experimental periods.

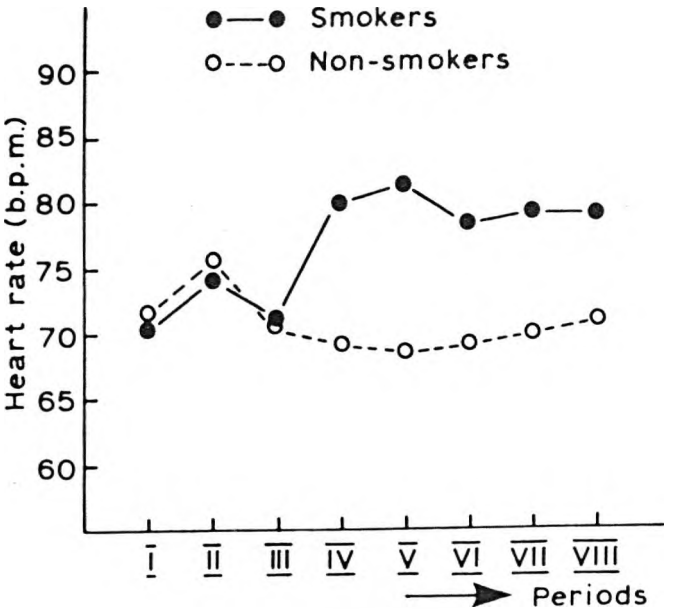

Fig. 6.15 Mean HR's for 'smokers' and 'non-smokers': Main periods (C) effect.

For smokers post-smoking 1 (i.e; period IV), HR values were noted to be significantly higher than the pre-smoking period levels (Duncan M.R.T, df = 112, MSe = 38.09, $p < 0.05$, see appendix G-i). HR was highest in the period following the first smoking (i.e; Lat 1). On the other hand, for non-smokers HR values were found to be higher in the two vigilance test periods and also in the first base-line period (probably due to higher anxiety levels in the beginning of the experimental session) than the other experimental periods (Duncan M.R.T, df = 56, MSe = 6.24, $p < 0.05$, see appendix K-i).

The comparison of HR trends for smokers and non-smokers throughout the experimental periods clearly shows that the elevated post-smoking 1 period HR observed for smokers is related to smoking and/or nicotine intake rather than to any other experimental factor, since for non-smokers a gradual decline in HR was noted between the periods corresponding to SM 1 and SM 2. The lower HR values noted in the deprivation condition (see section C), also supported this conclusion.

Significant changes in skin conductance levels throughout the experimental session were noted only for smokers (ANOVA, Periods (C) main effect, $F = 4.61$, $df = 7/112$, $p < 0.05$, see appendix G–ii). For non-smokers no significant change was noted in SCL's over the eight experimental periods (ANOVA, Periods effect, $F = 0.78$, $df = 7/56$, $p > 0.025$, NS, see appendix K–ii).

Figure 6.16 shows the SCL (micromhos) values throughout the experimental session for the two groups.

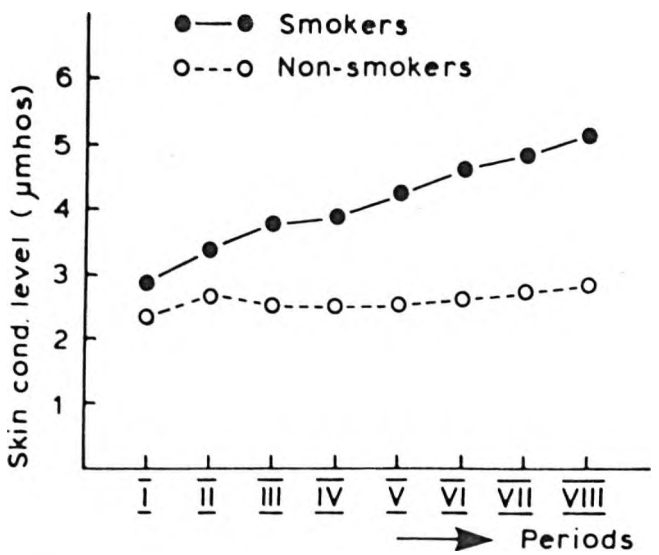

Fig 6.16 Mean SCL's for 'smokers' and 'non-smokers': Main periods (C) effect

For smokers a gradual increase in SCL up to the Lat 2 (i.e; VI), period was noted. All pairwise comparisons up to the Lat 2 period showed that in each period SCL was higher than the preceding one. However, Lat 2, SM 2, and Vig 2, skin conductance levels were not found to be significantly different (Duncan M.R.T, $df = 112$, $MSe = 0.998$, $p < 0.05$, see appendix G–ii). Thus, smokers were noted to have a gradual increase in SCL levels, from the first experimental period up to the period before their second cigarettes. As can

be seen from Fig. 6.16, the SCL for non-smokers during the experimental session showed no systematic change. This observation suggested that the increase in SCL observed for smokers is due to some factor related to smoking (i.e; nicotine intake, act of smoking or expectation to smoke). Although, more research is needed to delineate the factors responsible for the increase in SCL's, this result is consistent with the HR analysis and indicates that smoking induces an increase in physiological activation levels.

The analysis of changes in lability scores (i.e; NS. SCR), over the eight experimental periods revealed results similar to the ones noted for SCL. Smokers were noted to have significant changes in lability throughout the experimental session (ANOVA, Periods (C) main effect, $F = 6.19$, $df = 7/112$, $p < 0.001$, see appendix G-iii), whereas for non-smokers there was no significant change in lability (ANOVA, Periods effect, $F = 0.54$, $df = 7/56$, $p > 0.05$, see appendix K-iii).

Figure 6.17 shows the non-specific S.C. responses/minute for non-smokers and smokers over the eight experimental periods.

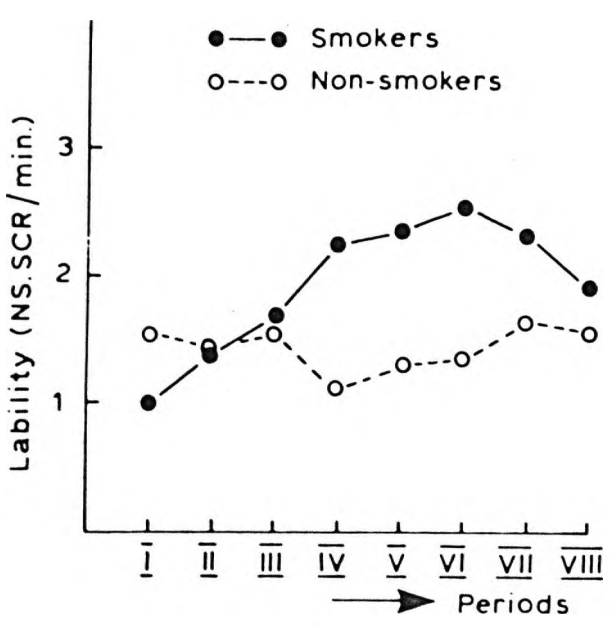

Fig. 6.17 Mean lability (NS.SCR): Main periods effect.

There was an increase in lability during and after the first smoking period to the period before the second cigarette (i.e; Lat 2) for smokers (Duncan M.R.T, df = 112, MSe = 1.72, $p < 0.05$, see appendix G-iii). Lability was noted to be highest in the Lat 2 period. Since, lability was found to be significantly lower in the Vig 2 (i.e; post-smoking 2) period as compared to the Lat 2 period which preceded the second cigarette, the increase in lability does not seem to be directly related to nicotine intake (i.e; drug effect). The decision to smoke a second cigarette (i.e; Lat 2) may have also contributed to the increase in lability.

Since, no significant change in lability over the eight experimental periods was noted for non-smokers, the changes observed for smokers seems to be related to smoking. However, as with SCL's, it is not clear which aspect (i.e; pharmacological or non-pharmacological) of smoking produces increases in lability. So, although the above finding suggests that smoking produces increases in activation levels further experimentation is needed to delineate the factors contributing to the changes noted in lability.

Respiratory activity (i.e; respiration rate (RR), and respiration amplitude (RA) was found to be altered significantly throughout the experimental session for smokers (RR: ANOVA, Periods (C) main effect, $F = 29.03$, df = 7/112, $p < 0.001$, see appendix G-iv; RA: ANOVA, Periods (C) main effect, $F = 27.37$, df = 7/112, $p < 0.001$, see appendix G-v), and non-smokers (RR: ANOVA, Periods effect, $F = 13.36$, df = 7/56, $p < 0.01$, see appendix K-iv; RA: ANOVA, Periods effect, $F = 2.47$, df = 7/56, $p < 0.05$, see appendix K-v).

Figure 6.18 illustrates the RR (per minute) values for the two groups over the eight experimental periods.

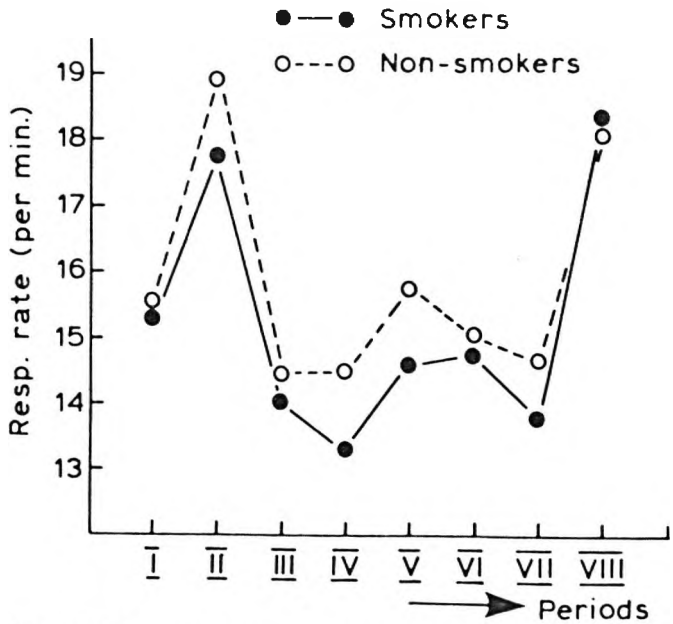

Fig. 6.18 Mean R.R.(per min.): Main periods effect.

For both the smokers (Duncan M.R.T, df = 112, MSe = 4.41, $p < 0.05$, see appendix G-iv), and non-smokers (Duncan M.R.T, df = 56, MSe = 0.61, $p < 0.05$, see appendix K-iv), respiration rates were noted to be significantly higher in the two vigilance test periods (i.e; periods II and VIII) as compared to all the other experimental periods. For non-smokers, there was no significant difference between the RR's in the other experimental periods, whereas for smokers RR was noted to be significantly lower in the first smoking period as compared to BL 1 period. Thus, the act of smoking seems to have slowed down the rate of respiration (probably due to inhalation or taking puffs) in smokers.

Figure 6.19 (overleaf) shows the mean respiration amplitudes (standard scores), for the two groups throughout the experimental periods.

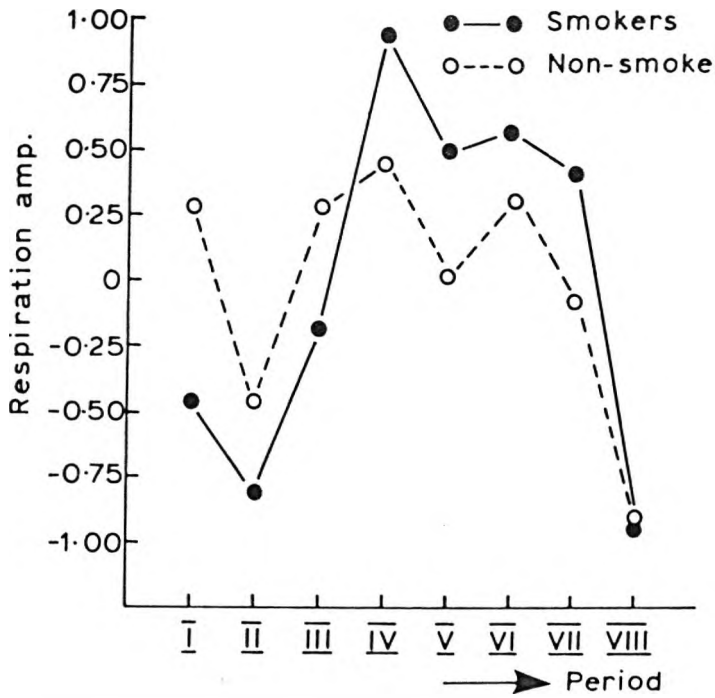

Fig. 6.19 Mean respiration amp.: Main periods (C) effect.

The trends of change noted for RA, were similar to those observed for RR, but in opposite direction. For smokers (Duncan M.R.T, df = 112, MSe = 0.638, $p < 0.05$, see appendix G-v), RA was found to be significantly lower in the two vigilance test periods as compared to all other experimental periods except BL 1. On the other hand, RA was significantly higher in the two smoking periods as compared with the base-line periods. For non-smokers (Duncan M.R.T, df = 56, MSe = 0.85, $p < 0.05$, see appendix K-v), RA in Vig 2 period was found to be significantly lower than the other experimental periods. Respiration amplitude in the other periods showed no systematic variation for this group.

The results presented above indicated that vigilance test (i.e; attending to stimuli) alters the respiratory activity and produces rapid and shallow breathing. This effect was noted for both the smokers and non-smokers. On the other hand smoking seems to produce high amplitude, slow rate breathing.

Table 6.7 gives an outline of the main periods effects noted for smokers and non-smokers.

Measure	SMOKERS	NON-SMOKERS
HEART RATE	Post-SM-1 heart rate values are higher than pre-SM-1 values	Higher H.R in the two vigilance test periods as compared with other periods.
SCL	Gradual elevation of SCL noted, which stabilises after the Lat 2 period	No significant change in SCL.
Lability	Higher lability in Lat 1 & 2 and SM 2 periods as compared to BL 2 values.	No significant change in lability
Resp. Rate	Higher in Vig 1 & 2 and lower in SM 1 & 2 as compared to BL 1.	Higher in Vig 1 & 2 as compared to BL 1. No significant diff. between the other periods.
Resp. Amplitude	Lower in Vig 1 & 2 and higher in SM 1 & 2 as compared to BL 1.	Lower in Vig 2 as compared to BL 1. Corresponding periods to SM 1 & 2 were not sign. different from BL 1.

Table 6.7 Summary of periods effects for smokers and non-smokers.

As can be seen from Table 6.7, changes in the direction of autonomic activation occurred during and after the first smoking period in smokers. Elevated H.R, SCL, and lability were observed for this group following the SM 1 period. These results were in line with the previous reports and showed that smoking increases physiological activation levels. For non-smokers

the changes in the physiological measures seemed to be related to the demands of the vigilance tests. Significant changes in HR, RA and RR (as compared to other periods), were only observed during the vigilance test periods. No significant change was noted for SCL and lability, which indicated that the changes in these measures observed in smokers were related to some aspect of the smoking behaviour rather than other aspects of the procedure.

For both groups similar respiratory activity patterns were observed during the two vigilance test periods. This finding indicated that attending to stimuli induces rapid and shallow breathing patterns. The changes in R.R and R. Amp. during the two smoking periods for smokers seems to reflect the act of puffing.

E: DIFFERENTIAL EFFECTS OF CIGARETTE SMOKING IN THE NORMAL SMOKING AND DEPRIVATION CONDITIONS (ON THE WHOLE SAMPLE)

Smoking after a period of abstinence has been reported to produce more marked effects on physiological activation levels than smoking an additional cigarette on a normal smoking day (see chapter 4). In the present experiment, heart rate was noted to be the only physiological measure that showed a differential effect of smoking in the two experimental conditions. In the following section changes in HR values in the normal and deprivation conditions will be presented and these changes will be discussed in relation to differences noted in some of the smoking topography measures (i.e; cigarette duration and inter-puff-interval).

RESULTS AND DISCUSSION

The analysis of HR values in the normal smoking and deprivation conditions over the eight experimental periods showed that HR was differentially affected by smoking in the two conditions (ANOVA, Conditions X Periods (BC) effect, $F = 9.05$, $df = 7/112$, $p < 0.001$, see appendix G-i).

Figure 6.20 shows the mean HR levels over the eight experimental periods in the deprivation and normal smoking conditions.

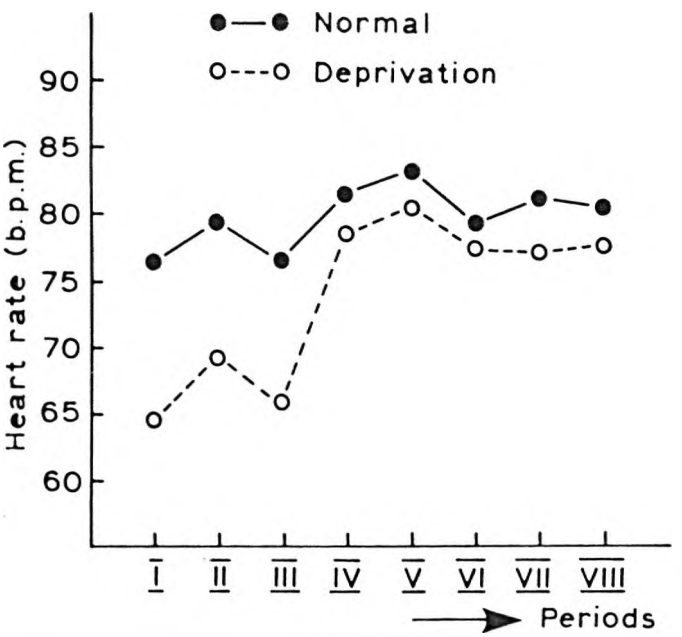

Fig. 6.20 Mean HR: Conditions x periods effect.

Pre-smoking 1 period (i.e; periods I - III), HR levels in the deprivation condition were noted to be significantly lower than the HR values in the corresponding normal smoking condition periods (Duncan M.R.T, df = 112, MSe = 17.34, $p < 0.05$, see appendix G-i, table 1). This finding suggests that smokers did abstain from smoking in the deprivation condition, and that this condition induced a decrease in physiological activation levels as manifested by lower pre-smoking HR levels. However, during and after the first smoking period there was no significant difference between the HR levels in the two experimental conditions (except the SM 2 period). In SM 2 period HR was found to be significantly higher in the normal smoking condition as compared to the deprivation condition. It is interesting to note that in the deprivation condition with only one cigarette, smokers achieve HR levels which were similar to those noted in the normal smoking condition. However, the higher HR noted in the SM 2 period of the normal smoking condition suggests a

cumulative effect of smoking on HR on a normal smoking day.

HR values were found to be significantly higher in all the post-smoking periods as compared to pre-smoking levels in the deprivation condition (see appendix G-i, table 2). In the normal smoking condition HR in the Lat 2 (i.e; VI) period was not found to be significantly higher than the BL 2 level (see appendix G-i, table 3). So, although smoking produces a marked increase in HR, in the normal smoking condition this effect is short-lived. HR was noted to return to base-line values in Lat 2 period.

Thus, in summary the main difference between the two experimental conditions is that smoking after a period of deprivation produces increases in HR that are maintained throughout the experimental session, whereas smoking on a normal smoking day produces an increase in HR which returns to base-line levels in approximately ten minutes (i.e; mean latency in the normal smoking condition for the whole sample). The differential effects of smoking in the two experimental conditions may be related to the way the cigarettes were smoked in these conditions. Ss may have titrated their nicotine intakes by smoking more intensely in the deprivation condition.

Several smoking topography measures yielded results indicating differential effects of experimental conditions on the way the first and second cigarettes of the experiment were smoked.

The time taken to smoke the first and second cigarettes of the experimental session was noted to be differentially altered in the deprivation and normal smoking conditions (ANOVA, Conditions X Periods (i.e; cig 1 versus cig 2) interaction, $F = 9,758$, $df = 1/16$, $p < 0.01$, see appendix G-viii).

The mean values for cigarette duration (seconds) are given in Table 6.8 (overleaf).

	Cig. 1	Cig. 2
Deprivation	393.30	356.10
Normal	353.30	386.70

Table 6.8 Mean cigarette durations (seconds).

Time taken to smoke the first cigarette of the deprivation condition was significantly longer than that of the normal smoking condition (Duncan M.R.T, df = 16, MSe = 2292, $p < 0.05$, see appendix G-viii). A negative correlation was noted between cigarette duration and nicotine intake as estimated from butt-nicotine analysis (see section F-ii). Thus, the longer duration of smoking in the deprivation condition may be reflecting a compensation for behavioural deprivation rather than nicotine deprivation.

Duration of smoking was noted to be significantly altered from the first to the second cigarettes in the deprivation condition. The second cigarette was smoked quicker than the first one. No significant alteration in smoking duration was noted in the normal smoking condition. The significant change in duration noted in the deprivation condition may indicate a behavioural adjustment.

The inter puff interval (IPI), was also noted to be differentially affected by the two experimental conditions (ANOVA, Conditions X Periods interaction, F = 10.24, df = 1/16, $p < 0.01$, see appendix G-vi).

Table 6.9 (overleaf) gives the mean IPI's (in seconds) in the normal smoking and deprivation conditions.

	CIG. 1	CIG. 2
DEPRIVATION	26.17	35.63
NORMAL	29.42	30.91

Table 6.9 Mean IPI's in the normal smoking and deprivation conditions.

In the deprivation condition IPI was noted to be shorter in the SM 1 period than the SM 2 period (Duncan, M.R.T, df = 16, MSe = 27.93, $p < 0.05$, see appendix G-vi). There was no significant difference in the IPI's of the first and second smoking periods in the normal smoking condition. This finding, is in agreement with the cigarette duration analysis and suggests that Ss were altering their smoking parameters from the first to the second smoking period in the deprivation condition. They smoked their first cigarettes for a longer period and took more frequent puffs from it than their second cigarettes. On the other hand, in the normal smoking condition they showed a consistent smoking behaviour.

The smoking topography measures discussed above provide information on the intensity of smoking behaviour. Although, it was noted that subjects smoked their first post-deprivation cigarettes more slowly than their first cigarettes in the normal smoking condition, there was no significant difference in IPI between the two experimental conditions. Nicotine intake as estimated from butt-nicotine analysis will be discussed in detail in section G. However, here it would be useful to mention that although no significant difference was noted between the nicotine intakes from the first cigarettes of the normal smoking (1.269 mg) and the deprivation (1.469 mg) conditions nicotine intake was slightly higher in the deprivation condition. So, although the smoking topography and nicotine analysis suggests that Ss might have smoked their first post-deprivation cigarettes more intensely than their first cigarettes in the normal smoking condition, the results are not conclusive.

The differential effects of smoking on HR in the two experimental conditions may also be related to the higher BL levels observed in the normal smoking condition rather than to differences in smoking parameters or nicotine intake. However, since the nicotine-butt analysis for the cigarettes smoked in the normal smoking condition was conducted only on 11 Ss, the results at present are not conclusive and more research is needed to investigate the reasons why smoking after abstinence produces more persistent changes in HR levels.

F: CORRELATIONAL ANALYSES: THE RELATIONSHIP BETWEEN SMOKING TYPOGRAPHY, TOPOGRAPHY AND PHYSIOLOGICAL EFFECTS OF SMOKING.

i) Smoking typography and physiological effects of smoking:-

In order to investigate the relationship between smoking typography and physiological effects of smoking the following measures were subjected to correlational analyses (Pearson r).

1) Pharmacological addiction score (PAS, Russell et al, 1974),
2) Addiction index score (ALS, from the smoking habits questionnaire),
3) Number of cigarettes smoked per day,

Pre- to post-smoking changes in the following physiological measures in the normal smoking condition (Change was expressed as the proportion of the SM 1 period values to the mean of BL 1 and 2 values; SM 1 / (BL 1 + BL 2) / 2).

4) Heart rate (HR),
5) Skin Conductance level (SCL),
6) Lability,
7) Respiration rate (RR),
8) Respiration amplitude (RA).

Pre- to post-smoking changes in the deprivation condition in;

9) Heart rate,
10) Skin conductance level,

11) Lability,

12) Respiration rate,

13) Respiration amplitude, and

14) Estimated nicotine intake (from the butt-nicotine analyses of the first post-deprivation cigarette).

The correlational matrix showing the relationships between the above 14 measures is presented in table 6.10 (overleaf).

The results of the correlational analysis indicated that the two smoking typography measures utilised in this experiment were positively related with each other ($r = 0.875$, $p < 0.01$), and with the daily cigarette consumption rate ($r = 0.773$ for PAS: and $r = 0.909$ for AIS, $p < 0.01$). Thus, these results suggested that smokers who score high on the pharmacological addiction scale also tend to score high on the addiction index scale which is mainly based on information about smoking topography (i.e; distribution of smoking frequency throughout the day, degree of inhalation, etc.). Furthermore, the high scorers on these scales tend to be heavy smokers. The relationship between daily consumption rate and addiction scores has also been noted in chapter 5.

This relationship points out that both of the addiction indices may indicate a general pharmacological addiction and might not be adequate in identifying different types of pharmacological addiction. If there are some light smokers who consistently smoke to achieve certain physiological states (i.e; low or high arousal) then they may also be regarded as pharmacologically addicted to smoking. Thus, it may be more useful to use the scores on different factors contributing to the pharmacological addiction dimension separately in order to assess the type of smoking in individual smokers.

CORRELATIONS (Pearson r)

NORMAL DAY

	PAS 1	AIS 2	No.Cigs 3	HR 4	SCL 5	Lab 6	R.R 7	R.A 8
2	0.875***							
3	0.773***	0.909***						
4	-0.172	-0.338	-0.259					
5	-0.116	-0.073	-0.162	-0.189				
6	0.378	0.437	0.318	-0.113	0.344			
7	-0.715***	-0.570**	-0.432	0.139	-0.022	-0.342		
8	0.424	0.391	0.363	0.047	-0.387	-0.097	-0.537*	
9	0.352	0.418	0.429	-0.023	-0.061	0.411	-0.344	0.283
10	-0.060	-0.067	-0.075	0.120	0.294	0.308	0.117	-0.050
11	-0.044	0.248	0.424	-0.181	-0.130	-0.017	0.264	0.154
12	-0.227	-0.130	-0.145	0.131	-0.041	0.156	0.137	-0.147
13	0.334	0.309	0.242	0.276	-0.220	-0.124	-0.225	0.607**
14	0.148	0.418	0.419	-0.309	-0.068	0.174	-0.228	0.141

(Nic. intake.

DEPRIVATION DAY

	HR 9	SCL 10	LAB 11	R.R 12	R.A 13
10	0.094				
11	-0.046	0.024			
12	-0.061	0.368	0.162		
13	0.369	-0.011	0.022	-0.452	
14	0.591***	-0.259	0.304	-0.317	0.374

MEANS	SDS
3.417	2.337
12.00	5.247
18.56	9.660
1.014	.2575
1.184	.2388
2.786	2.418
.8983	.2109
1.270	.3361
1.209	.1246
1.198	.0826
1.315	.8172
.9367	.1300
1.272	.3200
1.729	.7169

Table 6.10.

Correlations Matrix: Interrelationships between the typology measures and physiological changes accompanying smoking.

df = 16, two tailed test

* $p < 0.05$

** $p < 0.02$

*** $p < 0.01$.

Both of the addiction indices were noted to be negatively correlated with post-smoking changes in RR ($r = -0.715$, $p < 0.01$, for PAS; $r = -0.570$, $p < 0.02$, for AIS). The change scores were derived by dividing the SM 1 values by the mean of the two base-line values. Since, except the RR, all the physiological measures were noted to increase with smoking (see appendix F), the proportion of smoking- to pre-smoking values was used as a measure of change. Thus, the negative correlation between the addiction indices and RR, suggests that smokers scoring high on addiction had a larger decrease in RR from pre-smoking to smoking periods. This may either be due to higher base-line RR's for this group, or to more intense smoking parameters, or both. A negative relationship was noted between the RR and RA ($r = -0.537$, $p < 0.05$). A small positive correlation was also noted between PAS and RA ($r = 0.424$, $p < 0.1$). Although, this needs to be cautiously interpreted, it suggests that smokers scoring high on the PAS were showing high amplitude, slow rate of breathing in the smoking period as compared to base-line periods. Since RR and RA may be used as indices of inhalation, the results offer evidence to support the relationship between questionnaire scores and objective measures of the intensity of smoking.

A significant positive correlation was noted between the estimated nicotine intake from the first post-deprivation cigarette and change in HR in this condition ($r = 0.591$, $p < 0.01$). This finding lends support to the reliability of the butt-nicotine analysis and points out a relationship between nicotine presented to the smoker and nicotine absorbed. It also supports the dose dependant effect of nicotine on HR changes as reported by previous authors (see chapter 4).

ii) Smoking typography and topography:-

Smoking typography measures (i.e; PAS and AIS) and topography measures taken from the first post-deprivation smoking period were subjected to

a correlational analyses (Pearson r), in order to examine the relationship between the addiction indices, the way the cigarettes were smoked and nicotine intake.

The measures were: 1) PAS, 2) AIS3) Estimated nicotine intake, 4) Number of puffs, 5) Cigarette duration (secs), 7) Latency to the second cigarette, 8) Butt-length (mm).

Table 6.11 shows the correlational matrix describing the relationship between these eight measures.

	CORRELATIONS PAS 1	AIS 2	Nic.Int. 3	No. of Puffs 4	Cig. Durat. 5	Puff Durat. 6	Latency 7
2	0.876***						
3	0.147	0.412					
4	0.331	0.332	-0.006				
5	-0.173	-0.177	-0.632***	0.063			
6	0.343	0.402	0.400	-0.590***	-0.341		
7	-0.602***	-0.672***	-0.339	-0.555**	0.347	-0.110	
Butt L. 8	-0.276	-0.460	-0.521*	-0.447	-0.027	0.060	0.532*

MEANS	SDS
3.413	2.339
12.00	5.247
1.729	.7169
16.83	7.493
393.3	95.32
1.928	.8855
10.35	10.82
30.39	6.853

df = 16 (two-tailed test)
* $p < 0.05$
** $p < 0.02$
*** $p < 0.01$

Table 6.11 Correlations between smoking typography and topography measures.

Both of the addiction indices (i.e; PAS & AIS), were noted to be negatively related to the latency to next cigarette ($r = -0.602$, $p < 0.01$). This finding suggests that smokers who score high on the addiction indices smoke their cigarettes in quick succession. Since the measures were taken in the deprivation condition, these results may only be reflecting post-deprivation smoking behavicur. However, the short latency for this group is in agreement with their self-reports and provides a further behavioural evidence to support the typology classification.

Nicotine intake was noted to be negatively related to cigarette duration ($r = -0.632$, $p < 0.01$) and butt-length ($r = -0.521$, $p < 0.05$). This finding suggests that the quicker and the more (i.e; short butt-length) of the cigarette is smoked the more nicotine the smoker will obtain.

Latency was noted to be positively related to butt-length ($r = 0.532$, $p < 0.05$) and negatively correlated with number of puffs ($r = -0.555$, $p < 0.02$). This finding suggests that the shorter the latency is between two cigarettes, the shorter is the length of butt left and the higher is the puff rate. It is interesting to note that by only observing the puff-rate or butt-length of a single cigarette it may be possible to predict when a second cigarette will be smoked and whether the individual is a heavy smoker. However, since the measures used in this correlational analyses were taken from the first post-deprivation smoking period, these findings may only be applicable to post-deprivation smoking behaviour.

In conclusion the correlational analysis provided evidence to support the relationship between scores on the two addiction scales and some behavioural and physiological measures related to smoking. The relationship between HR changes and nicotine intake confirmed the reliability of the butt-nicotine analysis.

iii) Correlational analysis of the factor scores of Russell et al (1974) Smoking typology scale and the addiction index score;

In order to examine the relationship between the factor scores on the seven smoking factors of the smoking typology scale and the addiction index score a correlational analysis (Pearson r) was carried out.

The correlational matrix describing the relationship between the factors of the typology scale and the AIS are presented in Table 6.12.

CORRELATIONS	AIS 1	Autom. 2	Stim. 3	Addic. 4	Indulg. 5	Psycho-Soc. 6	Sensor. 7
2	0.723***						
3	0.850***	0.830***					
4	0.860***	0.809***	0.810**				
5	0.061	0.247	0.294	0.163			
6	0.053	0.335	0.327	0.260	0.592***		
7	0.320	0.254	0.322	0.265	0.432	0.092	
Sedative 8	0.758***	0.698***	0.659***	0.760***	0.007	0.066	0.027

Table 6.12 Correlations between the seven smoking factors of Russell et al (1974) Typology Scale and the addiction index score.
(df = 16, two-tailed test)

* $p < 0.05$,
** $p < 0.02$,
*** $p < 0.01$.

Factor scores on all of the three factors contributing to the pharmacological addiction dimension (PAD), (i.e; Automatic, Stimulation and Addictive) were found to be positively intercorrelated ($r = +0.809 - +0.830$; $p < 0.01$). None of the PAD factors were noted to be significantly correlated with the non-pharmacological addiction factors. These findings supported the independence of the pharmacological addiction dimension as proposed by Russell et al (1974).

As was also noted in the first experiment, all the PAD factors were found to be positively correlated with the sedative smoking factor ($r = +0.659 - +0.760$; $p < 0.01$). This finding once again pointed out that sedative smoking may also be included as a pharmacological addiction factor.

The addiction index score was significantly related to all the three PAD factors ($r = +0.723 - +0.860$; $p < 0.01$), and also with sedative smoking ($r = +0.758$, $p < 0.01$). Thus, the AIS based mainly upon the intensity (i.e; depth of inhalation, frequency and distribution of smoking throughout the day etc.) of smoking behaviour seems to be in agreement with the scores derived from all the 3 PAD factors of Russell et al.

So, from the correlations noted between the AIS and PAD factors and the intercorrelations between the three PAD factors we may conclude that smokers who state that they exhibit intense smoking behaviour (AIS), also state that they smoke to stimulate or sedate themselves and that they experience craving if cigarettes are not available. The verbal reports of the smokers to the above two questionnaires yield a fairly consistent picture of their smoking behaviour. Furthermore, the results of the correlational analysis presented in the previous sections, provided some behavioural evidence to support the reports of these smokers. These findings suggested that smokers who score high on these addiction indices tend to be heavy smokers and leave a short gap between their cigarettes.

The replies of smokers to the item "Would you consider yourself addicted to nicotine?, if yes, why?" of the smoking habit questionnaire are presented in appendix L. It is interesting to note that eight out of nine addicted smokers stated that they are addicted to nicotine and justified their answers by stating that they experience craving and withdrawal symptoms when cigarettes are not available. On the other hand, seven out of 9 non-addicted smokers stated that they do not consider themselves addicted and stated that

they would not experience withdrawal symptoms or feel an urge to smoke when cigarettes are not available.

All of these verbal responses indicate that it is possible to differentiate smokers by a number of questions. Smokers who have a high consumption rate seem to believe that they are addicted to nicotine and that they will experience craving and withdrawal symptoms with cessation. The correlational analysis provided some behavioural data that suggested an intense smoking behaviour (i.e; short latency to next cigarette, high consumption rate) for this group.

G: NICOTINE INTAKE: Differential effects of normal smoking and deprivation conditions on the addicted and non-addicted groups:-

Analysis of the SCL values suggested that smoking produced different effects on the SCL's of the addicted and non-addicted smokers (see sections A and B). Deprivation condition SCL's were noted to be significantly lower than the normal smoking condition values only for the non-addicted group. Similarly, post-smoking SCL's were significantly higher than the pre-smoking levels only for the non-addicted smokers. Although, the differential effects of smoking in the two groups may be related to differences in sensitivity to the effects of smoking, several smoking topography measures indicated that the addicted smokers were exhibiting a more intense (e.g; higher puff-rate, shorter butt-length) smoking behaviour than the non-addicted smokers. This finding suggests that the addicted smokers may have a higher nicotine intake than the non-addicted smokers. If this is so, then the differential effects of smoking on SCL's are more likely to be related to differences in sensitivity levels.

Nicotine intake as estimated from butt-nicotine analysis, is a more direct measure of nicotine extracted from the cigarettes than the

smoking topography measures. A positive correlation was noted (see section F) between estimated nicotine intake and HR changes following smoking. This finding confirmed the reliability of the butt-nicotine analysis.

In this section differences in nicotine intake between the addicted and non-addicted smokers in the two experimental conditions will be examined. Comparisons will also be made between the nicotine intake in the two laboratory smoking conditions and butts collected on a normal smoking day outside the laboratory (i.e; daily life).

RESULTS AND DISCUSSIONS

In order to examine the differences in nicotine intake between the addicted and non-addicted groups, the estimated nicotine intake from the two cigarettes smoked in the deprivation condition were analysed (ANOVA, 2 factors, A = Groups, B = Nicotine intake from first and second cigarettes, repeated measures on B, see appendix H-i).

No significant difference was noted between the groups (A; $F = 1.035$, $df = 1/16$, $p > 0.05$, NS), between the nicotine intake from the two cigarettes (B; $F = 0.993$, $df = 1/16$, $p > 0.05$, NS), or in the nicotine intake of the two groups from the first and second cigarettes (AB: $F = 0.433$, $df = 1/16$, $p > 0.05$, NS).

Mean nicotine intake from the two experimental cigarettes smoked in the deprivation condition by the two experimental groups are presented in Table 6.13 (overleaf).

Although, no statistically reliable difference was noted, it was seen that the addicted group extracted more nicotine than the non-addicted group from both of their experimental cigarettes. Both of the experimental

	Cig. 1	Cig. 2
Addicted (N = 9)	1.859	1.809
Non-Addicted (N = 9)	1.600	1.355

Table 6.13 Mean estimated nicotine intake (mg) for the addicted and non-addicted groups in the deprivation condition.

groups extracted more than expected amounts (i.e; standard smoking machine nicotine yield for the experimental cigarette is 1.50 mg) of nicotine from their first cigarettes (i.e; 1.859 and 1.600 mg), whereas only the addicted group extracted more than expected amount from their second cigarettes (i.e; 1.809 mg). Here, it is interesting to note that although the butt-length analyses showed that only the addicted smokers altered this smoking parameter from their first to second post-deprivation cigarettes & left longer butts from their second cigarettes there was no significant difference in the amount of nicotine they extracted from the two cigarettes. This finding points out that the smoking topography measures might not yield adequate and reliable information on nicotine intake, and it is necessary to employ more comprehensive measures of smoking topography, like puff volume, in order to gain information about nicotine intake. Although, the non-addicted smokers showed a more consistent smoking behaviour (i.e; no significant change in smoking topography from first to second cigarettes), the difference in nicotine intake between their two cigarettes seems to be larger.

In order to investigate the effects of experimental conditions on nicotine intake, and to examine whether the two experimental groups responded differentially to deprivation, nicotine intake from the first cigarettes of the normal and deprivation conditions were analysed (2 factors

ANOVA, unweighted means analysis, A = Groups B = Nicotine intake in normal and deprivation conditions, repeated measures on B, see appendix H-ii).

The mean values for the estimated nicotine intake are shown in Table 6.14.

	Normal	Deprivation
Addicted (N = 7)	1.483	1.727
Non-Addicted (N = 4)	0.895	1.017

Table 6.14 Mean estimated nicotine intake (mg) for the two experimental groups in the normal and deprivation conditions.

There were no significant difference between the groups (A; $F = 4.192$, $df = 1/9$, $p > 0.05$, NS), between the conditions (B; $F = 1.295$, $df = 1/9$, $p > 0.05$, NS), or in the responsivity of the two experimental groups to deprivation (A X B; $F = 0.165$, $df = 1/9$, $p > 0.05$, NS). Thus, these results did not support the view that smokers, especially addicted ones, will attempt to compensate for the effects of deprivation and will extract more nicotine from their cigarettes after a period of abstinence. However, although the difference between the groups or conditions did not reach significance, the mean values for the groups show that both of the groups had a higher nicotine intake in the deprivation condition and only the addicted group extracted more than expected amount of nicotine from their first post-deprivation cigarettes. Since, all the butts from the normal smoking condition were not collected, this analysis could not be performed on the whole sample. This makes it difficult to draw a firm conclusion on these results.

The lack of any reliable difference in nicotine intake between the normal and deprivation conditions could also be related to the experimental setting. Even in the normal smoking condition the Ss had to abstain from smoking for at least 40 minutes (i.e; time taken for the preparation of recording and vigilance test 1). For some Ss this might have represented a temporary deprivation period. In addition to this, being in a laboratory and having nothing else to do might have altered the smoking behaviour. Thus, the comparison of nicotine intake from the two laboratory conditions might not yield reliable data on post-deprivation nicotine titration.

In order to investigate this issue further, nicotine intake from the first post-deprivation cigarette and first daily life cigarette for the addicted and non-addicted groups were analysed (2 factor ANOVA, A = Groups, B = Nicotine intake, repeated measures on B, see appendix H-iii).

The mean nicotine intake (mg) values are presented in Table 6.15.

	1st Cig. DEPRIVATION	1st Cig. DAILY LIFE
Addicted (N = 9)	1.859	1.372
Non-Addicted (N = 9)	1.600	1.197

Table 6.15 Mean estimated nicotine intake (mg): Deprivation condition and daily life.

A significant difference was noted between the nicotine intakes in the deprivation condition and daily life (ANOVA, Conditions (B) effect, $F = 13.157$, $df = 1/16$, $p < 0.01$, see appendix H-iii). Both the addicted ($F = 7.868$, $df = 1/16$, $p < 0.02$) and the non-addicted ($F = 5.404$, $df = 1/16$, $p < 0.05$) groups

had a higher nicotine intake from their first post-deprivation cigarettes, than they did from their first cigarettes in daily life. These results suggested that both of the experimental groups were affected by deprivation and tried to compensate by smoking their first post-deprivation cigarettes more intensely. However, it is also necessary to examine the difference between nicotine intake in the normal laboratory condition and daily life, in order to account for the higher nicotine intake noted in the deprivation condition.

The difference between the estimated nicotine intake from the first cigarette of the normal laboratory smoking condition (N = 7 for addicted, N = 4 for non-addicted) and daily life were analysed (2 factor ANOVA, unweighted means analysis, A = Groups, B = Nicotine intake in normal and daily life conditions, see appendix H-iv).

Mean estimated nicotine intake (mg) for the two experimental groups are presented in Table 6.16.

	Lab. Normal	Daily Life
Addicted (N = 7)	1.483	1.294
Non-Addicted (N=4)	0.895	1.015

Table 6.16 Mean estimated nicotine intake (mg) for the two experimental groups in the laboratory normal smoking and daily life conditions.

No significant difference was noted between the groups (A; $F = 3.338$, $df = 1/9$, $p > 0.05$, NS), between the conditions (B; $F = 0.382$, $df = 1/9$, $p > 0.05$, NS), or in the responsivity of the two experimental groups to the conditions

(AXB; $F = 1.015$, $df = 1/9$, $p > 0.05$, NS). Thus, although nicotine intake was slightly higher for both groups in the laboratory normal smoking condition as compared to daily life, there was no reliable difference in nicotine intake between the two conditions.

Ashton, Stepney and Thompson (1978), noted that the estimated dose of nicotine intake (from butt nicotine analysis), in a laboratory condition was significantly higher than nicotine intake in daily life. They suggested that this may be due to either the abnormality of laboratory smoking condition or to pre-experimental cigarette deprivation. However, in the present analysis no significant difference was noted between the laboratory normal smoking and daily life nicotine intake. Therefore, the higher nicotine intake in the lab. deprivation condition seems to indicate a compensation for the effects of deprivation rather than the effects of laboratory smoking.

So, in conclusion the butt-nicotine analysis suggested that both groups of subjects had a significantly higher nicotine intake from the first post-deprivation cigarette as compared to the first cigarette of daily life. Although, the addicted group extracted slightly higher amounts of nicotine from their cigarettes in the two laboratory conditions and also in daily life than the non-addicted group, the difference between the groups was not statistically significant. These results suggest that the addicted smokers by smoking more frequently obtain a higher and continuous level of nicotine intake as compared to the non-addicted smokers. So, the difference between the two experimental groups seems to lie in nicotine intake over a period of time (e.g; day), rather than nicotine intake from a single cigarette.

H: DIFFERENTIAL EFFECTS OF CIGARETTE SMOKING AND DEPRIVATION ON HIGH AND LOW NICOTINE INTAKE GROUPS: HEART RATE ANALYSIS

The analysis of the butts from the first post-deprivation cigarettes showed that nicotine intake, ranged between 0.63 and 2.97 mg across

the subjects. A further analysis was carried out in order to examine the differential effects of smoking and deprivation on subjects with high and low nicotine intake. The analysis was limited to only the HR data because of the relationship noted between nicotine intake and heart rate changes (see section F and chapter 4).

Two groups of Ss were identified on the basis of their estimated nicotine intake from the first post-deprivation cigarette. Subjects whose nicotine intake was above the median value for the present sample were assigned to the high-nicotine-intake group (HNI: mean nic. intake = 2.33 mg; range = 1.88 to 2.97 mg; mean daily cig. consumption = 21.42, range = 2-30; 5 addicted and 4 non-addicted), and Ss whose nicotine intake was below the median were assigned to the low-nicotine-intake group (LNI: mean nic. intake = 1.14 mg, range = 0.63 to 1.72 mg; mean daily cig. consumption = 15.78, range = 5-25; 4 addicted and 5 non-addicted).

Mean HR values in the base-line (i.e; mean of BL 1 and 2 periods), first smoking and latency 1 periods for the LNI and HNI groups were analysed in order to examine the differential effects of smoking on these groups. The findings are presented below.

RESULTS AND DISCUSSIONS

The mean HR values for the HNI and LNI groups in the normal smoking and deprivation conditions over the base-line (1), first smoking (2), and first latency (3), periods are illustrated in Figure 6.21 (overleaf).

The analysis of the HR data indicated that the two groups were effected differentially by the experimental conditions and periods (ANOVA, Groups X Conditions X Periods (ABC) interaction, $F = 6.61$, $df = 2/32$, $p < 0.01$, see appendix I).

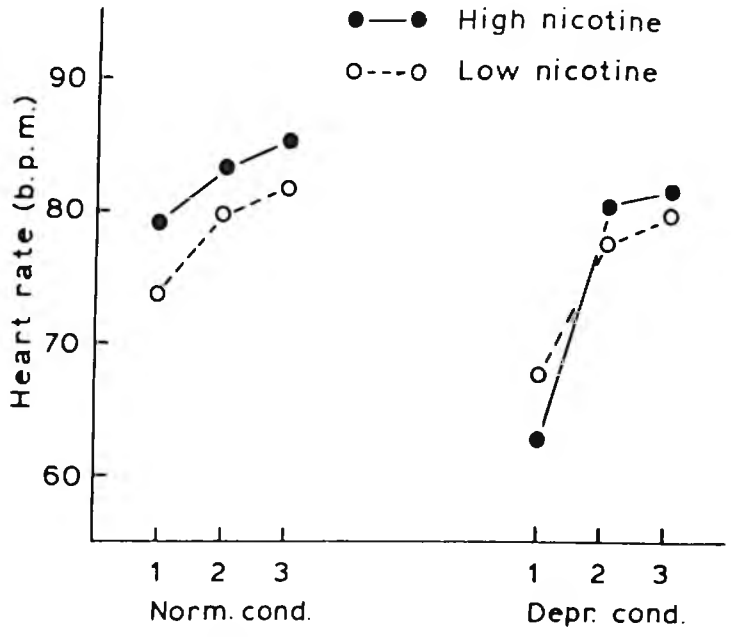

Fig. 6.21 Mean HR : High and low nicotine intake GRP's in the normal and deprivation conditions.

In the normal smoking condition the HNI group had a significantly higher HR in the BL 1 and Lat 1 periods as compared to the LNI group (Duncan, M.R.T, df = 32, MSe = 12.00, $p < 0.05$, see appendix I-i). However, there was no significant difference between the groups in the SM 1 period. These findings indicate that the HNI group exhibit a higher physiological activation level as indicated by higher HR levels prior to smoking. Although, the LNI group achieved HR levels similar to the ones noted for the HNI group by smoking one cigarette, the latter showed a higher HR level in the Lat 1 period following smoking.

Smoking in the normal condition was noted to increase the HR of LNI group significantly (i.e; HR was higher in SM 1 as compared to BL 1), whereas for the HNI group only the Lat 1 period HR was significantly higher than the BL 1 period value (see appendix I-i). These results suggested that the LNI group, either due to lower levels of tolerance to the effects of smoking and/or

nicotine or due to initially lower HR levels show marked changes in HR levels with smoking and achieve levels similar to the ones observed for the HNI group.

The first post-deprivation cigarette, which was used to classify the subjects into HNI and LNI groups, induced a greater percentage change in the HR of the HNI group. This finding supports the reliability of the nicotine butt analysis. It was noted that even though the HNI group had a higher nicotine intake than the LNI group, the two groups did not differ in HR in the SM1 and Lat 1 periods (see appendix I-ii), which again indicated that the HNI group needed a larger dose of nicotine to achieve HR levels similar to those of the LNI group.

A significant increase was noted in HR from the BL to the smoking period for both the HNI and LNI groups in the deprivation condition. It is interesting to note that smoking produced a significant increase in HR for the LNI group in both of the experimental conditions, whereas for the HNI group H.R. in the SM 1 period of the normal condition was not significantly higher than the resting level. This finding suggests that the LNI group may be more sensitive to the effects of smoking and shows a marked change in HR with smaller doses of nicotine.

An examination of differences between deprivation and normal smoking condition values for the two experimental groups, showed that for the HNI group HR was significantly lower in the BL and Lat 1 periods of the deprivation condition as compared to the normal condition (Duncan M.R.T, df = 16, MSe = 67.99, $p < 0.05$, see appendix I-iii). This finding indicates that although by smoking one cigarette in the deprivation condition the HNI group achieve HR levels similar to their normal smoking day levels, HR in the period following smoking (i.e; Lat 1), is higher in the normal condition. This may reflect the cumulative effect of smoking in a normal smoking day. On the other hand, although the LNI group also showed significantly lower resting HR levels in the deprivation condition, there was no difference between the HR levels in the SM 1 and Lat 1

periods of the normal and deprivation conditions (Duncan M.R.T, df = 16, MSe = 67.99, $p < 0.05$, see appendix I-iv). This result indicates that the LNI group, with low nicotine intake achieves HR levels similar to the ones observed in a normal smoking day. Furthermore, they show post-smoking (i.e; Lat 1) HR levels similar to those of a normal smoking day.

These results indicate that the relationship between nicotine intake and physiological response is not a simple one. The physiological effects of smoking and/ or nicotine as indicated by pre- to post-smoking HR changes seems to be determined by an interaction between nicotine dosage and certain organismic variables. It was seen that a group of smokers classified as having a low nicotine intake level achieve HR levels similar to those of a high-nicotine intake group.

On the other hand, after a period of deprivation the LNI group had a higher resting HR than the HNI group. All these findings suggest that if we define pharmacological addiction simply as dependance on the pharmacological effects of nicotine and experiencing withdrawal symptoms with cessation of smoking, then both of the groups may be regarded as pharmacologically addicted. In particular, both groups exhibited a withdrawal state: lower HR in the deprivation condition as compared to normal condition, and both groups showed similar physiological activation levels as indicated by HR levels following smoking. However, the HNI group seemed to require a higher level of nicotine intake to reach HR levels similar to the LNI group.

So, in conclusion, although the concept of pharmacological addiction seems to be a complex one, it may be possible to differentiate the degree of pharmacological addiction between smokers by levels of nicotine intake, physiological response and the frequency of seeking the effects of smoking. The findings suggest that there may be two types of smokers, possibly both of which

are pharmacologically addicted, one which smokes intermittently and experiences a marked change (i.e; an intense effect of smoking) with each cigarette, and the other who smokes cigarettes frequently in order to maintain a consistent blood nicotine level and the constant effect produced by this condition.

I: VIGILANCE TEST PERFORMANCE: ADDICTED AND NON-ADDICTED SMOKERS AND NON-SMOKERS

Cigarette smoking has been reported to prevent the decrement in performance over time commonly noted in vigilance tasks (see chapter 4).

In this section the differential effects of smoking on the vigilance test performance of the addicted and non-addicted groups will be examined and the results for smokers will be compared with the performance of non-smokers.

RESULTS AND DISCUSSIONS

The analysis of the vigilance test scores (tests 1 and 2) of the addicted and non-addicted smokers revealed that the groups differed in performance in the first (i.e; pre-smoking) and second (i.e; post-smoking) vigilance tests (ANOVA, 3 factors, Tests 1 and 2 (C) main effect, $F = 5.05$, $df = 1/16$, $p < 0.05$, see appendix J-i).

Further analysis indicated that only the addicted group showed a significant improvement in vigilance test performance from the first to the second test (Duncan M.R.T, $df = 16$, $MSe = 66.66$, $p < 0.05$, see appendix J-i). There was no significant difference for the non-addicted group between the two tests.

The mean vigilance test scores are presented in Table 6.17 (overleaf).

	Test 1	Test 2
Addicted	62.22	70.28
Non-Addicted	66.67	67.22

Table 6.17 Mean vigilance test scores for the addicted and non-addicted groups in tests one and two.

These results suggested that the addicted group might obtain more useful effects from smoking. Since no significant change was noted for the non-addicted group the improvement noted for the addicted group is not likely to be due to practice effect. Here, it needs to be pointed out that although there was no significant difference between the performance in the two tests for the non-addicted smokers, they maintained their performance at a stable level and did not show a decrement in performance that has been commonly reported and was observed in the present experiment for non-smokers (see below). However, this result may also be related to the type of test used in this study. Vigilance tests are usually longer and are given as a continuous test, whereas in the present study the tests were only 20 minutes long and were administered in two separate blocks.

The analysis of post-smoking changes in SCL levels showed that only the non-addicted smokers showed a significant post-smoking elevation in this measure (see section B). The non-addicted group had a significantly higher SCL level as compared to the addicted smokers in the post-smoking 1 periods. SCL may be used as an index of general activation. Considering these findings, the improvement noted for the addicted group from the pre-to-post-smoking vigilance test seems to be contradictory to what would be expected on the basis of the SCL data. However, SCL may only be reflecting a general level of activation, which may in fact be detrimental to performance in an attention task.

In order to compare the vigilance performance of smokers and non-smokers, a further analysis was carried out. The scores for the non-smokers (N = 9), and two groups of smokers (i.e; Deprived on first testing day; N = 8, and Normal smoking condition, first testing day; N = 10), for the first and second vigilance tests were analysed (ANOVA, 2 factors, A = Groups, B = tests 1 and 2, see appendix J-ii).

Figure 6.22 illustrates the vigilance scores for the three groups.

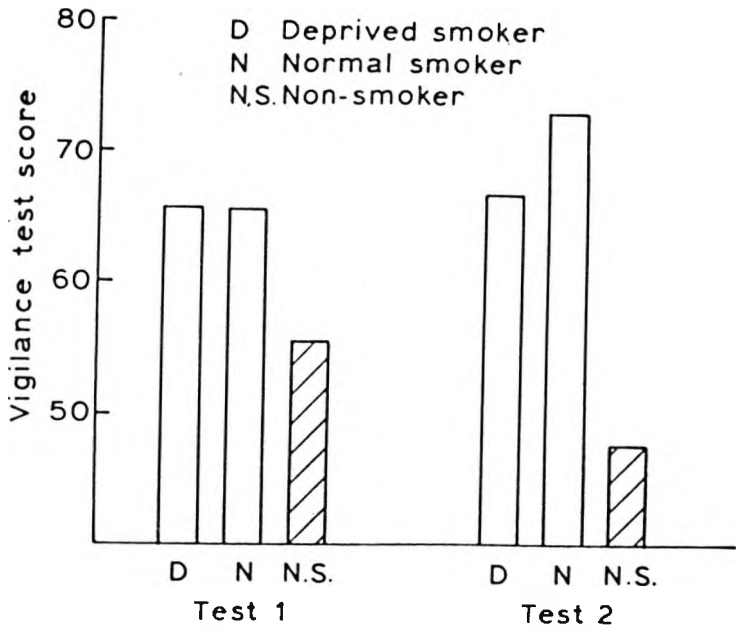

Fig. 6.22 Vigil. scores : Groups x tests (A x B) interaction.

The results indicated that there were differences in vigilance performance between the three groups (ANOVA, Groups (A) main effect, F = 4.675, df = 2/24, $p < 0.025$, see appendix J-ii). Both of the smoker groups (i.e; deprived and normal smokers) had a higher score in the second vigilance test (i.e; post-smoking) as compared to the performance of non-smokers in test two

(Duncan M.R.T, df = 24, MSe = 173.74, $p < 0.05$, see appendix J-ii). There was no significant difference between the three groups in the vigilance 1 performance. Amongst the normal smokers only the test 2 performance was superior to the test 1 performance of non-smokers.

Although, the non-smokers showed a slight deterioration in performance, the normal smokers an improvement and the deprived smokers a stable level of performance in the second test as compared to the first test, the difference between the first and second tests was not significant for any one of the experimental groups. These results suggest that smoking prevents the performance decrement commonly noted in vigilance tests and produces a stable level of performance or even some improvement. The results for non-smokers although not significant showed a slight deterioration in vigilance performance from the first to the second test.

These results are in line with the findings of other researchers (see chapter 4), and suggest that smokers may find smoking reinforcing because of its beneficial effects on attentional performance. It is interesting to note that although the duration of the vigilance test used in this experiment was much shorter (i.e; 20 minutes), than the ones reported in literature and that it was given in two separate blocks, it still showed the advantageous effects of smoking on attentional performance. It also provided evidence to support the differential effects of smoking on addicted and non-addicted smokers.

6.4. CONCLUSIONS

The aim of the present study was to investigate the differential effects of cigarette smoking and deprivation on pharmacologically addicted and non-addicted smokers as classified by Russell *et al* (1974), smoking typology scale. On the basis of items characterizing addicted smoking, it may be

expected that addicted group would be affected more markedly by deprivation (i.e; larger differences between normal and deprivation condition values), than the non-addicted group. Although, the findings of the study indicated that the addicted and non-addicted smokers are affected differentially by deprivation and smoking the results were contrary to the expectations.

The SCL values of the non-addicted smokers were noted to be significantly lower in the deprivation condition as compared to the normal smoking condition. On the other hand, there was no significant difference in SCL's between the two experimental conditions for the addicted group. In other words, a deprivation effect was only observed for the non-addicted smokers. Examination of all the effects of smoking on the two experimental groups, regardless of the conditions supported this conclusion. The change in SCL from base-line to post-smoking periods was only significant for the non-addicted group. Although, the non-addicted and addicted smokers showed similar SCL's in the pre-smoking periods, they differed in post-smoking SCL values with the non-addicted group having significantly higher SCL's. These findings indicate that the non-addicted group is affected markedly by smoking, and show higher levels of SC on a normal smoking day as compared to a deprivation day. In other words, deprivation produces lowered activation levels as indicated by SCL data in this group.

An examination of the topographical components of smoking behaviour for the addicted and non-addicted groups, indicated that the former smoked more intensely (i.e; larger changes in respiration rate, higher number of puffs and shorter latency to next cigarette, and although not significant had a slightly higher nicotine intake than the non-addicted group).

So, although differences in smoking topography suggest that the addicted smokers should exhibit higher physiological activation levels in the post-smoking periods than the non-addicted group, they do not.

These fihdings may be due to the lower tolerance levels of non-addicted smokers to smoking and/or nicotine. However, it needs to be pointed out that although a gradual increase in SCL was noted for smokers, this change did not appear to be dose related. Since, SCL is the only measure that differentiated the responsiveness of the two experimental groups to experimental manipulations the results need to be cautiously interpreted. Further research is needed to delineate the factors (i.e; pharmacological or non-pharmacological) contributing to the changes in SCLs following smoking.

Apart from the possibility of lower tolerance levels for nicotine in non-addicted smokers, a second explanation seems to be plausible in elucidating the findings. Non-addicted smokers tend to have a lower consumption rate. Thus, they have larger time gaps between their cigarettes as compared to the addicted smokers. These time-gaps can be regarded as temporary deprivation periods. The results of the present study showed that the first cigarette after a period of deprivation produces a marked increase in heart rate which persists throughout the experimental session, whereas HR in a normal smoking day returns to base-line levels in approximately 10 minutes following smoking. This effect may be due to the initially lower HR levels noted in the pre-smoking periods of the deprivation condition.

The marked change in physiological activation levels as indicated by the post-smoking increase in HR in the deprivation condition seems to be very crucial in understanding the smoking behaviour of the non-addicted smokers. A synthesis of the findings pointing out the differential responsivity of the addicted and non-addicted smokers to smoking and the effects of post-deprivation smoking suggests that there may be two types of smoking which are likely to be maintained by pharmacological rewards. The types are mainly differentiated in terms of the frequency and degree of occurrance of pharmacological effects.

Table 6.18 outlines the two types of pharmacological addiction.

TYPE	PHARMACOLOGICAL EFFECTS	WITHDRAWAL
I: Smoking to maintain a consistent blood nicotine level (heavy smokers)	A stable level of physiological activation	Withdrawal experienced any time the blood nic. level falls below a certain level
II: Smoking with low frequency (light smokers)	Intermittent, but marked changes in physiological activation levels (i.e; resembles post-deprivation smoking) with each cigarette.	Likely to experience withdrawal if abstains from smoking for long time periods or at certain physiological states (i.e; high or low arousal)

Table 6.18 Summary of two types of pharmacological addiction.

The addiction indices used in this study seem to be identifying the first type of pharmacological addiction. Further research is needed to understand the smoking behaviour of the second type. It is important to investigate whether light smokers, who are likely to have a significant increase in physiological activation levels after each cigarette are smoking consistently in specific situations (i.e; low-or high-arousal). At this stage it is difficult to know whether and to what extent the smoking behaviour of the second type proposed above is maintained by the marked effects of smoking that are likely to occur every time they smoke. It is necessary to investigate the role of nicotine for this group. In the present experiment it was noted that both groups had a

higher nicotine intake in the laboratory deprivation condition as compared to daily life nicotine intake. Since no significant difference was noted between lab. normal smoking and daily life nicotine intake, this finding does not seem to be related to the conditions of laboratory smoking. It suggests that the non-addicted smokers also show a compensatory smoking after a period of deprivation by titrating their nicotine intake. Thus, at present it seems advisable to view pharmacological addiction as either smoking to maintain a consistent blood nicotine level or smoking less frequently in order to achieve a marked effect after each cigarette.

Russell et al (1978), have also made a distinction between smokers in terms of "peak seekers" and "trough-maintainers". They have stated, "for those smokers who smoke less than one cigarette per hour and inhalers the predominant plasma profile is one of repeated high nicotine peaks, whereas the accumulation of nicotine in the body would suggest that those who smoke at least one cigarette every thirty minutes would tend to show peaks which are smaller relative to the absolute level...Very tentatively we would suggest that trough maintenance is the main motive for the addicted heavy smoker while optimal peak effects are more important to indulgent smokers who smoke less heavily". (pp. 345). The model presented in Table 6.18 is in line with Russell et al's suggestions. Type II smokers may be peak seekers (i.e; smoking for marked changes in physiological activation levels), whereas type I smokers may be trough maintainers.

It would be interesting to investigate whether there is a progression from smoking for peak effects to smoking in order to maintain a consistent blood nicotine level. Since on a behavioural level every smoker starts as a light smoker and the majority increase consumption to become heavy smokers, it would be interesting to investigate the characteristics of long-time light smokers.

A second important point that emerged from the findings was the interaction between nicotine intake and some organismic variables in determining physiological change. It was found that a group of low-nicotine intake smokers had a significant increase in heart rate in both the normal smoking and the deprivation conditions, whereas the high-nicotine intake group only exhibited a significant change in heart rate in the deprivation condition. The findings from this analysis pointed out that the level of nicotine intake may be dependant on individual needs and different dosages of nicotine may produce similar response levels in different individuals. Thus, the dose-response relationship needs to be examined in order to understand the motives of smoking. Some individuals may have lower nicotine intake, not because that they are not smoking for pharmacological motives, but simply because they need less nicotine to achieve marked physiological effects.

6.5 CHAPTER SUMMARY

The main aim of this study was to investigate the effects of cigarette smoking and deprivation on physiological activation levels and attentional performance of pharmacologically addicted (N = 9) and non-addicted (N = 9) smokers as classified by Russell et al (1974), smoking typology scale. Smoking parameters and nicotine intake (from butt-nicotine analysis), was also examined.

The results showed that smoking only produces a marked increase in skin conductance levels for the non-addicted smokers. Although, no significant difference was noted between the two experimental groups in nicotine intake, the addicted smokers showed more intense smoking parameters and had a shorter latency to their second experimental cigarettes than the non-addicted group.

A control group of non-smokers (N = 9), did not show significant changes in physiological activation levels in periods corresponding to smoking for the smokers. Thus, the changes in physiological activation levels observed for smokers are likely to be due to smoking and/or nicotine intake. Deprivation was noted to produce a decrease in heart rate for all smokers, whereas only the non-addicted group was noted to have a lower SCL in the deprivation condition as compared to the normal smoking day.

Smoking seemed to have prevented the decrement in vigilance test performance. Addicted smokers showed a superior performance in the post-smoking vigilance test, non-addicted smokers did not show any change in performance between the first and second tests, whereas non-smokers, though

not significant showed a deterioration in performance in the second test.

The implications of the findings for the concept of pharmacological addiction have been discussed and two types of smoking that may be maintained by pharmacological effects have been suggested.

The farther one pursues knowledge

The less one knows.

Tao Te Ching.

CHAPTER 7

OVERVIEW AND PROJECTIONS

The first part of this thesis provides an analysis of several research areas that are relevant to a general understanding of cigarette smoking. The second part describes an experimental investigation of certain issues raised by a synthesis of these research findings.

The modification, the maintenance, the effects and the types of smoking has been reviewed in the first part. Although these are closely interconnected areas, the majority of researchers interested in one field seemed to have overlooked the findings from other areas. However, it seems apparent that a thorough understanding of smoking behaviour can only be achieved by incorporating findings from research on different aspects of smoking behaviour. In treating cigarette smoking the emphasis has been on treatment methods rather than on smokers. Researchers employing methods derived from learning theory principles have held the view that smoking is a learned, maladaptive behaviour that can be modified or extinguished. On the other hand, researchers using group therapy, hypnosis, or multicomponent treatment packages have held an ambiguous, less defined model of smoking behaviour. Although, the treatment strategies that have been used in the smoking modification field have been shown to be effective in dealing with other clinical problems (e.g; phobias, obsessions, etc.), before we acquire a thorough and valid understanding of cigarette smoking, there seems to be little justification for applying these methods to the modification of this habit. The effectiveness of a treatment method cannot be assessed in isolation from "who" and "what" it is designed for. Researchers in smoking modification seem to have adopted very general answers to these questions.

However, research in smoking typologies indicates that smokers differ in their motives for smoking and/or in the occasions on which they usually smoke. This work suggests that we should not view smokers as a homogeneous group. If the factors maintaining the smoking behaviour differ between smokers, then a single treatment method is not likely to be universally effective for all smokers.

The smoking typology schema, used in this thesis suggests that smokers can be classified as pharmacologically addicted and non-addicted. Pharmacological addiction refers to smoking that is maintained (reinforced), by the pharmacological effects of smoking. Nicotine, delivered by cigarette smoking seems to be a powerful agent in producing changes in physiological and behavioural activation levels.

On the basis of the typology schema, it may be expected that nicotine is an important factor for smokers classified as pharmacologically addicted. Thus, investigating the role of nicotine in the maintenance of the smoking behaviour of pharmacologically addicted and non-addicted groups seems to be a fruitful line of research. Unfortunately, although much research has been conducted on the role of nicotine, very few researchers have investigated the role of nicotine for different types of smokers (e.g; light and heavy, see chapter 3). Thus, the research on the role of nicotine has also been plagued by the notion of a homogeneous group of smokers and a single, universal motive for smoking (i.e; nicotine). For this reason, the results, not surprisingly have been contradictory and inconclusive.

The discussion that follows centres about two major questions:

i - Can we identify types of smokers by methods other than self-reports?

ii - Can we get better treatment-outcome results by tailoring treatment methods to types of smokers?

These questions will be dealt with seperately in the light of the findings of the present study.

i - Can we identify types of smoking by methods other than self-reports?

At present, the classification schemas for identifying different types of smoking are based on the self-reports of smokers. These reports suggest that smokers differ in their motives for smoking and/or in the occasions on which they usually smoke. Although, the verbal reports of smokers provide valid data to classify them in terms of their subjective self-images, the objective validity of these reports needs to be investigated.

The present study dealt with this issue by investigating the effects of cigarette smoking, deprivation and nicotine intake on smokers classified (according to their verbal reports), as smoking primarily to obtain pharmacological effects and on smokers who report smoking for other reasons (e.g; social, sensorimotor, etc.). The pharmacologically addicted smokers report smoking in order to stimulate themselves and to avoid withdrawal symptoms. Since, these reasons seem to reflect a tendency to manipulate activation levels, physiological and behavioural measures indicative of activation were used to assess the self-reports of the smokers.

The present study yielded two interrelated results which suggested that the two groups responded differently to smoking.

Cigarette deprivation: Addicted versus non-addicted groups -

It was noted that deprivation produced a marked decrease in physiological activation levels (i.e; skin conductance level), for only the non-addicted smokers.

Effects of cigarette smoking: Addicted versus non-addicted groups -

Smoking was noted to produce a marked increase in physiological activation levels (i.e; SCL) only for the non-addicted group (exp. 2). The increase in sensitivity to detect flicker in light flashes after smoking was

only significant for the non-addicted group (exp. 1).

These results suggested that smoking has a marked effect on the physiological activation levels and sensory sensitivity of the non-addicted smokers. Similarly, the difference between the deprivation and normal day SCL values was only significant for the non-addicted smokers. No significant difference was noted between the two experimental groups in nicotine intake (as estimated from butt-nicotine analysis). Thus, the differential responsivity of the two groups to smoking is not likely to be due to differences in nicotine intake.

The differential effects of smoking on the two groups may be explained in two ways:

Tolerance to smoking and/or nicotine -

The addicted group was noted to have a significantly higher level of cigarette consumption than the non-addicted group. Thus, it seems plausible that the addicted smokers, due to a greater exposure to nicotine may have developed more tolerance to the effects of smoking and thus do not exhibit very marked physiological changes following smoking. Russell (1971), stated that tolerance to the effects of nicotine may be due to "an increased capacity to metabolize and excrete the drug (i.e; nicotine) as a result of enzyme induction mainly in the liver" and also adaptive changes at synapses. Beckett and Trigs (1967), have noted that non-smokers excrete unchanged nicotine in their urine in a greater proportion to a given dose than do smokers. On the other hand, Schachter et al (1977), noted a significant negative correlation between number of cigarettes smoked daily and urine alkalinity. This finding suggested that smokers with high consumption levels may be compensating for the high nicotine excretion rate (see chapter 3). The excretion rates of given doses of nicotine by light and heavy smokers would seem worthy of investigation.

Effects of smoking in relation to smoking frequency -

The findings of the present study may also be explained in terms of the longer time gaps between the cigarettes of the non-addicted group as compared to the addicted group. Since the non-addicted smokers tend to be light smokers they are likely to have long time gaps between their cigarettes.(1) In the present study it was noted that smoking after a period of deprivation has a pronounced effect on physiological activation levels (i.e; heart rate), which persists throughout the experimental session whereas, smoking a further cigarette, after a period of normal smoking produces transitory changes in activation levels. Since with cigarette deprivation a decrease in physiological activation levels is generally observed, the marked effect of post-deprivation smoking may be due to the initially low activation levels of deprivation conditions. The results of the present study on the effects of deprivation and marked effects of post-deprivation smoking are in line with the findings reported in literature (see chapter 4).

Thus, when we consider the smoking behaviour of the non-addicted smokers in the light of these findings, it seems that with each cigarette these smokers are likely to exhibit marked physiological changes, which resemble those observed with post-deprivation smoking. In other words, if we view the time gaps between cigarettes as temporary deprivation periods, then light smokers seem to have a smoking pattern that is likely to produce effects similar to post-deprivation smoking.

At this stage it would be useful to examine types of regular smoking in terms of the frequency of smoking. Fig. 7.1 (overleaf), outlines

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(1) - In the present study, addicted smokers had a significantly shorter latency to light their second cigarette as compared to the non-addicted smokers.

types of smoking in terms of frequency of smoking and the frequency and degree of physiological effects that are likely to be produced.

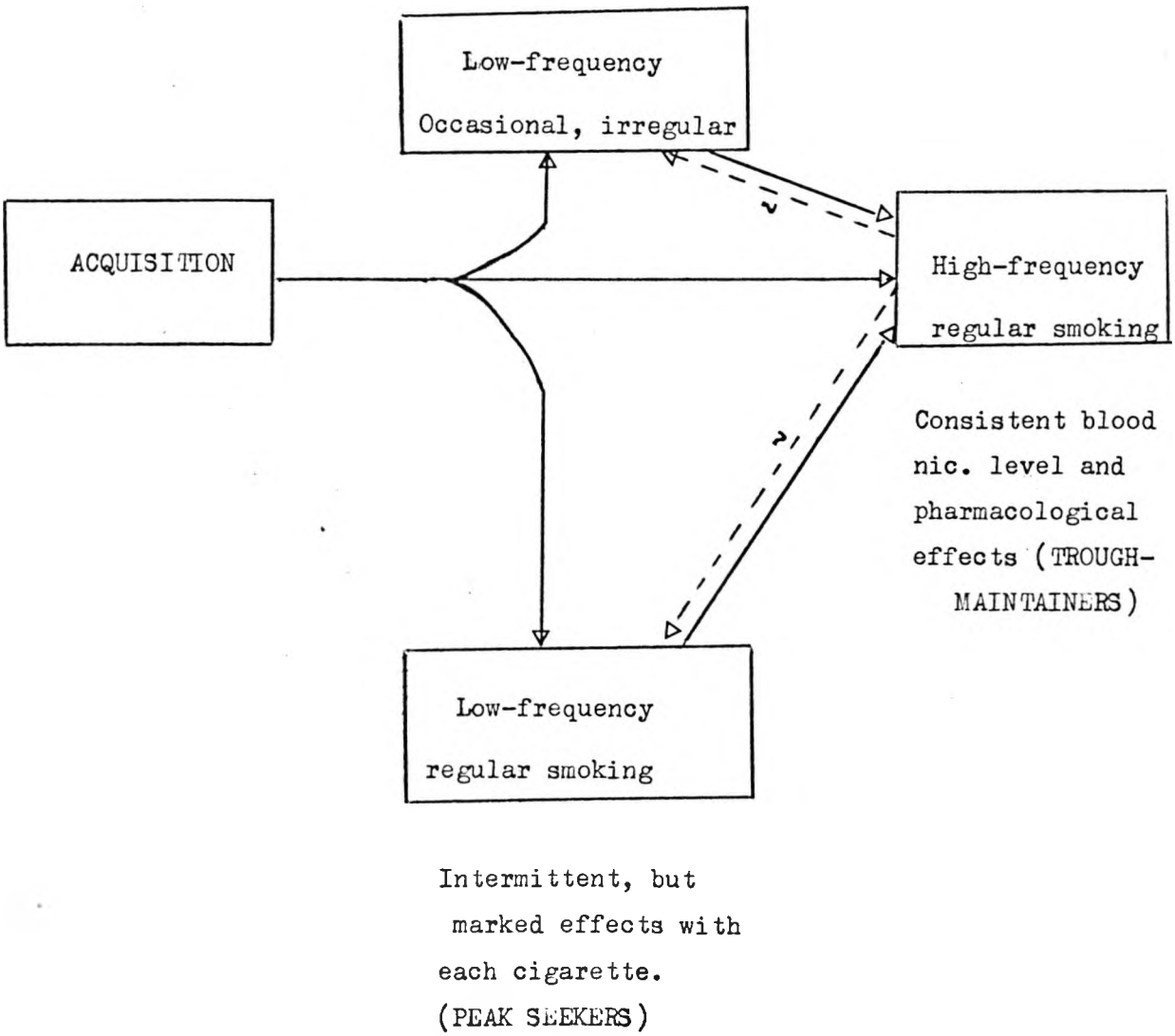

Fig. 7.1 Types of smoking: Frequency, regularity, and pharmacological effects.

The present study focussed on smoking behaviour after it is established as a regular habit. Therefore, the motives for acquisition will not be dealt with. However, it is important to investigate the time period that is necessary to develop a firmly established smoking habit. McKennell and Thomas (1967), have noted that the mean length of period between the first smoking and the onset of regular smoking is related to the age of first smoking. They reported that the younger the person is when he first experiments with smoking the slower will be his rate of becoming a regular smoker. These authors

have defined regular smoking as smoking one cigarette per day. However, regular smoking as used in Fig. 7.1, refers to progression from the acquisition stage to a stage where the smoking habit is established very firmly (i.e; light or heavy smoking).

Low-Frequency Smoking (i.e; Light-smokers)

Two types of light smoking have been distinguished in Fig. 7.1. One is the occasional smoker who smokes very infrequently and who does not have a regular consumption pattern, and the second is the light smoker who smokes regularly in low-frequency, every day or in specific physiological states (i.e; low or high arousal). The second type will be discussed in this section.

The present study demonstrated that the non-addicted group, who were also light smokers (X = 11 cig/day), showed a marked increase in SCLs following smoking and had significantly lower SCLs on deprivation days as compared to normal smoking days. Thus, these results suggested that smoking produced a marked effect on the physiological activation levels of this group.

Thus, the smokers classified as non-addicted by their verbal reports to a typology scale, either due to lower tolerance levels to nicotine or to the similarity of their smoking to post-deprivation smoking are likely to obtain intermittent, but marked effects from smoking. Such smokers are likely to be the "peak seekers" as suggested by Russell et al (1978). These authors have described peak seekers as those smokers who smoke less than 1 cig/hour and who inhale. The criteria provided by this definition for the frequency of smoking is very broad. Although, a consumption criterion may be useful, it seems necessary to include other factors in the classification of smoking that may be maintained by the intermittent but marked physiological effects. Consistency of frequency/day, in certain situations, and/or physiological states

may also be a useful index in identifying this type of smoking.

High-Frequency Smoking (i.e; Heavy smokers)

Heavy smokers, are those smokers who smoke regularly and with a high frequency throughout the day. The main motive maintaining their smoking behaviour seems to be the maintenance of a consistent blood nicotine level. These are likely to be smokers classified as pharmacologically addicted by the Russell et al (1974), smoking typology scale. Russell et al (1978), described this group as those smokers who smoke at least one cigarette every 30 minutes. Since, the plasma half-life of nicotine has been estimated to be approximately 30 minutes, this criterion is comparible with the motive of maintaining a consistent blood nicotine level. Russell et al (1978), suggested that the plasma blood profile of these smokers are likely to show peaks which are smaller relative to the absolute levels. The findings of the present study support this view. No significant change was noted in SCL of the addicted smokers after smoking.

Although, it was predicted that a period of cigarette deprivation would have a marked effect on the physiological activation levels of this group, this was not borne out by the SCL results. On the contrary, no significant difference was noted between the normal and deprivation day SCL values for the addicted group.

As was pointed out earlier (chapter 6), SCL was the only measure that suggested a differential responsivity of the two groups to smoking. Although, the sweat glands are innervated solely by the sympathetic nervous system and thus the increase in SCL may be interpreted as an increase in sympathetic activity, the lack of a direct dose-response relationship for this measure poses some difficulties for interpretation. Thus, the results need to be interpreted cautiously before further research is undertaken to

delineate the factors in cigarette smoking that contribute to the observed changes in SCL levels.

Fig. 7.1 illustrates how smoking may progress from one type to the other. The broken lines indicate that progression from heavy smoking to light smoking seems highly unlikely, unless the smoker is trying to cut down his consumption level. As has been noted in the method of gradual reduction (see chapter 1), with fewer cigarettes smokers are likely to experience marked effects with each cigarette, which may further reinforce the smoking behaviour. On the other hand progression from occasional smoking to light smoking and then to heavy smoking seems to be plausible.

The findings of the present study indicated that cigarette smoking produces physiological changes in the direction of increased physiological activation (i.e; smoking day values compared with deprivation condition values) for the whole sample. These results suggest that cigarette smoking produces pharmacological effects in all smokers (probably if they are inhalers). However, the degree of this effect seems to depend on the frequency of smoking. This suggests a need to re-examine the concept of pharmacological addiction. At the outset of this thesis, the author held the view that cigarette smoking may be predominantly reinforced by its pharmacological effects in some smokers. The aim was to identify this group and to apply a pharmacological treatment method for these smokers. However, in the light of the findings of the present study this goal seems rather simplistic.

Pharmacological effects, though in different degrees seem to be present in all smokers (i.e; addicted or non-addicted as classified by a typology scale). Thus, it seems more appropriate to represent the role of pharmacological effects along a continium as illustrated in Figure 7.2.(overleaf)

Light-Smokers regular smoking		Heavy-Smokers regular smoking
Motive: Intermittent but marked physiological effects		Motive: Continuously high nicotine intake, but less effect with each cigarette

Fig. 7.2 Range of pharmacological addiction.

It was noted that the two experimental groups did not differ significantly in nicotine intake. This result indicates that light and heavy smokers do not differ in nicotine intake from each cigarette but they do differ in nicotine intake per day.

Although, as outlined in Fig. 7.2 cigarette smoking may produce pharmacological effects, of different intensity in heavy and light smokers, this does not necessarily indicate that smoking is maintained by these effects. It seems more appropriate to view the factors maintaining smoking behaviour in terms of a hierarchy (i.e; pharmacological, social, sensorimotor, etc.). Delineation of the importance of different factors in reinforcing cigarette smoking in different types of smokers will aid the development of appropriate treatment strategies.

ii - Can we get better treatment-outcome results by tailoring appropriate treatment methods with types of smokers?

In order to devise appropriate treatment strategies we need to develop reliable methods to identify types of smokers according to the hierarchy of reinforcers maintaining their smoking behaviour.

The findings of the present study suggested that pharmacological effects of different intensity and frequency are produced in all regular smokers

(probably if they are inhalers). Thus all smokers may be dependant on the pharmacological effects of smoking to a certain extent. However, although the results pointed out that light smokers exhibit marked effects following smoking, this does not necessarily indicate that pharmacological effects are playing a predominant role in their smoking habit. In the following section several research issues raised by the present study will be presented.

Before further research is undertaken it seems premature to make suggestions about treatment methods that may be appropriate for modifying the smoking behaviour of different types of smokers.

Suggestions for further research:

i - Longtitudinal analysis of smoking behaviour in daily life:-

Measures: Smoking frequency (from detailed records)
Time and occasion of smoking (" " ")
Nicotine-intake (e.g; butt-nicotine analysis)
Smoking topography (Video-recording)
Physiological effects (Portable recording devices)
Subjective effects (e.g; mood ratings)

Duration of study: One day/week for at least six months.

It is important to investigate whether smokers exhibit a regular smoking behaviour (i.e; smoke consistently at regular time intervals or occasions). This is particularly crucial for light smokers. Although, a longtitudinal study on smoking habits may impose practical limitations and may be time consuming, reliable data on when individuals smoke, the physiological and psychological effects of smoking will prove to be invaluable to a thorough understanding of smoking behaviour. It may also provide information on the stability of smoking frequency and progression from light to heavy smoking.

The measures outlined above can be obtained by an automated system, by which every time the subject takes a cigarette from a cigarette holder this can trigger the video-recording and a short taped interview. The replies can be recorded on a separate tape recorder. Although, this type of measurement will limit the location of observation, it will still provide a reliable and comprehensive record of smoking and its effects.

ii - The role of nicotine for light and heavy smokers:-

As has been pointed out earlier, although much research has been undertaken to investigate the effects of nicotine manipulations on smoking parameters, very few investigators have examined the differential role of nicotine for light and heavy smokers (see chapter 3). The few studies conducted on these groups separately, have pointed out that nicotine is not an important factor for light smokers. However, in order to assess the effects of nicotine manipulations on light smokers, the manipulations must be introduced at appropriate time periods. Since light smokers have a low smoking frequency, first it is necessary to have a reliable record of when they smoke. This type of knowledge may be acquired either by asking them to keep a detailed record of the times they smoke or from the type of investigation outlined in section i, above. In other words, if a light smoker never smokes in the mornings, it is very unlikely that he will respond to nicotine manipulations introduced in the morning.

On the other hand, since heavy smokers tend to smoke regularly throughout the day it does not seem important at which time of the day the role of nicotine is assessed for this group. If they are smoking in order to maintain a consistent nicotine level then they are likely to respond to nicotine manipulations at any time during the day. However, since light-smokers only smoke at particular time periods they are only likely to respond to nicotine manipulations at these times if they are smoking to obtain nicotine.

In the present study it was noted that both of the experimental groups had a significantly higher nicotine intake from their first post-deprivation cigarettes as compared to normal daily life nicotine intake. Thus, both groups seemed to have titrated their nicotine intake to compensate for the deprivation period. So, the results suggested that nicotine is also an important factor for the light smokers. However, the higher nicotine intake after a period of deprivation may also be a result of a purely behavioural compensation (i.e; more puffs, longer cigarette duration, etc.). In the present study four non-addicted smokers refused to smoke the morning and afternoon cigarettes given to them, which they were asked to smoke a day before the first experimental session. They stated that they never smoke before the evening. Considering the fact that these smokers were asked to smoke two cigarettes during the experimental session points out a shortcoming of the present study. Although, these smokers did not object to smoking in the afternoon session, this certainly was not something they were accustomed to.

iii - Tolerance versus Low-baseline values:-

The marked changes in SCL levels observed only for the non-addicted smokers in the second study and the increase in sensitivity in the flicker fusion test for this group following smoking raised the issue of whether these smokers have a lower tolerance to the effects of smoking, or whether this result is due to the time gaps between their cigarettes.

If tolerance develops to the effects of smoking, then light smokers are also likely to develop tolerance to the doses of nicotine they inhale. On the basis of this view, marked effects are not likely to be observed in long-term light smokers. On the other hand, if the marked effect is related to the time-gaps between cigarettes then long-term light smokers are likely to exhibit marked changes in activation levels after smoking. On this basis it may also be

predicted that if heavy smokers leave gaps between their cigarettes, similar to the ones observed in light smokers then they also will show marked changes in activation levels following smoking.

In order to resolve this issue the physiological effects of smoking on the following groups may be investigated:

a - Long-term light smokers - smoking at their usual frequency.

b - Heavy smokers - smoking with time gaps between their cigarettes (e.g; matched to the time-gaps observed for light smokers)

c - Heavy smokers smoking as usual.

In the present study it would have been appropriate to ask the smokers when they had their last cigarette before the experimental session on the normal smoking condition. This would have provided data on the time-gap the two groups had between their last daily life and experimental cigarette.

So, in conclusion, the present thesis aimed at demonstrating a differential responsivity for pharmacologically addicted and non-addicted smokers as classified according to a typology scale, to smoking and deprivation. Although, the study revealed that the two groups are effected differentially by smoking, the findings were contrary to the expectations. Only the non-addicted group was noted to exhibit a marked change in activation levels after smoking. This finding suggested that pharmacological effects may also be reinforcing for the light smokers.

The results raised several research questions, which need to be investigated before any conclusions can be drawn about appropriate treatment strategies.for different types of smokers.

The present study needs to be replicated with a larger sample,

of both sexes, and preferably in natural settings. No subjective reports (e.g; mood ratings), were obtained in the present study (in order to prevent interference with the physiological recording). However, it would also be useful to examine the subjective effects of smoking and deprivation. Although, findings from controlled, laboratory research are invaluable, the generalizability of laboratory research findings to daily life smoking conditions can only be assessed by actually experimenting in natural life settings.

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APPENDICES

A:

i- Newspaper article.

ii-Letter to respondents.

iii-Smoking Typology test- Russell et al (1974) & Scoring.

iv-Smoking Habits questionnaire- Scoring for addiction index.

v-Smoking frequency record form.

vi-Instructions for the " Smoking" and " Deprivation" conditions.

vii-Letter to subjects and self-control techniques in giving up smoking (from; Yucesoy,N.(1976)).

B:

i-Instructions for the " Hand Steadiness" test.

ii -Instructions for the " Critical Flicker Fusion" test.

iii -Instructions for the " Peripheral Visual Field" test.

C: ANOVA tables for :

Pulse rate,

Systolic blood pressure,

Diastolic blood pressure,

Hand steadiness,

C.F.F. threshold,

Peripheral visual field,

Finger temperature.

D: Instructions for experiment II.

E: Computer programs for experiment II.

F: Histograms showing the physiological measures over the experimental periods in the smoking and deprivation conditions for each subject.

G: ANOVA and DUNCAN M.R.T tables : Addicted versus non-addicted groups.

i-Heart rate.

ii-Skin conductance level.

iii-Lability.

iv-Respiration rate.

v-Respiration amplitude.

vi-Inter-puff interval.

vii-Number of puffs.

viii-Cigarette duration.

ix-Latency.

x-Butt-length.

H: ANOVA and DUNCAN M.R.T. tables: Nicotine intake estimated from butt-nicotine analysis.

I: ANOVA and DUNCAN M.R.T. tables: High-versus-Low- Nicotine intake groups- Heart rate.

J: ANOVA and DUNCAN M.R.T. tables: Vigilance test scores.

i-Addicted versus non-addicted groups.

ii-Smokers versus non-smokers.

K: ANOVA and DUNCAN M.R.T. tables: NON-SMOKERS

i-Heart rate.

ii-Skin conductance level.

iii-Lability.

iv-Respiration rate.

v-Respiration amplitude.

L: Responses to the item, " would you consider yourself addicted to nicotine?".

App. A.i

CHANCE FOR SMOKERS WHO WANT TO STOP

HELP IS ON THE WAY for smokers who want to give up the habit and at the same time are willing to aid research.

Miss Nuray Yucesoy, a post-graduate student at Hull University, is studying how smokers behave when they cannot smoke and is looking for volunteers to help in her research.

She wants to hear from helpers, who will be sent a questionnaire and probably asked to attend the University's department of psychology on two consecutive days, and then, later, for about a month.

In that time Miss Yucesoy will study their behaviour when deprived of nicotine.

For some of the volunteers there will also be tests, under medical supervision, such as trying other ways of giving them the nicotine to which they are addicted.

"It is possible to give people tablets, and there is also a nicotine chewing gum But not all smokers are addicted to nicotine — some just need something to hold in their hand," Miss Yucesoy said.

The Turkish-born student, who is 25 and comes from Ankara, is using the research for her Ph.D. She has been in Hull for a year, and will spend another two years on her studies.

"Smoking is a dangerous habit and a social problem. Many people are encouraged to stop smoking after a lot of publicity, but in a month or so they go back. I want to trp to help them give up for longer than that," she said.

28.6.1977, HULL DAILY MAIL

A-ii.

9th November, 1977.

Dear

This is to inform you that you have been selected to participate in the research project on "The Effects and Methods of Giving Up Smoking".

As you already know, you will be required to attend the University twice, on consecutive days, for the purpose of testing. However, prior to these sessions I would like to have an introductory meeting, during which we can discuss the procedures to be followed and your smoking habits. I should be grateful if you could attend the Psychology Department of Hull University at <u>any time</u> between 1 p.m. and 5.00 p.m. on either Thursday, November 17th or between 10.30 a.m. and 5.00 p.m. on Friday, November 18th. If neither of these dates is convenient for you, would you please ring the Psychology Department so that we can arrange another date.

Thank you once again for your kind co-operation.

Yours sincerely,

Nuray Yucesoy

Research Student

Date:

Name:_____ Age: _____ Sex: _____ Cigarette Consumption:_____

Here are some statements about some of the reasons that people give for their smoking. Please indicate how much each statement applies to you by drawing a circle around the appropriate number.

UNCERTAIN or NOT AT ALL	A LITTLE	QUITE A BIT	VERY MUCH SO
0	1	2	3

1. I feel more attractive to the opposite sex when smoking 0 1 2 3
2. I like smoking while I am busy and working hard 0 1 2 3
3. Without a cigarette I don't know what to do with my hands 0 1 2 3
4. I want to smoke most when I am comfortable and relaxed 0 1 2 3
5. Part of the enjoyment of smoking comes from the steps I take to light up 0 1 2 3
6. Smoking helps to keep me going when I'm tired 0 1 2 3
7. I light up a cigarette whenever I talk on the telephone 0 1 2 3
8. I think I look good with a cigarette 0 1 2 3
9. I smoke more when I am unhappy 0 1 2 3
10. In the morning I usually smoke before having tea or coffee 0 1 2 3
11. I like a cigarette best when I am having a quiet rest 0 1 2 3
12. I smoke for the pleasure of offering and accepting cigarettes from other people 0 1 2 3
13. Part of the enjoyment of smoking is watching the smoke as I blow it out 0 1 2 3
14. I often smoke without really enjoying it 0 1 2 3
15. I smoke to keep my weight down 0 1 2 3
16. It is easier to talk and get on with other people when smoking .. 0 1 2 3
17. I smoke automatically without even being aware of it 0 1 2 3
18. I am very much aware of the fact when I am not smoking 0 1 2 3
19. I smoke more when I am worried about something 0 1 2 3
20. I get a definite lift and feel more alert when smoking 0 1 2 3

continued ..... P.T.O.

UNCERTAIN or NOT AT ALL	A LITTLE	QUITE A BIT	VERY MUCH SO
0	1	2	3

21. While smoking I feel more confident with other people 0 1 2 3

22. After meals is the time I most enjoy smoking 0 1 2 3

23. I smoke because I like the smell so much 0 1 2 3

24. Smoking cheers me up 0 1 2 3

25. When I have run out of cigarettes I find it almost unbearable until I can get them 0 1 2 3

26. I light up a cigarette when I feel angry about something 0 1 2 3

27. I light up a cigarette without realising I still have one burning in the ash tray 0 1 2 3

28. I feel I look more mature and sophisticated when smoking 0 1 2 3

29. I usually only smoke when I have something to drink (te, coffee, alcohol) 0 1 2 3

30. I smoke more when I am rushed and have lots to do 0 1 2 3

31. I smoke for the pleasure of having something to put in my mouth . 0 1 2 3

32. I am usually very careful not to run out of cigarettes 0 1 2 3

33. I smoke to calm my nerves 0 1 2 3

34. I find myself smoking without remembering lighting up 0 1 2 3

35. Smoking helps me to think and concentrate 0 1 2 3

36. I smoke much more when I am with other people 0 1 2 3

37. Handling a cigarette is part of the enjoyment of smoking it . .. 0 1 2 3

38. I get a real gnawing hunger to smoke when I haven't smoked for a while 0 1 2 3

39. I usually only smoke when I can really sit back and enjoy it .. 0 1 2 3

40. I feel guilty about my smoking 0 1 2 3

PLEASE CHECK THAT YOU HAVE ANSWERED EVERY ITEM

If there is any other important reason for your smoking please write it down here:

MAUDSLEY HOSPITAL SMOKERS CLINIC: SMOKING TYPOLOGY TEST

SCORING:

PSYCHOSOCIAL:

Item No: 1; 8; 12; 16; 21; 28; 36.

SENSORIMOTOR:

Item No: 5; 13; 23; 31; 37.

INDULGENT:

4; 11; 22; 29; 39.

SEDATIVE:

Item No: 9; 19; 26; 33.

STIMULATION:

Item No: 2; 6; 20; 24; 30; 35.

ADDICTIVE:

Item No: 3; 10; 14; 18; 25; 32; 38.

AUTOMATIC:

Item No: 7; 17; 27; 34.

PHARMACOLOGICAL ADDICTION DIMENSION: STIMULATION+ADDICTIVE+AUTOMATIC.

NON-PHARMACOLOGICAL ADDICTION DIMENSION: INDULGENT+PSYCHOSOCIAL+SENSORIMOTOR.

APPENDIX A-iv.

SMOKING HABITS QUESTIONNAIRE

Date .....

1. SURNAME: _____

   FIRST NAME: _____

2. ADDRESS: _____

   _____

   _____ Telephone Number: _____

3. AGE: _____ (DATE OF BIRTH: _____)

4. SEX: _____

5. MARITAL STATUS: a) Single ..... b) Married ..... c) Divorced.....

   d) Widowed ..... e) Separated .....

6. PRESENT OCCUPATION: _____

7. HUSBAND'S OR WIFE'S OCCUPATION: _____

8. Is the most important person you spend your time with a smoker? YES.....

   NO .....

9. For how many years have you been smoking regularly? _____

10. At present, how many cigarettes do you smoke a day? _____

11. What do you think is the lowest number of cigarettes you could manage to cut down to without much discomfort? (Please think carefully before you put down your estimate)

    _____

12. Have you ever tried to give up smoking completely?

    a) Yes..... How many times? .....

    b) No.....

P.T.O.

13. Have you ever tried to cut down the number you smoke? (as opposed to giving up completely).

a) Yes ..... How many times? .....

b) No .....

14. If you managed to stop smoking, for how long did you stop? (Please tick as appropriate).

Several Days	
Several weeks	
Several months	
Several years	

15. At present, do you want to stop smoking? Yes .....

No .....

16. If 'Yes', what are your reasons? (Please tick the ones you apply to yourself)

i) Health:

a) Chronic Bronchitis.....
b) Emphysema.....
c) Coronary Heart Disease.....
d) Arterial disease.....
e) Cancer.....
f) Tuberculosis of the lungs...
g) List any other condition .....
.....
.....

ii) Cost of cigarettes .....

iii) List other reasons, if any .....
.....
.....

17. Do you suffer from any physical illness related to smoking?

a) Yes..... Please name it .....

b) No.....

18. Do you smoke cigars or pipe?

a) Yes..... If so, how much?.....

b) No.....

19. What type of cigarettes do you usually smoke?

Manufactured with filter tips.....

Manufactured plain.....

Hand rolled.....

20. What brand of cigarettes do you usually smoke?.....

.....

21. When smoking do you inhale?

	Addiction Index Score
A lot	(3)
A fair amount	(2)
Just a little.....	(1)
Not at all	(0)

22. How far do you usually take the smoke in?

Hold it in the mouth.....	(0)
To back of throat.....	(1)
Partly into chest.....	(2)
Deeply into chest.....	(3)
Not known.....	(0)

23. Do you chain smoke?.... Yes:..... NO: ..... Sometimes .... Yes:(2); No:(0); Sometimes:(1)

If so, in what situations? .....

.....

.....

24. If you have not smoked for a while, do you tend to smoke two or three cigarettes in quick succession, to catch up and feel "normal" again?

Often.....	(3)
Sometimes.....	(2)
Rarely.....	(1)
Never.....	(0)

25. In the morning do you have your first cigarette, before tea or coffee?

Often.....	(3)
Sometimes.....	(2)
Rarely.....	(1)
Never.....	(0)

26. In the morning, how long after waking-up do you light your first cigarette?

Less than one minute.....	(3)
1-5 minutes.....	(3)
6-15 minutes.....	(2)
16-30 minutes.....	(2)
31-60 minutes.....	(1)
Over one hour.....	(0)

27. At night, how long is the usual period between finishing your last cigarette and going to sleep?

Less than one minute.....	(3)
1-5 minutes.....	(3)
6-15 minutes.....	(2)
16-30 minutes.....	(2)
31-60 minutes.....	(1)
Over one hour.....	(0)

28. Do you think you will suffer from any withdrawal symptoms if you give up smoking?

a) Yes..... please state them .....
.....
.....

b) No.....

29. Do you think that you are addicted to nicotine? ..... Yes: ... (1)

NO: .... (0)

Do not know:

... (0)

ADDICTION INDEX SCORE =

Questions: 21; 22; 23; 24; 25; 26; 27; 29.

APPENDIX A-v

INSTRUCTIONS:

This form will be used to record the number of cigarettes you smoke during a three days recording period.

The aim is to get an exact estimate of your smoking frequency. Please do not try to alter your smoking habits at this state. Go on smoking as normal. Try to be accurate in your recording. To help you in this, you can put a mark at the appropriate box of your record form whenever you light a cigarette.

Please bring this form to the next meeting.

RECORD FORM

Name: Miss / Mrs. / Mr. _____

	DATE	DAY	BEFORE BREAKFAST	BREAKFAST-LUNCH	LUNCH-EVENING MEAL	AFTER EVENING MEAL	TOTAL OF DAY
1							
2							
3							

For Office Use.

RS No_____

Grp _____

X _____

APPENDIX A-vi

SMOKING RESEARCH

SMOKING CONDITION

INSTRUCTIONS:

You can smoke as many cigarettes as you normally would, prior to the testing session. It would be very helpful if you can avoid taking alcohol and drugs and also try to have a good sleep, the night before the experimental date.

On your arrival, you will have a short resting period. During this period you will be asked to smoke a cigarette of your usual brand, so please remember to bring your cigarettes with you.

The testing session will take approximately one hour.

Many thanks for your co-operation.

DATE OF THE TESTING SESSION:

TIME:

APPENDIX A-vi

SMOKING RESEARCH

DEPRIVATION CONDITION

INSTRUCTIONS:

The experimental period starts from the evening prior to the testing date. At 12 p.m, you should stop smoking. After this time DO NOT SMOKE any cigarettes. You may have a craving and be tempted to smoke, but do not give in. Your deprivation from cigarettes is a very important factor for this study.

It would be very helpful if you can avoid taking alcohol and drugs and also try to have a good sleep, the night before the besting date.

The testing will take approximately one hour. After the testing you will be asked to smoke a cigarette of your usual brand, so please remember to bring your cigarettes with you.

Many thanks for your co-operation.

DATE OF TESTING:

TIME:

21st June, 1978.

Dear

I am very sorry for the delay in informing you of the outcome of the "Smoking Research Project".

The findings of the project has led me to reorientate my research interest from treatment of smoking to pure experimental investigation. So, unfortunately I will not be conducting any treatment sessions in future.

However, I am enclosing the procedures and instructions for a "Self-Control" method in giving up smoking, which had proved to be effective in my previous work. I hope it will also be helpful for you.

I would like to express my gratitude once more for your valuable contribution to this project.

Yours sincerely,

N. Yucesoy.

Note: A report on the
"Smoking Research Project"
is available in the Psychology
Dept. Library.

INTRODUCTION TO TREATMENT

There is plenty of evidence that if you smoke you are more likely to get lots of nasty diseases. Your chances of lung cancer are higher. Many studies show this. Research has also shown that smokers risk heart disease. They are also more likely to suffer from other diseases. Some of these are not serious, but many are. As well as this the chances are that they will die sooner. Cigarette prices also place a burden on the people who smoke.

Smoking is a habit. You have learned to smoke in many different occasions. For example; you may want to smoke when you have a cup of coffee. The presence of these things reminds you of smoking. That is why many people find it hard to give it up. There is no magical way to stop smoking. However, you can quite easily learn to control your smoking. You can do this by learning the techniques of "SELF-CONTROL".

WHAT IS SELF-CONTROL?

"Self-Control" is a person's effort to control his own behaviour. There is no general pressure. The individual decides to change a certain behaviour himself. He makes an action plan for the change he wants, to take place. He alters his environment systematically.

It is quite easy to learn these methods. They are based on ideas of how people learn to change their habits. Many people have said that the "Self-Control" techniques are useful.

Now, we can look at these methods. Four separate sheets of "Self-Control" techniques are enclosed. Now, you can go on to the next page. Read the techniques you will use during the first week.

SHEET NO: 1

"SELF-CONTROL" TECHNIQUES FOR THE FIRST WEEK

1. HOW TO SMOKE:

Smoking is a behaviour you can control. To prove to yourself that you can successfully alter your smoking behaviour, here are some rules to follow;

1 - Put your cigarette down, on the ashtray after every puff.

2 - Do not lift your cigarette, until you exhale and you are ready for the next puff.

3.- Introduce pauses into your smoking. At first, wait after the middle of the cigarette. Put your cigarette down and do not lift it for one minute. Then you can begin smoking again if you wish. Gradually try to put down your cigarette sooner and sooner after you start it.

4 - Wait one minute after you take out a cigarette before you light it. Do this only after you feel confident to pause immediately after you light a cigarette.

2. HOW TO RESIST TEMPTATION:

It is obvious that it is difficult to smoke if you do not leave cigarettes lying about or if you do not buy more than you need. Much smoking is "automatic". You may be unaware of the fact that you are going to smoke or you are in fact smoking.

Make it hard for yourself to get your cigarettes. Keep cigarettes only in one place in the house (or office), which is not handy. A trip to get a cigarette may prevent your smoking. You may change your mind while you are reaching for a cigarette.

Here are some rules to help you do this:

1 - Do not leave cigarettes lying about, within easy reach, in the house, in your car or in your office.

2 - Keep all cigarettes out of sight except when you are smoking. Store all your cigarettes in a cupboard, which is not easy to reach - preferably a locked one, that you have to unlock every time you want to smoke. Keep your ash tray and lighter in the same place and only get them when you are going to smoke.

3 - Do not buy more than one pack of cigarettes at a time.

Here is a brief outline of the techniques you will use. This will act as a handy reminder to you:-

1 - Put your cigarette down on the ashtray after every puff.

2 - Introduce pauses into your smoking. Starting at first, after the middle of the cigarette. Gradually try to put down your cigarette sooner and sooner. When you feel confident wait one minute before you light your cigarette. You may change your mind while you are waiting.

3 - Do not leave cigarettes lying around in the house, in the car or in your office.

4 - Keep all your cigarettes, ashtray and lighter in One special place, like a side-board.

5 - Do not buy more than one pack of cigarettes at a time.

SHEET NO: 2

"SELF-CONTROL" TECHNIQUES FOR THE SECOND WEEK

This week you will practice some additional techniques. It is important to continue using the ones you have learned last week as well as the ones discussed below.

1. WHERE AND WHEN TO SMOKE:

Restrict the places you smoke. Smoke only at one place. By setting aside a special place to smoke you will learn to smoke only at this place. You will slowly "unlearn" the habit of smoking at other places. At first you may find this difficult but gradually you will learn not to expect cigarettes at other places.

Here are some rules to help you:

1 - Smoke only at one special, distinct place. At home, (and also at your Office), choose a special chair for smoking.

2 - Try to restrict your smoking to certain times in each hour. For example; smoke only on the half hour and the hour.

3 - Do not smoke at any other place except, while sitting in the chair you have chosen for smoking.

4 - If possible, choose a chair where you will not feel very comfortable. A chair which is not in a handy position, will be very suitable for your purpose.

5 - Leave the chair after you finish smoking. Use this chair only for smoking.

II. SMOKING AS A "PURE-EXPERIENCE"

You are used to smoking on many different occasions - such as when

reading a book or listening to the radio, etc. These situations then may become times which remind you of smoking. So, if you separate smoking from all other activities, it will not continue to be associated with these times.

Here are some rules about this:-

1 - When you are smoking, do not do anything else,
e.g; Do not read, watch the television, etc. while smoking. Ask your friends and your family not to speak with you while you are smoking.

2 - Concentrate on smoking and nothing else. Make smoking a "pure-experience".

Here is a brief outline of the "self-Control" techniques you will practice this week, in addition to the ones you have learned the first week, to control your smoking:-

1 - Choose a special chair for smoking.
2 - Restrict smoking to certain periods only.
3 - Only smoke while sitting in this chair.
4 - If possible, choose a chair which is not in a handy position.
5 - Do not do anything else while you smoke, (like reading, watching the television, etc.).

SHEET NO: 3

"SELF-CONTROL" techniques for the third week:-

These will be the final techniques you will practice. You must use the techniques you have been practicing for the last two weeks together with these.

I: RESISTING TEMPTATION:

At times when you feel very tempted to smoke, do things that stop you from smoking at the same time. For example, it is difficult to smoke while having a bath, cleaning the floor, sitting in the non-smokers part of the cinema or bus. These are not foolproof. It is still possible to smoke while you are doing these things, but it is more difficult than if you are sitting down doing nothing but thinking of cigarettes. You can help yourself by saving these activities until you feel really tempted to smoke. Then go for a walk, have a bath, etc. This will help you stop smoking.

II: TO AVOID RELAPSES:

1) Tell everyone that you have stopped smoking. By this you will avoid people offering you cigarettes and also you will get support from others.

2) If possible get rid of all the ashtrays (put them out of sight), and lighters, cigarettes, which will remind you of smoking.

Here is a brief outline of this week's "Self-Control" techniques:-

1) When you feel very tempted to smoke, do something that will prevent you from smoking at the same time.

2) Tell everyone that you have given up smoking.

3) Get rid of all the cigarettes, ashtrays and lighters which will remind you of smoking.

SHEET NO: 4

This is our last meeting. Now, most of you have achieved total or partial control of your smoking habit. From this time onwards you have to keep up this control over your smoking. One important thing is to give up (or cut down), smoking and the second equally important task is to keep to this level.

Simply, go on using the "Self-Control" techniques. If, in future you have a bad day and smoke more than you do now, do not be discouraged. It is never too late, just start again with your techniques and always remind yourself that"YOU CAN CONTROL YOUR SMOKING", as you have done in the last month.

INSTRUCTIONS

This is a test of hand steadiness. Please follow these instructions.

1. Take the metal stylus into your right hand (or left if you are left-handed) and hold it in the aperture of the metal plate.

2. Your arm should not be supported. Do not rest your arm against your body or on the table.

3. Hold the stylus as steadily as you can, without touching the sides of the hole.

4. Every time the stylus touches the sides of the hole, you will hear a clicking sound.

5. You will be given a "Ready, Now" signal, indicating the start of the test period. When you hear this signal, you should already be holding the stylus in the hole. Just go on holding it steadily until the experimenter says "Stop".

6. You will have two practice trails, of twenty seconds duration each, before we start the experiment.

If you have any questions, please ask them now.

INSTRUCTIONS

This is an experiment to find out the way you perceive light flashes, with different frequencies.

The room will be darkened throughout the experiment. In order to get your eyes adapted to darkness, you will have to sit in this room for five minutes before we start.

Please follow these instructions:

1. Adjust your stool, so that you can look through the tube, with your right eye, at the light bulb comfortably. Your left eye will be occluded with an eye-patch.
2. You will be given a warning signal of "Ready, Now" before you are presented with a light flash. You will be shown a number of light flashes, each one lasting for one second.
3. All you have to do is to look at each light flash carefully, and then decide whether it was a "FLICKERING" OR A "STEADY" light.
4. So, you will hear a "Ready, Now" signal, and then see a light flash, and then you have to say either "FLICKERING" or "STEADY" depending upon your judgment. There is no right or wrong answer.

You will have two practice trials before we start the proper testing.

If you have any questions please ask them now.

B-iii

INSTRUCTIONS

This is an experiment to determine your peripheral visual field. Below are the procedures we will follow:

1. During the next experiment your left eye will be occluded with an eye patch.
2. Please adjust your stool, so that you can comfortably put your chin in the left cup of the double chin rest. Your head should be in a vertical position. Your arms should be resting on the table, on either side of the instrument.
3. Fixate your right eye at the cross, which is at the centre of the arc. Please, remember to look ONLY at the cross, and no other place on the arc all through the experiment.
4. You will be presented with a light-dot at different points on the arc. Fixating on the cross, you will be able to see some of these dots, and not the others.
5. You will hear a "Ready, Now" signal before you are presented with the light-dot.
6. All you have to do is to say "Yes" when you see it, and to say "NO" when you cannot see it,
7. You will be given some practice before we start the testing.

REMEMBER TO FIXATE AT THE CROSS ALL THE TIME.

If you have any questions, please ask them now.

APPENDIX C

PQAV2

Two factor analysis of variance with repeated measures on one factor.(B).

Factor A= Groups

A 1= Non-Addicted

A 2= Addicted

Factor B= Conditions

B 1=Smoking

B 2= Deprivation

POAV2

PULSE RATE

CELL MEANS

	SMOKING	DEPRIVATION
NON-ADD.	86.00000	69.66667
ADDICTED	82.66667	67.66667

SOURCE	SS	DF	MS	F	
BETW SUBJS	1522.000	11			
A	42.66669	1	42.66669	.2884183	NS
SUBJ W GPS	1479.333	10	147.9333		
WITHN SUBJ	2200.000	12			
B	1472.667	1	1472.667	20.32199	*p<.01
AB	2.666687	1	2.666687	.0367988	NS
B SWG	724.6666	10	72.46666		
A AT B 1	33.33331	1	33.33331	.3024802	NS
A AT B 2	12.00000	1	12.00000	.1088929	NS
SSWCELL	2204.000	20	110.2000		
B AT A 1	800.3333	1	800.3333	11.04416	*p<.01
B AT A 2	675.0000	1	675.0000	9.314629	*p<.025
SSBSWG	724.6666	10	72.46666		

F MAX SWG 1.857695

F MAX BSWG 2.032078

FINISH

POAV2

SYSTOLIC BLOOD PRESSURE

CELL MEANS

	SMOKING	DEPRIVATION
NON-ADD.	125.6667	117.0000
ADDICTED	111.0000	103.6667

SOURCE	SS	DF	MS	F
BETW SUBJS	2725.332	11		
A	1175.999	1	1175.999	7.590352*p<.025
SUBJ W GPS	1549.333	10	154.9333	
WITHN SUBJ	639.9999	12		
B	383.9987	1	383.9987	15.15785*p<.01
AB	2.668091	1	2.668091	.1053195 NS
B SWG	253.3331	10	25.33331	
A AT B 1	645.3334	1	645.3334	7.159764*p<.025
A AT B 2	533.3334	1	533.3334	5.917160*p<.025
SSWCELL	1802.667	20	90.13333	
B AT A 1	225.3333	1	225.3333	8.894741*p<.025
B AT A 2	161.3334	1	161.3334	6.368426*p<.05
SSBSWG	253.3331	10	25.33331	

F MAX SWG 12.99999

F MAX BSWG 8.500033

FINISH

P0AV2

DIASTOLIC BLOOD PRESSURE

CELL MEANS

	SMOKING	DEPRIVATION
NON-ADD.	63.66667	78.66667
ADDICTED	64.33333	67.33333

SOURCE	SS	DF	MS	F	
BETW SUBJS	2918.000	11			
A	170.6667	1	170.6667	.6212085	NS
SUBJ W GPS	2747.333	10	274.7333		
WITHN SUBJ	3196.000	12			
B	486.0001	1	486.0001	1.948677	NS
AB	215.9999	1	215.9999	.8660784	NS
B SWG	2494.000	10	249.4000		
A AT B 1	1.333313	1	1.333313	.0050877	NS
A AT B 2	385.3333	1	385.3333	1.470364	NS
SSWCELL	5241.333	20	262.0667		
B AT A 1	675.0000	1	675.0000	2.706496	NS
B AT A 2	27.00000	1	27.00000	.1082598	NS
SSBSWG	2494.000	10	249.4000		

F MAX SWG 20.86207

F MAX BSWG 130.2633

FINISH

PGAV2

HAND STEADINESS

CELL MEANS

	SMOKING	DEPRIVATION
NON-ADD.	38.33333	21.08333
ADDICTED	21.83333	32.58333

SOURCE	SS	DF	MS	F	
BETW SUBJS	8538.458	11			
A	37.50008	1	37.50008	.0441128	NS
SUBJ W GPS	8500.958	10	850.0958		
WITHN SUBJ	7145.500	12			
B	63.37525	1	63.37525	.1073043	NS
AB	1176.000	1	1176.000	1.991153	NS
B SWG	5906.125	10	590.6125		
A AT B 1	816.7499	1	816.7499	1.133817	NS
A AT B 2	396.7501	1	396.7501	.5507709	NS
SSWCELL	14407.08	20	720.3542		
B AT A 1	892.6875	1	892.6875	1.511461	NS
B AT A 2	346.6874	1	346.6874	.5869963	NS
SSBSWG	5906.125	10	590.6125		

F MAX SWG 1.467548

F MAX BSWG 5.836287

FINISH

POAV2

CRITICAL F.F. THRESHOLD

CELL MEANS

	SMOKING	DEPRIVATION
NON-ADD.	7.738333	7.575000
ADDICTED	7.000000	7.201667

SOURCE	SS	DF	MS	F	
A	1.853704	1	1.853704	8.402802*p	.025
B	.0022025	1	.0022025	.0099839	NS
AB	.1998343	1	.1998343	.9058450	NS
WITHN CELL	4.412109	20	.2206054		
TOTAL	6.467850	23			
A AT B 1	1.635401	1	1.635401	7.413238*p	.025
A AT B 2	.4181333	1	.4181333	1.895390	NS
SSWCELL	4.412109	20	.2206054		
B AT A 1	.0800257	1	.0800257	1.204836	NS
B AT A 2	.1220088	1	.1220088	1.836917	NS
SSBSWG	.6642041	10	.0664204		
F MAX	1.840556				

FINISH

PVF- TOTAL Peripheral Visual Field

CELL MEANS

	SMOKING	DEPRIVATION
NON-ADD.	2853.400	2904.417
ADDICTED	2685.333	2776.350

SOURCE	SS	DF	MS	F	
BETW SUBJS	5581590	11			
A	131544.6	1	131544.6	.2413643	NS
SUBJ W GPS	5450045	10	545004.5		
WITHN SUBJ	1103630	12			
B	30262.12	1	30262.12	.2825671	NS
AB	2396.625	1	2396.625	.0223781	NS
B SWG	1070971	10	107097.1		
A AT B 1	84738.37	1	84738.37	.2598931	NS
A AT B 2	49203.00	1	49203.00	.1509059	NS
SS CELL	6521017	20	326050.8		
B AT A 1	7806.125	1	7806.125	.0728883	NS
B AT A 2	24853.00	1	24853.00	.2320604	NS
SSBSWG	1070971	10	107097.1		

F MAX SWG 1.152524

F MAX BSWG 25.59816

FINISH

PGAV2

P.V.F : 0° Meridian

CELL MEANS

	SMOKING	DEPRIVATION
NON-ADD	57.08333	58.23000
ADDICTED	56.15000	55.73333

SOURCE	SS	DF	MS	F	
BETW SUBJS	403.5127	11			
A	17.64713	1	17.64713	.4573386	NS
SUBJ W GPS	385.8656	10	38.58656		
WITHN SUBJ	93.55173	12			
B	.7988586	1	.7988586	.0896719	NS
AB	3.665985	1	3.665985	.4115067	NS
B SWG	89.08688	10	8.908688		
A AT B 1	2.613495	1	2.613495	.1100529	NS
A AT B 2	18.69958	1	18.69958	.7874297	NS
SSWCELL	474.9525	20	23.74762		
B AT A 1	3.944672	1	3.944672	.4427893	NS
B AT A 2	.5204163	1	.5204163	.0584167	NS
SSBSWG	89.08688	10	8.908688		

F MAX SWG 1.042320

F MAX BSWG 2.065590

FINISH

PVF- 180° Meridian

CELL MEANS

	SMOKING	DEPRIVATION
Non-ADD	85.31333	84.27333
ADDICTED	81.14833	80.83667

SOURCE	SS	DF	MS	F	
BETW SUBJS	1984.936	11			
A	86.67389	1	86.67389	.4565956	NS
SUBJ W GPS	1898.264	10	189.8264		
WITHN SUBJ	170.4413	12			
B	2.736389	1	2.736389	.1639489	NS
AB	.7999878	1	.7999878	.0479307	NS
B SWG	166.9050	10	16.69050		
A AT B 1	52.04187	1	52.04187	.5039963	NS
A AT B 2	35.43164	1	35.43164	.3431356	NS
SSWCELL	2065.169	20	103.2584		
B AT A 1	3.244995	1	3.244995	.1944217	NS
B AT A 2	.2910156	1	.2910156	.0174360	NS
SSBSWG	166.9050	10	16.69050		

F MAX SWG 1.146471

F MAX BSWG 5.815425

FINISH

PUAV2

PVF- 90° Meridian

CELL MEANS

	SMOKING	DEPRIVATION
NON-ADD	40.73167	45.42000
ADDICTED	42.39833	44.79500

SOURCE	SS	DF	MS	F	
BETW SUBJS	1354.937	11			
A	1.629730	1	1.629730	.0120426	NS
SUBJ W GPS	1353.307	10	135.3307		
WITHN SUBJ	261.8252	12			
B	75.29770	1	75.29770	4.214769	NS
AB	7.875458	1	7.875458	.4408267	NS
B SWG	178.6520	10	17.86520		
A AT B 1	8.333283	1	8.333283	.1087925	NS
A AT B 2	1.171387	1	1.171387	.0152927	NS
SSWCELL	1531.959	20	76.59797		
B AT A 1	65.94119	1	65.94119	3.691041	NS
B AT A 2	17.23221	1	17.23221	.9645683	NS
SSBSWG	178.6520	10	17.86520		

F MAX SWG 1.200742

F MAX BSWG 11.12645

FINISH

PUAV2

PVF- 270° Meridian

CELL MEANS

	SMOKING	DEPRIVATION
NON-ADD.	69.79333	66.25167
ADDICTED	62.10667	63.23000

SOURCE	SS	DF	MS	F	
BETW SUBJS	963.2657	11			
A	172.0032	1	172.0032	2.173782	NS
SUBJ W GPS	791.2624	10	79.12624		
WITHN SUBJ	166.5781	12			
B	8.774109	1	8.774109	.7010163	NS
AB	32.64130	1	32.64130	2.607909	NS
B SWG	125.1627	10	12.51627		
A AT B 1	177.2541	1	177.2541	3.868381	NS
A AT B 2	27.39087	1	27.39087	.5977765	NS
SSWCELL	916.4251	20	45.82126		
B AT A 1	37.63013	1	37.63013	3.006497	NS
B AT A 2	3.785767	1	3.785767	.3024677	NS
SSBSWG	125.1627	10	12.51627		

F MAX SWG 2.236431

F MAX BSWG 1.550050

FINISH

INDEX FINGER TEMPERATURE (^{o}C)

CELL MEANS

	SMOKING	DEPRIVATION
NON-A	29.58333	31.08333
ADDICTED	32.00000	33.66667

SOURCE	SS	DF	MS	F	
BETW SUBJS	311.3333	11			
A	37.50026	1	37.50026	1.369457	NS
SUBJ W GPS	273.8330	10	27.38330		
WITHN SUBJ	141.5000	12			
B	15.04192	1	15.04192	1.189868	NS
AB	.0413971	1	.0413971	.0032747	NS
B SWG	126.4167	10	12.64167		
A AT B 1	17.52077	1	17.52077	.8754919	NS
A AT B 2	20.02083	1	20.02083	1.000417	NS
SSWCELL	400.2497	20	20.01248		
B AT A 1	6.750000	1	6.750000	.5339485	NS
B AT A 2	8.333252	1	8.333252	.6591893	NS
SSBSWG	126.4167	10	12.64167		

F_MAX SWG 9.566006

F MAX BSWG 17.27661

FINISH

PLEASE FOLLOW THESE INSTRUCTIONS VERY CAREFULLY

You will receive three king size filter cigarettes (placed in a tin) and three plastic bags marked as:

Morning

Afternoon

Evening

The day before the experiment:

Please smoke, one of the given cigarettes as your first cigarette of the day. Keep the butt in the plastic bag marked as " Morning ". Smoke the second cigarette as your first cigarette after lunch, and keep the butt in the plastic bag marked as " Afternoon ". Smoke the third cigarette as your first cigarette after dinner and keep the butt in the plastic bag marked as " Evening ".

Place the plastic bags in the tine and bring them with you to the experiment. Continue to smoke as usual throughout the day.

App. D.ii

DEPRIVATION CONDITION

INSTRUCTIONS :

Do not smoke any cigarettes after 12.00 p.m (midnight), the night before the experiment, till you come hear at 2.00 p.m, in the afternoon. Your abstention is very important and you will not be able to participate in the experiment unless you have deprived yourself of cigarettes. A test will be made to check your abstention. So, please remember not to smoke after midnight. If you feel this will be too difficult for you please say so now..

It would also be very helpful if you can avoid taking alcohol and drugs.

Many thanks for your co-operation.

SMOKING CONDITION

INSTRUCTIONS :

You can smoke as many cigarettes as you normally would prior to the testing session. However, it would be very helpful if you can avoid taking alcohol and drugs.

Many thanks for your co-operation.

App. D.iii

PROCEDURES FOR THIS EXPERIMENT

REST : When you see the rest light, please try to relax (without going to sleep). During one of the rest periods you will be informed that you are free to smoke. So, in that period you can have a cigarette whenever you wish to. Please smoke the cigarettes provided by the experimenter.

TRIAL : You will be given an attention test when the trial light is on. Instructions for this test will be given beforehand, and you will also have a practive trial.

Please try NOT TO MOVE AROUND during the experiment, since this will cause interference with the recording.

If you have any questions please ask them now.

The experiment will take approximately 2.oo hours.

App. D.iv

Please put a circle around all the cases of three odd digits occurring consecutively. For example: 5 7 3

3	7	3	8
6	5	6	4
9	8	2	1
5	9	6	7
1	5	5	6
8	2	8	2
4	4	1	7
8	3	2	5
2	7	6	1
7	5	9	8
4	8	1	4
5	5	4	9
6	7	3	1
8	8	5	
2	1	9	
7	4	6	
6	8	4	
5	4	7	
4	1	5	
7	6	8	
2	3	7	
9	4	9	
1	7	2	
4	8	4	
3	4	3	
5	9	7	
9	4	8	
6	5	5	
4	2	7	

COMPUTER PROGRAMS

E.i: "New.1" : For reading the magnetic tape and storing the data on mini disk.

Starts the tape-recorder and takes one sample per second. Converts the data into digital values. Computes the means and standard deviations for 10. sec. periods, whenever an event signal is detected from channel 4 or whenever 30 ten second samples are taken (i.e; 5 minutes), the tape is stopped automatically and the data is stored in a record on the data file in the mini disk. Whenever a signal for SCL is detected, the tape stops and the videoscreen shows "SCL = ? ", when the new offset value is given the tape starts again.

E.ii: " Getdat. Nur": For printing the data read and stored by "New.1 ".

Provides access to the data recorded in the "Data.1" file. By inserting the number of any record, data can be retrieved.

E.iii: "Getdat.Analysis": For calculating the means and standard deviations of the records (i.e; 5 minutes or less if period ends before) created by " New.1".

E.iv: "Mean": To create one record for each subject storing the means and standard deviations for each experimental period successively.

E.v: ""Getdat.Mean": Provides access to records created by the "Mean" program and prints means and standard deviations.

E.vi:"New.Graph": Uses the means of experimental periods for each subject and plots histograms of heart rate, respiration rate and amplitude and SCL and lability (see App.F).

E.vii: " Calibrate": Provides calibration histograms to check the accuracy of the range employed for the " New.Graph" program (see. App. F).

```
>>ENTER "NEW.1"

***END***
>>LIST

   10 SEMODE
   20 LET E6=0
   30 INPUT"RECORD NUMBER =",R2
   40 INPUT"SAMPLE TIME  ??  ",B
   50 INPUT"SKIN CONDUCTANCE LEVEL  ??  ",Z
   60 INPUT"NUMBER OF SAMPLES  ??  ",K
   70 INTEGER Y(3),E(3)
   80 SHORT M1(K),A1(K),R1(K)
   90 SHORT M3(K),L1(K)
  100 Y1=0 : Y2=0 : Y3=0 : E1=0 : E2=0 : E3=0
  110 L=0 : Y=0
  120 S=0 : T=0 : C=0 : S1=0 : S3=0
  125 M4=0
  130 M=0 : N=0 : U1=0 : U3=0 : U=0
  140 Z1=0
  150 L=L+1 : Y=Y+1
  160 IF L>K THEN 800
  170 OUT%0018%,132
  190 C=C+1
  200 X=INP(%0018%)
  210 IF X<128 THEN 200
  220 F1=F
  230 F=INP(%001C%) : F=INP(%001C%)
  240 IF F<128 THEN 260
  250 F=F-256
  251 F2=F1-F
  252 IF F2<0 THEN F2=F2*-1
  253 IF F2<5 THEN 400
  260 IF F<100 THEN 290
  270 GOTO 400
  290 IF F>65 AND F<80 THEN 306
  300 GOTO 340
  306 PRINT
  307 A6=A6+1
  308 IF A6<100 THEN 307
  310 E6=E6+1
  320 PRINT"EVENT=",E6,"F=",F
  330 GOTO 800
  340 IF F<25 AND F>15 THEN 365
  350 GOTO 400
  365 OUT(%0018%),0
  367 PRINT
  368 A6=A6+1
  369 IF A6<100 THEN 368
  370 INPUT"SKIN CONDUCTANCE  ?? ",Z
  375 PRINT"F=",F
  380 OUT%0018%,132
  400 A=INP(%0019%)
  410 IF A<128 THEN 425
  420 A=A-256
  425 M2=A0
  430 A0=(127.0-A)/3.04+45.0
  432 M3=M2-A0
  434 IF M3<0 THEN M3=M3*-1
  435 IF M3>15 THEN 460
```

```
450 S1=S1+D1/M
460 Y3=Y2
470 Y2=Y1
480 Y1=Y0
490 Y0=INP(%001A%)
500 IF Y0<128 THEN 520
510 Y0=Y0-256
520 IF Y0<=0 THEN 540
530 GOTO 560
540 M=M+Y0
550 GOTO 570
560 N=N+Y0
570 IF Y2<Y3 AND Y2<Y1 THEN 590
580 GOTO 600
590 T=T+1
600 E3=E2
610 E2=E1
620 E1=E
630 E=INP(%001B%)
640 IF E<128 THEN 660
650 E=E-256
660 E=(22.0-E)*4/188.0+Z
670 D3=E-S3
680 S3=S3+D3/C
690 IF E2>E3+0.03 AND E2>E1+0.03 THEN 710
700 GOTO 720
710 U=U+1
720 IF C>=B THEN 740
730 GOTO 190
740 M1(L)=S1
750 A1(L)=(N-M)/B
760 R1(L)=T
770 M3(L)=S3
780 L1(L)=U
790 GOTO 120
800 OUT%0018%,0
810 REM TO DISCFILE DATA
820 Z3=K*20
830 OPEN\1,Z3\"DATA.1"
840  FOR K1=1 TO K
850  PUT\1,R2,Z1\M1(K1),A1(K1),R1(K1),M3(K1),L1(K1)
860  Z1=Z1+20
870  NEXT K1
880 CLOSE\1\
882 PRINT
883 A6=A6+1
884 IF A6<100 THEN 883
885 T3=(L-1)*B/60
886 PRINT"REC.TIME= ",T3
890 R2=R2+1
900 PRINT"RECORD #  ",R2
910 MAT M1=0 : MAT A1=0 : MAT R1=0 : MAT M3=0 : MAT L1=0
915 A6=0
920 GOTO 100
930 END
```

```
>>ENTER "GETDAT.NUR"

***END***
>>LIST

   10 REM TO GET DATA FROM DISC AND ANALYZE
   30 INPUT"NUMBER OF SAMPLES  ??  ",K
   35 INPUT"RECORD NUMBER  ??  ",R2
   40 Z3=K*20
   50 OPEN\1,Z3\"DATA.1"
   60 SHORT M1(K),A1(K),R1(K),M3(K),L1(K)
   70  FOR K1=1 TO K
   80  GET\1,R2,Z2\M1(K1),A1(K1),R1(K1),M3(K1),L1(K1)
   90  Z2=Z2+20
  100  NEXT K1
  110 CLOSE\1\
  120 SET 0,60 : SET 1,10
  130  FOR K1=1 TO K
  140  PRINT K1,M1(K1),A1(K1),R1(K1),M3(K1),L1(K1)
  150  NEXT K1
  155 MAT M1=0 : MAT A1=0 : MAT R1=0 : MAT M3=0 : MAT L1=0
  156 Z2=0
  160 GOTO 35
  170 END

>>
```

```
  RUN
NUMBER OF SAMPLES  ??  30
RECORD NUMBER  ??  1
1          86.2829    21.8       3          7.88936    3
2          86.3158    15.7       2          7.99574    0
3          86.1842    18.1       2          7.92979    0
4          86.25      16.1       3          7.9766     0
5          86.3487    12.7       3          8.19574    2
6          86.3158    17.9       2          8.18085    0
7          86.25      16.6       3          8.14894    1
8          86.1513    17.9       4          8.24255    0
9          86.1513    15.5       3          8.31702    0
10         86.25      15.3       3          8.49362    0
11         86.25      14.5       2          8.4383     1
12         86.2829    21.3       3          8.39787    0
13         86.1513    13.6       3          8.57447    0
14         84.5724    28.9       2          8.98511    0
15         75.5592    18.8       3          8.72979    1
16         71.0526    16.9       3          8.55319    0
17         68.2566    16.5       3          8.5234     0
18         65.477     15.3       3          8.42128    0
19         68.4868    16.4       3          8.42766    1
20         69.3421    17.1       3          8.41277    0
21         68.3224    17.4       4          8.44468    1
22         70.2303    14.4       3          8.49787    0
23         70.6908    17.4       3          8.6234     1
24         67.2368    16.2       4          8.54255    0
25         70.4276    16.9       4          8.61277    0
26         69.7368    16.4       3          8.69149    1
27         84.0132    31.3       2          9.2234     0
28         86.25      24.1       1          9.1617     2
29         86.2829    13.7       3          9.15957    1
30         86.1513    16.4       3          9.07021    1
RECORD NUMBER  ??
```

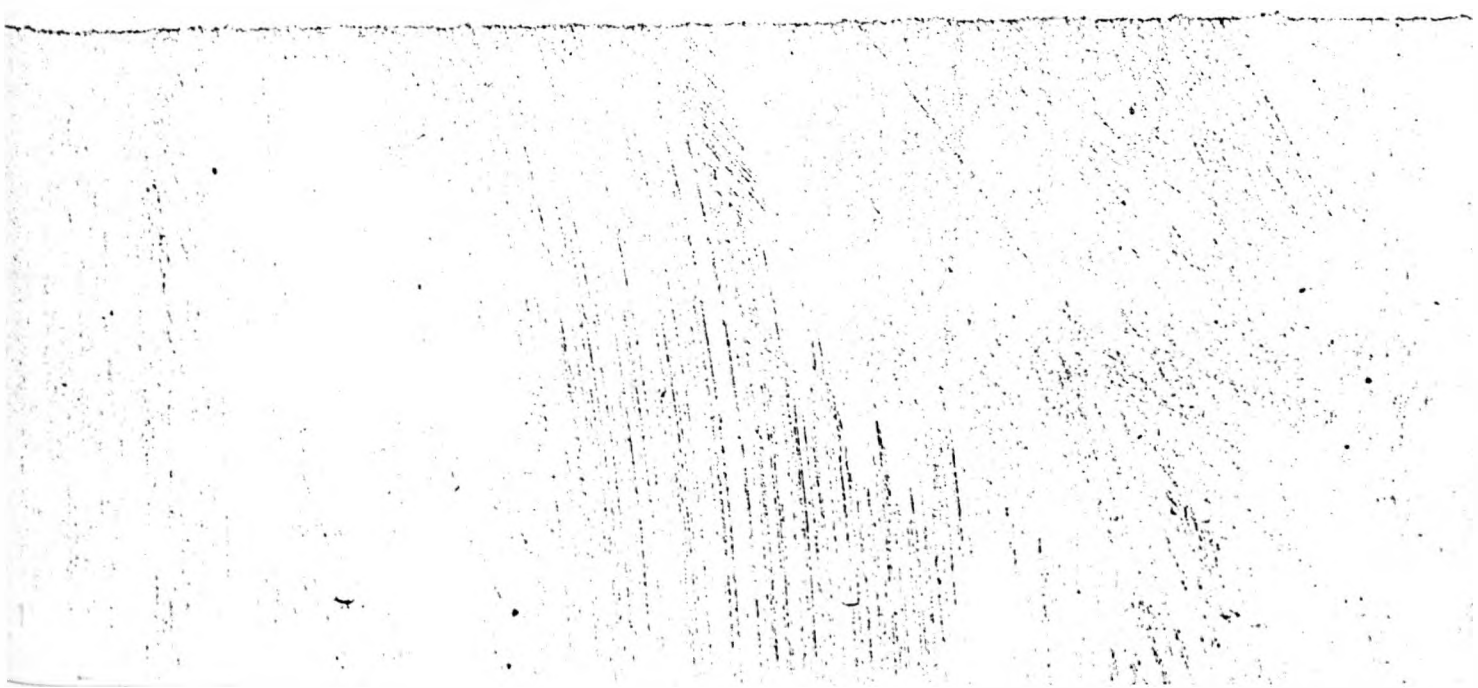
```
***MP***                                                    APR-80.111
>>LIST

   10 REM TO GET DATA FROM DISC AND ANALYZE
   11 SFMODE
   15 INPUT"NUMBER OF SAMPLES  ??  ",K
   16 Z3=K*20
   20 SFMODE
   30 INPUT"RECORD NUMBER  ??  ",R2
   40 K=30
   50 Z3=K*20
   60 OPEN\1,Z3\"DATA.1"
   70 SHORT M1(K),A1(K),R1(K),M3(K),L1(K)
   80 SHORT M4,D4,M5,D5,M6
   85 SET 0,60 : SET 1,10
   86 S4=0 : S5=0 : S6=0 : S7=0 : S8=0
   87 U4=0 : U5=0 : U6=0 : U7=0 : U8=0
   90 SHORT D6,M7,D7,M8,D8
  100  FOR K1=1 TO K
  110  GET\1,R2,Z2\M1(K1),A1(K1),R1(K1),M3(K1),L1(K1)
  120  Z2=Z2+20
  130  NEXT K1
  140 CLOSE\1\
  145 INPUT"NUMBER OF ANALYS SAMPLE  ??",K
  150 SET 0,60 : SET 1,10
  160 S4=0 : S5=0 : S6=0 : S7=0 : S8=0
  170 U4=0 : U5=0 : U6=0 : U7=0 : U8=0
  180  FOR K1=1 TO K
  190  D4=M1(K1)-S4
  200  S4=S4+D4/K1
  210  U4=U4+D4*(M1(K1)-S4)
  220  D5=A1(K1)-S5
  230  S5=S5+D5/K1
  240  U5=U5+D5*(A1(K1)-S5)
  250  D6=R1(K1)-S6
  260  S6=S6+D6/K1
  270  U6=U6+D6*(R1(K1)-S6)
  280  D7=M3(K1)-S7
  290  S7=S7+D7/K1
  300  U7=U7+D7*(M3(K1)-S7)
  310  D8=L1(K1)-S8
  320  S8=S8+D8/K1
  330  U8=U8+D8*(L1(K1)-S8)
  340  NEXT K1
  350 M4=S4
  360 V4=U4/(K-1)
  370 D4=SQR(V4)
  380 M5=S5
  390 V5=U5/(K-1)
  400 D5=SQR(V5)
  410 M6=S6
  420 V6=U6/(K-1)
  430 D6=SQR(V6)
  440 M7=S7
  450 V7=U7/(K-1)
  460 D7=SQR(V7)
  470 M8=S8
  480 V8=U8/(K-1)
  490 D8=SQR(V8)
  500 PRINT"M.H.R=",M4,"S.D",D4
  510 PRINT"M.R.AMP=",M5,"S.D=",D5
  520 PRINT"M.R.RATE=",M6,"S.D=",D6
  530 PRINT"M.S.COND=",M7,"S.D=",D7
  540 PRINT"M.LAB=",M8,"S.D=",D8
  550 MAT M1=0 : MAT A1=0 : MAT R1=0
  560 MAT M3=0 : MAT L1=0
  570 Z2=0
```

```
  DSK "@"

DISK #A
>>RUN
NUMBER OF SAMPLES  ??  30
RECORD NUMBER  ??  36
NUMBER OF ANALYS SAMPLE  ??30
M.H.R=     61.6292    S.D        2.0565
M.R.AMP=   17.5233    S.D=       3.62622
M.R.RATE=  2.33333    S.D=       0.546672
M.S.COND=  2.54397    S.D=       0.0596879
M.LAB=     0.0666667  S.D=       0.253708
RECORD NUMBER  ??  61
NUMBER OF ANALYS SAMPLE  ??30
M.H.R=     65.5748    S.D        4.4287
M.R.AMP=   15.01      S.D=       4.87049
M.R.RATE=  2.66667    S.D=       0.660895
M.S.COND=  1.40121    S.D=       0.0854861
M.LAB=     0.166667   S.D=       0.379049
RECORD NUMBER  ??  57
NUMBER OF ANALYS SAMPLE  ??30
M.H.R=     78.5858    S.D        3.21968
M.R.AMP=   10.68      S.D=       1.13119
M.R.RATE=  2.76667    S.D=       0.430183
M.S.COND=  6.14532    S.D=       0.165674
M.LAB=     0.366667   S.D=       0.614948
RECORD NUMBER  ??
***30 ESCAPE***

>>
```



```
>>ENTER "MEAN"

***END***
>>LIST

   10 SFMODE
   20 SHORT B1(25)
   40 INPUT"SUBJECT NUMBER  ",A2
   50 PRINT"RECORD NUMBERS ?"
   60 INPUT B1(A)
   70 IF B1(A)<0 THEN 90
   80 A=A+1 : GOTO 60
   90 REM TO GET DATA FROM DISC AND ANALYZE
   99 R=A
  100 A1=A-1
  102 PRINT"NUMBER OF RECORDS=",R
  105 SHORT M4(R),M5(R),M6(R),M7(R),M8(R),S9(R)
  110 SHORT D4(R),D5(R),D6(R),D7(R),D8(R)
  130  FOR A=0 TO A1
  140  K=30
  150  Z3=K*20
  160  Z2=0
  180  SHORT M1(K),A1(K),R1(K),M3(K),L1(K)
  230  R2=B1(A)
  240  OPEN\1,Z3\"A:DATA.1"
  250   FOR K1=1 TO K
  260   GET\1,R2,Z2\M1(K1),A1(K1),R1(K1),M3(K1),L1(K1)
  270   Z2=Z2+20
  280   NEXT K1
  290  CLOSE\1\
  310  S4=0 : S5=0 : S6=0 : S7=0 : S8=0
  320  U4=0 : U5=0 : U6=0 : U7=0 : U8=0
  330   FOR K1=1 TO K
  340   D4=M1(K1)-S4
  350   IF M1(K1)<10 THEN 505
  360   S4=S4+D4/K1
  370   U4=U4+D4*(M1(K1)-S4)
  380   D5=A1(K1)-S5
  390   S5=S5+D5/K1
  400   U5=U5+D5*(A1(K1)-S5)
  410   D6=R1(K1)*6-S6
  420   S6=S6+D6/K1
  430   U6=U6+D6*(R1(K1)*6-S6)
  440   D7=M3(K1)-S7
  450   S7=S7+D7/K1
  460   U7=U7+D7*(M3(K1)-S7)
  470   D8=L1(K1)*6-S8
  480   S8=S8+D8/K1
  490   U8=U8+D8*(L1(K1)*6-S8)
  500   NEXT K1
```

```
501  GOTO 510
505  IF K1<3 THEN K1=3
510  K=K1-1
520  M4(A)=S4
530  V4=U4/(K-1)
540  D4(A)=SQR(V4)
550  M5(A)=S5
560  V5=U5/(K-1)
570  D5(A)=SQR(V5)
580  M6(A)=S6
590  V6=U6/(K-1)
600  D6(A)=SQR(V6)
610  M7(A)=S7
620  V7=U7/(K-1)
630  D7(A)=SQR(V7)
640  M8(A)=S8
650  V8=U8/(K-1)
660  D8(A)=SQR(V8)
661  S9(A)=K
662  NEXT A
670 Z5=R*44
680 OPEN\1,Z5\"B:MEAN.D"
681 PRINT A1
682 Z4=0
683  FOR A=0 TO A1
690  PUT\1,A2,Z4\M4(A),M5(A),M6(A),M7(A),M8(A),D4(A),D5(A),D6(A),D7(A
692  Z4=Z4+44
695  NEXT A
700 CLOSE\1
720 END
```

```
  ENTER "GETDATMEAN"

***END***
>>LIST

    2 INPUT"SUBJECT NUMBER ??",A2
    5 INPUT"NUMBER OF RECORDS ??",A
   10 SHORT M4(A),M5(A),M6(A),M7(A),M8(A),S8(A)
   20 SHORT D4(A),D5(A),D6(A),D7(A),D8(A)
   24 Z2=0
   25 A1=A*44
   30 OPEN\1,A1\"B:MEAN.D"
   35 Z2=0
   40  FOR K1=0 TO A-1
   50  GET\1,A2,Z2\M4(K1),M5(K1),M6(K1),M7(K1),M8(K1),D4(K1),D5(K1),D6(K
   60  Z2=Z2+44
   70  NEXT K1
   75 CLOSE\1\
   83 PRINT USING"####.###","REC.NO","NUMB.SAMP","M.H.R","M.R.AMP","M.H
   90  FOR K1=0 TO A-1
  100  PRINT USING"####.###",K1+1,S8(K1),M4(K1),M5(K1),M6(K1),M7(K1),M8(
  102  PRINT USING"###.###","STAND.DEV","          ",D4(K1),D5(K1),D6(K1)
  110  NEXT K1
  120 END

>>
```

SCR

```
ENTER "NEW.GRAPH"

***END***
>>LIST

  2 INPUT"SUBJECT NUMBER ??",A2
  5 A=21
 10 SHORT M4(A),M5(A),M6(A),M7(A),M8(A),S8(A)
 20 SHORT D4(A),D5(A),D6(A),D7(A),D8(A)
 25 A1=A*44
 30 OPEN\1,A1\"B:MEAN.D"
 35 Z2=0
 40  FOR K1=0 TO A-1
 50  GET\1,A2,Z2\M4(K1),M5(K1),M6(K1),M7(K1),M8(K1),D4(K1),D5(K1),D6(
 60  Z2=Z2+44
 70  NEXT K1
 75 CLOSE\1\
 80  FOR V9=0 TO 200
 83  V8=V9
 90  NEXT V9
100 INPUT"TYPE 1 FOR GRAPH , 2 FOR PRINT ???",V8
110 IF V8=2 THEN 600
120 REM D/A CONVERSION
130  FOR K1=0 TO A-1
131  IF M4(K1)<10 THEN 284
140  M4(K1)=((80-M4(K1))*1.5)+3
150  IF M4(K1)<0 THEN M4(K1)=M4(K1)+256
160  IF M4(K1)>255 THEN M4(K1)=255
170  M7(K1)=((8-M7(K1))*7.5)+6
180  IF M7(K1)<0 THEN M7(K1)=M7(K1)+256
190  IF M7(K1)>255 THEN M7(K1)=255
200  M8(K1)=((6-M8(K1))*10)+4
210  IF M8(K1)<0 THEN M8(K1)=M8(K1)+256
220  IF M8(K1)>255 THEN M8(K1)=255
230  M6(K1)=((18-M6(K1))*5)+2
240  IF M6(K1)<0 THEN M6(K1)=M6(K1)+256
250  IF M6(K1)>255 THEN M6(K1)=255
260  M5(K1)=((25-M5(K1))*4)+10
270  IF M5(K1)<0 THEN M5(K1)=M5(K1)+256
280  IF M5(K1)>255 THEN M5(K1)=255
283  GOTO 290
284  M4(K1)=0 : M7(K1)=0 : M8(K1)=0 : M6(K1)=0 : M5(K1)=0
290  NEXT K1
300 REM TO DRAW THE GRAPH
310  FOR X1=0 TO 10
320  X2=INP(%0018%)
330  IF X2<128 THEN 320
340  OUT%0019%,65 : OUT%001A%,70 : OUT%001B%,63
350  OUT%001C%,65 : OUT%001D%,70
360  NEXT X1
```

APP-E.vi Cont.

```
370  FOR K1=0 TO A-1
380  X2=INP(%0018%)
390  IF X2<128 THEN 380
400  OUT%0019%,65 : OUT%001A%,70 : OUT%001B%,63
410  OUT%001C%,65 : OUT%001D%,70
420   FOR X3=0 TO 9
430   X4=X3
440   NEXT X3
450  X2=INP(%0018%)
460  IF X2<128 THEN 450
461  IF M4(K1)=0 THEN 511
470  OUT%0019%,M4(K1)
480  OUT%001A%,M7(K1)
490  OUT%001B%,M8(K1)
500  OUT%001C%,M6(K1)
509  OUT%001D%,M5(K1)
510  GOTO 520
511  OUT%0019%,65
512  OUT%001A%,70
513  OUT%001B%,63
514  OUT%001C%,65
515  OUT%001D%,70
520  NEXT K1
530  FOR X1=0 TO 10
540  X2=INP(%0018%)
550  IF X2<128 THEN 540
560  OUT%0019%,65 : OUT%001A%,70 : OUT%001B%,63
570  OUT%001C%,65 : OUT%001D%,70
580  NEXT X1
590 GOTO 650
600 PRINT USING"###.###","REC.NO","NUM.SAMP","M.H.R","M.R.AMP","M.R.F
610  FOR K1=0 TO A-1
620  PRINT USING"####.###",K1+1,S8(K1),M4(K1),M5(K1),M6(K1),M7(K1),M8
630  PRINT USING"####.###","STAND.DEV","      ",D4(K1),D5(K1),D6(K1),D
640  NEXT K1
650 GOTO 2
660 END
```

```
>>ENTER "CALIBRATE"

***END***
>>LIST

   10 SFMODE
   40 INPUT"SUBJECT NUMBER  ",A2
   50 INPUT"NUMBER OF RECORDS",R
   99 A1=R-1
  105 SHORT M4(R),M5(R),M6(R),M7(R),M8(R),S9(R)
  110 SHORT D4(R),D5(R),D6(R),D7(R),D8(R)
  130  FOR A=0 TO A1
  140  INPUT M4(A),M5(A),M6(A),M7(A),M8(A)
  150  INPUT D4(A),D5(A),D6(A),D7(A),D8(A),S9(A)
  160  NEXT A
  670 Z5=R*44
  680 OPEN\1,Z5\"B:MEAN.D"
  681 PRINT A1
  682 Z4=0
  683  FOR A=0 TO A1
  690  PUT\1,A2,Z4\M4(A),M5(A),M6(A),M7(A),M8(A),D4(A),D5(A),D6(A),D7(A
  692  Z4=Z4+44
  695  NEXT A
  700 CLOSE\1\
  720 END

>>
```

App. F

HISTOGRAMS: Showing the heart rate, respiration rate and amplitude, skin conductance level and lability for the whole experimental session (normal and deprivation conditions for smokers).

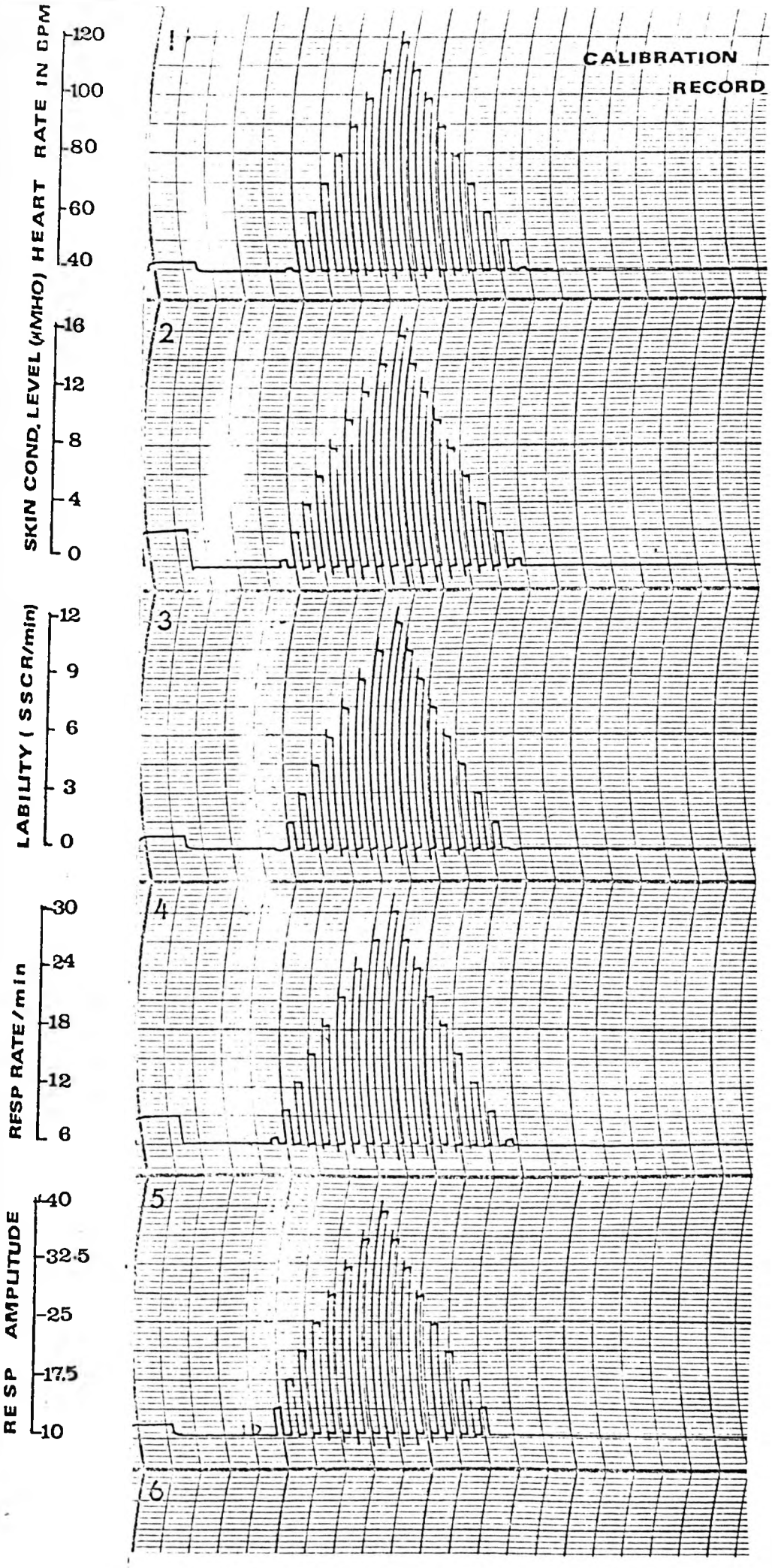
Histograms are based on mean values for consecutive records (5 minutes or less).

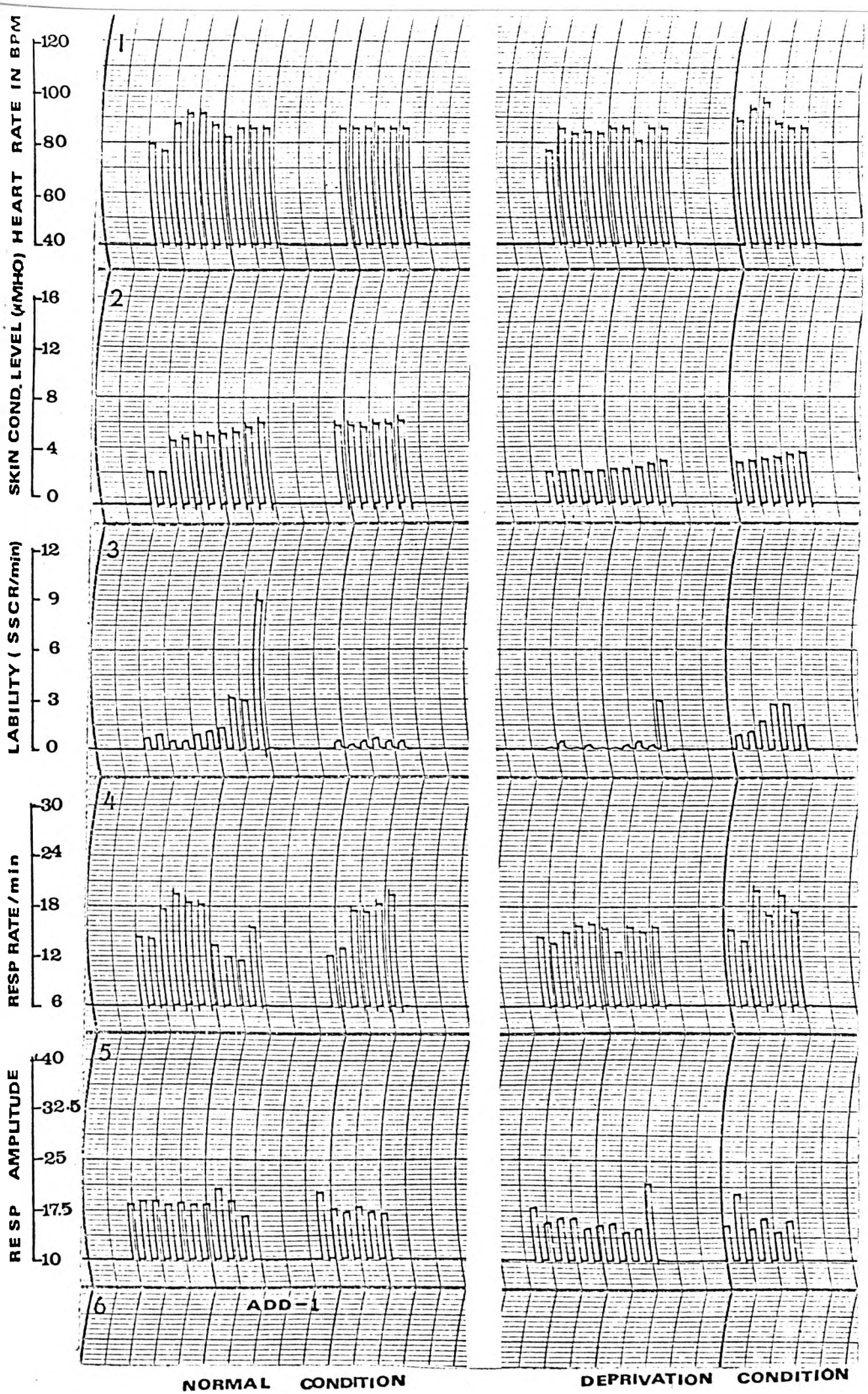
The bars of the histograms correspond to the following experimental periods:

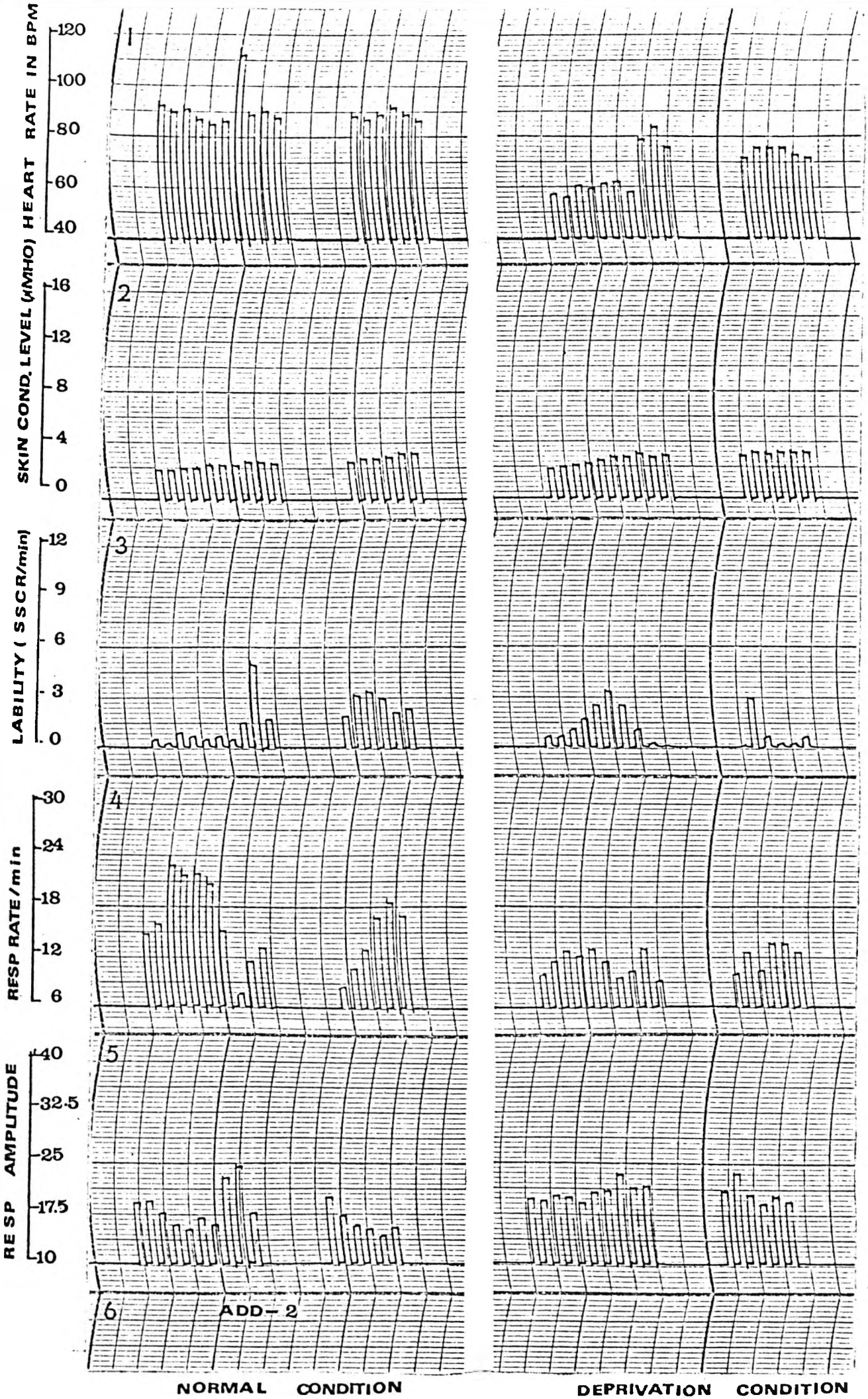
EXPERIMENTAL PERIOD	BAR NUMBERS
Base-line I	1 & 2
Vigilance test I	3,4,5,6
Base-line II	7
Smoking I*	8 & 9
Latency**	10,11,12,13,14
Smoking II*	15 & 16
Vigilance test II	17,18,19,20

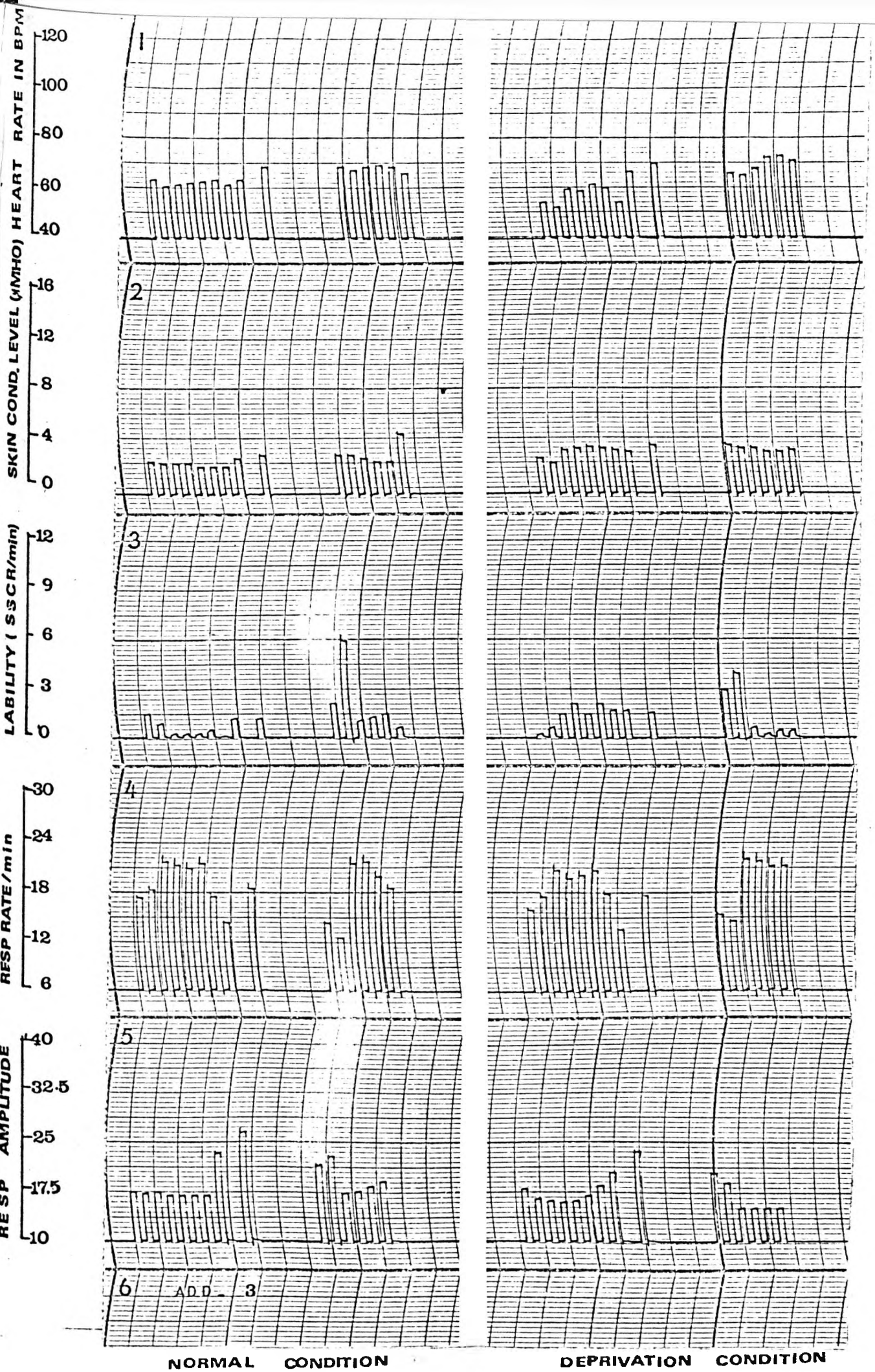
* If the smoking periods had less than two records then a straight line can be seen for bars 9 and/or 16.

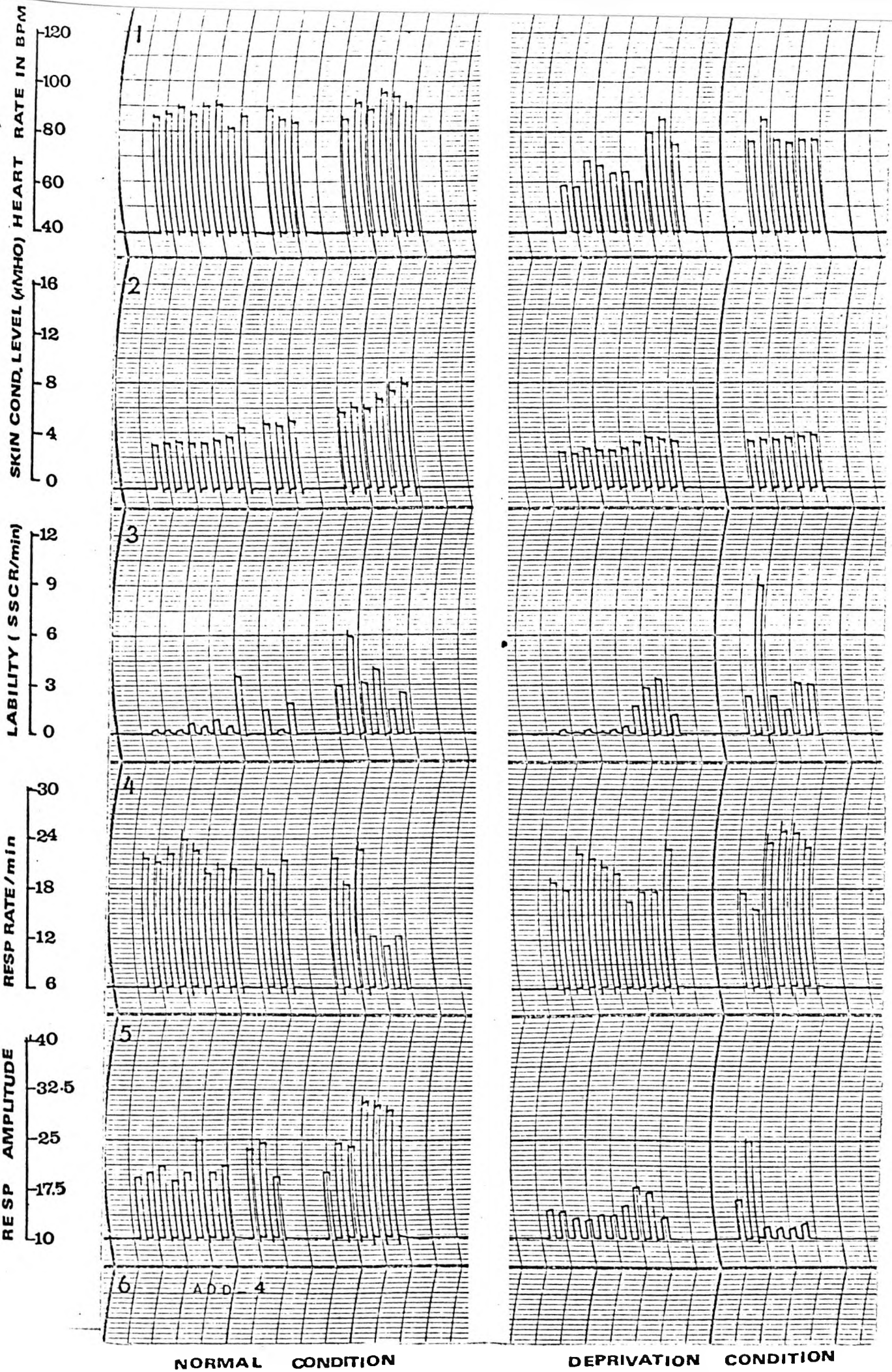
** If the latency to the second cigarette was less than 30 minutes (i.e; 5 five minutes records), then a straight line can be seen in the middle of the histogram. This indicates that the subject had less than 5 records for the latency period, (i.e; the longer the straight line, the shorter was the latency).

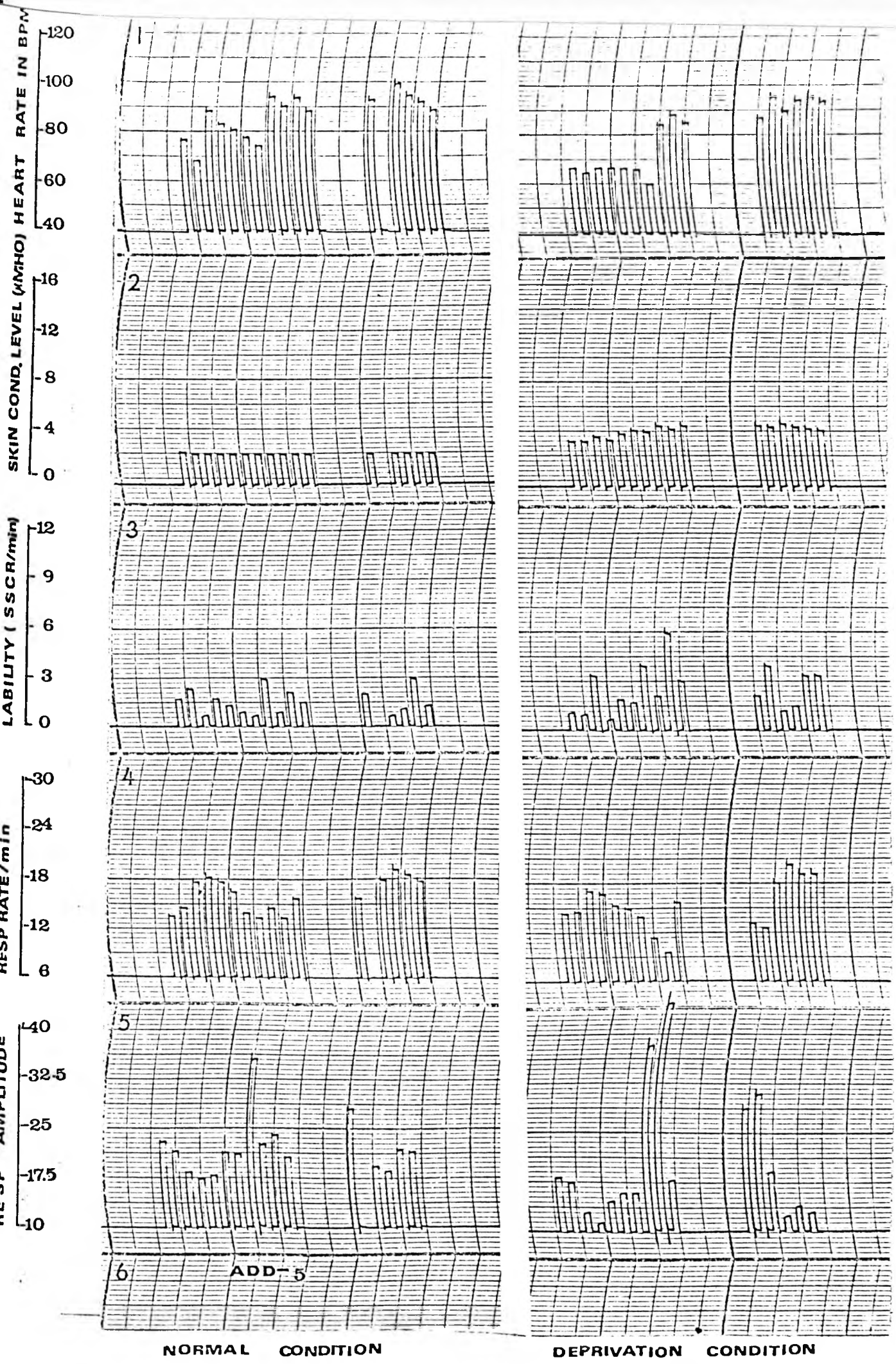

CALIBRATION
RECORD
1
2
3
4
5
6
HEART RATE IN BPM
120
100
80
60
40
SKIN COND. LEVEL (MHO)
16
12
8
4
0
LABILITY (SSCR/min)
12
9
6
3
0
RESP RATE/min
30
24
18
12
6
RESP AMPLITUDE
40
32.5
25
17.5
10

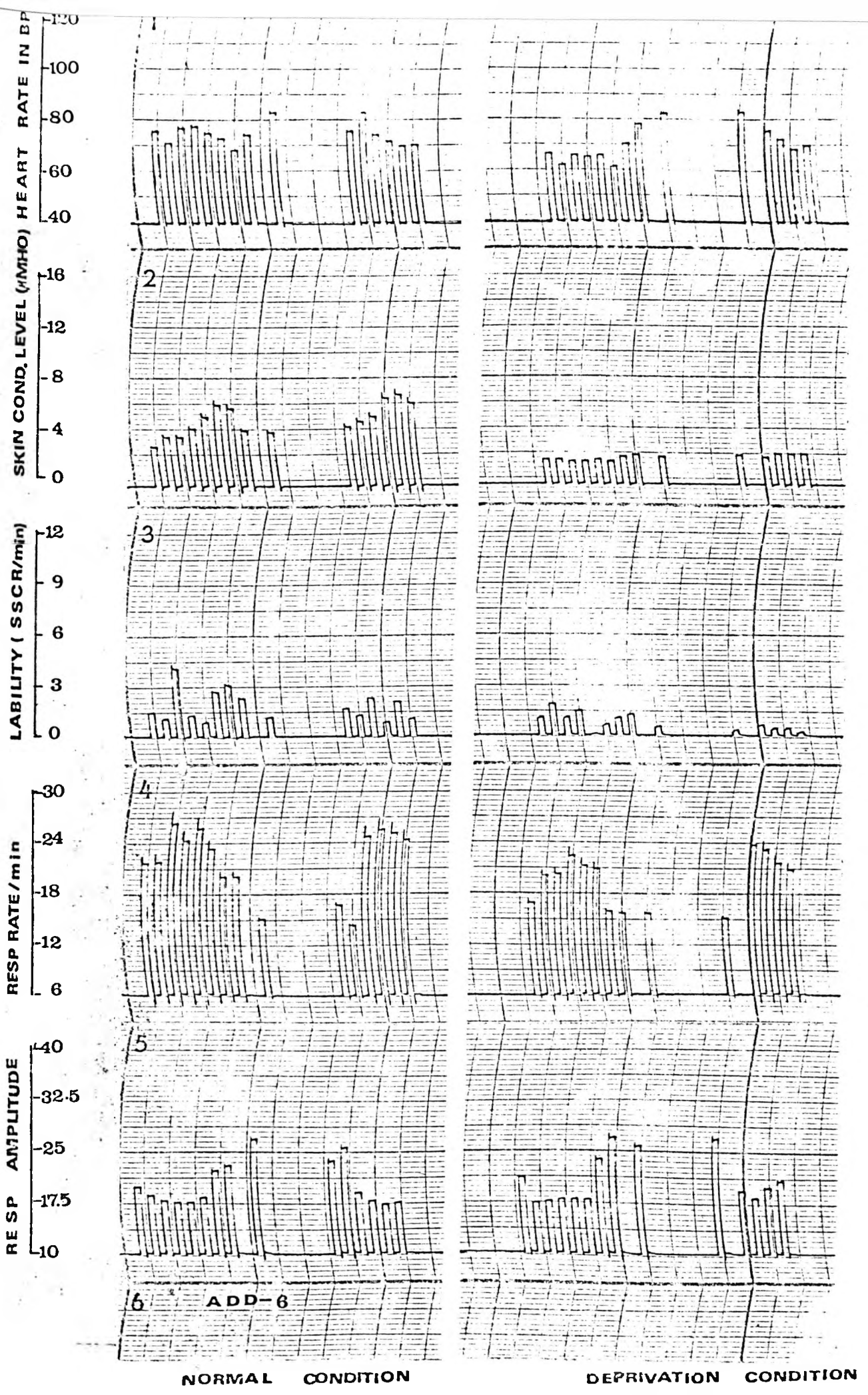

1
HEART RATE IN BPM
120
100
80
60
40
2
SKIN COND. LEVEL (μMHO)
16
12
8
4
0
3
LABILITY (SSCR/min)
12
9
6
3
0
4
RESP RATE/min
30
24
18
12
6
5
RESP AMPLITUDE
40
32.5
25
17.5
10
6
ADD-1
NORMAL CONDITION
DEPRIVATION CONDITION

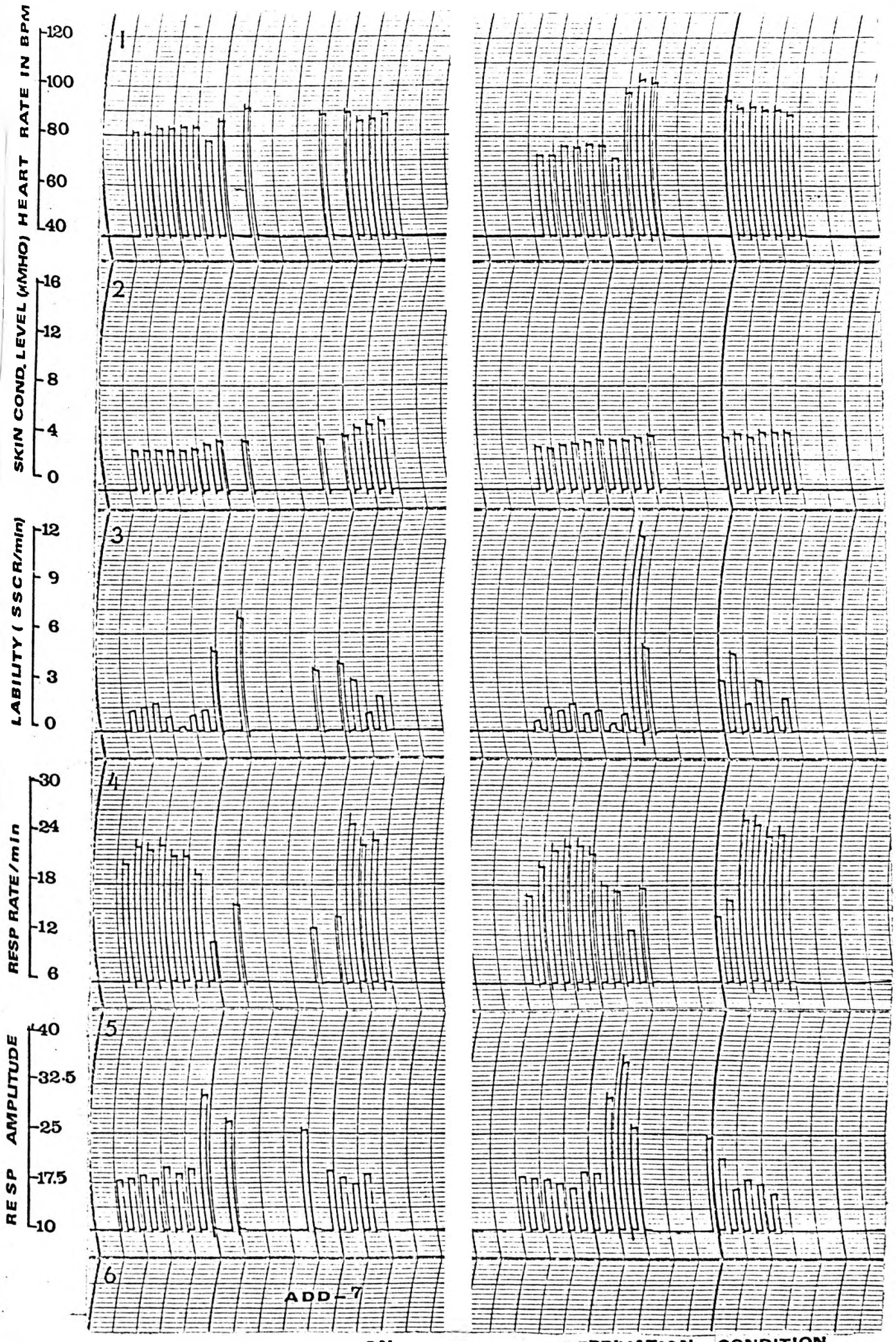

1
HEART RATE IN BPM
120
100
80
60
40
2
SKIN COND. LEVEL (μMHO)
16
12
8
4
0
3
LABILITY (SSCR/min)
12
9
6
3
0
4
RESP RATE/min
30
24
18
12
6
5
RESP AMPLITUDE
40
32.5
25
17.5
10
6
ADD– 2
NORMAL CONDITION
DEPRIVATION CONDITION

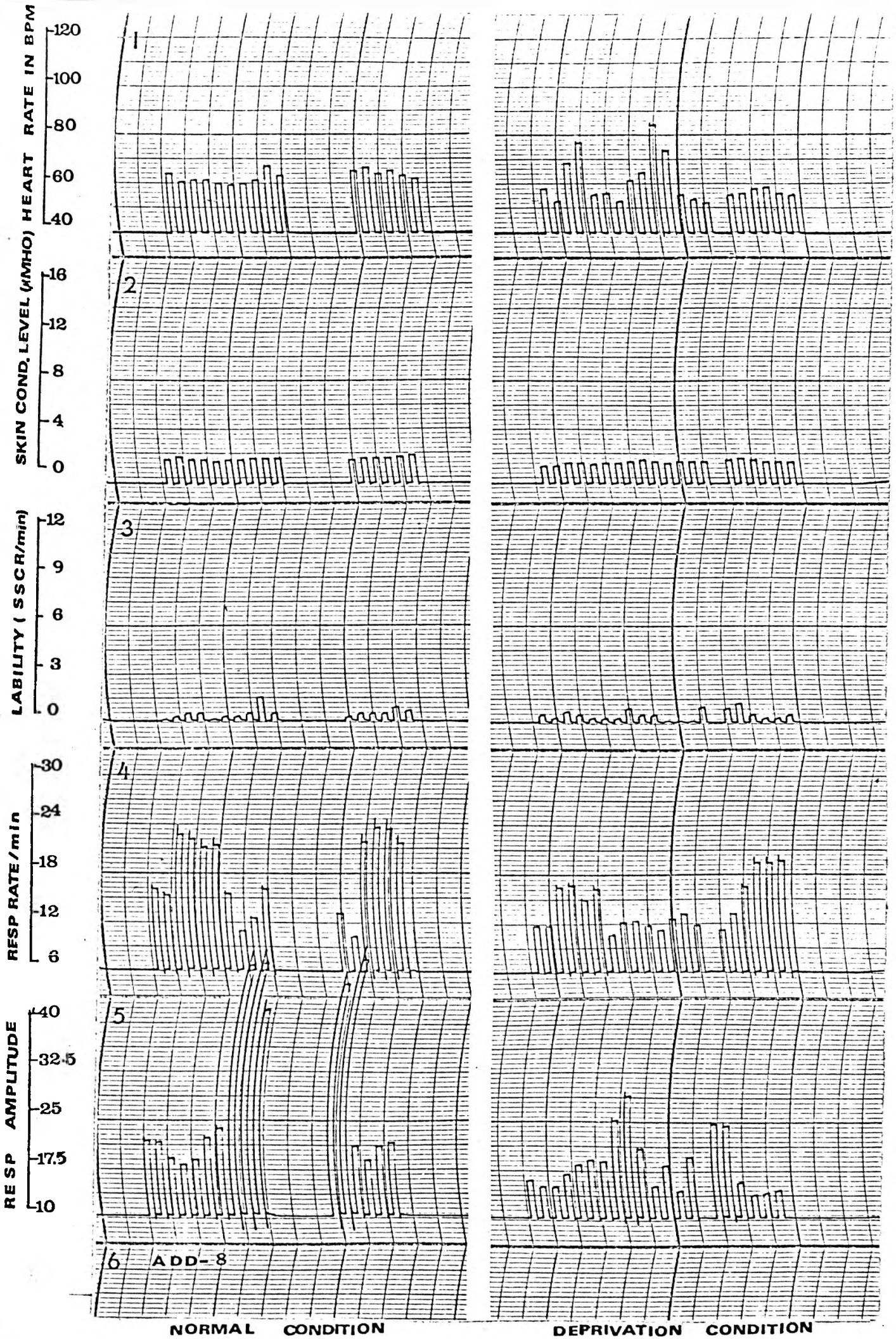

1
HEART RATE IN BPM
120
100
80
60
40
2
SKIN COND. LEVEL (MHO)
16
12
8
4
0
3
LABILITY (SSCR/min)
12
9
6
3
0
4
RESP RATE/min
30
24
18
12
6
5
RESP AMPLITUDE
40
32.5
25
17.5
10
6
ADD - 3
NORMAL CONDITION
DEPRIVATION CONDITION

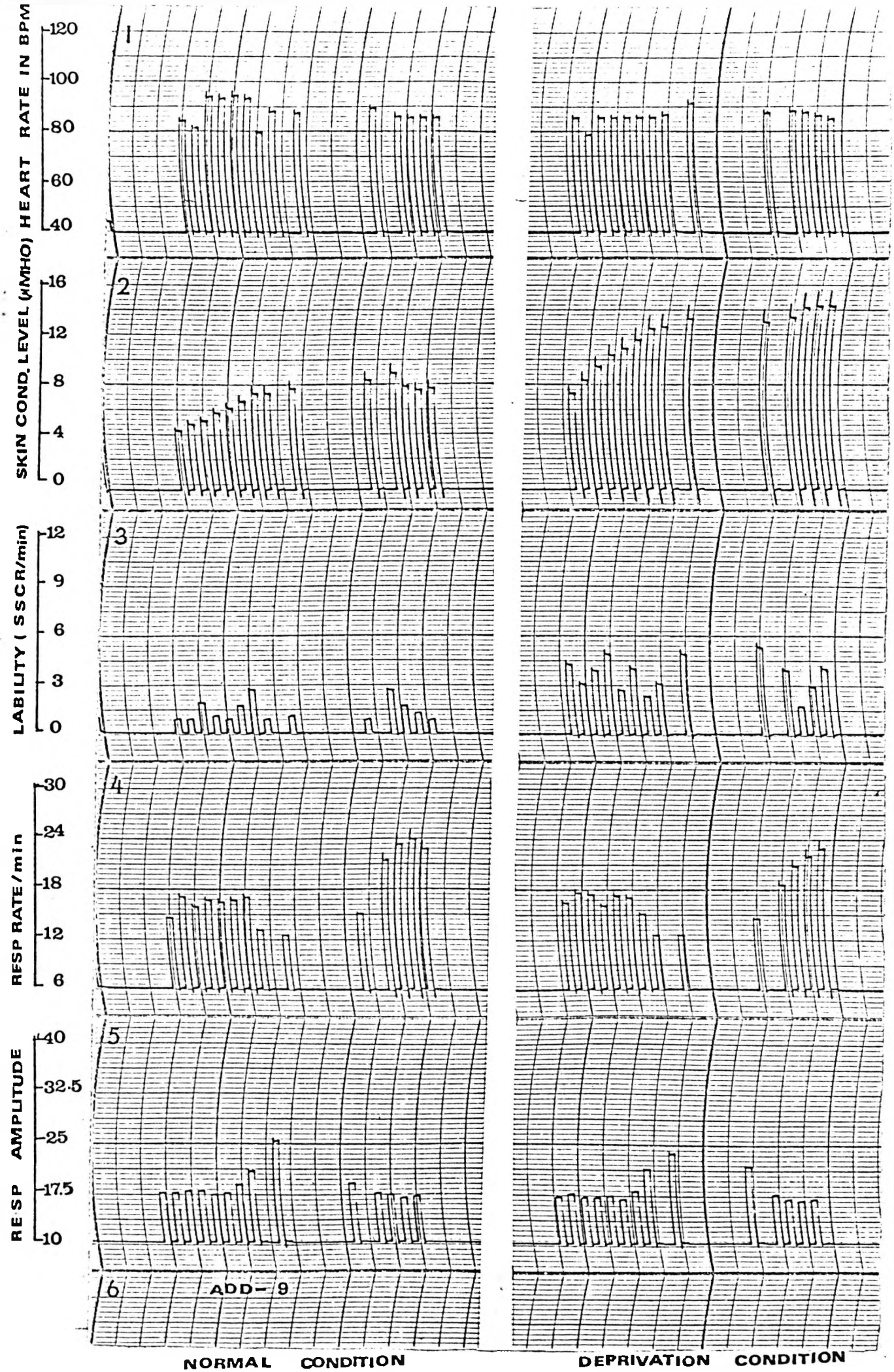

HEART RATE IN BPM
120
100
80
60
40
1
SKIN COND. LEVEL (μMHO)
16
12
8
4
0
2
LABILITY (SSCR/min)
12
9
6
3
0
3
RESP RATE/min
30
24
18
12
6
4
RESP AMPLITUDE
40
32.5
25
17.5
10
5
6
ADD - 4
NORMAL CONDITION
DEPRIVATION CONDITION

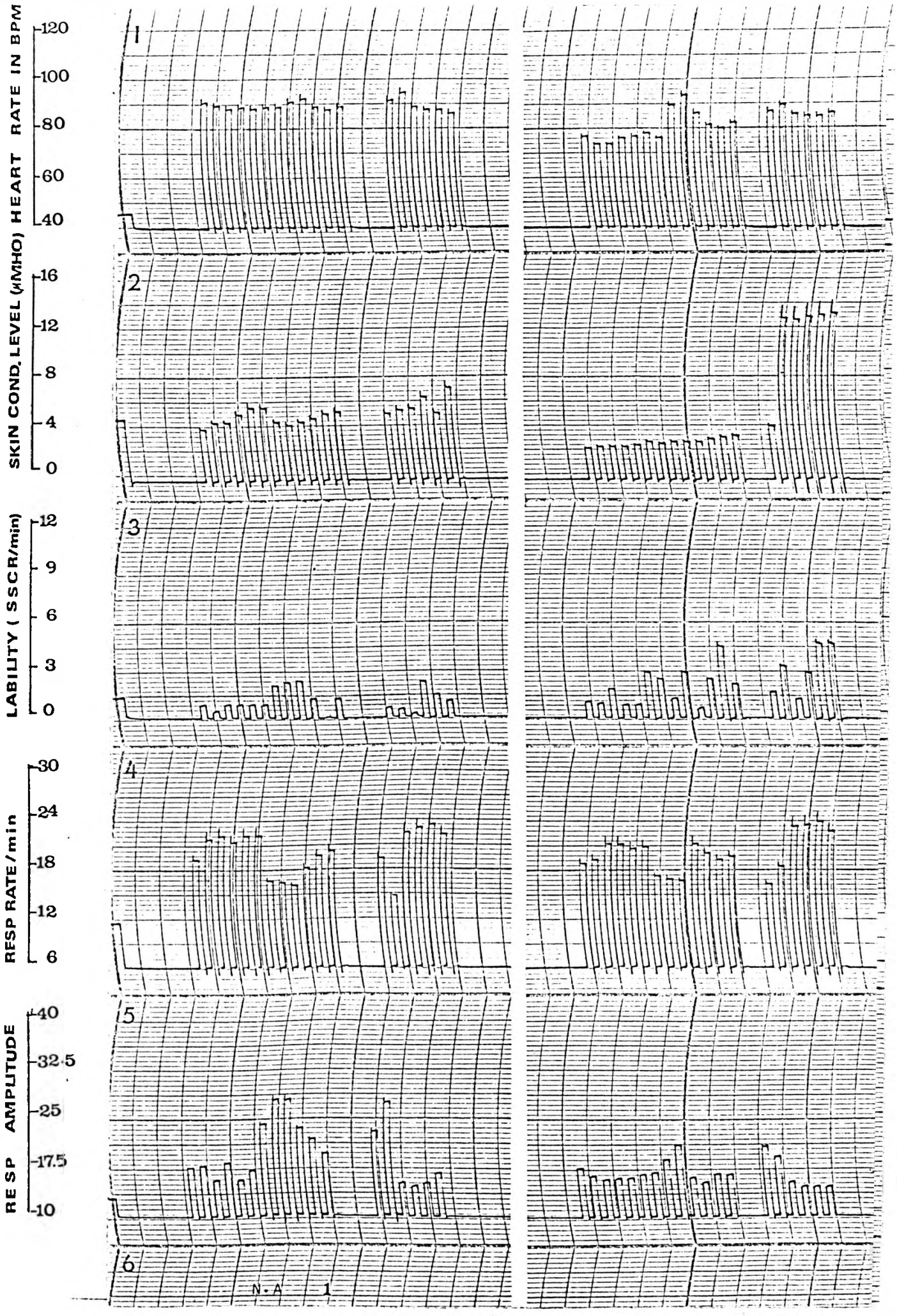

HEART RATE IN BPM
120
100
80
60
40
SKIN COND. LEVEL (μMHO)
16
12
8
4
0
LABILITY (SSCR/min)
12
9
6
3
0
RESP RATE/min
30
24
18
12
6
RESP AMPLITUDE
40
32.5
25
17.5
10
1
2
3
4
5
6
ADD-5
NORMAL CONDITION
DEPRIVATION CONDITION

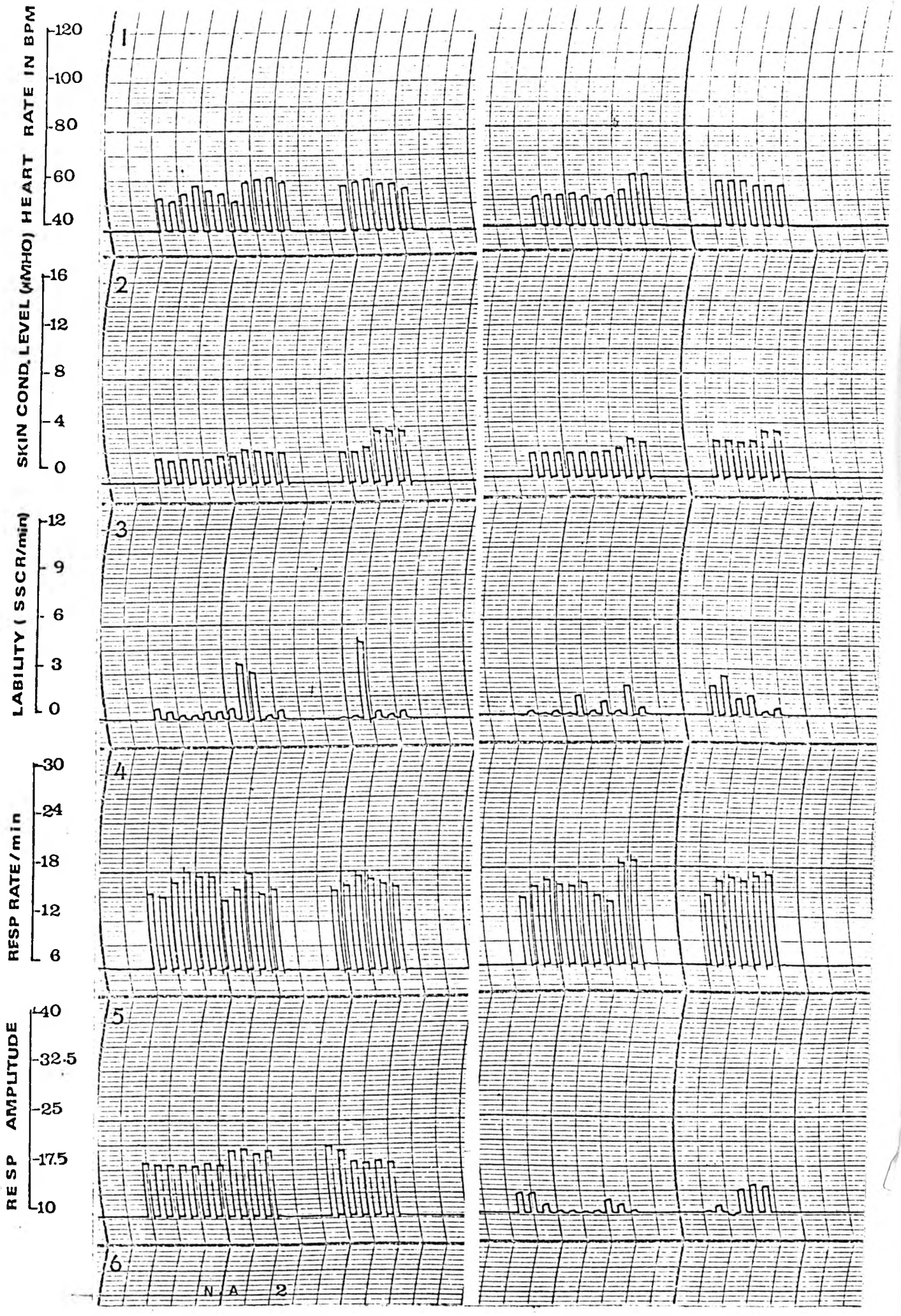

HEART RATE IN BP
120
100
80
60
40
1
SKIN COND. LEVEL (μMHO)
16
12
8
4
0
2
LABILITY (SSCR/min)
12
9
6
3
0
3
RESP RATE/min
30
24
18
12
6
4
RESP AMPLITUDE
40
32.5
25
17.5
10
5
6
ADD-6
NORMAL CONDITION
DEPRIVATION CONDITION

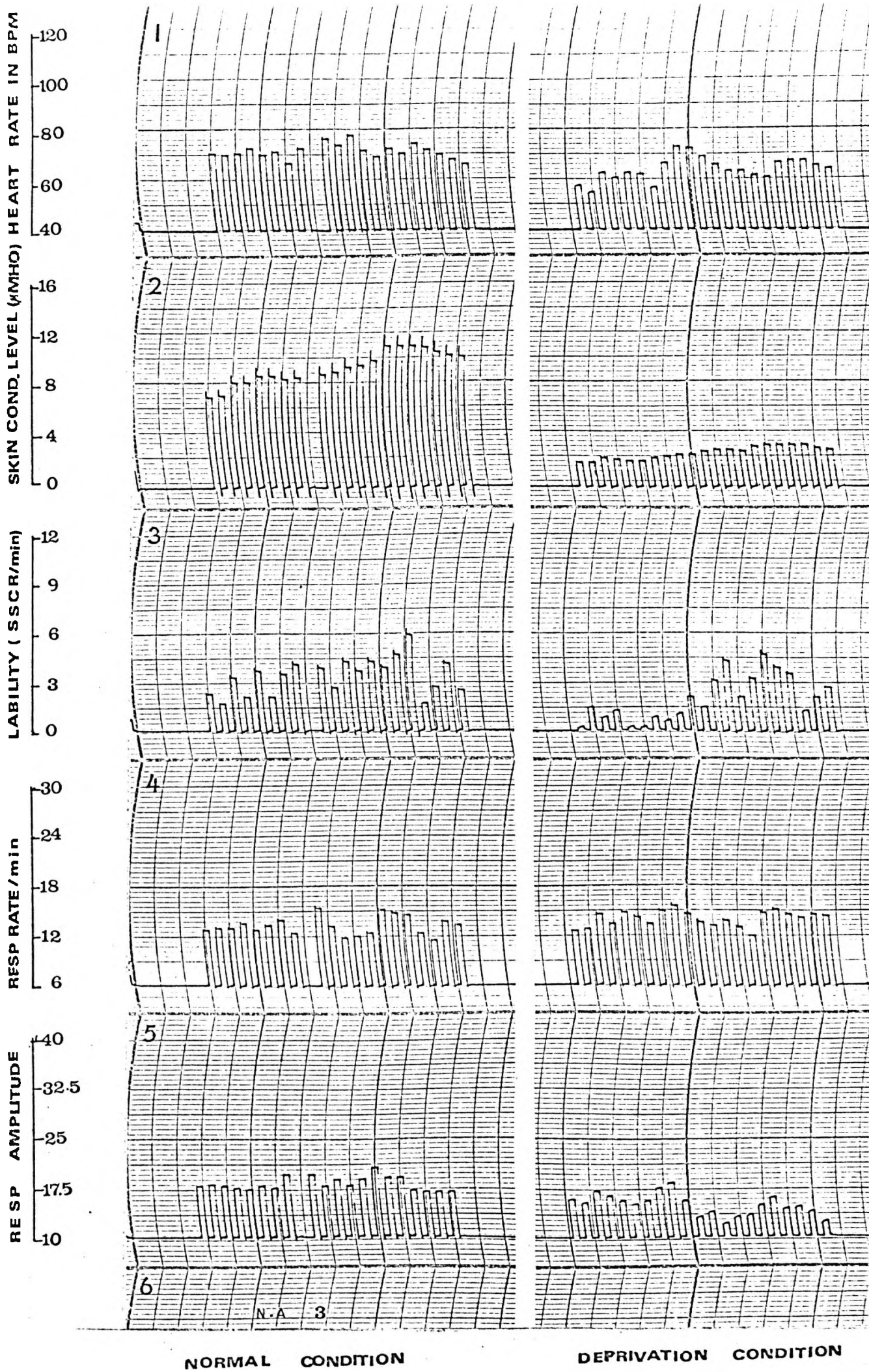

1
2
3
4
5
6
HEART RATE IN BPM
120
100
80
60
40
SKIN COND. LEVEL (μMHO)
16
12
8
4
0
LABILITY (SSCR/min)
12
9
6
3
0
RESP RATE/min
30
24
18
12
6
RESP AMPLITUDE
40
32.5
25
17.5
10
ADD-7
NORMAL CONDITION
DEPRIVATION CONDITION

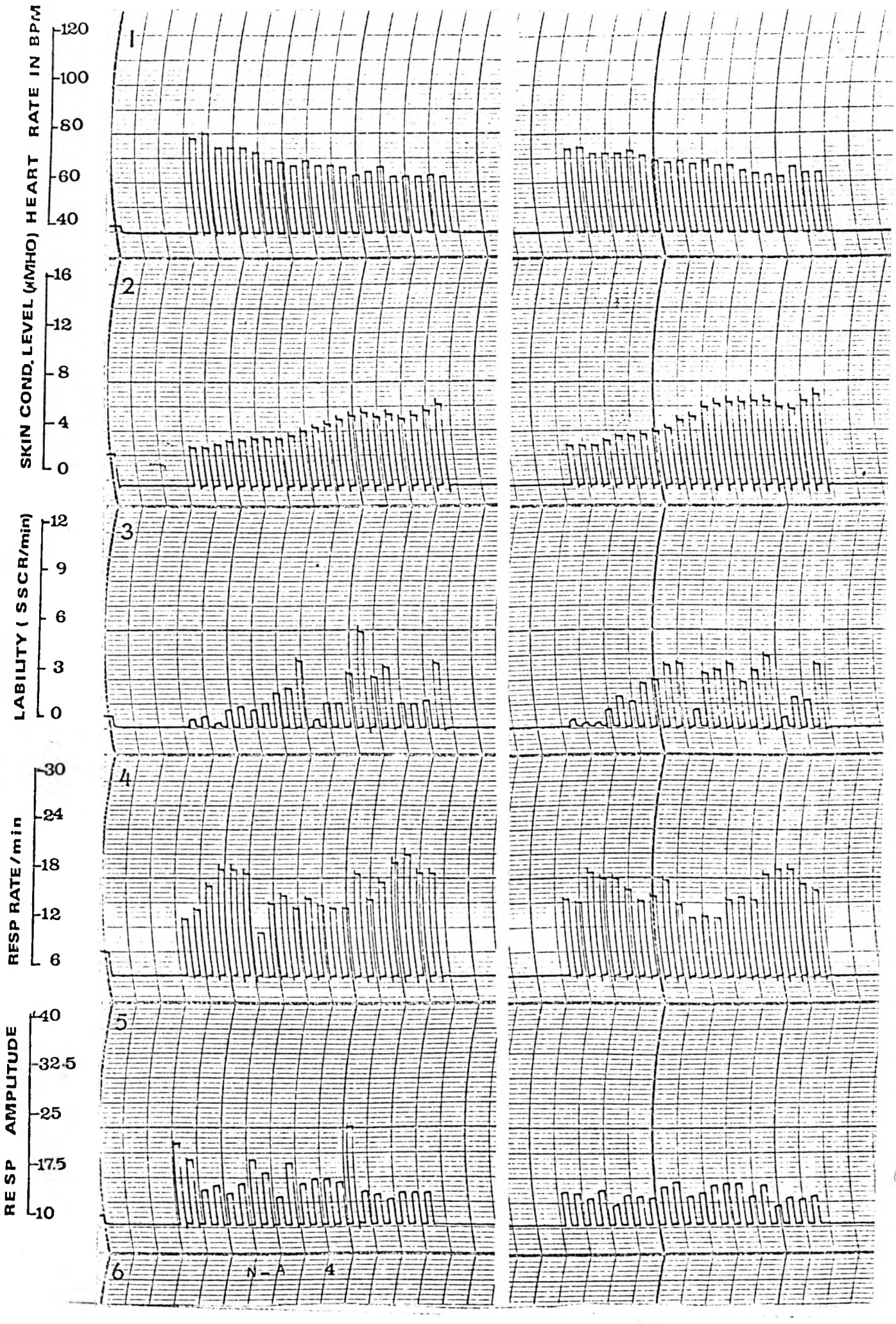

HEART RATE IN BPM
120
100
80
60
40
1
SKIN COND. LEVEL (μMHO)
16
12
8
4
0
2
LABILITY (SSCR/min)
12
9
6
3
0
3
RESP RATE/min
30
24
18
12
6
4
RESP AMPLITUDE
40
32.5
25
17.5
10
5
6
ADD-8
NORMAL CONDITION
DEPRIVATION CONDITION

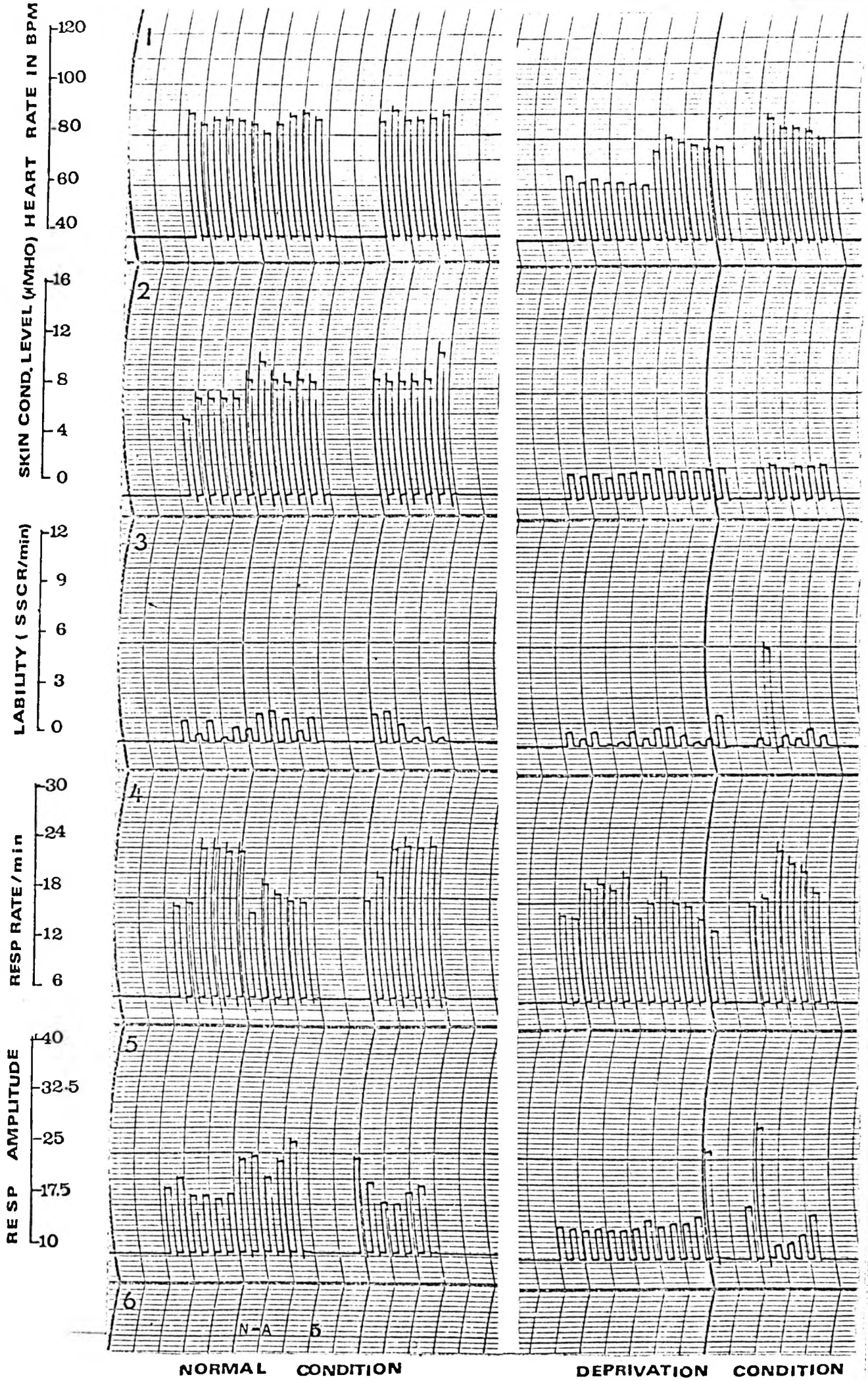

HEART RATE IN BPM
120
100
80
60
40
1
SKIN COND. LEVEL (MHO)
16
12
8
4
0
2
LABILITY (SSCR/min)
12
9
6
3
0
3
RESP RATE/min
30
24
18
12
6
4
RESP AMPLITUDE
40
32.5
25
17.5
10
5
6
ADD-9
NORMAL CONDITION
DEPRIVATION CONDITION

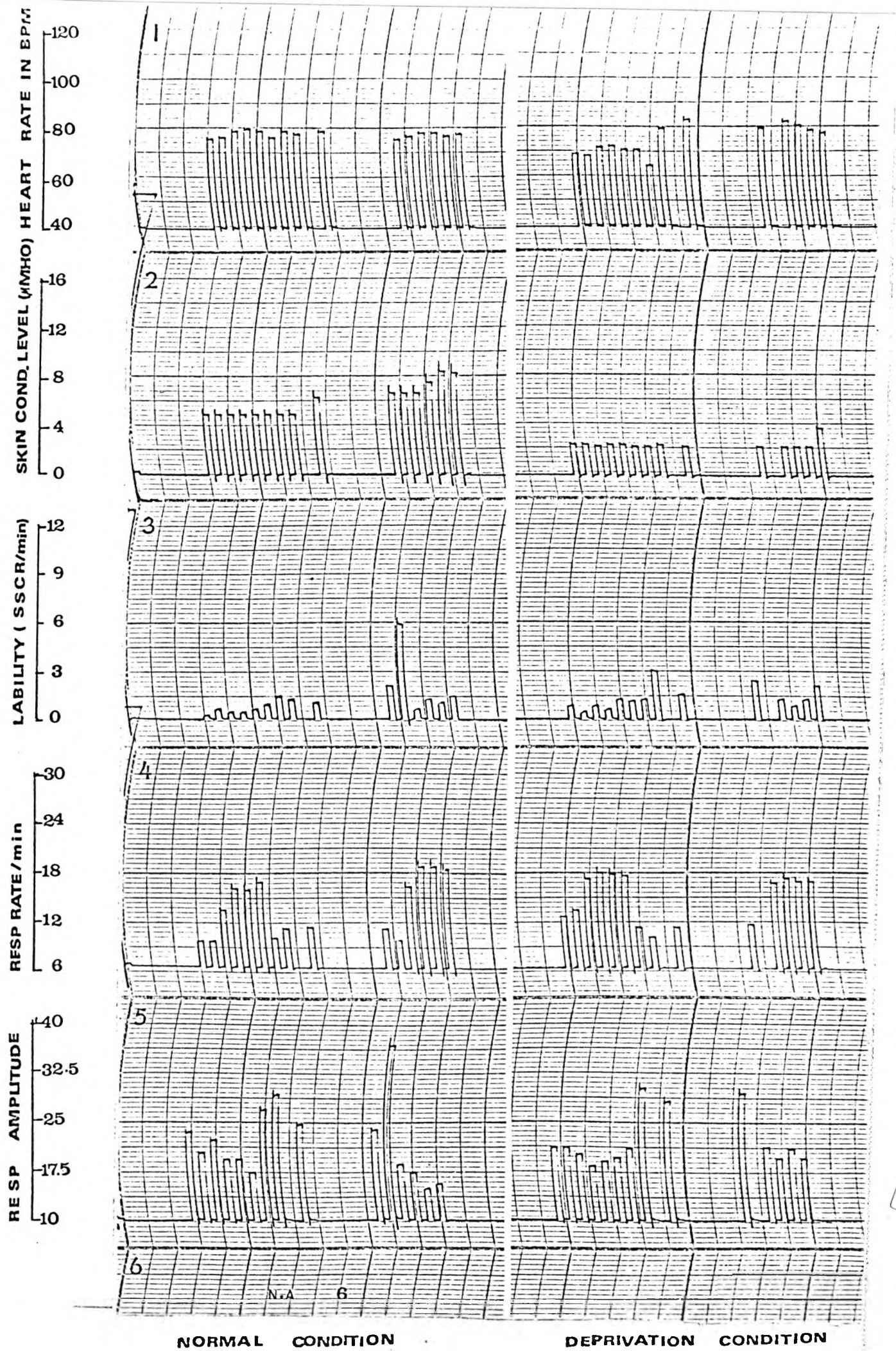

HEART RATE IN BPM
120
100
80
60
40
SKIN COND. LEVEL (μMHO)
16
12
8
4
0
LABILITY (SSCR/min)
12
9
6
3
0
RESP RATE/min
30
24
18
12
6
RESP AMPLITUDE
40
32.5
25
17.5
10
1
2
3
4
5
6
N.A 1
NORMAL CONDITION
DEPRIVATION CONDITION

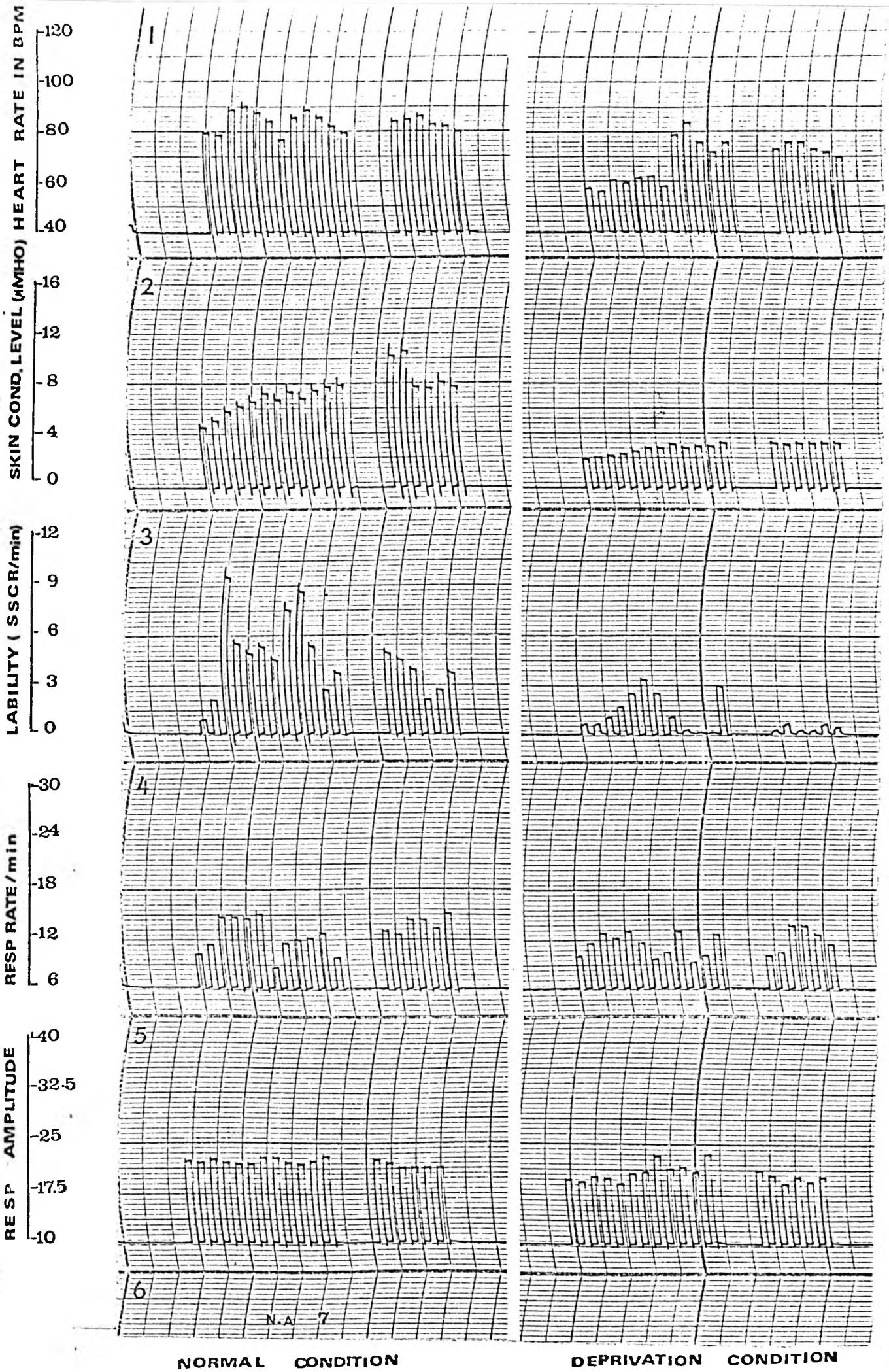

HEART RATE IN BPM
120
100
80
60
40
1
SKIN COND. LEVEL (μMHO)
16
12
8
4
0
2
LABILITY (SSCR/min)
12
9
6
3
0
3
RESP RATE/min
30
24
18
12
6
4
RESP AMPLITUDE
40
32.5
25
17.5
10
5
6
N A 2
NORMAL CONDITION
DEPRIVATION CONDITION

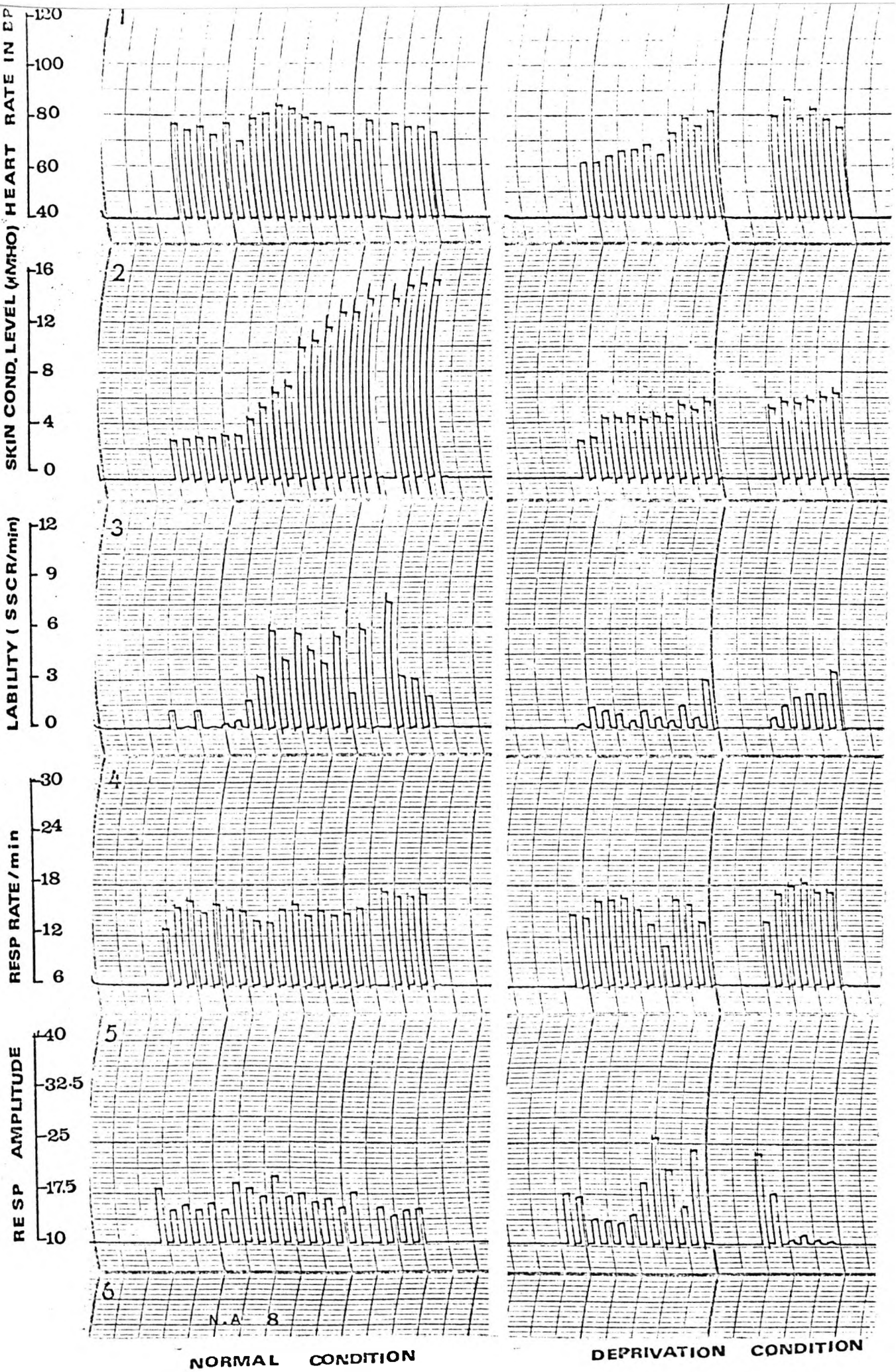

1
HEART RATE IN BPM
120
100
80
60
40
2
SKIN COND. LEVEL (μMHO)
16
12
8
4
0
3
LABILITY (SSCR/min)
12
9
6
3
0
4
RESP RATE/min
30
24
18
12
6
5
RESP AMPLITUDE
40
32.5
25
17.5
10
6
N.A 3
NORMAL CONDITION
DEPRIVATION CONDITION

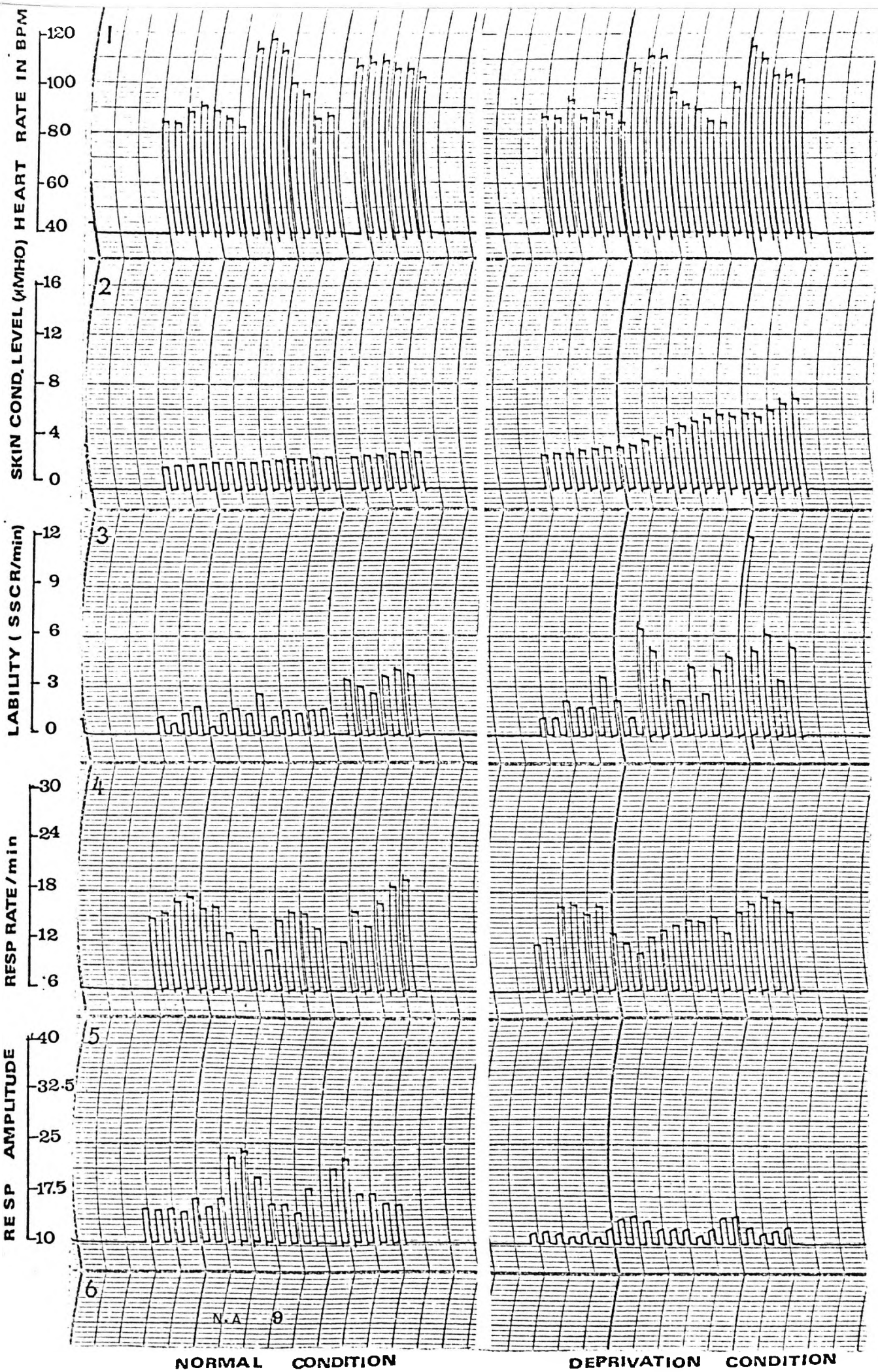

HEART RATE IN BPM
120
100
80
60
40
SKIN COND. LEVEL (μMHO)
16
12
8
4
0
LABILITY (SSCR/min)
12
9
6
3
0
RESP RATE/min
30
24
18
12
6
RESP AMPLITUDE
40
32.5
25
17.5
10
1
2
3
4
5
6
N – A 4
NORMAL CONDITION
DEPRIVATION CONDITION

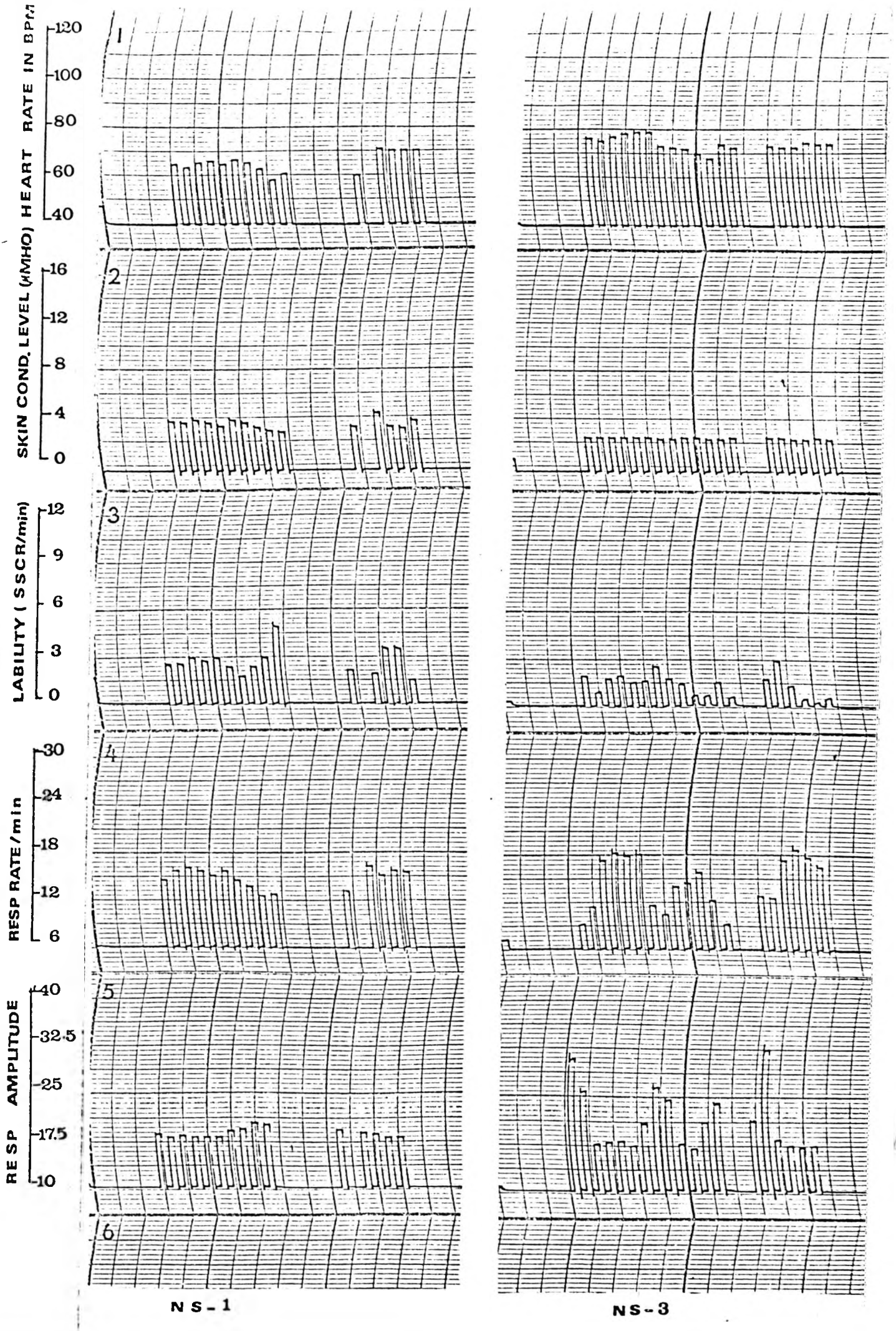

HEART RATE IN BPM
120
100
80
60
40
SKIN COND. LEVEL (μMHO)
16
12
8
4
0
LABILITY (SSCR/min)
12
9
6
3
0
RESP RATE / min
30
24
18
12
6
RESP AMPLITUDE
40
32.5
25
17.5
10
1
2
3
4
5
6
N-A 5
NORMAL CONDITION
DEPRIVATION CONDITION

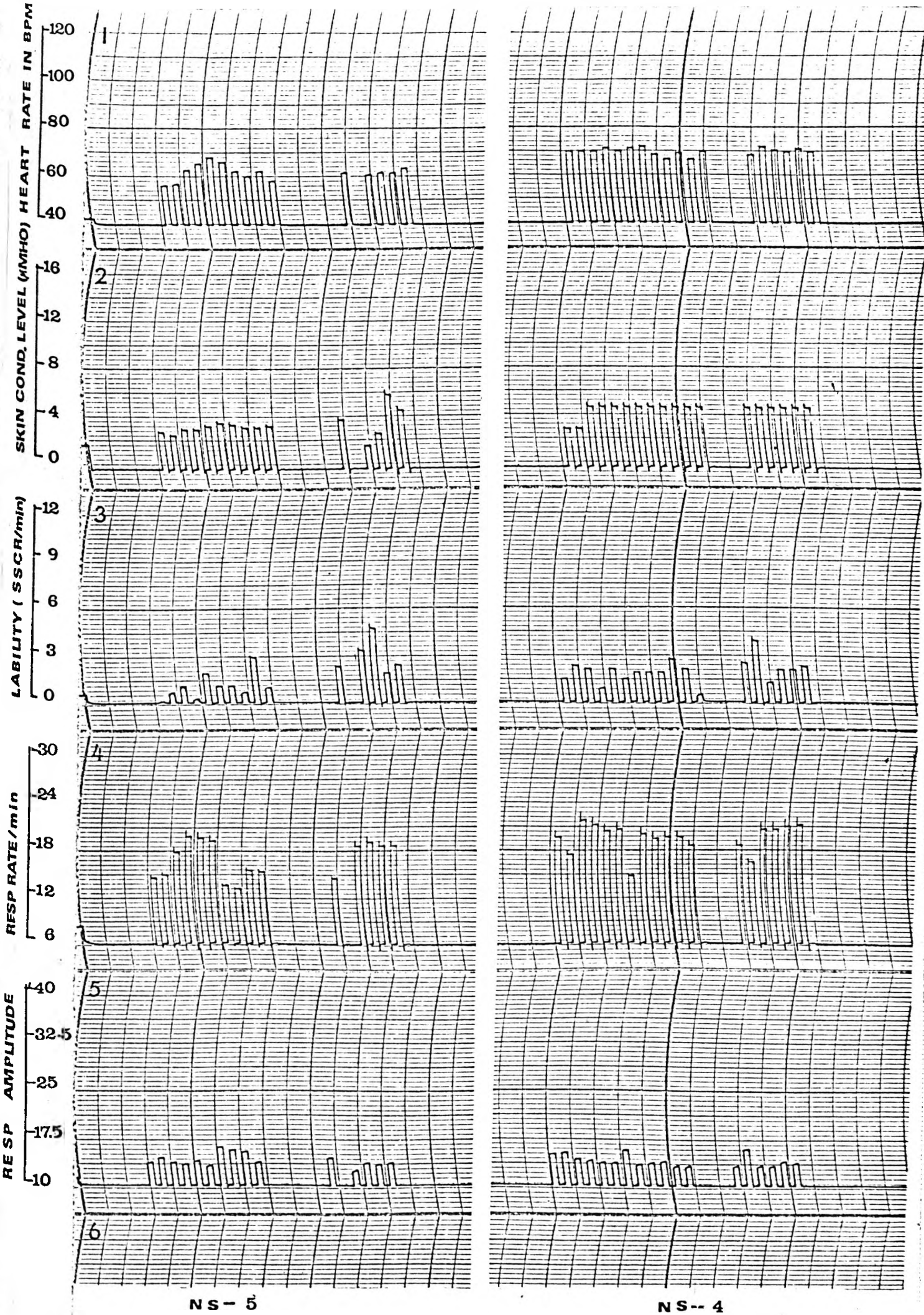

1
HEART RATE IN BPM
120
100
80
60
40
2
SKIN COND. LEVEL (MHO)
16
12
8
4
0
3
LABILITY (SSCR/min)
12
9
6
3
0
4
RESP RATE/min
30
24
18
12
6
5
RESP AMPLITUDE
40
32.5
25
17.5
10
6
N.A 6
NORMAL CONDITION
DEPRIVATION CONDITION

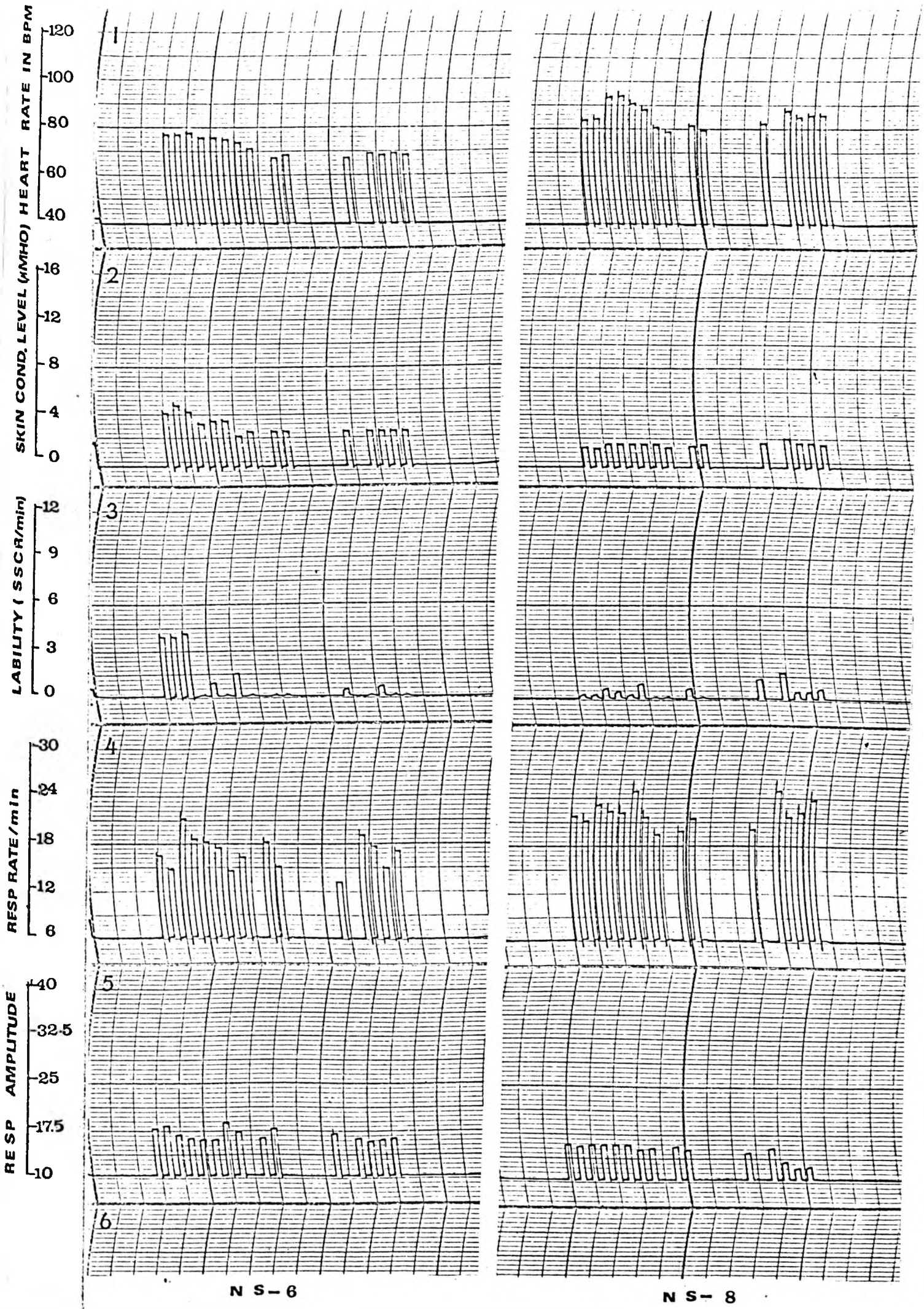

HEART RATE IN BPM
120
100
80
60
40
1
SKIN COND. LEVEL (μMHO)
16
12
8
4
0
2
LABILITY (SSCR/min)
12
9
6
3
0
3
RESP RATE/min
30
24
18
12
6
4
RESP AMPLITUDE
40
32·5
25
17·5
10
5
6
N.A 7
NORMAL CONDITION
DEPRIVATION CONDITION

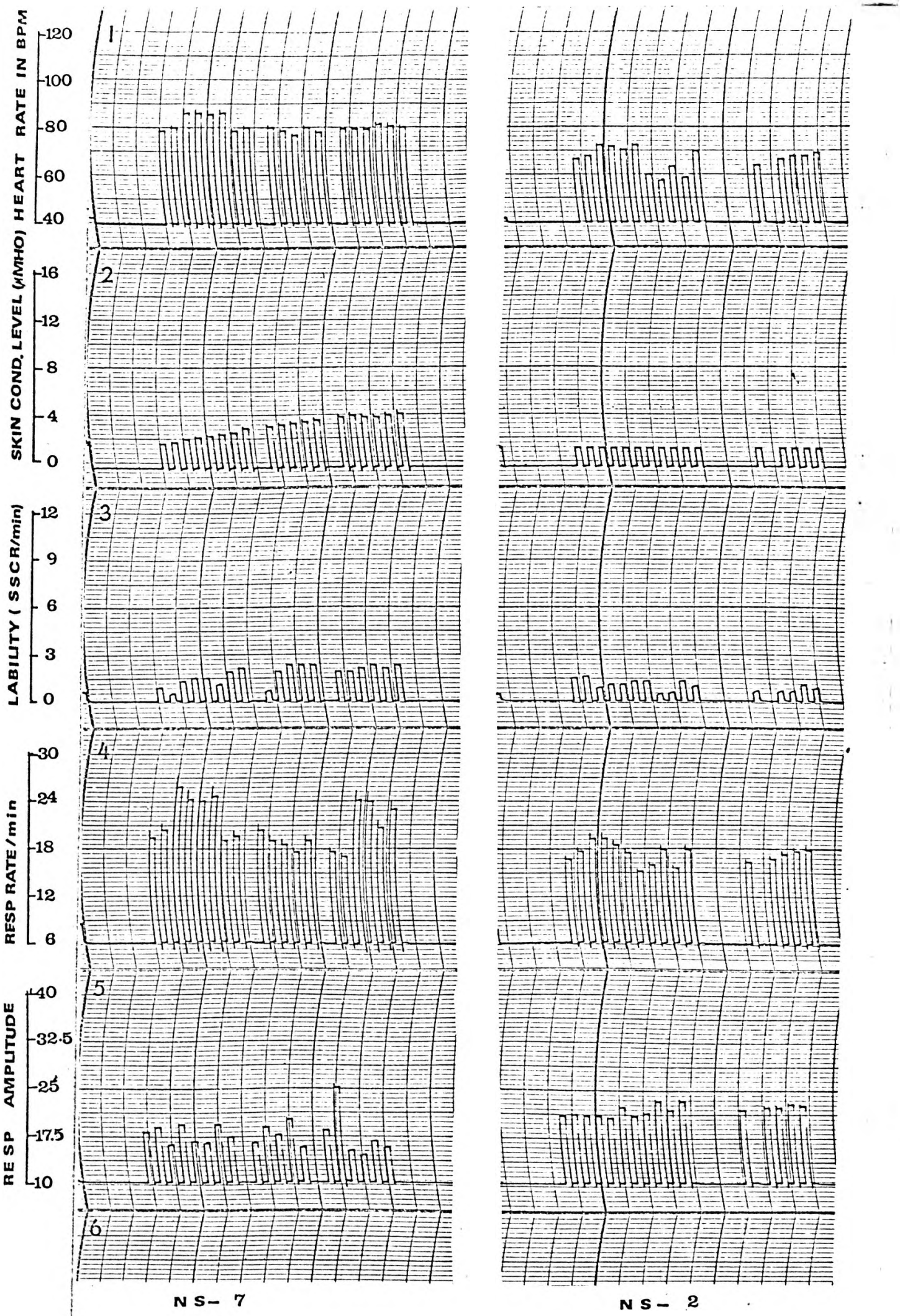

HEART RATE IN BP
120
100
80
60
40
1
SKIN COND. LEVEL (μMHO)
16
12
8
4
0
2
LABILITY (SSCR/min)
12
9
6
3
0
3
RESP RATE/min
30
24
18
12
6
4
RESP AMPLITUDE
40
32.5
25
17.5
10
5
6
N.A 8
NORMAL CONDITION
DEPRIVATION CONDITION

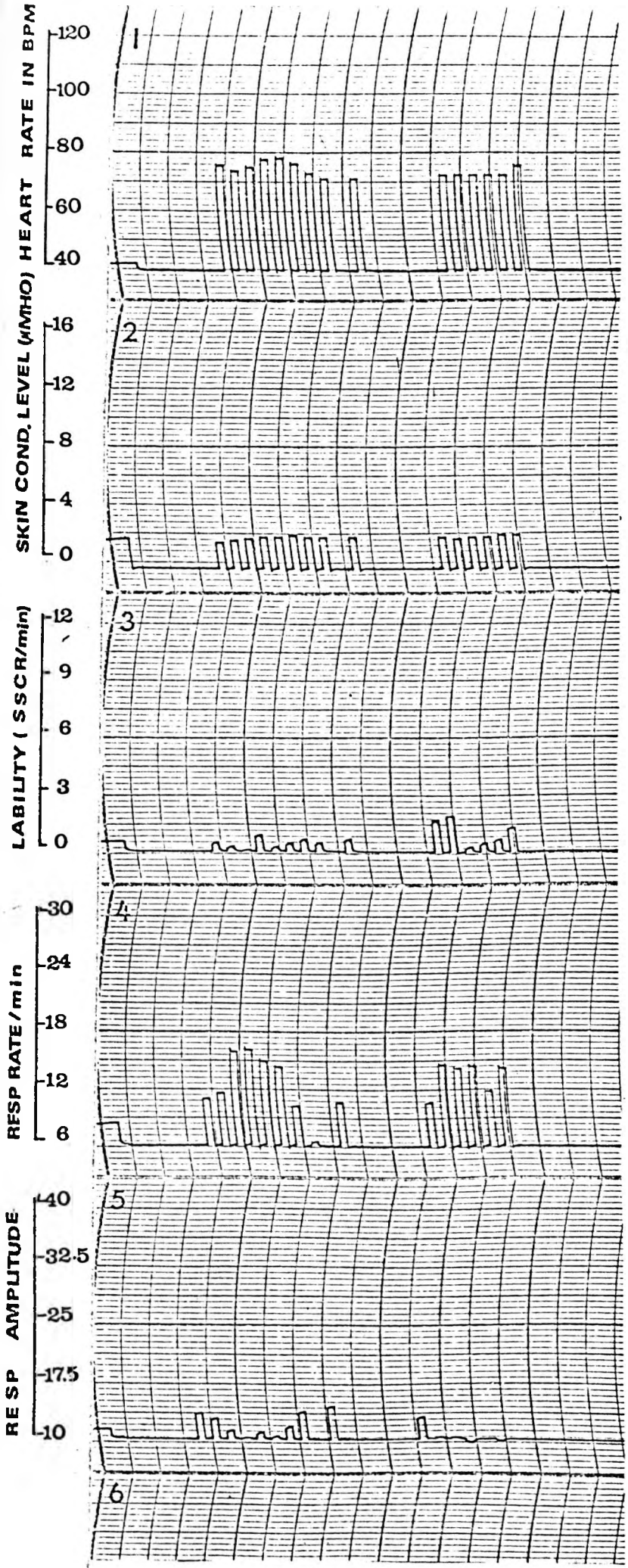

HEART RATE IN BPM
120
100
80
60
40
1
SKIN COND. LEVEL (μMHO)
16
12
8
4
0
2
LABILITY (SSCR/min)
12
9
6
3
0
3
RESP RATE/min
30
24
18
12
6
4
RESP AMPLITUDE
40
32.5
25
17.5
10
5
6
N.A 9
NORMAL CONDITION
DEPRIVATION CONDITION


HEART RATE IN BPM
120
100
80
60
40
1
SKIN COND. LEVEL (μMHO)
16
12
8
4
0
2
LABILITY (SSCR/min)
12
9
6
3
0
3
RESP RATE / min
30
24
18
12
6
4
RESP AMPLITUDE
40
32.5
25
17.5
10
5
6
NS - 1
NS - 3


HEART RATE IN BPM
120
100
80
60
40
1
SKIN COND. LEVEL (MHO)
16
12
8
4
0
2
LABILITY (SSCR/min)
12
9
6
3
0
3
RESP RATE/min
30
24
18
12
6
4
RESP AMPLITUDE
40
32.5
25
17.5
10
5
6
NS– 5
NS-- 4


HEART RATE IN BPM
120
100
80
60
40
1
SKIN COND. LEVEL (MMHO)
16
12
8
4
0
2
LABILITY (SSCR/min)
12
9
6
3
0
3
RESP RATE/min
30
24
18
12
6
4
RESP AMPLITUDE
40
32.5
25
17.5
10
5
6
N S – 6
N S – 8


HEART RATE IN BPM
120
100
80
60
40
1
SKIN COND. LEVEL (μMHO)
16
12
8
4
0
2
LABILITY (SSCR/min)
12
9
6
3
0
3
RESP RATE/min
30
24
18
12
6
4
RESP AMPLITUDE
40
32.5
25
17.5
10
5
6
NS– 7
NS– 2


HEART RATE IN BPM
120
100
80
60
40
1
SKIN COND. LEVEL (μMHO)
16
12
8
4
0
2
LABILITY (SSCR/min)
12
9
6
3
0
3
RESP RATE / min
30
24
18
12
6
4
RESP AMPLITUDE
40
32.5
25
17.5
10
5
6

NS- 9

App. G

3 factors ANOVA- with repeated measures on two factors (B & C).

A=Groups (A_1= Addicted; A_2= Non-Addicted)

B=Conditions (B_1=Normal; B_2=Deprivation)

C=Experimental periods (i.e; BL 1; Vig I; BL 2; SM 1; Lat 1; Lat 2; SM 2; Vig II).

* $p<0.05$; ** $p<0.025$; *** $p<0.001$; NS, $p>0.05$.

App. G.i : HEART RATE

SOURCE	SOS	DF	MS	F
BETWEEN SUBJECTS	29941.9404	17		
A	294.5771	1	294.5771	0.1591
ERROR (A)	29647.3632	16	1852.9602	
WITHIN SUBJECTS	17889.1313	270		
B	2456.4792	1	2456.4792	12.6128***
AB	0.2502	1	0.2502	0.0013
ERROR (B)	3116.1691	16	194.7606	
C	4675.8083	7	667.9726	17.5331***
AC	263.9991	7	37.7142	0.9899
ERROR (C)	4266.9538	112	38.0978	
BC	1098.7740	7	156.9677	9.0495***
ABC	68.0129	7	9.7161	0.5602
ERROR (BC)	1942.6847	112	17.3454	

DUNCAN MULTIPLE RANGE TEST FOR "C",PERIODS EFFECT:(df=112, MSe=38.09, 0=NS, 1=Signif. p 0.05.

BL I	BL II	VIG I	Lat II	VIG II	SM II	SM I	LAT I
70.490	71.240	74.360	78.510	79.330	79.400	80.020	81.980
70.490	0	1	1	1	1	1	1
71.240		1	1	1	1	1	1
74.360			1	1	1	1	1
78.510				0	0	0	1
79.330					0	0	0
79.400						0	0
80.020							0

App.G.i. Cont.

DUNCAN M.R.T: CONDITIONS (B) X PERIODS (C) INTERACTION (df=112, MSe=17.34,

0=NS; 1=Signif. $p < 0.05$)

N=Normal condition

D=Deprivation condition.

PERIODS	I	II	III	IV	V	VI	VII	VIII
NORMAL	76.21	79.39	76.72	81.36	83.36	79.51	81.46	80.70
DEPRIV.	64.77	69.32	65.77	78.68	80.60	77.52	77.35	77.96

Mean HR values in the two experimental conditions over the 8 experimental periods.

	N.I (76.21)	N.III (76.72)	N.II (79.39)
D.I(64.77)	1	1	1
D.III(65.77)	1	1	1
D.II (69.32)	1	1	1

Table (G.i).1 Pre-smoking: Normal versus Deprivation values.

	D.IV 78.68)	D.V (80.60)	D.VI (77.52)	D.VII (77.35)	D.VIII (77.96)
D.III (65.77)	1	1	1	1	1
D.V (80.60)	0		0	1	0

Table (G.i).2 Deprivation condition: Effects of smoking.

	N.IV (81.36)	N.V (83.36)	N.VI (79.51)	N.VII (81.46)	N.VIII (80.70)
N.III (76.72)	1	1	0	1	1
N.V (83.36)	0		1	0	0

Table (G.i).3 Normal Smoking condition: Effects of smoking

App. G.ii- SKIN CONDUCTANCE LEVEL

SOURCE	SOS	DF	MS	F
BETWEEN SUBJECTS	1000.1559	17		
A	28.0560	1	28.0560	0.4618
ERROR (A)	972.0999	16	60.7562	
WITHIN SUBJECTS	1171.4534	270		
B	124.9121	1	124.9121	3.8111
AB	151.1046	1	151.1046	4.6103*
ERROR (B)	524.4096	16	32.7756	
C	137.2316	7	19.6045	19.6218***
AC	19.4954	7	2.7851	2.7889**
ERROR (C)	111.8442	112	0.9986	
BC	8.4898	7	1.2128	1.5118
ABC	4.1129	7	0.5876	0.7324
ERROR (BC)	89.8530	112	0.8023	

DUNCAN M.R.T: GROUPS (A) X CONDITIONS (B) INTERACTION-

df=16; MSe=32.77; $p < 0.05$; 0=NS; 1=Signif.

		ADD.Norm.	ADD.depr.	N.A. Norm
	Means	3.735	3.867	5.808
N.A. Depr.	3.042	0	0	1
ADD. Norm.	3.735		0	0
ADD. Depr.	3.867			0

APP. G.ii

SKIN CONDUCTANCE- GROUPS X PERIODS (AC) INTERACTION

DUNCAN M.R.T- df=112, MSe=0.9986, 1= significant at<0.05 level; 0=NS

A=Addicted; NA=Non-Addicted

AC MEANS

	C 1	C 2	C 3	C 4	C 5	C 6	C 7	C 8	
Addicted	2.758	3.331	3.757	3.865	4.037	4.047	4.121	4.492	3.801
Non-Addicted	3.089	3.526	3.884	3.994	4.389	5.266	5.506	5.747	4.425
	2.924	3.428	3.821	3.929	4.213	4.657	4.813	5.119	

	NA I	A II	NA II	A III	A IV	NA III	NA IV	A V	A VI	A VII
A I 2.758	0	0	1	1	1	1	1	1	1	1
NA I 3.089		0	0	0	1	1	1	1	1	1
A II 3.331			0	0	0	0	0	0	0	1
NA II 3.526				0	0	0	0	0	0	0
A III 3.757					0	0	0	0	0	0
A IV 3.865						0	0	0	0	0
NA III 3.884							0	0	0	0
NA IV 3.994								0	0	0
A V 4.037									0	0
A VI 4.047										0
A VII 4.121										

	NA V	A VIII	NA VI	NA VII	NA VIII (5.747)
A I	1	1	1	1	1
NA I	1	1	1	1	1
A II	1	1	1	1	1
NA II	1	1	1	1	1
A III	0	0	1	1	1
A IV	0	0	1	1	1
NA III	0	0	1	1	1
NA IV	0	0	1	1	1
A IV	0	0	1	1	1
A VI	0	0	1	1	1
A VII	0	0	1	1	1
NA V		0	1	1	1
A VIII			1	1	1
NA VI				0	0
NA VII					0
NA VIII					

App.G.ii-Cont.

SKIN CONDUCTANCE- PERIODS (C) EFFECT.

DUNCAN M.R.T.- df=112, MSe=0.9986, $p < 0.05$; 1= signif, 0=NS.

							Vig II
	2.924	3.428	3.821	3.929	4.213	4.657	4.813	5.119
BL I	2.924	1	1	1	1	1	1	1
VIG I	3.428		0	1	1	1	1	1
BL II	3.821			0	0	1	1	1
SM I	3.929				0	1	1	1
LAT I	4.213					0	1	1
LAT II	4.657						0	0
SM II	4.813							0

App. G.iii- LABILITY (NS.SCR)

SOURCE	SOS	DF	MS	F
BETWEEN SUBJECTS	154.6247	17		
A	1.6095	1	1.6095	0.1681
ERROR (A)	153.2152	16	9.5760	
WITHIN SUBJECTS	532.9540	270		
B	7.5434	1	7.5434	1.0551
AB	2.8581	1	2.8581	0.3998
ERROR (B)	114.3936	16	7.1496	
C	74.6944	7	10.6706	6.1975***
AC	9.9123	7	1.4161	0.8218
ERROR (C)	192.9904	112	1.7231	
BC	14.5033	7	2.0719	2.0756
ABC	4.2562	7	0.6080	0.6091
ERROR (BC)	111.8023	112	0.9982	

DUNCAN M.R.T. - LABILITY: PERIODS (C) EFFECT

(df=112, MSe=1.72, p (0.05; 1=signif, 0=NS)

								LAT II
	1.014	1.401	1.703	1.975	2.269	2.349	2.388	2.569
BL I	1.014	0	1	1	1	1	1	1
VIG I	1.401		0	0	1	1	1	1
BL II	1.703			0	0	0	1	1
VIG II	1.975				0	0	0	1
SM I	2.269					0	0	0
SM II	2.349						0	0
LAT I	2.388							0

APP. G.iv: RESPIRATION RATE

SOURCE	SOS	DF	MS	F
BETWEEN SUBJECTS	1654.7939	17		
A	125.6641	1	125.6641	1.1625
ERROR (A)	1729.1299	16	108.0706	
WITHIN SUBJECTS	1936.3418	270		
B	25.0488	1	25.0488	3.6201
AB	7.8609	1	7.8609	1.1359
ERROR (B)	110.7109	16	6.9194	
C	896.1494	7	128.0213	29.0170***
AC	81.6486	7	11.6632	2.6445
ERROR (C)	493.9541	112	4.4103	
BC	24.5791	7	3.5113	1.4665
ABC	31.6125	7	4.5446	1.9239
ERROR (BC)	264.5635	112	2.3622	

APP. G.iv: DUNCAN M.R.T.: RESPIRATION RATE- PERIODS (C) EFFECT

(df=112, MSe=4.41, $p < 0.05$, 1=signif, 0=NS).

								VIG II
	13.310	13.810	14.050	14.620	14.790	15.290	17.800	18.490
SM I	13.310	0	0	1	1	1	1	1
SM II	13.810		0	0	0	1	1	1
BL II	14.050			0	0	1	1	1
LAT I	14.620				0	0	1	1
LAT II	14.790					0	1	1
BL I	15.290						1	1
VIG I	17.800							0

DUNCAN M.R.T. RESPIRATION RATE- GROUPS X PERIODS (AC) INTERACTION

(df=112, MSe=4.41, $p < 0.05$, 1=signif, 0=NS)

AC MEANS

		C 1	C 2	C 3	C 4	C 5	C 6	C 7	C 8	
Add.	A 1	16.49	18.97	15.18	13.28	14.98	15.17	13.70	19.68	15.93
Non-Add	A 2	14.09	16.64	12.93	13.33	14.26	14.41	13.91	17.29	14.61
		15.29	17.80	14.05	13.31	14.62	14.79	13.81	18.49	

	A I 16.49	A II 18.97	A III 15.18	A IV 13.28	A V 14.98	A VI 15.17	A VII 13.70	A VIII 19.68
N.A I(14.09)	1							
N.A II(16.64)		1						
N.A III(12.93)			1					
N.A IV (13.33)				0				
N.A V (14.26)					1			
N.A VI (14.41)						1		
N.A VII(13.91)							0	
N.A VIII(17.29)								1

Addicted (A) versus non-addicted (N.A) groups: Comparison of RR's in each experimental period.

	A IV	A VII
	13.28	13.70
A III (15.18)	1	1
A IV (13.28)		1

Addicted (A) group: RR's in the two smoking periods and base-line two period.

	N.A IV	N.A VII
	13.33	13.91
N.A III(12.93)	0	1
N.A IV (13.33)		1

Non-addicted (N.A) group: RR's in the two smoking periods and base-line two.period.

APP. G.v: RESPIRATION AMPLITUDE (STANDARD SCORES)

SOURCE	SOS	DF	MS	F
BETWEEN SUBJECTS	1.1911	17		
A	0.0875	1	0.0875	1.2686
ERROR (A)	1.1036	16	0.0690	
WITHIN SUBJECTS	251.0589	270		
B	0.0539	1	0.0539	0.8146
AB	0.1369	1	0.1369	2.0696
ERROR (B)	1.0586	16	0.0662	
C	122.1774	7	17.4539	27.3713***
AC	3.4729	7	0.4961	1.7[illegible]
ERROR (C)	71.4826	112	0.6377	
BC	0.7214	7	0.1031	0.[illegible]
ABC	4.1657	7	0.5951	1.39[illegible]
ERROR (BC)	47.8500	112	0.4272	

DUNCAN M.R.T. RESPIRATION AMPLITUDE- PERIODS (C) EFFECT

(df=112, MSe=0.638, p < 0.05, 1=Signif, 0=NS)

Vig I	Vig II	BL I	BL III	SM II	LAT I	LAT II	SM I
-0.879	-0.779	-0.477	-0.215	0.448	0.512	0.569	0.978
-0.879	0	0	1	1	1	1	1
-0.779		0	1	1	1	1	1
-0.477			0	1	1	1	1
-0.215				1	1	1	1
0.448					0	0	0
0.512						0	0
0.569							0

App. G.vi: INTER-PUFF INTERVAL

SOURCE	SOS	DF	MS	F
BETWEEN SUBJECTS	18721.4404	17		
A	1416.7158	1	1416.7158	1.3099
ERROR (A)	17304.7245	16	1081.5453	
WITHIN SUBJECTS	5692.2725	54		
B	9.7109	1	9.7109	0.0899
AB	7.0693	1	7.0693	0.0654
ERROR (B)	1728.3408	16	108.0213	
C	539.3428	1	539.3428	3.2744
AC	38.0682	1	38.0682	0.2311
ERROR (C)	2635.7441	16	164.7340	
BC	286.0859	1	286.0859	10.2417 ***
ABC	0.1865	1	0.1865	0.0067
ERROR (BC)	446.9877	16	27.9330	

DUNCAN M.R.T.- INTER-PUFF INTERVAL: CONDITIONS X PERIODS (BC) INTERACTION

(df=16, MSe=27.93, $p < 0.05$, 1=Signif, 0=NS)

	C MEANS		
	C 1	C 2	
Normal	26.17	35.63	30.90
Depriv.	29.42	30.91	30.16
	27.79	33.27	

C 1=cig 1

C 2=Cig 2

	29.42	30.91	35.63
26.17	0	1	1
29.42		0	1
30.91			1

APP. G.vii. NUMBER OF PUFFS

SOURCE	SOS	DF	MS	F
BETWEEN SUBJECTS	2412.2776	17		
A	3.5557	1	3.5557	0.0236
ERROR (A)	2408.7222	16	150.5451	
WITHIN SUBJECTS	569.0000	54		
B	26.8889	1	26.8889	2.3055
AB	24.5000	1	24.5000	2.1006
ERROR (B)	186.6111	16	11.6632	
C	84.5000	1	84.5000	14.4221 **
AC	43.5556	1	43.5556	7.4361 *
ERROR (C)	93.9444	16	5.8715	
BC	8.0000	1	8.0000	1.3084
ABC	2.7222	1	2.7222	0.4452
ERROR (BC)	98.2778	16	6.1424	

**$p < 0.01$; * $p < 0.025$.

App.G.vii. Cont.

DUNCAN M.R.T. - NUMBER OF PUFFS: GROUPS X PERIODS (AC)

INTERACTION

(df=16, MSe= 5.871, $p < 0.05$, 1=Signif. 0=NS)

	Means	14.67 (Add-Cig 1)	15.28	17.28
Add- Cig 2	13.56	0	0	1
N.A - Cig 2	14.67		0	1
N.A - Cig 1	15.28			1

APP. G.viii. CIGARETTE DURATION

SOURCE	SDS	DF	MS	F
BETWEEN SUBJECTS	404183.5684	17		
A	33411.1251	1	33411.1251	1.4416
ERROR (A)	370772.4455	16	23173.2777	
WITHIN SUBJECTS	165540.7724	54		
B	396.6806	1	396.6806	0.1279
AB	8213.3473	1	8213.3473	2.6484
ERROR (B)	49634.3224	16	3101.2639	
C	66.1250	1	66.1250	0.0242
AC	2346.1250	1	2346.1250	0.8579
ERROR (C)	43757.9998	16	2734.8750	
BC	22366.1250	1	22366.1250	9.7538**
ABC	2161.6810	1	2161.6810	0.9426
ERROR (BC)	36672.4679	16	2292.0292	

(** $p < 0.01$)

DUNCAN M.R.T - CIGARETTE DURATION: CONDITIONS X PERIODS (BC)

INTERACTION

(df=16, MSe=2292.03, p<0.05, 1=Signif, 0=NS)

B1.C1	B2.C2	B1.C2	B2.C1
353.300	356.100	366.700	393.300
353.300	0	0	1
356.100		0	1
366.700			0

C1=Cig. 1 B1=Normal condition

C2=Cig. 2 B2=Deprivation condition .

APP. G.ix. LATENCY

2 Factors ANOVA: repeated measures on B

A=Groups (A_1= Addicted, A_2= Non-addocted)

B=Conditions (B_1= Normal , B_2= Deprivation)

MEANS

	B_1	B_2
A_1	3.4588	4.4044
A_2	16.4644	16.2944

App.G.ix. Cont. ANOVA table:

SOURCE	SS	DF	MS	F
BETW SUBJS	3004.031	17		
A	1394.525	1	1394.525	14.[illegible]**
SUBJ W GPS	1609.506	16	100.5941	
WITHN SUBJ	619.3296	18		
B	1.353308	1	1.353308	.0351979
AB	2.800018	1	2.800018	.0728251
B SWG	615.1763	16	38.44852	
A AT B 1	761.1501	1	761.1501	19.94842**
A AT B 2	636.1745	1	636.1745	9.150762**
SSWCELL	2224.682	32	69.52133	
B AT A 1	4.023335	1	4.023335	.1046421
B AT A 2	.1300049	1	.1300049	.0033813
SSBSWG	615.1763	16	38.44852	

(** p<0.01)

APP. G.x: BUTT LENGTH

2 Factors ANOVA, repeated measures on one factor.

A=Groups (A_1=Addicted; A_2= Non-addicted)

B=Cig 1 & 2 (B_1= Cig 1, B_2= Cig 2) (** p<0.01)

SOURCE	SS	DF	MS	F
BETW SUBJS	1320.472	17		
A	17.36107	1	17.36107	.2131646
SUBJ W GPS	1303.111	16	81.44444	
WITHN SUBJ	494.5000	18		
B	164.6944	1	164.6944	6.736723**
AB	30.25005	1	30.25005	1.615739
B SWG	299.5556	16	18.72222	
A AT B 1	46.72223	1	46.72223	.9326897
A AT B 2	.6388855	1	.6388855	.0177481
SSWCELL	1604.667	32	50.08333	
B AT A 1	163.6555	1	163.6555	3.376601**
B AT A 2	26.63889	1	26.63889	1.436602
SSBSWG	299.5556	16	18.72222	

APP. H: NICOTINE INTAKE ESTIMATED FROM BUTT NICOTINE ANALYSIS

H.i : ADDICTED versus NON-ADDICTED GROUPS: NICOTINE INTAKE IN THE DEPRIVATION CONDITION

SOURCE	SS	DF	MS	F
BETW SUBJS	18.78072	17		
A	1.141334	1	1.141334	1.035259
SUBJ W GPS	17.63939	16	1.102462	
WITHN SUBJ	3.423151	18		
B	.1950670	1	.1950670	.9930194
AB	.0850725	1	.0850725	.4330753
B SWG	3.143012	16	.1964382	
A AT B 1	.3016055	1	.3016055	.4644014
A AT B 2	.9247999	1	.9247999	1.423974
SSWCELL	20.78240	32	.6494500	
B AT A 1	.0112506	1	.0112506	.0572727
B AT A 2	.2688886	1	.2688886	1.368820
SSBSWG	3.143012	16	.1964382	

A=Groups (A_1=Addicted, A_2=Non-addicted)

B=Cigarettes (B_1= Cig. 1, B_2= Cig. 2)

APP. H.ii : NICOTINE INTAKE: NORMAL VERSUS DEPRIVATION CONDITIONS CIG. 1 :

ADDICTED AND NON-ADDICTED GROUPS

A=GROUPS

B=CONDITIONS

SOURCE	SS	DF	MS	F
BETW SUBJS		10		
A	2.190086	1	2.190086	4.191800 NS; p>0.05
SUBJ W GPS	4.702222	9	.5224691	
WITHN SUBJ		11		
B	.1848194	1	.1848194	1.294676
AB	.0235653	1	.0235653	.1650768
B X SWG	1.284781	9	.1427534	

APP. H.iii: NICOTINE INTAKE: DEPRIVATION DAY FIRST CIG. AND DAILY LIFE:

ADDICTED AND NON-ADDICTED GROUPS

(* p<0.05, **p<0.025, ***p<0.01)

SOURCE	SS	DF	MS	F
BETW SUBJS	10.06301	17		
A	.4246684	1	.4246684	.7049649
SUBJ W GPS	9.638345	16	.6023965	
WITHN SUBJ	3.965151	18		
B	1.782224	1	1.782224	13.15719***
AB	.0156257	1	.0156257	.1153559
B SWG	2.167301	16	.1354563	
A AT B 1	.3016055	1	.3016055	.8175221
A AT B 2	.1386890	1	.1386890	.3759259
SSWCELL	11.80565	32	.3689264	
B AT A 1	1.065800	1	1.065800	7.868217**
B AT A 2	.7320499	1	.7320499	5.404325*
SSBSWG	2.167301	16	.1354563	

APP. H.iv: NICOTINE INTAKE: LAB. NORMAL SMOKING AND DAILY LIFE:

ADDICTED AND NON-ADDICTED GROUPS

A=Groups

B=Conditions

SOURCE	SS	DF	MS	F	
BETW SUBJS		10			
A	.9570103	1	.9570103	3.338446	NS, P>0.05
SUBJ W GPS	2.579970	9	.2866634		
WITHN SUBJ		11			
B	.0059842	1	.0059842	.0381985	
AB	.1211846	1	.1211846	.7735502	
B X SWG	1.409943	9	.1566603		

APP. I- HIGH VERSUS LOW NICOTINE INTAKE GROUPS : HEART RATE

3 Factor ANOVA (A=Groups; B=Conditions; C=periods,(BL 1; SM 1; Lat 1), repeated measures on B & C).

SOURCE	SOS	DF	MS	F
BETWEEN SUBJECTS	12198.8671	17		
A	123.9375	1	123.9375	0.1642
ERROR (A)	12074.9297	16	754.6831	
WITHIN SUBJECTS	6965.5625	90		
B	847.7266	1	847.7266	12.4673*
AB	159.9844	1	159.9844	2.3528
ERROR (B)	1087.9375	16	67.9961	
C	2575.6328	2	1287.8164	32.1927**
AC	22.4922	2	11.2461	0.2811
ERROR (C)	1280.1094	32	40.0034	
BC	448.8828	2	224.4414	18.7008**
ABC	158.7422	2	79.3711	6.6133*
ERROR (BC)	384.0547	32	12.0017	

(* $p<0.01$; **$p<0.001$)

APP.I.i- DUNCAN M.R.T : NORMAL SMOKING CONDITION

(df=32, MSe=12.00, $p<0.05$, 1=Signif, 0=NS)

L= Low-nicotine intake group

H= High-nicotine intake group.

L BL 1	L SM 1	H BL 1	L Lat 1	H SM 1	H Lat 1
73.100	79.660	79.830	81.560	83.070	85.150
73.100	1	1	1	1	1
79.660		0	0	0	1
79.830			0	0	1
81.560				0	1
83.070					0

APP. I. Cont.
ii)

DUNCAN M.R.T: DEPRIVATION CONDITION

(df=32, MSe=12.00, p<0.05, 1=Signif, 0=NS) (L=Low-nic. intake grp; H=High-nic.intake grp)

H BL 1	L BL 1	L SM 1	L Lat 1	H SM 1	H Lat 1
62.620	67.570	77.340	79.910	80.030	81.290
62.620	1	1	1	1	1
67.570		1	1	1	1
77.340			0	0	1
79.910				0	0
80.030					0

I.iii: NORMAL VERSUS DEPRIVATION CONDITIONS:

a-HIGH-NICOTINE INTAKE GROUP: DUNCAN M.R.T

(df=16, MSe=67.99, p<0.05, 1=Signif, 0=NS)

D=deprivation; N=Normal smoking condition

D BL 1	N BL 1	D SM 1	D Lat 1	N SM 1	N Lat 1
62.620	79.830	80.030	81.290	83.070	85.150
62.620	1	1	1	1	1
79.830		0	0	0	1
80.030			0	0	1
81.290				0	1
83.070					0

iv-b: LOW-NICOTINE INTAKE GROUP: DUNCAN M.R.T

(df=16, MSe=67.99, p $<$ 0.05, 1=Signif, 0=NS)(D=Deprivation; N=Normal condition)

BL 1 D	BL 1 N	SM 1 D	SM 1 N	Lat 1 D	Lat 1 N
67.570	73.100	77.340	79.660	79.910	81.560
67.570	1	1	1	1	1
73.100		1	1	1	1
77.340			0	0	1
79.660				0	0
79.910					0

APPENDIX. J:

J.i: VIGILANCE TEST PERFORMANCE

3 Factors ANOVA (A=Groups; Addicted and Non-Addicted;
B=Conditions; Normal and deprivation;
C= Test 1 versus test 2, repeated measures on B & C).

SOURCE	SOS	DF	MS	F
BETWEEN SUBJECTS	7910.0703	17		
A	8.6797	1	8.6797	0.0176
ERROR (A)	7901.3906	16	493.8369	
WITHIN SUBJECTS	5581.2500	54		
B	0.3477	1	0.3477	0.0028
AB	0.3477	1	0.3477	0.0028
ERROR (B)	1993.0547	16	124.5659	
C	333.6797	1	333.6797	5.2151 *
AC	253.1289	1	253.1289	3.9559
ERROR (C)	1956.9414	16	66.0586	
BC	153.1250	1	153.1250	1.4712
ABC	125.3437	1	125.3437	1.2043
ERROR (BC)	1665.2813	16	104.0801	

(* p $<$ 0.05)

APP.J.i. Cont.

DUNCAN M.R.T: GROUPS X PERIODS (AC) MEANS

(df=16, MSe=66.66, $p<0.05$, 1=Signif, 0=NS)
Add= Addicted; N.A= Non-Addicted.

A Test 1	N.A Test 1	N.A Test 2	A Test 2
62.220	66.670	67.220	70.280
62.220	0	0	1
66.670		0	0
67.220			0

APP. J.ii: VIGILANCE TEST PERFORMANCE: SMOKERS AND NON-SMOKERS

2 Factors ANOVA (A=GROUPS: Non-smokers; deprived smokers and normal smokers)

SOURCE	SS	DF	MS	F
BETW SUBJS		26		
A	3116.841	2	1558.421	4.675445*
SUBJ W GPS	7999.688	24	333.3203	
WITHN SUBJ		27		
B	.1766438	1	.1766438	.0010167
AB	522.5713	2	261.2857	1.503815
B X SWG	4169.965	24	173.7485	

(* $p<0.025$)

APP. J.ii. Cont.

DUNCAN M.R.T: GROUPS X TESTS (AB) INTERACTION

(df=24, MSe=173,74, p $<$0.05, 1=Signif, 0=NS)

NS=Non-Smoker

DS= Deprived -Smoker

N.SM= Normal-Smoker

NS	NS	N.SM	DS	DS	N.SM
test 2	test 1	test 1	test 1	test 2	test 2
47.780	55.550	65.500	65.620	66.250	73.000
47.780	0	1	1	1	1
55.550		0	0	0	1
65.500			0	0	0
65.620				0	0
66.250					0

APPENDIX: K

NON-SMOKERS: PHYSIOLOGICAL MEASURES

One factor ANOVA (repeated measures, Periods)

App.K.i: HEART RATE

SOURCE	SS	DF	MS	F
BET. SUB	3797.54	8		
WIT. SUB	706.72	63		
COLUMN	357.33	7	51.05	[illegible] **
RESID	349.39	56	6.24	
TOTAL	4504.25	71		

(** p<0.01)

DUNCAN M.R.T: FOR HEART RATE: PERIODS EFFECT

(df= 56, MSe=6.24, p <0.05, 1=Signif. , 0=NS)

V	IV	VI	VII	III	I	VIII	II
68.589	69.330	69.920	70.050	71.110	72.240	73.680	76.010
68.589	0	0	0	0	1	1	1
69.330		0	0	0	1	1	1
69.920			0	0	0	1	1
70.050				0	0	1	1
71.110					0	0	1
72.240						0	1
73.680							1

APP.K.ii: SKIN CONDUCTANCE LEVEL

SOURCE	SS	DF	MS	F
BET. SUB	75.87	8		
WIT. SUB	14.15	63		
COLUMN	1.26	7	0.18	0.78 NS
RESID	12.89	56	0.23	
TOTAL	90.02	71		

APP.K.iii: LABILITY (NS. SCR)

SOURCE	SS	DF	MS	F
BET. SUB	28.57	8		
WIT. SUB	36.42	63		
COLUMN	2.31	7	0.33	0.54 NS
RESID	34.11	56	0.61	
TOTAL	64.99	71		

APP.K.iv: RESPIRATION RATE

SOURCE	SS	DF	MS	F
BET. SUB	766.14	8		
WIT. SUB	294.31	63		
COLUMN	184.08	7	26.30	13.35**
RESID	110.23	56	1.97	
TOTAL	1060.44	71		

(**$p < 0.01$)

APP. k.iv. Cont-

DUNCAN M.R.T: RESPIRATION RATE: PERIODS (df=56, MSe=0.61, p < 0.05, 1=Signif. 0=NS)

III	IV	VII	VI	I	V	VIII	II
14.470	14.490	14.700	15.060	15.520	15.890	18.100	18.940
14.470	0	0	0	0	0	1	1
14.490		0	0	0	0	1	1
14.700			0	0	0	1	1
15.060				0	0	1	1
15.520					0	1	1
15.890						1	1
18.100							0

APPENDIX: K.v- RESPIRATION AMPLITUDE: (STANDARD SCORES)

SOURCE	SS	DF	MS	F
BET. ROW	0.07	8		
WIT. ROW	62.31	63		
COLUMN	14.68	7	2.10	2.47 *
RESID	47.62	56	0.85	
TOTAL	62.38	71		

(* p < 0.05)

DUNCAN M.R.T: RESPIRATION AMPLITUDE: PERIODS

(df=56, MSe=0.85, p < 0.05, 1=Signif. , 0=NS)

VIII	II	VII	V	III	I	VI	IV
-0.950	-0.470	-0.050	0.030	0.280	0.280	0.320	0.480
-0.950	0	0	1	1	1	1	1
-0.470		0	0	0	0	0	0
-0.050			0	0	0	0	0
0.030				0	0	0	0
0.280					0	0	0
0.280						0	0
0.320							0

App. L

WOULD YOU CONSIDER YOURSELF AS ADDICTED TO NICOTINE ?..IF YES WHY ?

ADDICTED SMOKERS

A need exists which smoking supplies.

Even a morning without is unpleasant.

I don't like not having any cigarettes.

Necessity to smoke.

I will go out at night (late) if I run out, to buy more.

I feel that my body needs nicotine.

Because of the withdrawal symptoms (irritability, fiddling), which occur when I have tried to give up.

Craving for cigarettes

Do not know.

NON-ADDICTED SMOKERS

Dont't worry much when I don't have any.

I don't think you can be and I don't feel it.

Can stop without withdrawal symptoms.

Withdrawal symptoms are mental not physical and can be broken by effort.

Because I don't have the urge to smoke if I'm without cigarettes.

I feel that I can easily give up smoking.

I don't often feel that I need a cigarette.

2- Do not know.