AN INVESTIGATION INTO THE ADDITIVE EFFECTS OF TWO BEHAVIOURAL TECHNIQUES IN THE MODIFICATION OF TOBACCO-SMOKING BEHAVIOUR

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For my Parents.

ABSTRACT

A review of the pertinent literature led to the identification of certain issues in the behavioural modification of cigarette smoking which required investigation and clarification.

A three-way factorial design was therefore utilized to assess:

1) the effectiveness of a self-control treatment package, derived from a comprehensive model of smoking behaviour, as compared to a more complex, multi-element package, comprising additional, therapist-administered techniques.

2) The viability of long-term "controlled" smoking, at 25% of baseline smoking rate, as opposed to total abstinence from cigarettes.

and 3) the differential responses of "heavy" and "light" smokers to the above treatment packages and with the alternative aims of abstinence or smoking reduction.

Comprehensive assessment on self-report, physiological and personality measures at pre-, mid- and post-treatment and at three-, six- and twelve-month follow-up, allowed the study of a number of other important issues, namely, the role of expectancy, motivation and personality characteristics in determining response to treatment and the effects of modified smoking behaviour upon lung-function and body-weight.

Eight treatment groups and two control groups were used in the study, each comprising of six randomly allocated subjects.

Regardless of the treatment package utilized and baseline or target smoking-rate, all treatment groups responded equally well to intervention: the statistically significant reductions in smoking-rate evident at mid-treatment assessment were still evident at one-year follow-up. (Changes in self-reported smoking-rate were corroborated by objective measurement in the form of serum-thiocyanate blood-sampling). However, an analysis of the relative rates of success of "abstainers" and "reducers", in attaining and maintaining their targets, in the long term, showed that total abstinence was more easily maintained than reduced smoking (at 25% of baseline-rate). The control groups demonstrated no significant changes in smoking-rate, over time.

Certain positive predictive factors were identified, notably, a high level of expectancy and low scores on measures of psychopathological symptomatology, at pre-treatment.

Although no changes were apparent in respiratory functioning, for any of the treatment groups, all such groups increased significantly in body-weight and maintained this increase at follow-up. "Anxiety" and "craving" measures demonstrated "inverted-U" shaped changes over time, related to initial decrease in smoking rate and later movement in the direction of baseline.

Certain anomalous results were discussed and some methodological difficulties and shortcomings identified; then followed recommendations for increasing the effectiveness of intervention for smoking and a discussion of the implications of the study for future research.

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Neil McQuire
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1. REVIEW OF THE LITERATURE

a) Introduction

Over the last two decades, increasingly sophisticated attempts have been made to modify cigarette smoking behaviour. This increase in sophistication has been not only with regard to treatment methods, but applies also to techniques of measurement and assessment, to the nature and conceptualization of smoking behaviour, the subsequent development of appropriate models of smoking and to the increased recognition of certain "non-specific" factors which influence the outcome of efforts at modification.

A vast amount of literature has accumulated as a result of the continued interest in this field. Behavioural work accounts for the greater part of this literature. Using multiple searches, including Psychological Abstracts, Orleans, et al (1981) compiled a topical bibliography, covering the years 1969-1979, of research into behavioural approaches to smoking cessation. Over 350 references were listed. Research has continued at a similar rate (although with different emphases) over the last five years.

Lichtenstein (1982) draws attention to the fact that over twenty comprehensive reviews of the smoking-cessation literature have been produced (eg. Bernstein and McAlister, 1976; Lichtenstein and Danaher, 1976; Raw, 1978; Frederiksen & Simon, 1979; Pechacek, 1979) and recommends that "because the smoking literature is now so vast, broad reviews should be forsaken in favour of detailed analyses of specific issues".

It is therefore intended, in this review, to follow Lichtenstein's recommendation and to focus primarily on the issues especially relevant to the present research study. The central issues are the nature of smoking behaviour, appropriate goals of intervention, germane and efficient methods of assessment and treatment and certain non-specific factors (McFall and Hammen, 1971) influencing treatment outcome. The side-issue of weight increase as a result of smoking reduction or cessation is also of interest, and this topic will be reviewed in some detail.

b) The Nature of Smoking

(i) Historical Perspective and Health Risks

In the early sixteenth century it was claimed that tobacco cured headaches, coughs, asthma, gout, stomach pains, constipation, flatulence, kidney-stones, arthritis, toothache and hemoptysis. In addition, tobacco was recommended for the treatment of syphilis, consumption, epilepsy and blindness. In 1604, however, the harmful effects of tobacco were already being recognized; James I published a "counter blaste to tobacco". Later in the seventeenth century, Pope Urban VIII condemned tobacco use, threatening excommunication for offenders, and physicians began to link tobacco usage with disorders such as heart pain, asthma, cough and "ulcers of the lungs".

Vogt (1982) quotes a contemporary, anonymous poet :

"Tobacco is an evil weed
It was the devil sowed the seed;
It stains your fingers, burns your clothes,
And makes a chimney of your nose".

In the 1870's, Horace Greely referred to smoking as "fire at one end and a fool at the other". (Harris, 1978).

In the 1920's, cigarettes surpassed pipes, cigars and chewing-tobacco in popularity. The national consumption of cigarettes, in the United Kingdom, steadily increased until 1945, for both men and women (although the proportion of males smoking has always been higher) and then decreased sharply when cigarette prices increased after World War II. Since then, the proportion of male smokers in the population has steadily declined (from 65% to 42%) but female consumption has remained relatively stable (at about 40%). It is encouraging to note that, in 1962, 1971 and 1977, when reports were published by the Royal College of Physicians, tobacco consumption decreased; however, these decreases were both minimal and ephemeral (see Fig. 1.1).

It is estimated that, at present, there are eighteen million cigarette smokers in Britain.

The harmful physical effects of tobacco smoking are well documented (Doll and Peto, 1976; R.C.P. 1962, 1971, 1977, W.H.O., 1975; U.S.D.H.E.W., 1964, 1971, 1973, 1974, 1975, 1976, 1979).

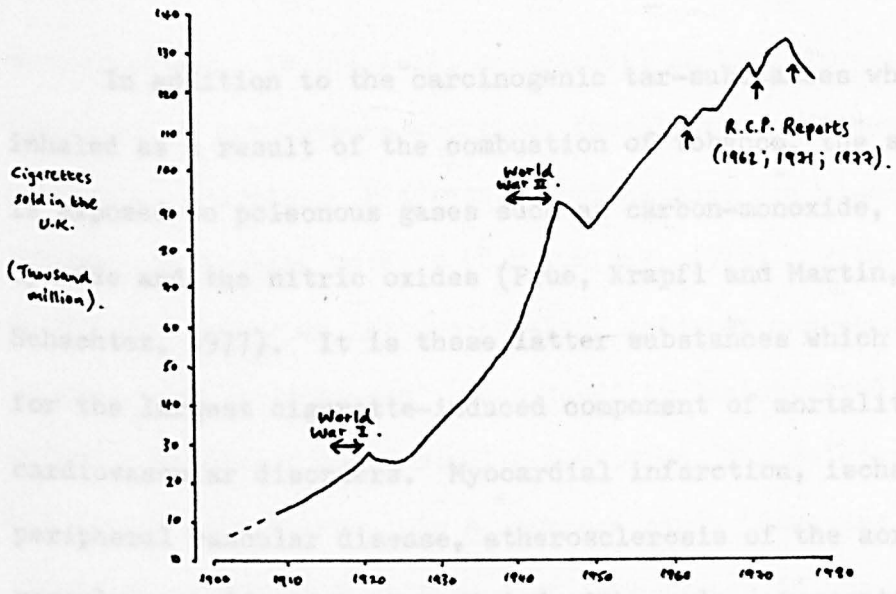

Fig. 1.1

The consumption of cigarettes in the U.K. 1900-1980

(Adapted from Ashton, H. and Stepney, R. 1982)

A recent report by the U.S.D.H.E.W. (1979) noted that smokers of high-tar cigarettes have an annual mortality rate 70% greater than non-smokers; low-tar cigarettes increase the risk by 50%. The relative risk (mortality rate of smokers divided by the mortality rate of non-smokers) is greatest in the 35-55 age range. Individuals who stop smoking exhibit a declining relative risk, which, after fifteen years, approaches 1.0.

In the United Kingdom, tobacco accounts for 10 to 20% of all deaths (R.C.P. 1983); this amounts to a figure greater than 100,000, annually.

In addition to the carcinogenic tar-substances which are inhaled as a result of the combustion of tobacco, the smoker is exposed to poisonous gases such as carbon-monoxide, hydrogen cyanide and the nitric oxides (Prue, Krapfl and Martin, 1981; Schachter, 1977). It is these latter substances which account for the largest cigarette-induced component of mortality - cardiovascular disorders. Myocardial infarction, ischaemic peripheral vascular disease, atherosclerosis of the aorta and vascular complications associated with oral contraceptives have all been clearly attributed to cigarette smoking (U.S.D.H.E.W. 1979). Lung cancer and chronic obstructive lung disease follow the cardiovascular category as causes of death. It is reported that smokers suffer more frequently from bronchitis, emphysema, sinusitis, peptic and duodenal ulceration and tooth and gum disease, than do non-smokers. Some evidence also exists for the existence of tobacco amblyopia; Victor (1970) suggested that this disorder may result from the incomplete detoxification of the cyanide present in tobacco smoke, by certain, prone individuals.

Finally, it is necessary to comment briefly on two further harmful consequences of smoking; impaired foetal development and foetal mortality and the effects of smoke on non-smokers ("passive smoking"). Infants born to smokers are lighter (by 100 grams), shorter (by 1 cm) and smaller in head-circumference than those born to comparable non-

smoking women. As the level of maternal smoking increases, the risks of spontaneous abortion, foetal death and neonatal death also increase. Placental complications are also more frequent in smoking mothers (U.S.D.H.E.W. 1979). With regard to the phenomenon of passive-smoking, evidence has accumulated to show that "side-stream" (as opposed to "mainstream") smoke, although differing in chemical composition, has deleterious effects on non-smokers. In a Japanese study (Hirayama, 1981) it was shown that the incidence of lung cancer in non-smoking wives whose husbands smoked in excess of 20 cigarettes a day, was twice as great as wives of men who did not smoke. Similar results have been obtained in Greece (Trichopoulos et al, 1981) and the United States (Garfinkel, 1981). The evidence concerning cardiovascular damage is rather more sparse, although Aronow (1978) has shown that non-smokers suffering from coronary heart disease are liable to experience angina more readily, when exposed to cigarette smoke, and Bocanegra and Espinosa (1980) observed that the symptoms of Raynaud's disease (spasm of the small arteries of the fingers) were relieved in two female non-smokers who were separated from their smoking husbands.

(ii) Models of Smoking Behaviour

It is clear from the foregoing account that continued efforts to help individuals cease (or, at least, reduce) their smoking behaviour are of paramount importance. The

most essential pre-requisite of any programme of treatment hoping to achieve (and maintain) this aim is that its rationale should be derived from a viable and comprehensive model of smoking behaviour.

Many models of smoking behaviour have been put forward, some emphasizing psychological, some sociological, and, more recently, pharmacological factors. The more comprehensive models take all of these factors into account. Lichtenstein and Danaher (1976) correctly remarked that psychological models were, at least then, "long on theory and short on data" (p.83), in contrast to pharmacological models, which were "data rich and perhaps theory poor". Some of the major models of smoking will now be briefly reviewed.

(a) The psychoanalytic model

Freud (1905) considered smoking to be basically sexual in nature, being related, dynamically, to oral behaviours such as thumb-sucking and suckling. Green (1923) saw smoking as having rather less obscure sexual connotations, saying that pipes were unconsciously identified with phalli. The relatively modern phenomenon of the woman smoker, although not commented on by either of the above theorists, would perhaps have been explained in terms of phallic symbolism and penis envy, although this is speculation. Bergler (1946), similarly, perceived smoking as being linked with libidinous oral gratification, this

perhaps being a less extreme view than that adopted by Brill (1922), who saw it as a substitute for masturbation.

The scientific validity of psychoanalytic explanations of smoking is questionable. McArthur, Waldron and Dickinson (1958) did produce some evidence that the ease of stopping smoking may be related to the individual's weaning history, but, even conceding the validity of the "oral personality", Howe and Summerfield (1979) were able to provide only minimal support for the hypothesis that smoking is associated with more general "orality".

There does seem to be a clear association between smoking and other oral behaviours such as alcohol and coffee consumption (Matarazzo and Saslow, 1960; Borgatta and Evans, 1968); however, this association can perhaps be more plausibly attributed to the fact that nicotine (see below), alcohol and coffee are all drugs which can affect the level of cortical arousal in some way and which may each therefore be used to effect desirable psychological changes in the same individual. This alternative explanation is consistent with the view of Stepney (1979), who has described smoking as a "psychological tool" and also with Eysenckian theory (Eysenck, 1973).

Psychoanalytic models of smoking behaviour are perhaps the best vindication of Lichtenstein and Danaher's description of psychological models as being theory-rich and data-poor.

(b) The "ethological" model

Morris (1977) views smoking as a "displacement activity", being analagous to animal behaviours such as scratching and pawing the ground. He sees smoking as the overt manifestation of "inner conflict", especially when the behaviour occurs when the organism is under stress. (There is considerable evidence that smoking frequency can be related to level of stress, as detailed in (c) below). Morris rightly describes smoking as being "much more than a question of inhaling smoke", this behaviour being only one of those constituting the act of cigarette smoking (other elements being taking the cigarette from the packet, lighting it, etc.).

Morris thus believes that smoking is essentially a stress-reduction mechanism. Some support has been afforded to this view by Schachter, et al (1977b) and by Comer and Creighton (1978), who found that smokers exposed to high levels of stress smoked more cigarettes and took more puffs, respectively.

More anecdotal, but perhaps more striking evidence

for the validity of construing smoking as a displacement or stress reducing activity is that the number of cigarettes consumed in Israel at the time of the Yom Kippur War (1973) increased markedly; reference to Figure 1:1 reveals peaks in the U.K. cigarette consumption at the times of World Wars I and II (although these apparent peaks may be artefacts due to the decrease in post-war smoking for economic, rather than psychological, reasons).

(c) Smoking as a means of arousal control

This model views smoking (or, more precisely, the absorbtion of nicotine into the body) as a behaviour leading to the individual's obtaining positive psychological effects. It has not been suggested that every cigarette smoked is "used" by the smoker in this way, but that occasional cigarettes are, and that operant learning principles (Skinner 1938, 1953), and especially intermittent schedules of reinforcement, are therefore implicated. Smoking is thus construed as being, essentially, an operantly conditioned and operantly maintained behaviour.

The beneficial psychological effects which are gained from smoking are apparently not uni-directional, where the level of CNS arousal is concerned. Somewhat paradoxically, nicotine as a drug can have both activating

and sedating properties, depending on dosage, (small doses being stimulating and large doses depressing), and it seems that an experienced smoker can regulate the intake of nicotine, without being conscious of this, to achieve the optimum benefit in a given situation. Mangan and Golding (1978) found, for example, that the proportion of EEG alpha rhythm was increased by smoking when subjects were stressed by bursts of white-noise, but was decreased by smoking in a situation of mild sensory deprivation. Ashton, et al, (1978), also found a dose-response relationship with nicotine. The implication here is that smoking can have a normalizing, regulatory function with regard to cerebral activation.

It will be recalled that, in describing the psychoanalytic model of smoking, an alternative explanation for the association between smoking and other "oral" behaviours was offered by Eysenckian theory. The view of smoking as being related to arousal control can also, clearly, be associated with certain aspects of Eysenck's theory of personality (Eysenck, 1947). Eysenck describes the extravert (who has a relatively low level of cortical arousal) as being stimulus-hungry, whereas the introvert is seen as stimulus-shy. Thus extraverts, it is suggested, will have a slower rate of nicotine intake, to increase their level of CNS activation, and introverts, conversely,

will strive for a larger, depressant dose (Eysenck, 1973). Evidence supporting this view is rather sparse, however, and further research is needed.

In conclusion, the model of smoking as a means of arousal control (or as a "psychological tool" (Stepney, 1979)) has certainly gained some scientific support, being based solidly on physiological phenomena, which are rather more easily observed and measured than are purely psychological variables. Interestingly, this is the only model of smoking which postulates that smokers actually have an advantage over non-smokers, the latter lacking an instrument to regulate their arousal level as necessary, in response to environmental demands.

(d) Smoking as an addictive behaviour

Dictionary definitions of the word "addiction" fail to differentiate the term adequately from the term "habit". A habit-forming drug is not necessarily needed by the regular user to satisfy a physiological dependence; rather, psychological needs are those that require fulfilment. In contrast, an addictive drug is one which leads to the user's being dependent through primarily pharmacological mechanisms.

The weight of evidence supporting the view that nicotine, the pharmacological agent contained in tobacco which is of the greatest psychological importance to the smoker

is, in fact, an addictive drug, has grown over the years. The fact that many smokers themselves recognize smoking as "a form of slow-motion suicide" (Stepney, 1979) but nevertheless persist with the behaviour, is evidence, albeit anecdotal, for the addictive nature of tobacco. A strong proponent of this model, Russell, has said that "if it were not for the nicotine in tobacco smoke people would be little more inclined to smoke cigarettes than they are to blow bubbles or light sparklers" (Russell, 1971, p.7). The same author goes so far as to say that "Cigarette smoking is probably the most addictive and dependence-producing form of object-specific self-administered gratification known to man" (Russell, 1974). What is the scientific evidence for the above view? The essential difference between the addiction model and the previously discussed arousal-control model is that the latter suggests that smoking occurs to obtain psychological benefits, whereas the former views smoking as a behaviour which prevents psychological distress, in the form of withdrawal symptoms. Thus one area in which controlled studies have been carried out relates to the aversive consequences of nicotine deprivation; if nicotine is a truly addictive drug, then it follows that the same, or similar, symptoms will be evident in different individuals undergoing deprivation and that a "withdrawal syndrome" should therefore be identifiable.

Several authors have found support for such a syndrome (Guilford, 1966; Wynder, et al, 1967; Horn, 1970). Brecher (1972) and Larson and Silvette (1971) described symptoms, following abstinence from nicotine, which included irritability, restlessness, poor concentration, lightheadedness, insomnia, tremor and increased hunger. At the physiological level, a decrease in heart-rate and blood-pressure have been noted (Knapp, Bliss and Wells, 1963), and similar findings were reported by Weybrew and Stark (1967), who demonstrated that both mood changes and physiological changes were reversed when smoking was resumed. Heimstra, Bancroft and DeKock (1967) found that, during a simulated driving task, deprived smokers made more errors in tracking and vigilence than did subjects who were permitted to smoke. Kleinman, Vaughn and Christ (1973) noted impaired paired-associate learning ability as a result of 24 hour abstinence from smoking. Finally, Shiffman and Jarvik (1976) found, using trend analysis procedures, "U-shaped" trends in both physiological and psychological symptom-clusters, as a function of days after smoking cessation, implying that the withdrawal symptoms first increased but then decreased in severity, over time.

In contrast to the above evidence for a clearly defined withdrawal syndrome, the Surgeon General's Report of 1964

(U.S.D.H.E.W. 1964) concluded that "no characteristic abstinence syndrome occurs" and that reports on the duration of symptoms were inconsistent, ranging from days to months. Although the evidence, on balance, seems to be in favour of the existence of a characteristic syndrome, it must nevertheless be acknowledged that scientific investigation limited to this area has tended to overlook (or ignore) the possibility that it may not be the physiological effects of nicotine that are missed by the incipient ex-smoker, but, instead, some, or all, of the discrete, psychological elements of smoking as a behaviour. Examples of these elements would be the sight of the familiar packet, lighting the cigarette, the smell, taste and sight of the smoke (the "sensorimotor ritual" to use Russell's words (Russell, 1980)) and the opportunity of satisfying oral and manual needs (see sections (a) and (b) above).

A second and perhaps more sophisticated way of examining the validity of a nicotine-addiction model of smoking is to look at the effects on smoking behaviour when nicotine intake is externally manipulated. Raw (1978) remarks that "smokers seem to smoke in such a way as to maintain a certain level of nicotine in the blood, and there is supporting evidence that intravenous injections of nicotine depress smoking", (eg. Armitage, 1973;

Jarvik, 1973). As well as lending support to the view of nicotine as being a drug upon which (the majority of) smokers are dependent, Raw's statement reflects the essential belief of those who support the notion of "nicotine regulation". This phenomenon, the investigation of which has led to some contradictory research findings, will be discussed in some detail below. A review of the research into this particular type of evidence for the addiction model of smoking would, at this point, therefore, be superfluous.

In conclusion, considerable evidence exists for construing smoking as being an addictive behaviour. However, it is unlikely (and, indeed, it would be naive to assume) that the difficulty experienced by the majority of smokers in abstaining is related purely to physiological factors. Raw (1977) has accurately pointed out that authors who see theories about the role of nicotine and of psychological factors as alternatives (Bernstein, 1969; Yates, 1975) would, perhaps, be better viewing them as being complementary to one-another. The present author agrees that a realistic model of smoking behaviour needs to take into account a wide range of factors.

(e) The affect-management model

Various elements of the previously mentioned models are incorporated in this model, which was proposed by

Tomkins (1966). It is suggested here that smoking only becomes a regular behaviour when it is reinforced by a desirable change in affect - either positive affect enhancement or negative affect reduction. An implication here is that there are different types of smokers, whose psychological benefits obtained from the behaviour differ. Ikard and Tomkins (1973) produced some tentative validity data in support of this model, but considerable overlap between types was apparent.

(f) The habit model

Hunt and Matarazzo (1970) suggested that smoking was a habit rather than an addiction. They defined habit as "a fixed behaviour pattern overlearned to the point of becoming automatic and marked by decreasing awareness and increasing dependency on secondary, rather than primary, reinforcement" (p.67). In contrast to model (e), above, Hunt and Matarazzo do not acknowledge the role of affect in smoking but see it as an operantly overlearned behaviour. Certainly, when one takes into account the sheer number of trials experienced by the regular smoker (a 20-a-day smoker will draw on a cigarette and inhale the smoke in excess of a million times over a ten-year period) this model seems to have good face-validity. However, it appears, as do many models, to be rather simplistic, ignoring cognitive, social and physiological variables.

(g) A behavioural contingency model

It is clear from the foregoing account of the major models of smoking behaviour which have been proposed over the years, that a degree of conceptual over-simplification has taken place. Smoking is a complex behaviour and it must be re-emphasized that an adequate model (which should lead logically to an equally adequate treatment approach) needs to be multi-faceted, acknowledging that smoking behaviour is maintained by multifarious factors.

Composite models have been described by Dunn (1973), Russell (1974) and Raw (1977), who see smoking as being initiated primarily by psychosocial reinforcers but being maintained by a learned dependence on nicotine. It can be argued that psychological factors are as significant (and perhaps even more so) than physiological factors in the maintenance of smoking, although there now seems to be little doubt that nicotine plays an important role.

Frederiksen and Simon (1979) have developed a comprehensive model based on the behavioural contingency (Kanfer and Phillips, 1970), this concept requiring a behaviour to be viewed both in terms of its antecedents, or precursors, and its consequences. Three separate response systems are suggested, relating respectively

to overt behaviours, covert (cognitive) behaviours and physiological behaviours. Thus, with regard to smoking, examples of overt antecedents would be buying a packet of cigarettes, taking one out of the packet and lighting it, whereas overt consequences may be extinguishing the cigarette, beginning a difficult task, switching out the light to go to sleep, and so forth. Covert antecedents may take the form of self-statements regarding the need for a cigarette or positive cognitions concerning smoking behaviour; covert consequences may be self-evaluative statements such as "I feel more relaxed now" or "I can make that 'phone call now". At the physiological level, antecedents to smoking behaviour will perhaps be a depleted nicotine level, an increased level of muscle tension or lowered blood-pressure, whereas physiological consequences will be a reversal of these states along with an increase in the level of certain substances in the body, such as carbon-monoxide and thiocyanate (see below).

In addition to considering the individual's behaviour, with respect to the above response systems, whilst smoking, it is also essential to pay attention to the environmental stimuli which are salient at the time. As a result of both operant and classical learning

mechanisms, the smoker will inevitably associate certain environmental events with smoking. Examples of such events would be watching T.V., interacting socially, being in the 'pub, driving or having eaten a meal. Frederiksen and Simon illustrate this model of smoking behaviour diagramatically, showing the inter-relationships between the "smoking event" per se, its antecedents and consequences and the concomitant situational events. For this purpose, the example of a cigarette smoked during a coffee-break is used. This illustration is reproduced in Fig. 1:2.

Frederiksen and Simon go on to point out that, using this model, "there are numerous points at which interventions can be aimed" and, moreover, that "interventions aimed at only a single component of this model are incomplete" (p.482). The present author agrees with this view and, as such, the present experiment is designed to utilize treatment techniques which are individually appropriate to different components of the above model. Evidence will be presented that treatment strategies derived from this type of comprehensive model of smoking behaviour are indeed effective.

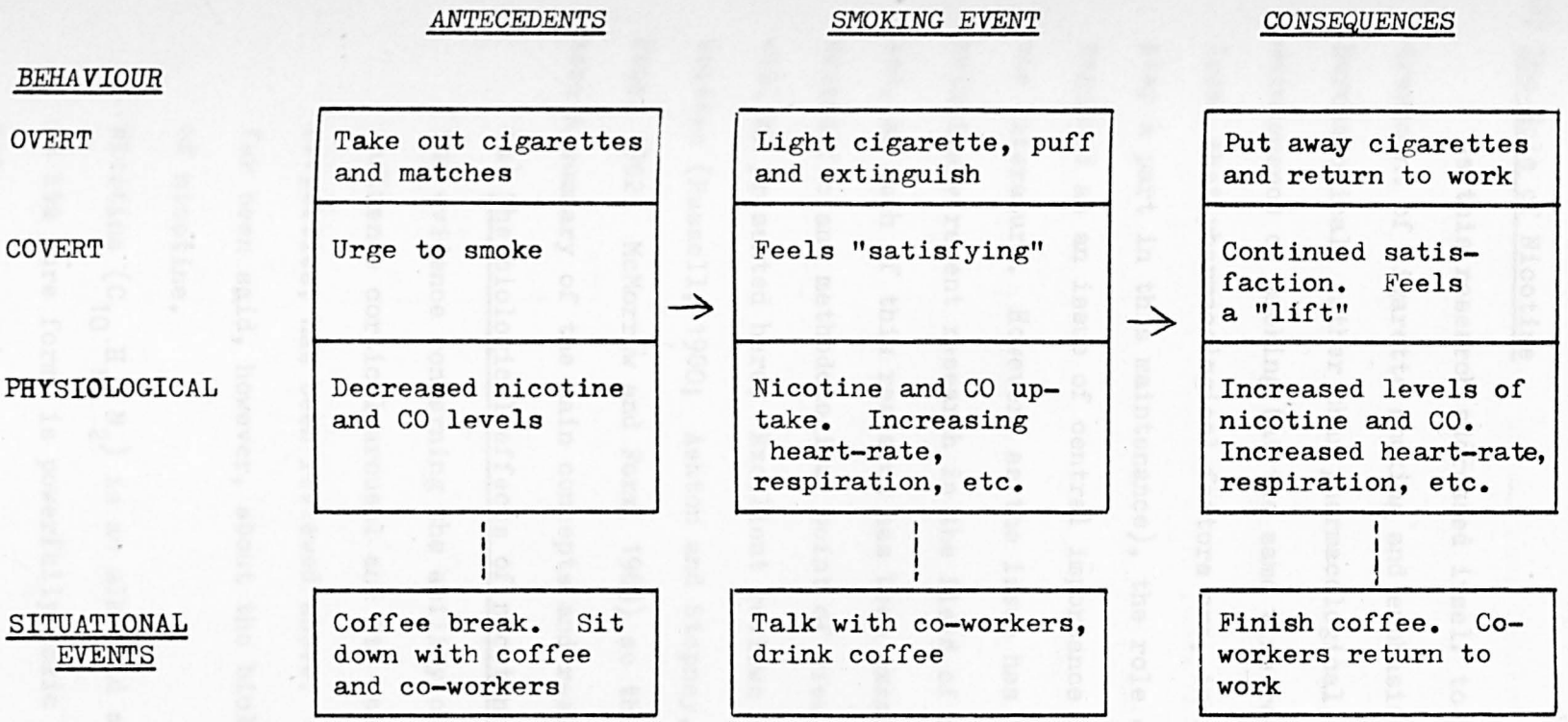

Fig. 1:2

An illustration of the behavioural contingency model (from Frederiksen L.W. and Simon S.J., 1979)

(iii) The Role of Nicotine

As this research addressed itself to the behavioural treatment of cigarette smoking and emphasized the importance of psychological, rather than pharmacological, factors in the maintenance of smoking (at the same time recognizing, nevertheless, that pharmacological factors are, in fact, likely to play a part in this maintenance), the role of nicotine is not regarded as an issue of central importance in this review of the literature. However, as the issue has been the subject of relatively recent research in the field of cigarette smoking and, as much of this research has been exemplary from a scientific and methodological point of view, a brief review will be presented here. Excellent reviews have already been written (Russell, 1980; Ashton and Stepney, 1982; Moss and Prue, 1982; McMorrow and Foxx, 1983) so this account will be more a summary of the main concepts and research findings

(a) The biological effects of nicotine

The evidence concerning the ability of nicotine to influence cortical arousal and its having addictive properties, has been reviewed above. Little has so far been said, however, about the biological effects of nicotine.

Nicotine (C_{10} H_{14} N_2) is an alkaloid substance, which, in its pure form, is powerfully toxic (Larson, Haag and Silvette, 1961). Upon combustion, nicotine clings to

particles of tar, which, when inhaled as smoke, reach the lung alveoli, whereupon the nicotine is absorbed rapidly into the bloodstream. After each inhalation of tobacco smoke, a "bolus" of nicotine reaches the brain within seven or eight seconds (Russell and Feyerabend, 1978); blood nicotine level peaks after a few minutes of smoking, having risen sharply, and then decreases rather more slowly. Successive cigarettes further increase the level of nicotine in blood plasma. This cumulative process is illustrated in Fig. 1:3. The diagram is a simplified adaptation of a Figure by Ashton and Stepney (1982).

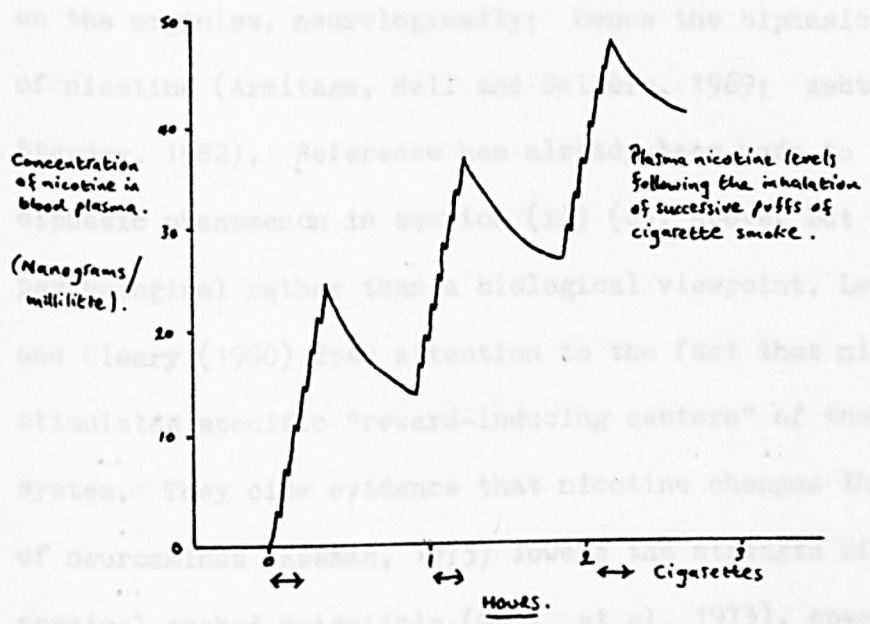

Fig.1:3 The absorption of nicotine into the bloodstream (adapted from Ashton, H. & Stepney, R. (1982)).

The primary pharmacological reason why cigarettes need to be smoked regularly over a given time period is that nicotine has a half-life of only 40-80 minutes (Russell and Feyerabend, 1978); blood nicotine level needs to be constantly replenished, in order to forestall the aversive consequences of not smoking.

Nicotine exerts both direct and indirect effects on nervous system activity. Its molecular structural similarity to acetylcholine (ACh), an important neuro-transmitter, allows nicotine to behave as a chemical with excitatory properties. However, as the "life" of nicotine molecules is longer than that of ACh molecules, larger doses of nicotine eventually block the synaptic receptor-sites, preventing the natural transmission of ACh, and this results in a depressant effect on the organism, neurologically; hence the biphasic effects of nicotine (Armitage, Hall and Sellers, 1969; Ashton and Stepney, 1982). Reference has already been made to this biphasic phenomenon in section (ii) (c), above, but from a psychological rather than a biological viewpoint. Leventhal and Cleary (1980) draw attention to the fact that nicotine stimulates specific "reward-inducing centers" of the nervous system. They cite evidence that nicotine changes the levels of neuroamines (Essman, 1973) lowers the strength of cortical evoked potentials (Hall, et al. 1973), speeds heart rate (Armitage, 1973) and acts on the inhibitory (Renshaw)

cells in the dorsal spinal column to produce muscular relaxation. Autonomic and somatic effects such as mentioned here can, it must be emphasized, be bi-directional, depending on the dosage of nicotine (this being determined by such variables as depth, duration and volume of smoke inhalation).

Russell (1980) listed some of the indirect biological effects of nicotine as follows : "increase in heart-rate and blood pressure, release of epinephrine from the adrenal medulla and 11-hydroxycorticosteroids from the adrenal cortex, increase in serum free fatty acids and triglycerides, inhibition of stomach contractions and gastric secretions, delay in the emptying time of the stomach, impairment of pyloric competence with increase in duodenogastric reflux, increase in the activity of the colon, inhibition of appetite, and an effect of reducing body weight by some process over and above the effect on appetite" (p.254-5). Russell lists these effects in the context of their relevance to psychosomatic problems. He goes on to quote evidence that nicotine influences hypothalamic electrical self-stimulating behaviour, (Domino, 1973) and thus has some influence on the hypothalamic reward-system; this is in agreement with Leventhal and Cleary's statement with regard to "reward-inducing" centres. In addition to the peripheral and central effects described, nicotine also possesses an ability to stimulate the release

of certain hormonal substances from the CNS (Husain, et al, 1975; Cryer, et al, 1976; Winternitz and Quillen, 1977). Growth hormone, cortisol, vasopressin and neurophysin are all produced at a greater rate as a result of smoking (the increase in cortisol being a result of increased quantities of adreno-corticotrophic hormone (ACTH)). The finding that the release of beta-endorphin is associated with that of ACTH (Jaffe, 1980) would seem to have important implications where the nature of nicotine as a primary reinforcer is concerned, endorphins being of some significance in relation to the "reward" or "pleasure" centres of the brain.

It can be seen, to conclude, that the biological effects of nicotine are widespread and numerous. It should be clear, from this brief account, that nicotine is a potent drug, and that the diversity of its somatic effects, many of which are perceived as pleasurable by the smoker, lends itself to the development of a withdrawal state, upon cessation of smoking. As mentioned previously, effective treatment approaches need to take into account the physiological aspects of nicotine-withdrawal.

(b) Research on nicotine regulation

A concept of increasing interest over recent years has been that of nicotine regulation. The essence of this concept is that a smoker will maintain a characteristic level of plasma nicotine, by altering his smoking behaviour, if the availability of nicotine is externally manipulated. Regulation may be upwards or downwards, depending on whether nicotine is

made either less or more available, respectively. The existence of regulation has been assumed to support the premise that smoking is an addictive behaviour.

Some terminological confusion has occurred in this area of study. Some authors have used the terms "nicotine regulation" and "nicotine titration" synonymously (Ashton, Stepney and Thompson, 1979; Moss and Prue, 1982) whereas others (McMorrow and Foxx, 1983) have deliberately adhered to one term alone, to avoid ambiguity. Although both "regulation" and "titration" are appropriate terms, "regulation" is perhaps more descriptive and will therefore be used in this account. Further confusion has arisen from the synonymous use of "nicotine regulation" and "nicotine compensation". McMorrow and Foxx (1983) have drawn attention to the obfuscation of the significance of the results of various studies, as a result of the failure, in many cases, to clarify which of these two processes was implicated. These authors maintain that "compensation" should refer, not to the biochemical process of regulation, but to the changes which may occur in smoking behaviour (topographically), as a result of modified nicotine availability. Thus, compensation may, or may not, be a means of regulation; it is not tantamount to regulation.

The experimental investigation of nicotine regulation has taken a number of forms. McMorrow and Foxx (1983) have categorized the various manipulation procedures, which have been used in these investigations, as follows :-

(i) nicotine "preloads"

(ii) nicotine substitutes

(iii) shortened cigarettes

(iv) brand-switching

and (v) ventilation/ dilution of cigarette smoke

Moss and Prue (1982) have defined a separate category, namely,

(vi) indirect nicotine manipulation

The experimental evidence for and against the concept of nicotine regulation will be discussed using these categories as a framework

(i) Nicotine "preloads"

Johnston (1942) conducted the earliest experiment related to the regulation of nicotine. Intravenous injection of nicotine was found to decrease the desire to smoke, resulting in a depressed smoking frequency. Although this study lacked methodological sophistication (it was not, for example, a 'double-blind' experiment), some support was lent to the hypothesis that, if the body level of nicotine is "artificially" increased, smoking frequency will decrease as a result. Johnston's study is the only one of its kind; other workers (see below) have since used intravenously administered nicotine to investigate nicotine regulation, but as a substitute, rather than a "preload".

(ii) Nicotine substitutes

Lucchesi, Schuster and Emley (1967) observed a 30% decrease in the number of cigarettes smoked in a group of smokers who were given an intravenous infusion of nicotine of 22 mg., over a period of six hours. A control group, receiving intravenous saline, showed no significant decrease. The subjects were, in

this experiment, unaware of the phenomenon under investigation. The same authors obtained similar results in a separate experiment, in which the injection of nicotine was staggered, in an attempt to reproduce the effects of smoking a number of cigarettes. This research supported the regulation hypotheses. Kumar, et al (1977), however, attempting to mimic the effects of "real" smoking by more discrete injections of nicotine (introduced into the body once a minute, for a period of five seconds), failed to find support for the nicotine regulation hypothesis, no significant decline in smoking frequency being observed. However, the subjects in this experiment were not allowed to smoke naturally; rather, they smoked through a cigarette holder and were not required to light the cigarettes themselves - a somewhat unfamiliar way of smoking. This factor perhaps weakens the validity of the conclusions drawn.

An advantage of providing a nicotine substitute via injection is that, as with smoke inhalation, the pharmacological effects are relatively immediate; this similarity enables comparisons to be more easily made. However, injections are, of course, intrusive and can only be administered in a laboratory setting. The emphasis has therefore been placed, in attempts to verify the nicotine regulation hypothesis through using nicotine substitution techniques, on the oral administration of nicotine. Although this method of administration is dissimilar to cigarette smoking in terms of immediacy of effect, it is a less intrusive/obtrusive method and smoking behaviour can, as a consequence, be observed in more natural environments.

As with nicotine injections, the hypothesis is that, if a smoker

is administered nicotine orally (nicotine tartrate contained in tablets or chewing-gum), his need to smoke to obtain nicotine will decrease. Regulation should be evident (at least by inference) by the occurence of compensatory behaviours, (such as less cigarettes being consumed, longer inter-puff intervals, less deep inhalation, etc.) Support has been lent to this hypothesis by Jarvik, Glick and Nakamura (1970) (the support here being minimal), Kozlowski, et al, (1975) and Russell, et al, (1976). It must be re-emphasized, however, that the absorption of nicotine orally is not analagous to absorption through inhalation, so the conclusions which can be drawn from these studies are tentative.

(iii) Shortened cigarettes and (iv) Brand-switching

As both these methods of investigation entail the subject's smoking modified cigarettes, this is perhaps the most accurate and "natural" means of attempting to confirm the nicotine regulation hypothesis. Russell, et al (1975), Sutton, et al (1978), Ashton, Stepney and Thompson (1979), have all shown that, if smokers switch to a brand of cigarettes yielding less nicotine than their usual brand, compensatory behaviours occur. (Conversely, the latter authors also demonstrated that compensation of an opposite nature (i.e. smoking less cigarettes, etc.) occurred when the new brand had a higher nicotine yield). Similarly, Schachter (1977) found that "heavy" smokers smoked 25.3% more cigarettes and light-smokers 17.5% more, when the level of nicotine in their cigarettes was lowered. However, this study

may be criticized in that adequate pre-intervention baseline measures were not taken. The study by Ashton and her colleagues did not have this shortcoming and was, in fact, methodologically very sound, the only criticism perhaps being the short duration of the experiment (three days).

On the whole, the studies mentioned here, and others (eg. Frith, 1971), have produced results which uphold the regulation hypothesis.

(v) Ventilation/Dilution of cigarette smoke

Again, nicotine regulation seems to have taken place in experiments which have held constant the brand of cigarette smoked, at the same time as artificially decreasing nicotine yield. Ashton, et al, (1970), used high retention filters, Freedman and Fletcher (1976) employed nicotine-free additives and Sutton, et al (1978) altered yield by utilizing special cigarette holders; all of these researchers observed an increase in smoking behaviour (ie. compensation).

(vi) Indirect nicotine manipulation

To date, only two studies have attempted to demonstrate the existence of the regulation phenomenon by the indirect manipulation of nicotine level in the body. Stoleman, et al (1973) observed that cigarette consumption and puff-frequency both increased following the administration of mecamylamine, a nicotine-antagonist, (although there were some contradictory findings amongst the results obtained). Schachter, Silverstein and Perlick (1977) found that smoking increased as a result of stress and attributed this to the earlier finding (Schachter, Koslowski and Silverstein, 1977) that stress increases urine pH, which in turn

leads to increased nicotine excretion; this excreted nicotine therefore needs to be replaced.

Thus, taken as a whole, the research findings tend to lend credence to the nicotine regulation hypothesis. Moss and Prue (1982) and McMorrow and Foxx (1983) commented on a number of deficiencies in this field of enquiry, which may limit the generalizability of the results from the laboratory to "applied" settings. On closer inspection, the evidence produced needs to be viewed with some caution.

The comments made by these reviewers are lengthy and detailed and it is not appropriate to re-iterate these here in full. In summary, however, the main criticisms are as follows :-

(i) Baseline data has generally been inadequate. When accurate baselines have been obtained, less support has been offered to the regulation hypothesis (eg. Goldfarb, et al, 1970).

(ii) Studies have typically been of short duration. Longer term studies (eg. thirty days plus - Jaffe , et al, 1981; Martin, et al, 1981) have failed to support the hypothesis.

(iii) Only one study (Forbes, et al, 1976) matched groups on baseline nicotine consumption. This study also failed to support the hypothesis.

(iv) Direct measurement of nicotine levels has been lacking; levels have often been inferred from, for example, carboxy-haemoglobin (COHb) levels in blood samples (Hill and Marquardt, 1980) or thiocyanate levels in blood, urine or saliva samples (Prue, et al, 1981). (See section on assessment and measurement, below).

(v) With regard to the studies utilizing cigarette manipulation procedures, little attention has been paid to holding constant variables such as taste, ease of draw, appearance, and so forth, whilst varying nicotine yield. Such factors obviously have an impact on the smoker's desire to smoke.

(vi) Studies which have used direct biochemical measures have often relied on a single (perhaps unrepresentative) sample; the mean of a number of samples over a period of time would lead to more accurate measures.

(vii) There is still some uncertainty as to whether biochemical measures can, in fact, reliably distinguish smokers from non-smokers (Butts, Kuehnemann and Widdowson, 1974; Paxton and Bernacca, 1979). Valid and reliable measurement is a quint-essential element in research of the kind in question.

In addition to these comments, it is necessary to bear in mind the effects of individual subject characteristics on the results of experiments of this nature (for example, motivation, type of smoker (Tomkins, 1966; Ikard, et al, 1969) and individual response to the demand characteristics of the experiment), and, finally, the fact that, even in well-designed experiments, only limited support has usually been provided for the nicotine regulation hypothesis. To illustrate this point, which is of paramount importance, Schachter (1977) found that a 77% reduction in nicotine level produced only a 17-25% increase in cigarette consumption. The hypothesis of regulation is thus only partially sustained, here, and, again, this is a typical experimental result.

It can clearly be seen, therefore, that, at the present time, the evidence for nicotine regulation is by no means conclusive. Further, methodologically-sound research is needed before any firm conclusions concerning the process of regulation can be reached.

Finally, with regard to the concept of nicotine regulation, the implications with respect to health-risks warrant some discussion. If regulation does, indeed, occur when smokers switch to brands containing less nicotine, or if the number of cigarettes smoked is reduced, then, any argument that changing smoking behaviour in such a way should lead to a decreased health risk is without foundation. As nicotine and tar content of cigarettes are highly correlated ($r = 0.96$) (Goldfarb et al, 1976), nicotine regulation would lead to incidental tar regulation; additionally, compensatory behaviours would also lead, logically, to an increase in exposure to carbon monoxide, hydrogen cyanide and nitric oxide (see earlier section - Health Risks of Smoking), the harmful gases contained in tobacco smoke. Intervention goals such as reduced or controlled smoking (Frederiksen, Peterson and Murphy, 1976; Frederiksen, 1979; Colletti, Supnick and Rizzo, 1982; Foxx and Axelroth, 1983; Glasgow, Klesges and Vasey, 1983) would therefore be unacceptable from a health-risk perspective.

However, as described above, the evidence that nicotine regulation does occur is not conclusive and, moreover, several other factors need to be taken into account in determining whether smoking reduction is a viable alternative to cessation. As mentioned previously, work on regulation has taken place in laboratory rather than "applied" settings; there is some evidence from more applied studies that smokers can, in time, adapt to lower-

nicotine cigarettes (and thus, presumably, to a lower rate of smoking), (Cherry and Forbes, 1972; Freedman and Fletcher, 1976). Again, regulation studies have tended to be of relatively short duration. Further, the likelihood of nicotine regulation's occurring may be minimized by instructing smokers how to smoke to avoid upward compensatory behaviours; as far as the present author is aware, the effects of such experimental instructions have not yet been investigated. Finally, Foxx and Brown (1979) found that subjects maintained low-nicotine cigarette smoking at 2½ year follow-up and that rate did not increase significantly from baseline, as a consequence; changes in other topographical variables were, however, not monitored.

Thus, in summary, there is not yet sufficient evidence available to preclude treatment efforts which aim to establish a lower level of smoking in subjects, as an alternative to total abstinence. It will be seen, from this review, that, indeed, reduced smoking may, in the long term, be a more realistic goal than abstinence, in view of the traditionally high relapse-rate of those who choose the latter goal.

c) Assessment and Measurement

Of the characteristics which distinguish the behavioural approach from other psychological intervention strategies, perhaps the most important is the emphasis which is placed upon accurate measurement of the particular behaviour concerned. To be able to determine whether a particular mode of intervention has been effective in modifying a behaviour, precise and reliable assessment of this behaviour is of vital importance.

This principle applies to the assessment of smoking behaviour no less than any other behaviour: the effectiveness of any treatment method can only be evaluated if accurate measurement techniques are used.

A number of pertinent issues will be discussed in this review, namely: the measurement of abstinence (i.e. the percentage of subjects who have stopped smoking) versus the measurement of smoking rate (i.e. the percentage of baseline smoking level); the measurement of smoking behaviour, per se; the use of self-monitoring as an assessment technique, with special reference to the phenomenon of reactivity; and the use of indirect, physiological measures as a means of validating self-report data.

(i) Abstinence vs. Rate

It will be seen from the review of treatment methods, in a later section, that smokers who do not achieve the goal of abstinence have tended generally to return to their baseline levels of smoking within a relatively short period of time. (Exceptions will, however, be pointed out). Consequently, it could be argued that abstinence measures (eg. the percentage of a group of smokers who stop smoking completely) are the only ones of any value in this field of research. However, any treatment programme which is able to justify a goal of reduced smoking will obviously find this type of measure inappropriate and, in such a case, the rate of smoking (eg. the number of cigarettes smoked per day) is the only alternative kind of measurement.

Where a decision needs to be made as to which of the above measures to employ, in any particular treatment programme where a choice is available, several factors need to be taken into account. First, abstinence tends to be less susceptible to the reactivity effects of self-monitoring (Kazdin, 1974) (see below) than does smoking rate. Secondly, abstinence can more easily be confirmed by observers than can a reduction in rate (Schmahl, Lichtenstein and Harris, 1972; Lichtenstein et al, 1973) and is also more

easily detected through the use of biochemical validation techniques (see section (iv) below). Thirdly, however, a marked disadvantage of using abstinence measures in research is that, being a nominal scale datum, less powerful, non-parametric statistical analyses are required and statistical differences are therefore less likely to be yielded than when rate is used in assessment (parametric methods being necessary in the latter case), (Lichtenstein et al, 1973; Lichtenstein and Danaher, 1976; Pechacek, 1979). Abstinence is therefore a less sensitive indicator of differential treatment effects.

On balance, although total abstinence from smoking is obviously the more desirable goal, where appropriate, rate is the datum of choice as long as reactivity effects are, as far as possible, controlled for and biochemical validity measures are taken to corroborate rate reports. Some authors, however, (eg. Raw, personal communication, 1981) would disagree with this contention, believing that rate is an inappropriate dependent variable.

A recent trend in the literature (eg. Colletti and Stern, 1980) has been to report both abstinence and rate data wherever that is possible, and the present author agrees with this practice.

(ii) <u>The Measurement of Smoking Behaviour, per se</u>.

The most widely used method of assessing smoking behaviour, or, more precisely, smoking frequency, has been that of self-monitoring (McFall, 1978; Frederiksen, Martin and Webster, 1979; Frederiksen and Simon, 1979; Merbaum and Rosenbaum, 1980). This method warrants separate discussion and this will be the purpose of section (iii) below.

The simplest method of "measurement" is the self-report questionnaire

Eiser and Sutton (1977) have used this in survey research and McFall and Hammen (1971) as a means of evaluating the effects of other measurement techniques. The questionnaire has also been used as an easy-to-administer follow-up technique (Delahunt and Curran, 1976). This method's simplicity is appealing, but it's limitations are severe: reported data is retrospective, and therefore unlikely to be accurate and such self-reports are open to deliberate falsification.

Laboratory measurement of smoking behaviour is a poor alternative to self-reporting by questionnaire. Observation of smoking under controlled conditions is undoubtedly a more scientific approach, but the representativeness and generalizability of results obtained in such a way would be questionable. (Reference may be made here to the criticisms which have been levelled at nicotine regulation research methodology; a similar lack of generalizability to the "natural" environment is, it will be remembered, a major deficit of this research).

A further, somewhat more efficient, technique of measurement is to use external monitoring of some kind. Azrin and Powell (1968) used an ingenious method of counting the number of cigarettes smoked; they designed a cigarette case which recorded the number of times it was opened. However, subjects were less than honest in this experiment, taking more than one cigarette at a time from the case, accepting cigarettes from other people, or simply not using the case. Powell and Azrin (1968), in another external-monitoring investigation, used "participant observers". This involved the subjects' designating some individual in their environment (for example, a spouse) as the observer, the task

of whom was to report on the number of cigarettes smoked. Again, however, this method was fraught with difficulties; for example, the observers were unable to observe the subject continuously and they too, were open to bias and distortion in their reporting. It has therefore been suggested that this type of measurement is used only to corroborate data obtained in other ways (Schmahl, Lichtenstein and Harris, 1972; Best, 1975; Frederiksen et al, 1975); Frederiksen and Simon (1978b), in this respect, employed highly skilled "professional staff" as corroborators, whereas Best (1975), Katz et al (1977) and Lando (1977) used minimally trained friends of the subjects.

A more accurate method of measurement is that of unobtrusive, naturalistic observation. With external-monitoring, the subject may, or may not, be aware that he/she is being observed. With unobtrusive measurement, this eventuality is precluded. The confounding effect of reactivity (see below) is removed, and this is an advantage of using such methods. However, as indirect assessment is necessary (eg. counting used cigarette butts (Auger, Wright and Simpson, 1972)), the accuracy of such an approach is again questionable. Webb et al (1966) suggested a number of other unobtrusive methods that may be used in assessment. Examples are looking for stains on the fingers, smelling breath or clothing and searching for cigarette packets or other smoking paraphernalia (matches, cigarette lighter, etc.) in the individual's environment. It is clear that such techniques are, again, unlikely to yield highly accurate measures of smoking behaviour.

(iii) Self-monitoring and reactivity

As indicated at the beginning of the previous section, self-monitoring has been the most widely used method of assessment in the field of smoking research. In a review of forty behavioural studies on smoking reduction, Frederiksen and Simon note that 95% used some form of self-report procedure, a large proportion (83%) of this percentage being accounted for by self-monitoring methods.

Frederiksen, Martin and Webster (1979) draw attention to the fact that "self-monitoring" is a complex activity, comprising two separate steps: first, the discrimination of the occurrence (or incipient occurrence) of the behaviour in question and, secondly, the recording of this behaviour in some way. Measurement error, these authors correctly point out, can enter into either (or both) of these two stages. Frederiksen and his colleagues listed a number of dimensions on which recording procedures can vary and cited studies to exemplify these variations. The most significant variables described are : (a) the nature of the information recorded, e.g., number of cigarettes smoked (Barton and Barton, 1978), time of each cigarette (Frederiksen and Frazier, 1977; McGrath and Hall, 1976), and situational factors associated with smoking (Brockway et al, 1977; Dericco, Brigham and Garlington, 1977; Epstein and Collins, 1977); (b) the nature of the recording device, e.g., wrist-counters (Katz, Heiman and Gordon, 1977), pocket-counters (Levinson et al, 1971), index cards (Gordon and Hall, 1973), booklets (Lando, 1975) and slips attached to cigarette packets (Brockway et al, 1977); (c) the timing of the recording, e.g., before lighting the cigarette (Rozensky, 1974; Frederiksen, Epstein and Kosevsky, 1975);

(d) the schedule of recording, e.g., continuous, daily or weekly (Frederiksen et al, 1975); and (e) the schedule of returning data to the experimenter, e.g., daily (Frederiksen and Simon, 1978a), weekly (Danaher, 1977) or longer (Norton and Barske, 1977). Frederiksen, Martin and Webster go on to say that, taking into account the variations which are possible in methods of self-monitoring, "(there exists) much potential for differential control of self-monitoring accuracy ..... ". (p. 656)

It is clear from the above account that self-monitoring is an extremely flexible method of assessment and places little demand on the time of the experimenter. However, there is a large demand on subjects and the use of self-monitoring in trials may lead to inaccurate recording, or, at worst, a high subject attrition-rate. (This latter factor is, perhaps, the bête-noir of smoking research and deserves separate discussion later).

With regard to the accuracy/reliability of self-monitoring procedures, McFall (1970) found only a 0.61 index of reliability between self-monitored and observer reports. In contrast, Frederiksen, Epstein and Kosevsky (1975) and Epstein and Collins (1977) reported reliabilities of 0.85+.

Two of the above examples of the variables related to self-monitoring merit elaboration, as they are of particular theoretical and practical interest. Rozensky's (1974) study, with respect to the timing of the recording, revealed that the reactivity effect was greater (i.e., smoking decreased more) when the subject completed the record before the smoking event, in contrast to post-smoking recording.

Kazdin (1974) regarded the recording of an undesirable behaviour such as smoking as an aversive event and, conversely, "non-recording" behaviour as a positive event. Thus, the requirements of recording early in the response-chain of smoking would be more likely to inhibit the behaviour of smoking, whereas, in effect, post-smoking recording occurs too late in the chain to influence the behaviour. In a similar vein, McFall (1970) found that self-monitoring cigarettes smoked led to an increase in smoking, whereas self-monitoring resistance to urges to smoke decreased the level of smoking.

Frederiksen et al's study (1975) compared continuous with intermittent monitoring and found that continuous recording resulted in greater smoking reduction than did intermittent monitoring.

To re-iterate, self-monitoring is not a simple concept, nor is it a unitary method of assessment. It can take many forms and further research is necessary to clarify which combination of the variables mentioned leads to the higher degree of accuracy in assessment.

The more detailed account of Rozensky's and Frederiksen et al's experiments implicate the phenonemon of reactivity and this will now be discussed at greater length.

McFall defines reactivity as "... the tendency for certain experimental measurement operations to function as an unintended independent source of influence on the behaviors being measured". (McFall, 1970, p.135). With his colleague, Constance Hammen (McFall and Hammen, 1971) McFall noted that, despite giving specific instructions to a group of smokers not to alter their smoking behaviour during a 96 hour baseline period, a

significant ($p<0.01$) decrease in smoking did occur. The correlation between S's original estimates of their smoking rate and their observed rate was low ($r = 0.55$). However, this low correlation could, perhaps, in part, at least, have been due to the subjects' inaccurate estimates of their original rate.

With regard to smoking behaviour, reactivity can, according to the exact behaviour being monitored and recorded, exert its influence bi-directionally. As mentioned earlier, McFall (1970) found that self-monitoring cigarettes smoked led to an increase in smoking, but self-monitoring resistance to urges to smoke led to a decrease. Interestingly, this effect was not apparent in McFall and Hammen's study; here, subjects who monitored "negatively", as well as those who monitored "positively", smoked less. Other factors, such as motivation and demand characteristics are, almost certainly, implicated here, and these will be discussed at a later point in this review. Suffice it to remark here that motivation to reduce smoking or to comply with the experimenter's (implied) wishes is a significant factor in determining the strength of reactive behaviour; although, like McFall (1970) and McFall and Hammen (1971), a number of researchers have reported decreased smoking rates as a result of self-monitoring,(Lawson and May, 1970; Rozensky, 1974; Frederiksen et al, 1975), Epstein and Collins (1977) found that, with non-motivated subjects, reactive effects were minimized. Lipinski et al (1975) compared motivated and non-motivated subjects who self-monitored and only the motivated subjects decreased their rate of smoking. Nelson reviewed this area a year later (Nelson, 1977) and she concluded that cognitive-motivational factors are

of considerable importance where reactivity is concerned. It has been noted here that self-monitoring smoking behaviour (as opposed to "non-smoking" behaviour) has been found to lead to an increase in smoking rate and, in contrast, that self-monitoring either of these two behaviours has decreased smoking (McFall and Hammen, 1971). Kantorowitz, Walters and Pezdek (1978) also found that negative self-monitoring as well as positive self-monitoring reduced subjects' initial smoking rates. These contrasting findings suggest that further research is needed in this area. One final comment on reactivity: where testing the effectiveness of treatment procedures is concerned, reactivity need not be seen as a confounding variable. McFall (1970) clarifies this point succinctly: " ... it is not vital that undistorted base-rate data be obtained; rather, it is only necessary that the data be stable and that such base-rates be sensitive to subsequent experimental interventions" (p.141).

(iv) Physiological correlates of smoking behaviour

Assessment techniques such as external-monitoring, direct observation and self-monitoring clearly cannot be regarded as fully reliable means of measuring smoking behaviour. Although some authors would disagree (Schinke, Blythe and Dolleck (1978), for example, maintaining that the close correspondence between self-report and objective measures of smoking obviates the need for the latter) the most widely held view is that some type of objective measurement is essential, in order to check the reliability of other measures. Further, measurement of the physiological correlates of smoking behaviour is of vital importance from the health standpoint, as a measure of rate

alone may not be sufficient to establish whether the "reduced" smoker is, in fact, any healthier than formerly. Protagonists of the nicotine regulation hypothesis would see some form of biochemical index of smoking behaviour as essential, as part of assessment.

Frederiksen and Simon (1979) have suggested classifying physiological measures as either "molecular" or "molar". Molecular changes in body chemistry tend to be of short duration and therefore relate to recent smoking history. Such measures, therefore, are of value in establishing the reliability ofless objective measures, as well as being significant in their own right. Molar measures, on the other hand, refer to gross body changes and are related more to long-term smoking; these cannot be used as reliability checks, but are of undeniable importance where the physical health of the individual smoker is concerned. We are concerned here more with the former type of measure; however, as the present study assessed subjects' pulmonary function and gross body weight, in an attempt to detect any relatively long-term changes, these two molar functions will be discussed in (b), below.

(a) Physiological measurement at the molecular level

(i) Nicotine

The measurement of blood nicotine levels has been found to corroborate self-reported smoking (Falkman et al, 1975; Russell, Feyerabend and Cole, 1976). However, this method is intrusive and expensive (Lichtenstein, 1982). Urinary nicotine analysis does not have the former drawbacks and has been found to discriminate successfully between smokers and non-smokers (Paxton and Bernacca, 1979); it is still, however, an expensive method

requiring the use of sophisticated laboratory equipment, and is, therefore, not the assessment technique of choice.

(ii) Alveolar Carbon Monoxide (CO_a)

Exhaled alveolar air can be analysed to yield a carbon monoxide level, which is raised as a result of cigarette smoking (Jones et al, 1958; Horan, Hackett and Linburg, 1978). Again, this method has been reported to discriminate between smokers and non-smokers (Ringold et al, 1962; Goldsmith, Terzaghi and Hackney, 1963; Lando, 1975a; Rawbone, Coppin and Guz, 1976) but when applied to smokers only, the correlation between smoking rate and CO has tended to be only moderate ($r = 0.59$, Lando, 1975 .; $r = 0.48$, Vogt et al, 1977). The measure lacks reliability in that CO_a levels can be increased through factors other than smoking (e.g., exposure to road-traffic exhaust fumes); its main advantage, however, is its lack of intrusiveness and ease of analysis. The use of CO_a as a measure of smoking behaviour is, therefore, well suited to large scale, epidemiological surveys, perhaps better than to scientific research studies (Vogt et al, 1977). Research on CO_a measurement has been reviewed by Frederiksen and Martin (1979).

(iii) Carboxyhaemoglobin (COHb)

This substance, which is the result of carbon-monoxides being absorbed into the bloodstream and binding with oxygen, has been found to be a relatively reliable indicator of the subject's smoking behaviour (Castledon and Cole, 1974; Wald et al, 1975; Brockway, 1978; Dawley, Ellithorpe and Tretola, 1976). The drawbacks of the intrusiveness of taking blood samples and the liability of COHb levels' being affected by sources of CO other

than smoking, however, apply here; additionally, COHb has a relatively short half-life, generally estimated to be between two and five hours; thus, smokers who had abstained from cigarettes for this length of time would be assessed as being non-smokers (i.e., the number of false-negatives in assessment is high).

(iv) Thiocyanate (CN)

Thiocyanate is a metabolite of cyanide and the end-product of the body's detoxification of cyanide compounds (Boxer and Rickards, 1952; Pettigrew and Fell, 1972; Brockway, 1978). As one of the gases produced by the combustion of tobacco is hydrogen cyanide, the inhalation of tobacco smoke inflates the natural level of CN in the body. Courant (1967) pointed out that moderate levels of CN in the body appear to serve necessary biological functions (such as the prevention of oral cavity disease) and that cyanide compounds are available in foods such as broccoli, garlic , cabbage, turnips and horseradish. However, excessive levels, such as caused by cigarette smoking, have been found to be related to stomach cancer (Lederer, 1976) and hydrogen cyanide itself has been cited as the primary ciliatoxic agent in tobacco smoke (USDHEW, 1979).

There is little doubt that serum thiocyanate (SCN) levels in blood samples can distinguish between smokers and non-smokers (Butts, Keuhnemann and Widdowson, 1974; Vogt, 1977). Moreover, significant positive correlations have been established between SCN levels and reported rate of smoking ($r = 0.46$, Butts et al, 1974; $r = 0.48$, Vogt et al, 1977; $r = 0.54$, in the present study). Despite the disadvantage of the intrusiveness of sampling, SCN

as a measure has several distinct assets: analysis is relatively easy (Butts et al, 1974); samples can be frozen and stored; and the half-life of SCN in the body is far longer than that of CO or COHb (ten to fourteen days) (Densen et al, 1967).

An alternative method of CN analysis has been the use of urine and saliva samples, rather than blood samples and this, too, has been found to correlate positively with smoking rate reported (Maliszewski and Buss, 1955; Barylko-Pikielna and Pangborn, 1968). The higher sensitivity of these measures is advantageous, in that smokers and non-smokers may more easily be discriminated, but problematic in that urine and saliva samples are more easily contaminated by thiocyanate absorbed gastrically, as a result of eating the high-cyanide content foods mentioned above.

In conclusion, therefore, although Prue, Martin and Hume (1980) suggest in their review that saliva CN samples are the measure of choice, there does appear to be rather more evidence in favour of using blood-samples.

(b) Physiological measurement at the molar level

(i) Pulmonary function

Considerable evidence has accrued to show that pulmonary functions deteriorate as a function of prolonged smoking (Peterson, Lonergen and Hardinage, 1968) and, conversely, that lung function improves with reduced smoking (Bode et al, 1975; Gordon et al, 1975; Buist et al, 1976; McCarthy, Craig and Cherniak, 1976).

Using a Vitalograph Spirometer (Drew and Hughes, 1969) to assess lung-function produces three data : a) FEV_1 (forced-expiratory

volume) - the volume of air which can be exhaled in one second; b) FVC (forced vital capacity) - the volume of air which can be contained in the lungs; and c) FEV_1/FVC - the ratio between these two measures. Norms are available, which take into account an individual subject's height and age (factors which affect pulmonary function); current status and changes over time are, therefore, easily identified.

Paxton and Scott (1981) have also demonstrated improved lung function as a result of cessation of smoking (for two months) (These authors, incidentally, suggested using feedback of improvements in function, specifically, FEV_1, as a reinforcer of continued non-smoking behaviour).

A separate type of pulmonary measure which has been found to be related to cigarette smoking (Cotes, 1975) is the carbon monoxide "transfer factor" (TF). This index is a function of both the diffusing capacity of the alveolar capillary membrane and the rate at which carbon monoxide combines with haemoglobin, in the alveolar capillaries to produce COHb (see above).

(ii) Gross body weight

Cross-sectional studies have shown that smokers, in general, weigh less than do non-smokers (Karvonen et al, 1959; Bjelke, 1971; Khosla and Lowe, 1971; Goldbourt and Medalie, 1977). Whether this is because smokers have different eating habits (Birch, 1975) or because their metabolic activity differs in some way (Comroe, 1960) is still an unclear issue. Lincoln (1969) found that "heavy" smokers (undefined) in fact consumed about 575 calories a day more than non-smokers, suggesting the involvement of metabolic factors. Moreover, when smokers and

non-smokers were matched on calorie consumption, as well as on height and age, smokers were found to weigh 2.9 kg less than non-smokers.

Animal studies attempting to elucidate the reasons for this phenomenon have been typically inconclusive (Münster and Bättig, 1975; Schechter and Cook, 1976). Human studies, on balance, tend to favour metabolic theories (Lincoln, 1969; Gaudet and Hugli 1969), at least when "naturalistic" observations are made. Laboratory studies, however, have identified food consumption differences as being of relevance. For example, Grunberg (1982) demonstrated that nicotine administration (in animals) and cigarette smoking (in humans) were accompanied by a decreased consumption of sweet-tasting, high caloric foods, whereas consumption of other foods did not change.

Of more relevance to the present study, longitudinal studies have shown, unequivocally, that smokers who stop smoking increase in weight. Brožek and Keys (1957) observed an average 8.2 lb increase in weight in subjects who had stopped smoking, over a period of five years. (The control group, who were matched on important variables, decreased, nonsignificantly, in weight). Glauser et al (1970) found that a group of subjects who stopped smoking increased in weight by an average of 11.4 lb., in a period of one month. They also noted a number of metabolic changes, i.e., statistically significant decreases in the protein-bound iodine level, oxygen consumption, heart-rate, thirty-minute postprandial blood glucose level and serum-calcium level, and concluded that these changes "may be one of the reasons for the weight gain observed". (p.377).

Comstock and Stone (1972), in a long-term (six year) study, concluded that there was a dose-response relationship between smoking and changes in weight and (subscapular) fatness. Their subjects (n = 501) were aged between 40 and 59 years and were all males; even amongst the smokers, there was a tendency to increase in weight over the 6 year period, but this was significantly less than amongst ex-smokers. (Comstock and Stone make the incidental but important point that, although higher body weight is a recognized health-risk factor, it is healthier to be heavier and a non-smoker than to be lighter but to smoke; subcutaneous fatness is not especially associated with an increased risk of mortality (Comstock, Kendrick and Livesay, 1966) and, moreover, mortality and morbidity are decreased when smoking is stopped (see earlier section on health consequences of smoking)).

Yet further evidence has been produced in support of weight increase as a result of smoking cessation, by Hickey and Mulcahey (1973), Garvey, Bosse and Seltzer (1974), Gordon et al (1975) and Blitzer, Rimm and Giefer (1977). The latter authors conducted their study on women (previous longitudinal studies having been on males) and collected a vast amount of data (they used 57,032 subjects.) The duration of their observations was 25 years. Blitzer and his colleagues found that "light" smokers (i.e., who smoked approximately 10 cigarettes daily) gained about 5lb after ceasing smoking; "heavy" smokers, however, (40+ cigarettes a day) gained, on the average, 30lb. These gains were permanent (still being evident at 25 year follow up).

Gross body weight, in conclusion, appears to be a reliable correlate of smoking behaviour and is, therefore, along with lung-function, an important molar measure.

(v) Other issues in assessment

To conclude this section reviewing the measurement and assessment of smoking behaviour, certain minor issues warrant brief discussion.

Firstly, the measurement of covert, as opposed to overt, behaviours has been rather neglected. Cognitive aspects of smoking such as "craving" or "urges" (Chapman et al, 1971; Shiffman and Jarvik, 1976; Harrington, 1978), "images" (Berecz, 1972) and "self-statements" (Steffy, Meichenbaum and Best, 1970) should ideally be taken into account when assessing smoking behaviour comprehensively. These factors have obvious implications in designing appropriate treatment programs and are of relevance where the use of such treatment techniques as covert-sensitisation (Cautela, 1967) and coverant-control (Homme, 1965) is concerned. (See methods of Treatment, below).

Secondly, situational variables would seem to be of some importance (Epstein and McCoy, 1975). Many smokers tend to smoke more at certain times of the day (Hoffman-Tennov, 1972; Epstein and Collins, 1977), and during certain activities (Hoffman and Boyko, 1969; Griffiths et al, 1976); these factors also need to be taken into account in treatment.

Thirdly, McFall (1978) has raised the issue of when to assess subjects, recommending that the dependent variable (i.e.

smoking rate) "(should) be carefully assessed during treatment as well as before and after". McFall (correctly) maintains that: "Only in this way will it be possible to examine closely how the treatment exerted its effect on smoking" (p.708) and says that assessment during treatment allows investigation of the treatment process as well as the treatment outcome. Additionally, McFall emphasises that the same measures should be used at all assessment points. Finally, Shipley, Rosen and Williams (1982) have drawn attention to the need for consistency in the research literature with regard to three matters: a) the procedure for classifying people who smoke after treatment but are abstinent at follow-up; b) the duration of the measurement interval used to determine abstinence or smoking rate; and c) procedures for classifying people who use marijuana or tobacco products other than cigarettes. Shipley and his colleagues, after reviewing the literature and finding wide variations in the data-reporting behaviour of researchers, made the following recommendations, respective to the above issues: a) If subjects who "slip" after treatment, then again stop smoking, this should be noted in the research report, rather than being ignored; however, the practice of viewing subjects who have had a short-term relapse after a treatment programme as being "non-abstinent" should be discouraged, as this "inhibits research attention to an important question : Does a brief relapse always lead to a return to regular smoking (see Marlatt and Gordon, 1980)?" (p.301); b) one week should be

the minimum period of measurement, whether this be at baseline, post-treatment or follow up; shorter intervals are regarded as overlooking the fact that social events which may be related to smoking rate tend to occur in weekly cycles; longer intervals are seen as being "awkward at pre-treatment"; and c) if subjects switch to smoking any other harmful substance - including marijuana (which is now established as being a harmful drug (Petersen, 1980)) - this should be reported in the text or in footnotes; however, "the correspondence between abstinence and a cigarette smoking rate of zero should be preserved by ignoring cigarette substitutes when determining abstinence". (p.302).

d) Methods of Treatment

An overall evaluation of the effects of therapeutic intervention with regard to cigarette smoking will be presented at the end of this section. First, the main methods of treatment which have been employed will be reviewed evaluatively. As the present research is concerned with the behavioural treatment of cigarette smoking, this approach will be the primary concern of this review. Non-psychological (and non-behavioural) methods will be discussed only superficially. As indicated in the introduction, several, excellent and comprehensive reviews already exist (Bernstein and McAlister, 1976; Lichtenstein and Danaher, 1976; Raw, 1978; Frederiksen and Simon, 1979; Pechacek, 1979), so the present review will not be over-detailed.

There are a number of ways in which methods of treatment of smoking behaviour may be categorised. One obvious way is to differentiate between psychological and non-psychological methods; another way would

be to categorise according to whether the methods aim at dealing with the antecedents of smoking behaviour, the behaviour itself, or its consequences (e.g. Frederiksen and Simon, 1979). Yet another method may be to differentiate between cognitive and non-cognitive interventions.

The present author, however, has chosen a novel method of categorisation, namely, treatment methods which are "administered" or carried out by the therapist, directly ("Therapist Controlled Methods") versus methods which are "self-administered", as a result of therapist instructions ("Self-control strategies"). The reason for this type of differentiation is two-fold: firstly, such a distinction carries a logical-simplicity, which other methods of categorisation may lack; and, secondly, this distinction is of relevance to the design of the present experiment, which attempted, among other things, to establish whether a combination of these two types of intervention was more effective, as a treatment "package", than the use of one group of methods, alone (i.e. "self-control" methods).

It must be remarked, however, that this distinction is not always absolutely clear-cut. Certain methods which are, initially, therapist-administered may, at a later stage in treatment, be used with at least some effect, independently by the subject. This would apply particularly to cognitive techniques of modification. However, the categorisation employed allows individual methods to be placed clearly and logically within one or other of the categories.

(i) Therapist controlled methods

(a) Punishment and Aversion Therapy

(i) Electric shock

Although, in keeping with the current Zeitgeist in behavioural therapy, electrical aversion therapy is now a seldom used technique, the method has been very widely used in the past

as an attempt to modify cigarette smoking behaviour. Results, on the whole, have been disappointing. Powell and Azrin (1968), using a portable aversive-conditioning device (Whaley, Rosenkranz and Knowles, 1969), treated three subjects, with initially high success. However, as soon as the smoking/shock contingency was removed, all subjects immediately resumed smoking at their baseline rates. Pope and Mount (1975) used a similar device with better results: 63% of their subjects were still abstinent at 1 year follow-up. However, this was an uncontrolled study, so other, non-specific factors may well have been responsible for the treatment effect; moreover, Pope and Mount relied on self-report as a measure of improvement.

Koenig and Masters (1965) earlier obtained results which have come to be seen as typical of outcome studies in the field of smoking modification. They compared the effectiveness of electrical aversive conditioning with "supportive counselling" and systematic desensitization (Wolpe, 1958); no main treatment effect was found; all groups, regardless of treatment condition, decreased their smoking rate significantly, but almost all subjects had relapsed at 6 month follow-up. A rather higher success rate (40% abstinence at follow-up) was obtained in a similar study by Ober (1966).

Andrews (1970) compared the effects of punishing different responses (eg. touching a cigarette, inhaling smoke) using electric shock. No main effect was observed, but, interestingly, Andrews noted that non-contingent shock was no less effective

than contingent shock. Russell, Armstrong and Patel (1976) confirmed this result, comparing contingent shock with non-contingent shock, no-shock smoking, simple support and a no-treatment control group. All treatment methods were equally effective and more so than the no-treatment group. The fact that non-contingent shock has been found to be as effective as contingent shock seems to suggest that a strict conditioning explanation is less important than an explanation in terms of non-specific factors in treatment, such as motivation, expectancy, etc.

With reference to another variable in the use of electrical aversion therapy, it is noteworthy that Berecz (1972) found that self-administered shocking of cognitions related to smoking was more effective than shocking smoking behaviour, per se, at least for "heavy" smokers (20+ cigarettes per day). This finding, too, emphasises that cognitive variables are important in determining subjects' response to treatment. In a more recent study, Berecz (1974) reported that shocking subjects' imagining having an urge to smoke was even more effective than shock contingent on imagining smoking itself, the three subjects in the former condition being abstinent at two year follow-up and the three in the second condition having initially abstained but ultimately relapsed. The "n" in this study, however, was obviously rather low.

Chapman, Smith and Layden (1971) obtained an impressive abstinence rate of 91%, using a combination of electric-shock and self-management training. With a high level of therapist-

support post-treatment, a 1 year follow-up abstinence rate of 54% was reported, which is a relatively high rate.

In summary, the use of electrical-aversion therapy, in laboratory settings and as the sole method of treatment, has not been found to be effective in modifying smoking behaviour. However, when used as part of a treatment package (Chapman et al, 1971) or when applied to covert, rather than overt smoking behaviours (Berecz, 1972, 1974), rather more support has been produced for this technique. It would appear, however, that non-specific factors have played some part in the achievement of the higher success-rates. (Russell et al, 1976).

(ii) Cigarette smoke, rapid smoking and satiation

Each of these three techniques uses cigarette smoke itself as the aversive stimulus. (Strictly speaking, satiation techniques are not "aversive" but are classified along with the other methods because of the type of stimulus used). Wilson and Davi son (1969) recommended that, in aversion therapy, the noxious stimulus should, ideally, be of the same modality as the target behaviour, so these approaches are, in this respect, more suited to the problem behaviour of cigarette smoking than for example, electrical aversion therapy.

Overall, results have been far more positive than is the case with electrical-aversion; however, varying degrees of success have been reported. Wilde (1964) pioneered the use of hot, smoky air as an aversive stimulus (with cool, mentholated air as the "avoidance" stimulus). Of seven smokers thus treated, three became abstinent, one reduced to two cigarettes per day

and one began smoking a pipe. At one year follow-up, however, (Wilde, 1965), all had relapsed. Franks, Fried and Ashem (1966) refined Wilde's procedure and obtained remarkably similar end-of-treatment results and these were, in this case, unchanged at 6 month follow-up. Both Wilde's and Franks et al's studies neglected to use control groups. In contrast, Grimaldi and Lichtenstein (1969) compared contingent (i.e. whilst smoking) hot, smoky air administration with non-contingent administration and a yoked control group, which received the experimental procedure without the noxious stimulus. All three groups improved equally and significantly and all three had relapsed, to some extent, at 3 month follow-up. Grimaldi and Lichtenstein (1969) concluded that "contingent punishment is of limited value in the control of smoking" (p.275) - tones, here, of the conclusions relating to electrical aversion therapy (see above). Lublin and Joslyn (1968) were the first to use the aversion technique of rapid-smoking. This method entails the subject's inhaling deeply, every six seconds or so, until no more can be tolerated and nausea is produced, whereupon a short break is taken before the next smoking session begins. Sessions are continued, again, until no more can be tolerated. Lublin and Joslyn achieved an abstinence rate of 19% at one year follow-up. Keutzer (1968) obtained a six month follow-up rate of only 8.5%, using rapid-smoking, but Schmahl, Lichtenstein and Harris (1972) reported an end-of-treatment abstinence rate of 100% and a six month follow-up rate of 60%. Similar rates to these latter were also reported a year later by Lichtenstein et al (1973) and, later still, by Harris and Lichtenstein (1974). These

last three reported studies were all typified by the subjects' being given a high degree of social support and high expectations of success, by the therapist. In the study by Schmahl et al (1972), treatment success was actually correlated negatively with the number of "conditioning" sessions administered, suggesting that factors other than conditioning may have been responsible for the treatment outcome. In the absence of a high level of support and expectation, results of rapid-smoking studies have been rather less impressive (Curtis, Simpson and Cole, 1973; Kopel, 1974). Sutherland et al (1975) actually reported that, at three month follow-up, subjects were smoking at 102% of their baseline rate. Levenberg and Wagner (1976) found rapid-smoking to be more effective than either relaxation or systematic desensitization at post-treatment, but not at four month follow-up. (The rapid-smoking abstinence rate at this latter assessment point was a mere 11%). In contrast, Lando (1976) reported a six month abstinence rate of 43% for rapid-smoking.

Frederiksen and Simon (1979) have suggested that the wide variation in the reported effectiveness of rapid-smoking as a method of treatment is perhaps due to certain procedural variations, such as length of inter-puff intervals, number of trials and sessions, spaced (as opposed to massed) treatment and location of trials (laboratory versus home-based). The differing results also seem to be strongly determined by the presence or absebce of such non-specific factors as expectancy and the social behaviour of the therapist (Schmahl et al, 1972). Bernstein and McAlister (1976) conclude that ".. when unsupplemented

by social support, positive expectations and the like, rapid smoking is not clearly superior to other approaches;", but that, when such factors are included, it "appears to be one of the more powerful initial abstinence techniques available". (p.95).

The technique of satiation was introduced by Resnick (1968a, 1968b) and is closely related to that of rapid-smoking. Resnick required his subjects to either double or triple their baseline smoking rate for a week and then to attempt to stop. Both treatment groups showed a significant abstinence rate at four month follow-up (63%); a control group showed a 20% rate. Sushinsky (1972) failed to replicate Resnick's positive results and Clairborn, Lewis and Humble (1972) failed to find any difference in rate between the treatment and control groups in a further replication (the control groups here, unlike in Resnick's Study, being given a convincing treatment rationale and thus, presumably, having a higher expectation of success). Marrone, Merksamer and Salzberg (1970) reported that 60% of a group of subjects who were required to chain-smoke for 20 hours were abstinent at four month follow-up, where only 18% of the group who did the same for 10 hours were abstinent. (At one month follow-up, no difference was observed between these groups, although both were better than the control group, in terms of success). Marrone et al's impressive results have not been confirmed, no further experiments using this particular satiation technique having been conducted, probably due to the extreme aversiveness of the technique. Marrone et al reported that more

than 50% of their subjects vomited during the treatment period. With the possible exception of the study by Marrone et al (1970), there is little evidence which supports the effectiveness of satiation as a treatment technique. In line with the conclusions of Bernstein and McAlister (1976) concerning rapid-smoking, satiation is probably only effective when utilized in an environment which generates high expectations of success.

(iii) Taste aversion, emetics and other aversive stimuli

Little use has been made of taste-aversion procedures in attempts to modify cigarette smoking behaviour. This is surprising for two reasons: firstly, Wilson and Davi son's (1969) contention that the aversive stimulus should be in the same modality as the target behaviour would suggest that taste is an appropriate sensory modality on which to concentrate and, secondly, the experimental evidence for the use of taste-oriented techniques is still inconclusive. Marston and McFall (1971) found only temporary reductions is smoking rate, using Pronicotyl tablets and Whitman (1972) observed no difference between treatment and control groups at six month follow-up, using a preparation with similar, aversive properties. However, Seltzer (1975) reported an abstinence rate of 82%, using asafoetida lozenges. Further research is required. Only one study (a single case study) exists in the literature concerning the use of an emetic as an aversive stimulus. Raymond (1964) successfully treated a fourteen year-old boy using apomorphine to induce vomiting in association with cigarette smoking. The subject was reported to be abstinent

at one year follow-up. Again, further investigation seems to be warranted here.

Similarly, Kentzer's (1968) study appears to be the only one which has used breath-holding as an aversive technique. She found this technique to be equal, in effectiveness, to other methods of treatment and more effective than no-treatment.

Finally, Green (1964) attempted to modify smoking in a group of mentally retarded subjects by using white-noise (over on-going music) as the aversive stimulus. No reduction in smoking behaviour was apparent in this experimental group; it would seem likely that this result was due to the lack of motivation on the part of the subjects to reduce their rate of smoking (see McFall and Hammen, 1971).

(iv) Covert sensitization

Covert sensitization as a treatment method (Cautela, 1967, 1970, 1971), unlike the above methods of aversion therapy, is concerned solely with cognitive behaviour. It involves the imaginal pairing of a noxious stimulus with "approach" behaviours (e.g. taking a cigarette from a packet) and the pairing of a pleasant stimulus with "avoidance" behaviours (e.g., stubbing a cigarette out).

Wagner and Bragg (1970) found that a combination of covert sensitization and systematic desensitization was more effective than systematic desensitization alone. The degree of smoking reduction thus achieved, however, was only minimal. Sachs, Bean and Morrow (1970) reported covert sensitization to be more effective as a method of treatment than "self-control" techniques, subjects in the former group obtaining a higher degree of success

in reducing smoking than either the latter group or an attention-placebo control group. Despite this apparent support of the technique, Weiss (1974) and Wisocki and Rooney (1974) failed to find support for covert-sensitization's being any more effective than other treatment methods, and noted that only minimal smoking reduction occurred as a result of treatment. In contrast, once again, and in line with the results of Sachs et al (1970) and Cautela (1970) himself, Sipich, Russell and Tobias (1974), in a well-controlled study, found covert sensitization to lead to a reduction in smoking, greater than that obtained by a minimal treatment group.

Lichtenstein and Danaher (1976) commented: "the overall evidence in support of covert sensitization in the modification of smoking behaviour appears to be relatively weak", but that "the economy and portability of the procedure suggest ... that it deserves additional empirical study" (p.104-105). These authors suggested that covert sensitization may be more effective if the method were employed beyond the point of abstinence. Furthermore, a number of authors have drawn attention to certain methodological inadequacies in studies which have investigated the effectiveness of covert-sensitization. Raw (1978), in his review, has said that "the absolute effectiveness (of covert sensitization) is difficult to judge because so few of the studies mentioned report abstinence rates" (p.456) and Mahoney (1974) has suggested that a lack of standardized treatment procedures has perhaps contributed to the results' being largely negative. Frederiksen and Simon (1979) remark that covert

sensitization combined with other procedures as part of a package may lead to more favourable results. In conclusion, there yet appears to be room for further research into the potential of covert sensitization as a treatment technique in smoking modification programmes.

To summarise, some types of aversion therapy appear to be useful in the short-term modification of smoking behaviour and at least one method, covert sensitization, may still prove (given the design of methodologically sound studies) to be effective in maintaining initial reductions in smoking rate, or abstinence. Further research is warranted in this case; additionally, the techniques of taste-aversion, emetically induced vomiting and satiation (see Marrone et al, 1970) deserve further investigation.

As a footnote to this section on Aversion Therapy, mention may be briefly made of recent attempts to use the technique of covert-extinction (Cautela, 1971) as a means of modifying smoking behaviour. Although not actually an aversive technique, covert extinction does involve the dissociation of the target behaviour from its usual, subjectively pleasant connotations, imaginally, and is therefore discussed at this point. Götestam and Melin (1974), achieving some success in applying covert-extinction with amphetamine addicts, attempted to modify cigarette smoking behaviour in the same way (Götestam and Melin, 1983). They were disappointed in their results, which failed to provide support for the technique. Fagerström, Götestam and Melin (1983) also found little support, but, as subject compliance and attendance for treatment were minimal, no firm conclusions were drawn from

this study. This technique may have some potential, but further, controlled studies are needed to show this.]

b) Operant conditioning

This short section is concerned with the positive reinforcement of non-smoking behaviour, rather than the punishment of smoking behaviour. This latter behavioural approach has been reviewed in the preceding section. The section is short because, generally speaking, positive reinforcement as an individual technique in smoking modification research is now rarely used. Bernstein and McAlister (1976) point our that the effects of the technique have typically been difficult to evaluate because of confounding with other techniques, but that initial changes in smoking behaviour do seem to be brought about by positive reinforcement in the form of social approval and/or monetary rewards (Tighe and Elliott, 1968; Winett, 1971; Axelrod et al, 1971).

Brockway et al (1977) in a well-controlled study, used positive social reinforcement as one of a number of treatment methods. Although the effects of this reinforcement are difficult to separate from those of the other treatment techniques, the results obtained, at least up to 6 month follow-up, (when a reduction of approximately 45% in rate was apparent), were encouraging.

Nehemkis (1969) and Janis and Hoffman (1970) employed positive social reinforcement in the context of a "buddy system", where subjects were placed in dyads and encouraged to reinforce one another's improvements. Nehemkis' results were disappointing,

but those of Janis and Hoffman were more favourable, high-contact dyads maintaining significant reductions at 1 year follow up.

In a recent study, Paxton and Scott (1981) investigated the hypothesis that improvements in lung function, fed verbally back to subjects, would reinforce non-smoking behaviour. Although improvements in FEV, (see section on Assessment and Measurement, above) were evident, Paxton and Scott concluded that it was "not clear from the data .. whether the important consequence [of having stopped smoking] was the verbal feed-back given after test sessions or whether is was naturally occurring changes such as an improved ability to walk or run". Further research along these lines would appear to be of considerable value.

c) Pharmacological treatment methods

(i) Psychotropic drugs

The rationale underlying the use of psychotropic drugs in the modification of smoking behaviour is that these may serve either to reduee the anxiety (ostensibly) associated with smoking, or to provide a substitute for the stimulating effects of smoking (see section on the Biological Effects of Nicotine, above). Studies relating to the first category of drugs have been conducted by Turle (1958) (hydroxyzine), Schwartz and Dubitzky (1969) (meprobate), Whitehead and Davies (1964) (diazepam) and Graff et al (1966) (chlordiazepoxide); in all cases, the drug was either ineffective or no more effective than a placebo. In the second category, Whitehead and Davies (1964) found methylphenidate to be no better than a placebo drug. Hansel

(1954) claimed some support for dextroamphetamine and, more recently, Miller (1971) reported that benzedrine sulphate led to abstinence in 90% of a group of subjects of up to 6 months duration; however, both of these studies were methodologically unsound.

(ii) Lobeline

Lobeline is an alkaloid obtained from an Indian tobacco plant (Lobelia inflata) and which has been used as a nicotine substitute, because of its pharmacological similarity to nicotine. Davi son and Rosen (1972) have reviewed the literature on the experimental use of lobeline and neither studies before (Dorsey, 1936; Bartlett and Whitehead, 1957; Edwards, 1964; Ford and Ederer, 1965), nor since (Brengelmann and Sedlmayr, 1975) that review have provided any support for its effectiveness in modifying cigarette smoking.

(iii) Nicotine chewing gum

Ferno, Lichtneckert and Lundgren (1973) developed a chewing-gum which contained nicotine and which has been shown to be capable of satisfying the smoker's need for nicotine, when substituted for cigarettes (Russell, Feyerabend and Cole, 1976). Russell, Wilson, Feyerabend and Cole (1976) found only limited support for the use of "Nicorette" (the propietory name for the chewing-gum) as a treatment method, but stronger support has been since produced by Raw et al (1980), who reported that, at one year follow-up, chewing-gum treatment was more effective than behavioural treatment. Similarly, Fagerstrom (1980) reported a 63% abstinence rate at six month follow-up for a

chewing-gum group, as compared to 45% for a "behavioural counselling" placebo group. This recent evidence suggests that the use of nicotine chewing gum may prove to be a powerful method of treatment in the future, especially when combined with behavioural treatment strategies (Lichtenstein, 1982).

d) Hypnosis

Hunt and Bespalec (1974), in their review of the smoking literature, considered hypnosis to be one of the more effective techniques of modifying smoking behaviour. In contrast, Bernstein and McAlister (1976) maintained that hypnosis had yet to be shown to be effective as a method of treatment. The disparity between these views is a reflection of the disparity between the results of investigations which have been reported. For example, Von Dedenroth (1964a, 1964b, 1968) reported a 94% abstinence rate, at 6 year follow-up of 1,000 subjects, whereas Cohen (1969) reported an abstinence rate of 0%. Hypnosis is a generic term and does not represent any standard technique or set of techniques. This is one very obvious reason for the wide variation in the reported success of hypnosis. In other words, different investigators have used different methods. What does seem to be clear, however, is that hypnosis, however used, incorporates a number of non-specific factors, such as social support, expectancy (on the part of the therapist, in some cases, as well as the subject) and motivational influences, and it is likely that these influence smoking behaviour as much as the hypnotic process or state itself.

Many studies of hypnosis as a means of modifying smoking behaviour have been deplorable, from a methodological point of view. Crasilneck and Hall (1968) did not describe the procedure used and Cohen (1969) reported that no standard procedure was, in fact, utilized. Von Dedenroth (1964a, 1964b, 1968) used no control group in this ambitious study and did not report on the attrition rate over the follow-up period of six years. Successes have even been reported, in the literature, without data being presented (Arons, 1961; Erikson, 1964). As was the case with Von Dedenroth's work, rather more recent studies (Kline, 1970; Nuland and Field, 1970; Orr, 1971; Spiegel, 1970) failed to use control groups, so their generally high reported abstinence rates need to be viewed with extreme caution.

Those studies which have employed superior methodology and design (Edwards, 1964; Perry and Mullen, 1975) have failed to demonstrate that hypnosis is any more effective than placebo treatment.

In conclusion, because hypnosis as a technique is so intricately bound with a number of non-specific treatment factors (therapist "warmth", high expectancy, etc.) it is difficult to ascertain which of the properties of treatment have led to decreases in smoking rate or abstinence, when this has been reported. Hypnosis seems to be no more (nor, however, any less) effective than placebo treatment.

e) Sensory deprivation

Only a limited amount of work has taken place with this method. Suedfeld (1969) produced evidence that individuals subjected

to a period of sensory deprivation are made more susceptible to "persuasive communications". He extended this principle to dealing with smoking behaviour and found that initial abstinence rates were as high as 100%. However, in a study with F. Ikard (Suedfeld and Ikard, 1974) this same, initial abstinence rate compared poorly with a one year follow-up rate of 28%.

Interestingly, Suedfeld's studies suggested that the presence or absence of a persuasive, anti-smoking message did not influence outcome, but that the sensory deprivation, per se, was the important treatment variable.

Raw (1978) explains this by suggesting that "the short term effect is due to the absence of smoking cues for 24 hours and the long-term effect to the encouragement gained from the short-term effect". He concludes, as did Bernstein and McAlister (1976) in their earlier review, that, as sensory deprivation seems no more effective than other approaches (whilst certainly exerting some positive effect on smoking behaviour), the expense of using this technique is not justified.

f) Relaxation training and systematic desensitization

Little evidence exists for the effectiveness of relaxation training as a therapeutic technique, at least when used in isolation, for cigarette smoking. As smoking is not simply an anxiety-reducing mechanism (Ikard, Green and Horn, 1969), this is not surprising. Some claims have, however, been made for the value of systematic desensitization (Wolpe, 1958)

as a method of treatment, the rationale being that certain, anxiety-provoking situations are associated with smoking. Koenig and Masters (1965) found systematic desensitization (SD) to be as effective as aversive-conditioning and noted that SD was associated with the greatest initial decrease in rate; however, this effect was lost at 6 month follow-up. Morganstern and Ratcliffe (1969) reported that 87% of their subjects decreased their smoking rate significantly throughout treatment and that 38% were abstinent; but they did not provide follow-up data. Kraft and Al-issa (1967) treated a group of alcoholic patients for social-anxiety, using SD and noted that they reduced their cigarette consumption. The reductions reported by individuals were still evident at follow-up (from 8 months to 2 years after treatment), but no subjects had abstained.

Wagner and Bragg (1970) noted that relaxation alone was not effective, when compared to a relatively effective combination of covert sensitization and SD, and this finding was confirmed, more recently, by Levenberg and Wagner (1976).

In summary, relaxation training alone seems to be ineffective as a method of treatment. The same applies to SD, but this latter technique may have some value when used as part of a treatment "package" (Wagner and Bragg, 1970; Gerson and Lanyon, 1972).

As a footnote to this section, mention needs to be made of a study by Ravensborg (1976), who used "focussed muscular relaxation" to "subdue cigarette cravings and avoid smoking". This technique was based on that of Bernstein and Borkovec

(1973); subjects were encouraged to focus their relaxation on their "tension and craving spots". A significant ($p<0.01$; n = 40) decrease in rate was observed at four month follow-up (a reduction of approximately 30%). Although no control group was used in this study, the method has some face validity and its use as an adjunct to other methods of treatment warrants some attention.

g) Role playing and modelling

Janis and Mann (1965) used emotional role playing in an attempt to modify smoking attitudes and behaviour, heavy smokers playing the role of cancer patients. This group reduced their rate of smoking more than did a control group, who simply listened to a recording of a treatment session. Mann and Janis (1968) reported encouraging follow-up data after eighteen months. Mausner and Platt (1971) conducted a similar study, where subjects were required to play the role of a doctor informing a smoker that he/she had lung cancer, or the role of the patient, in the same vignette. The subjects in the former role decreased their rate of smoking to some degree, although this reduction was minimal. This technique has received surprisingly little attention in recent years. Similarly, a technique utilized by Brockway et al (1977), as part of a treatment package, which involved subjects' learning more adaptive and assertive "non-smoking" responses to be used in social situations, showed some promise, but has since been neglected.

Colletti and Kopel (1979) conducted a study which investigated the relative efficacy of experiencing one of three different

maintenance strategies following treatment for smoking reduction. These strategies were modelling (successful previous subjects acting as models for prospective reducers), participant observing and self-monitoring. Colletti and Stern (1980) conducted a 2 year follow-up study on these subjects and, although the self-monitoring group was the most successful (37.5% of baseline rate, which, incidentally, was not significantly higher than the groups end-of-treatment rate), the modelling group had maintained some reduction (66.7% of baseline).

h) Education and Persuasion

The dissemination of information about the harmful effects of smoking to large groups of smokers, for example, on a national level, seems to have led to minimal changes in smoking behaviour (Bernstein, 1969). However, observable decreases have occurred, if only temporarily, as a consequence of well-publicised information such as the reports of the Royal College of Physicians (1962, 1971, 1977). (see Fig 1.1).

Mair (1970) exposed a group of 265 smokers to films, lectures and group discussions on the harmful effects of tobacco. Abstinence rate at 3 month follow-up was a moderate 40%, but at one year follow-up was only 19%, no better than would be expected as a result of non-specific treatment. No control group was used in this study. On a smaller scale, Schauble, Woody and Resnikoff (1967) found that an education plus medication group had a post-treatment abstinence rate of 48%, which compared to 21% for medication alone. No follow-up data were reported.

"Scare tactics", such as those used by Leventhal and Watts (1966) and Leventhal, Watts and Pagano (1967) have proved to be ineffective in modifying smoking behaviour (Frederiksen and Simon, 1969). Frederiksen and Simon (1979) cite Hochbaum (1975) as concluding that fear-induction strategies may change smoking attitudes, but do not appear to change smoking behaviour, and go on to say that there is a danger of such strategies "boomeranging", heightening anxiety levels and perhaps increasing smoking behaviour as a result.

More "gentle" persuasion and advice to stop smoking, from a figure in authority, was found to be an effective method of treatment, by Raw (1976). He found that smokers who were advised to stop by a chest-physician reduced more than did a minimal-treatment control group (39% as opposed to 17% reduction at 3 month follow-up).

i) Psychoanalysis and "counselling"

Bergler (1946) claimed that psychoanalysis was effective in helping five smokers to stop. No data was provided to support this claim. Bergler's rationale is described (exceptionally briefly) by Raw (1977, 1978) and must, in the light of current knowledge, be interpreted with caution.

"Counselling" is a generic term and is the treatment "method" which has been traditionally used in anti-smoking clinics. Bernstein (1969), in his review, concluded that "most clinics represent a great deal of wasted time and effort" (p.431) having found no support for counselling, whether individual or group, in the literature. Studies have been typically uncontrolled (Ejrup, 1963[4+b] McFarland, 1965; Lawton, 1967).

Bernstein and McAlister (1976) point out that the "deliberate and isolated use of the nonspecific treatment factors contained in clinic settings produce post-treatment results comparable to those of clinics ... " (p.91), citing the research of Bernstein (1970), Lichtenstein et al (1973) and Sipich, Russell and Tobias (1974) as support for this statement.

(ii) Self-control methods

Self control methods are those which are administered by the subject, rather than the therapist, and in the natural environment. There is little evidence that such methods, used in isolation, are effective in the modification of smoking behaviour, but, when a "package" of self-control techniques is employed, results have been found to be more favourable. Self-control treatment packages will be discussed after individual techniques are detailed. For the purposes of this review, the classification strategy of Lichtenstein and Danaher (1976) will be used; these authors identified three types of self-control strategies :

a) Environmental Planning

b) Behavioural Programming

and, c) Cognitive Control

The nature of the first two of these categories was originally described by Thoresen and Mahoney (1974).

a) Environmental Planning

(i) Stimulus control

By virtue of associational learning mechanisms, smoking behaviour usually becomes psychologically linked with certain environmental situations or stimuli. Stimulus control involves associating

smoking with specific stimuli in the environment and then fading these novel stimuli.

One method of doing this is by increasing the stimulus interval. Powell and Azrin (1968) found that, by using a cigarette case which automatically locked itself and consistently increased the duration of being locked was effective in bringing about an initial reduction in smoking rate. However, once subjects stopped using the case, rates returned to baseline. Upper and Meredith (1971) obtained similar results using pocket timers, as did Bernard and Efran (1972). Both Shapiro et al (1971) and Levinson et al (1971) reported that significant reductions were reported when a variable interval schedule of cued smoking was used; however, attrition rates were high in these studies and a "barrier" was reached at a rate of about 12 cigarettes a day, subjects being unable to reduce further. Levinson et al (1971) believed that this "floor" effect was due to the appearance of withdrawal symptoms. Bernstein and McAlister (1976), however, suggest that it is due to the fact that, as less cigarettes are smoked, the reinforcement value of cigarettes increases and makes those that remain harder to relinquish. Bernstein and McAlister therefore recommend immediate cessation as opposed to gradual reduction. Flaxman (1978) has provided evidence to support this contention, although she recommended abrupt quitting on a target date, rather than immediately.

A second method of stimulus control is "hierarchial reduction". This technique entails the subjects' eliminating smoking, systematically, in situations where smoking is a high

probability behaviour. Pumroy and March (1966) and Gutmann and Marston (1967), although finding some short-term benefit using this method, did not find significant improvement at follow-up. Sachs, Bean and Morrow (1970) applied hierarchial reduction to thoughts and feelings concerning smoking but found the method no better than an attention-placebo control condition at one month follow-up. Marston and McFall (1971) used four sections of the day as hierarchy items and required subjects to stop smoking in each of these (starting with the easiest); again, no differences emerged, at 6 month follow-up, between this experimental group and a control group (both groups smoking at 69% of baseline). Flaxman (1978) in the study mentioned above, employed hierarchial reduction, but found little support for its effectiveness. The technique has since been used by Brockway et al (1977), as part of a package; the package was found to lead to significantly greater reductions in smoking than a control condition, up to 6 months post-treatment, but not at one year follow-up. Recently, Hills (1983) found hierarchial reduction to be effective in modifying smoking, but only provided follow-up data up to one month after treatment. This author, incidentally, noted a greater improvement in subjects who dealt with "hard" situations before "easy" ones, than vice-versa.

A third method of stimulus control is "deprived response-performance", which requires that smoking only occurs in situations bereft of stimulating properties. It is analagous to "time-out" procedures in other fields of behaviour modification (Blackham & Silberman, 1975). Nolan (1968)

and Roberts (1969) found this technique to be successful in eliminating smoking, using a "smoking chair" in uncomfortable surroundings. However, these were uncontrolled, n = 1 studies. Greenberg and Altman (1976) reported similar success more recently, but, again, only two subjects were used. However, control subjects were employed and abstinence in the experimental "group" was still evident at one year follow-up.

(ii) Contingency contracting

Contingency contracting, the second type of environmental planning strategy, requires the smoker to agree to accept certain consequences for smoking or non-smoking behaviour; such consequences may be either social or otherwise. Social contracting, as a therapeutic technique, has been investigated by a number of authors. Tighe and Elliott (1968), Lawson and May (1970) and Bornstein et al (1975) all showed that reductions in smoking can, at least initially, be brought about by this method. Similarly, Nehemkis and Lichtenstein (1971), using married couples who smoked, reported good short-term results, but considerable relapse at 6 month follow-up. The "buddy system", reviewed earlier, has also been found to lead to some reduction in smoking (Jan is and Hoffman, 1970). The use of "deposit contracting", where the return of monetary deposits is made contingent on non-smoking, has also been investigated. Such response cost techniques have been utilized by Nurnberger and Zimmermann (1970), (who used material possessions, rather than money, as a deposit), Bornstein et al (1975) and Elliott and Tighe (1968). The latter authors reported relatively good results - a post-treatment abstinence rate of 84% and a long-term follow-up rate of 37.5%. Winnett

(1973) found, in a better controlled study, that contingent repayment was more effective than non-contingent repayment (six month follow-up abstinence rates being 50% and 23.5% respectively).

Paxton (1977), in an unpublished paper, has recommended the use of deposit contracting on both theoretical and clinical grounds. He especially notes that the technique is well suited to maintaining reduction/abstinence, as deposits can be returned over a long period. Paxton cites a study by Lando (1977), in which the technique was used, effectively, as part of a more comprehensive package.

b) Behavioural Programming (operant control)

(i) Self reward

Few studies have investigated the self-administration of (tangible) rewards as reinforcement for non-smoking behaviour. Lando (1977) used the technique as part of a successful treatment package (76% of experimental subjects, as opposed to 35% of controls, being abstinent at six month follow-up). Murray and Hobbs (1981) found self-reward alone to result in only minimal smoking reduction at post-treatment and 3 month follow-up, and no significant reduction at 3 year follow-up. In contrast, they found a combination of self-punishment and self-reward to be more effective than either method used alone, the reduction here being still significant at 3 year follow-up.

(ii) Self punishment

This operant method has been more widely used than self-reward. Johnson (1968) found no difference between an aversive breath-holding group and a control group. Ober (1968) and

Whitman (1969) found no support for self-administered electric shock as a technique. Axelrod et al (1974) met with greater success, although their study involved only two subjects. The first was required to tear up a dollar-bill for each cigarette she smoked beyond a (gradually reduced) daily limit: she was abstinent at two year follow-up. The second contributed money to charity contingent on smoking and, at one year follow-up, smoked "only in stressful situations". (Watson and Tharp, (1972), incidentally, recommended that in this second type of self-punishment procedure, it would be more effective to donate the money to one's most hated charity organization). Finally, reference to the study by Murray and Hobbs (1981) in the preceding section, will show that a combination of self-punishment and self-reward would appear to be an effective technique in the self-modification of smoking behaviour.

c) Cognitive Control

(i) Coverant control

"Coverant (covert operant) control" (Homme, 1965) is a specialized form of operant conditioning which permits the subject to manage his own reinforcement contingencies (Keutzer, 1968). It is based on the differential probability hypothesis of Premack (1965) and, where smoking is concerned, involves the covert reinforcement of anti-smoking cognitions, in the hope that an increase in the frequency of such conditions will lead to a decrease in the frequency of smoking. The method was used, successfully, with two subjects by Tooley and Pratt (1967), in combination with other procedures.

Keutzer (1968) found coverant control to be equal in effectiveness to other behavioural techniques and more effective than a control condition, but Lichtenstein and Keutzer (1969) found, at six month follow-up, that all treatment groups had relapsed to approximately 75% of baseline smoking rate. Very similar results were obtained by Johnson (1969) and Lawson and May (1970), who also compared coverant control with other behavioural techniques. Danaher (1974) found the familiar pattern of results, namely, that different coverant control designs all resulted in smoking reduction by the end of treatment and did not differ from one another, but that, at (8 month) follow-up, pervasive relapse had occurred.

(ii) Self instructional training

Chapman et al (1971) and Miller and Gimpl (1971) used "self-instructional training" (Meichenbaum and Cameron, 1974), as one element of a treatment package. This method entailed subjects' giving themselves frequent, positive instructions, concerning their daily smoking goals. Although decreased smoking rates were reported in both of these studies, the effects of self-instruction cannot be separated from the impact of the other strategies used.

(iii) Thought stopping

This final, cognitive technique, developed by Cautela (1970) has been used in only one study in the literature. Wisocki and Rooney (1974) found the method to be more effective than a placebo procedure at post-treatment, but no more so, at four month follow-up.

d) Self-monitoring

Reference to the earlier section on Assessment and Measurement will draw attention to the fact that self-monitoring is a reactive procedure and may, therefore, be used as a treatment method in its own right (McFall, 1970; McFall and Hammen, 1971; Kantarowitz, Walters and Pezdek, 1978). Elaboration at this point of the present review would be repetitious and superfluous.

e) Self-control treatment packages

As smoking is a complex, multidetermined behaviour and has numerous, personal idiosyncratic facets (Best, 1975; Frederiksen and Simon, 1979), the treatment "package" would appear to be an appropriate approach to treatment. Few self-control techniques have been shown to be effective when employed individually but it has been hoped that "combining procedures may yield a unique and more powerful product (a catalytic effect)" (Lichtenstein and Danaher, 1976, p.117). Thus, most treatment packages sacrifice precision and specificity for breadth and generality (Merbaum and Rosenbaum, 1980). A number of authors have advocated the use of self-control manuals either as a substitute for or an adjunct to standard treatment (Harris and Rothberg, 1972; Conway, 1977; Danaher, 1977).

The results obtained by studies using self-control packages have been relatively impressive, though not uniformally so. Brengelmann (1973), using a package consisting of no less than 37 individual techniques, and encouraging gradual

reduction as opposed to abrupt quitting or reduction, reported a 58% abstinence rate at 2 month follow-up. Flaxman (1974) found that setting a target-date for quitting led to a package treatment's resulting in a 50% abstinence rate at 6 month follow-up. (Lichtenstein and Danaher (1976) present a selected list of self-control package studies, classifying the various elements utilized under the headings used in this review. This list is reproduced in Table 1.1 and represents the main studies conducted up to, and including, 1974). Studies have continued, more recently, again with varying results. Delahunt and Curran (1976) compared self-control with negative-practice (satiation) and with a combination of the two techniques. At 6 month follow-up, the combination group had attained a 70% reduction from baseline and an abstinence rate of 56%. (These figures were not matched by either of the two methods used alone). Danaher (1977), in contrast to the promising findings above, found that, although a self-control package led to some reduction in smoking, this compared poorly with rapid-smoking plus discussion.

Blittner, Goldberg and Merbaum (1978) compared a cognitive self-control group with a "stimulus instruction" group and a waiting list control. Their results favoured the former group, who had reduced their rate of smoking by 33% at six month follow-up. Merbaum, Avimier and Goldberg (1979) found that a self-control package was an effective maintenance technique, following treatment by rapid-smoking and covert-

Study	Environmental Planning	Behavioral Programming	Cognitive Control	Substitute Behavior
Brengelmann (1973)	Hierarchical reduction Deprived response Deposit system	-	-	-
Chapman et al (1971)	Hierarchical reduction Deprived response Deposit system	-	Emotional response routine Self-instruction	Time-structured activity
Conway (1974)	Hierarchical reduction Deprived response	Self-reward for non-smoking and self-control	Self-instruction	-
Flaxman (1974)	Miscellaneous stimulus control procedures Social contracts	Self-reward for non-smoking	Emotional response routine	Time-structured activity Relaxation
Harris & Rothberg (1972)	Hierarchical reduction	Self-reward for non-smoking Self-punishment for smoking	Miscellaneous cognitive control procedures	Time-structured activity Relaxation

Table 1.1

Self-Control Treatment Packages up to, and including, 1974.

(From Lichtenstein, E. and Danaher, B.G. 1976)

Study	Environmental Planning	Behavioral Programming	Cognitive Control	Substitute Behavior
Marston & McFall (1971)	Hierarchical reduction	Self-reward for non-smoking	Self-instruction	Eating Relaxation
Miller & Gimpl (1971)	-	Self-reward for non-smoking and self-control	Self-instruction	-
Morrow et al (1973)	Deprived response	Self-satiation	Self-instruction	Eating Physical and/or quiet activity
Ober (1968)	Miscellaneous stimulus control procedures	Self-reward for non-smoking	Self-instruction	Time-structured activity
Pomerleau & Ciccone (1974)	Hierarchical reduction Miscellaneous stimulus control procedures	Self-satiation	Self-instruction and imagery	Exercise Relaxation
St. Pierre & Lawrence (1974)	Increasing stimulus interval Reverse hierarchical reduction	Self-reward for non-smoking Self-satiation	Self-instruction and imagery	-

Table 1.1

Self-Control Treatment Packages up to, and including, 1974., (continued.)

(From Lichtenstein, E. and Danaher, B.G. 1976)

sensitization; 6 month follow-up abstinence rate was approximately 45%.

Finally, as mentioned previously, Murray and Hobbs (1981) found that a combined (admittedly rather small) treatment package of self-punishment plus self-reinforcement was more effective than either strategy used alone; and Buchkremer (1982), using a package consisting of ten separate methods, reported abstinence rates of 85% and approximately 30% at post-treatment and three year follow-up respectively (smoking-rate reductions for the (controlled) group being 91% and 33% at the same assessment points).

It can be seen from the abstinence/reduction figures quoted in these studies that, on the whole, self-control treatment packages appear to be more effective in modifying smoking behaviour than treatment methods used in isolation. Lichtenstein and Danaher's (1976) statement would, therefore, seem to be vindicated to some degree.

(iii) Multicomponent interventions

As was concluded in the preceding section, the hope that self-control treatment packages may be a more effective means of modifying cigarette smoking behaviour than individual techniques, has been partly borne out. It would seem plausible, therefore, to suggest that multi-component intervention programmes, being comprised of a combination of self-control and other, therapist-administered techniques, may be still more effective. The research evidence supports this suggestion, as will be seen from a brief description of pertinent studies.

Chapman et al (1971) achieved a 12 month follow-up abstinence rate of 54%, using a combination of electrical aversion therapy and a self-control package.

Morrow et al (1973) quoted a 46% abstinence rate one year after the end of treatment, using a self-control package and rapid smoking. An identical rate (at 11 month follow-up) was reported by Pomerleau and Ciccone (1974), again using a combination of self-control and aversive procedures.

In a study designed to test the issue of additive effects, Tongas, Patterson and Goodkind (1976) found that a group subjected to rapid-smoking, covert-sensitization and social reinforcement for reducing the rate of smoking, achieved a higher abstinence rate at 1 year follow-up (77%) than groups using only one of these techniques.

Elliott and Denney (1978) found a multi-component package (which included applied relaxation, rapid smoking, self-reward training, self-punishment, covert-sensitization, emotional role-playing, systematic desensitization and cognitive restructuring) to be superior to rapid smoking alone, a non-specific treatment and a control condition. At six-month follow-up, the multicomponent group had an abstinence rate of 45% and a smoking rate of 41% of baseline level. Unfortunately, the "package" in this programme consisted of so many individual methods that no conclusions can be drawn as to which methods were most important; perhaps the very complexity of the programme was responsible for the favourable follow-up figures, the "catalytic" effect being exploited to a large degree.

Probably the most impressive series of studies in the literature, in terms of outcome, (with the exception of those studies which were anecdotal or poorly controlled) is that conducted by H. Lando and his colleagues. Lando (1977) developed a two-stage program consisting of a preliminary period of satiation, followed by seven self-control training sessions (designed to maintain the abstinence hopefully resulting from first-stage intervention). At six month follow-up, a 76% abstinence rate was reported for the treatment group, in contrast to a (still relatively high) rate of 35% for a control group who received only the initial stage of treatment. Lando and McCullough (1978) successfully replicated this study, which yielded a 71% abstinence rate at six month follow-up. Lando (1978) went on to conduct a study with three treatment stages: stimulus control training, followed by satiation and then by self-control training (this latter component being of shorter duration than in the 1977 study). The treatment group responded no better than did the control group; additionally, there was a considerable relapse rate and a high attrition rate. Lando concluded that the complexity of the treatment program detracted from group-cohesiveness, which in turn led to the poor results obtained, and, more importantly, that the continued and extended use of self-control techniques, after treatment, is essential. This latter conclusion was confirmed by Lando et al (1979). Finally, Hughes et al (1981) have reported on the Multiple

Risk Factor Intervention Trial (MRFIT), which was designed to help middle-aged male smokers, who were at risk for cardiovascular disease, to stop smoking. This multi-component programme consisted of a large number of behavioural, non-behavioural and educational strategies and succeeded in bringing about an abstinence rate of 46%, at a follow-up period of four years, in a group of 4,103 subjects. (Abstinence was biochemically confirmed). A control group (Neaton et al, 1981) achieved a 27% abstinence rate.

It is interesting to note that the more successful multi-component interventions tend to include an aversive element (see studies described above). Lichtenstein (1982) has described this as unfortunate, because of the costly screening procedures entailed. Less expensive (and potentially harmful) aversive procedures, such as covert-sensitization, should, perhaps, therefore be considered as elements to be used in such programmes.

Keeping in mind the failure of Lando's (1978) study, due partly to the overcomplexity of the multicomponent package used, and the statement by Franks and Wilson (1975) that "more is not always better" (p.409), it may be nevertheless concluded, in the light of the favourable results obtained, to date, from multicomponent studies, that such interventions are probably the most promising type at present available. Bernstein and McAlister (1976) noted that "the multicomponent approach ... would seem to warrant further and more rigorous evaluation" (p.97) and, more recently, Frederiksen and Simon (1979) stated that :

"the area of multielement treatment packages requires a great deal of study. Although many researchers ... have suggested the need for attacking smoking behaviour along a number of dimensions, ... the increased efficacy of multielement approaches has yet to be substantiated. Nevertheless, logic would argue that a comprehensive, carefully designed multielement treatment package would hold a great deal of promise, particularly when some of the treatment components are self-administered in the natural environment by the smoker himself/herself".

(Frederiksen and Simon, 1979, p.536-537)

(iv) An overall evaluation of the efforts made, over the last twenty years, or so, to modify smoking behaviour (with any semblance of permanence), can, perhaps, best be presented, by quoting from a number of important papers and reviews which have appeared, since 1969.

These quotations appear here in chronological order :

"Smoking behaviour ... is incredibly resistant to long-term modification. ... The basic problem in the modification of smoking behaviour revolves about long-term maintenance of non-smoking, not about production of immediate, short-term behaviour change. The latter is accomplished by a variety of treatments ... but is followed, in the majority of cases, by a return to pre-treatment rates"

(Bernstein, 1969, p.435).

"The good news is that almost any intervention can be effective in eliminating or drastically reducing smoking behaviour. The bad news is that these changes tend to be relatively short-lived; data from the vast majority of controlled smoking modification research have presented an all-too-familiar pattern of immediate and dramatic reduction in cigarette consumption ... followed by relapse ... within a twelve month period"

(Bernstein and McAlister, 1976, p.89-90).

"A prevailing note of pessimism is reflected in many of the literature reviews in this area ... The crucial question becomes how to prevent or at least minimize post-treatment relapse"

(Lando, 1977, p.361).

" ... (a) Virtually any treatment program is capable of reducing smoking levels to 30% or 40% of baseline; (b) a return to about 75% of baseline is commonly observed from 3 to 6 months after treatment; (c) seldom more than 13% of the subjects in any treatment program are completely abstinent after a 3- to 6-month follow-up period; and (d) of those subjects who are abstinent at the end of treatment, less than one third manage to maintain non-smoking 3-6 months later"

(Elliott & Denney, 1978, p.1330).

> " ... it is clear that current methods for dealing with smoking are probably no more effective than those devised by smokers trying to manage the problem on their own"
>
> (Leventhal and Cleary, 1980, p.396).

> "Treatment approaches ... have been disappointingly unsuccessful. ... The vast majority of investigations have demonstrated no differential success among treatments, with the standard outcome of treatment and placebo conditions being significant reduction in cigarette consumption by the end of treatment and considerable relapse at follow-up ... ".
>
> (Murray and Hobbs, 1981, p.63).

It is clear from these comments, spanning twelve years, that little progress has been made in the field of the behavioural modification of smoking. The same pattern of results - epitomized by rapid positive change, as a result of treatment, with a drift back towards baseline - has continued to occur.

Recent work, using self-control packages (Flaxman, 1974; Delahunt and Curran, 1976; Blittner, Goldberg and Merbaum, 1978; Merbaum, Avimier and Goldberg, 1979; Buchkremer, 1982) has suggested that this pattern may, at last, be being broken and this seems to be even more so with regard to comprehensive, multicomponent treatment packages (Morrow et al, 1973; Tongas et al, 1976; Elliott and Denney, 1978; Lando, 1977; Lando and McCullough, 1978). Such treatment approaches recognize that smoking is a behaviour of some complexity and are, more often than not, based on a realistic and comprehensive model of smoking behaviour. They take into account cognitive and physiological factors, as well as overt smoking behaviour, and pay attention to the environmental determinants of smoking. Moreover, the maintenance of non-smoking is treated as being perhaps the most important goal of intervention.

It is hoped that reviews over the next twelve years will draw more positive conclusions than the selection quoted above.

e) Goals of intervention - the alternative of reduced smoking

This issue has already been discussed to some extent in the earlier section on nicotine regulation research. To re-iterate the conclusion drawn : "there is not yet sufficient evidence available to preclude treatment efforts which aim to establish a lower level of smoking in subjects, as an alternative to total abstinence". (p.35)

From the foregoing account of treatment outcome studies, it is clear that a problem of primary importance is the frequent, high relapse rate for those subjects who attempt to abstain. Reduced, or controlled, smoking, therefore would seem to be a viable alternative goal, worthy of investigation. The concept is analagous to the, now widely accepted, goal of "controlled" or "social" drinking, for former alcoholics (Strickler et al, 1976).

As was pointed out earlier, there is no firm evidence that subjects who reduce their rate of smoking indulge in compensatory smoking behaviours which maintain their nicotine and tar intake at the same level (Freedman and Fletcher, 1976; Martin et al, 1981). It has been argued (Ross, 1976) that reduced smoking in the form of smoking low tar/ low nicotine brands of cigarettes, may actually be more harmful than smoking at baseline level, due to the increased exposure to the poisonous gases contained in cigarettes (carbon-monoxide, hydrogen cyanide, nitrous oxide) (Prue, Krapfl and Martin, 1981); low tar/nicotine cigarettes have, in some cases, been shown (Ross, 1976) to contain more of these gases than non-filter, higher tar/nicotine brands. However, Foxx and Brown (1979) noted that the "five cigarette brands that ranked lowest on combined triple-gas ... ratings were also among the lowest in tar and nicotine (content)". Ross' argument, therefore, does not seem fully supported.

A further argument, in favour of reduced smoking, and partially explaining the traditionally high failure rate of most abstinence-oriented programs, is that many smokers find it exceptionally difficult to smoke at a rate (including a rate of zero) below their "stuck point" (Foxx and Brown, 1979). Levinson et al (1971) suggested that this point was a function of the smoker's addiction to nicotine, further reduction resulting in the appearance of withdrawal-symptoms (and often in dropping-out of treatment).

An incidental point related to the concept of reduced smoking, raised by Colletti, Supnick and Rizzo (1982) is that there has been an almost exclusive reliance, in the research literature, on using abstinence-rate as an outcome measure. This measure is (although, arguably, the only meaningful one - Raw, personal communication, 1981) seen as being rather coarse, and Colletti and his colleagues welcome the use of smoking-rate data, as such data permit researchers to evaluate whether subjects who have failed to abstain have undergone any improvement whatsoever, allowing a further evaluation of outcome.

A number of studies have provided support for the viability of reduced smoking as an alternative goal. One of the earliest, (Bernard and Efran, 1972), was aimed at comparing reduction versus elimination, using pocket-timers (a stimulus-control technique). The reduction group who were "not urged to eliminate smoking altogether" (p.400), was the most successful. Paradoxically, this group demonstrated a 40% abstinence rate at two month follow-up (a rather short follow-up); Bernard and Efran's interpretation of this result was that subjects in this group achieved, or surpassed, their established goal (of a reduced rate) and therefore felt successful and reinforced themselves; this reinforcement

eventually procured total abstinence. In contrast, subjects in the "elimination" group, having had higher standards set for them, were "rarely able to feel good about their performance".

Frederiksen et al. (1976) reported that 70% of subjects using a contingency contracting procedure were smoking at 50%, or less, of their initial rate, at six month follow-up and concluded that "it may be possible to develop controlled smoking as an alternative to abstinence" (p.196).

Schinke, Blythe and Doueck (1978), using a "multifaceted approach", reported that reduced smoking was maintained at six month follow-up and Elliott and Denney (1978), in a well-controlled study using a multi-component treatment package, achieved a 41% rate of baseline smoking, also at six months after the end of treatment. Blittner, Goldberg and Merbaum (1978) obtained a 70% reduction at 3 month follow-up, using cognitive self-control techniques (this result being superior to a stimulus control group and a no-treatment control condition).

More recently, Foxx and Brown (1979) showed that a nicotine fading/ self-monitoring treatment led to a decreased rate in 50% of cases at 18 month follow-up; Foxx, Brown and Katz (1981), following up this group one year later (2½ years after the end of treatment) reported that improvements were still maintained.

Colletti, Supnick and Rizzo (1982) found, at long-term follow-up (four years), that subjects who had been treated with a comprehensive, non-aversive behavioural treatment, using stimulus-control and other self-control techniques, demonstrated a 56% rate of baseline smoking rate, on average.

Foxx and Axelroth (1983) extended the earlier study of Foxx and Brown (1979) and reported, at 12 month follow-up, mean reductions of

82% and 85% for nicotine and tar intake, respectively, and a 28% reduction in rate.

To conclude, reduced smoking seems to be a realistic goal, for some smokers at least. Although total abstinence is, from a health viewpoint, the goal of choice, "safer" smoking is a goal which may well apply to those who are either unable or unwilling to stop smoking completely (Frederiksen, 1979).

Glasgow, Klesges and Vasey (1983) have indicated the need for "further development of controlled smoking procedures" (p.144). Finally, Russell (1974) said that "Rather than anti-smoking, the aim should be towards achieving acceptably safe, light to moderate, controlled smoking", and, further, that "With this more feasible goal, success is not only possible but probable" (p.256).

f) Non-specific factors in treatment

At several points in this review, allusion has been made to "non-specific" factors in treatment. As stated, a recurrent theme in the results of experiments designed to reduce or bring about the abstinence of cigarette smoking has been the equivalence in response to "treatment" of placebo-treatment or control groups, compared to groups receiving treatment per se. Additionally, it has often seemed that, no matter what the treatment, most subjects can easily reduce or abstain from smoking, in the short term (McFall and Hammen, 1971; Elliott and Denney, 1978; Raw, 1978) but relapse in the long-term (Raw, 1977).

It has been suggested that these findings can be explained by reference to non-specific factors in treatment (McFall and Hammen, 1971) and such factors thus deserve some mention, in their own right, as it is crucial that these are recognized (and, ideally, controlled for) in any treatment study.

(i) Self-monitoring

The issue of self-monitoring has already received extensive coverage in this review and attention has been drawn to the procedure's ability to influence the target behaviour. Reference has also been made to the use of self-monitoring as a method of treatment, per se; further discussion is therefore unnecessary.

(ii) Motivation

McFall and Hammen (1971) conducted a study designed to elucidate possible non-specific factors in treatment. Clear evidence was obtained regarding the role of motivation as a determinant of smoking reduction, in that subjects' "self-reported pre-treatment motivation to stop smoking was significantly related to whether or not they actually stopped smoking by the end of treatment" (p.85). McFall and Hammen were especially impressed with the fact that, although all subjects in the study rated themselves at the positive end of the motivation scale, it was possible to discriminate finely between them.

More recently, Raw (1976) found, with chest-clinic patients advised to stop smoking, that a measure of motivation predicted change in smoking behaviour and the same author (Raw, 1978) has related motivation to stop smoking and degree of dependence on cigarettes, orthogonally (see Fig. 1.4). Raw suggests that motivation to stop may be measured by questionnaire and "by the number of hurdles jumped during assessment" (p.476) and that degree of dependence can be inferred from severity of withdrawal symptoms, regularity of smoking pattern, blood nicotine levels, etc.

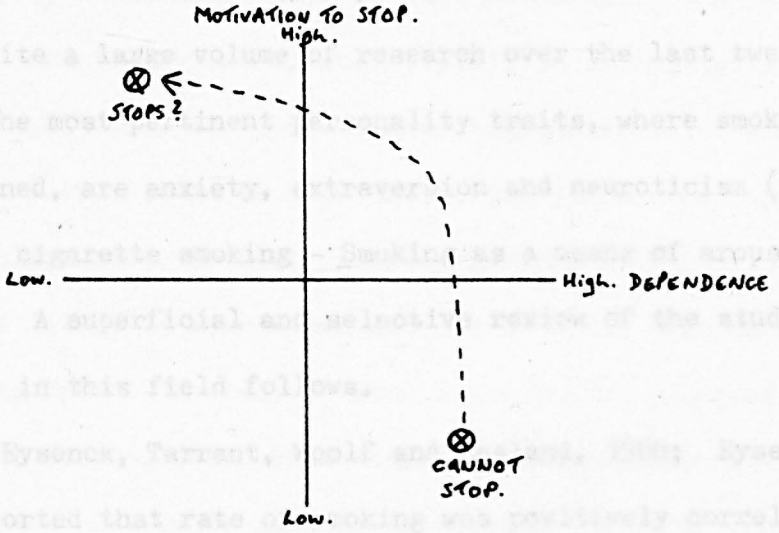

Fig. 1.4
Motivation, dependence, and predicted ability to stop smoking
(Adapted from M. Raw, 1978, p.476)
(Diagram compliments of Michael Russell)

Raw goes on to ask whether all treatment successes come from the top left-hand quadrant of this diagram, implying that, if people can be "pushed" into this quadrant, by having their motivation increased and their degree of nicotine dependence reduced (with, for example, nicotine substitutes), then they may find cessation easier. This approach certainly warrants investigation.

(iii) Personality

The issue of personality has been of interest, in connection with cigarette smoking behaviour, on two counts. Firstly, many attempts have been made to ascertain whether the concept of the "smoker's personality" has any validity; in other words, do cigarette smokers consistently exhibit certain personality characteristics? Secondly, are any particular characteristics associated with the ability or inability to respond to treatment for smoking - otherwise stated, can personality attributes be used to predict outcome?

Neither of these questions can yet be answered with certainty, this despite a large volume of research over the last twenty years. The most pertinent personality traits, where smoking is concerned, are anxiety, extraversion and neuroticism (viz. Models of cigarette smoking - Smoking as a means of arousal control). A superficial and selective review of the studies conducted in this field follows.

Eysenck (Eysenck, Tarrant, Woolf and England, 1960; Eysenck, 1963) reported that rate of smoking was positively correlated with degree of extraversion. This finding was consistent with Eysenck's cortical arousal model of extraversion (Eysenck, 1967), which describes extraverts as being constitutionally low in cortical arousal and, therefore, stimulus hungry (smoking being a satisfying stimulus). (This model overlooks the fact that nicotine, in larger doses, has sedating, rather than excitatory, properties (Ashton and Stepney, 1982)). Although a number of subsequent studies supported Eysenck's findings, (Feather, 1963; Kissen, 1964; Lefcourt, 1965; Tacon, 1965), Keutzer (1968) found that the smokers in their study "did not deviate from the published norms for normal adult populations on the (factor) of Extraversion ..." (p.147). Keutzer came to the same conclusion with regard to the "Neuroticism" dimension of personality (Eysenck, 1967), her subjects achieving "normal" scores, compared to the general population, (Eysenck (1967) suggesting that smokers, on the whole, were more neurotic than non-smokers, using cigarettes to reduce their autonomic arousal). Similarly, where trait anxiety was concerned, Keutzer found no differences between her group and "normal" subjects.

Smith (1970) found smokers to be more extraverted than non-smokers; and Cherry and Kiernan (1976), in a longitudinal study on 2,753 people, reported that high neuroticism scorers were more likely to smoke than those with low scores, that deep-inhalers were the most neurotic group and that extraverts were more likely to smoke than introverts - all this data supporting Eysenck's theory. From a predictive point of view, Cherry and Kiernan found that stable (non-neurotic) extraverts were the most likely to give up smoking of their own accord (this being in contrast to Eysenck's speculations).

Rae (1975) found smoking to be associated with extraversion, but not with neuroticism. In contrast, Floderus (1974), in a Swedish study, found exactly the opposite: smokers did not differ from non-smokers in terms of extraversion, but were significantly more neurotic.

McCrae, Costa and Bossé (1978) found no differences between a group of heavy smokers and a group of non-smokers in degree of extraversion, but that heavy smokers were significantly higher than non-smokers on both measures of neuroticism and anxiety (which are closely related concepts).

More recently, Chatterjea, et al (1979) reported that "consumption of nicotine was directly related to the level of "trait" anxiety ..." (p.205), but that neuroticism was not a determinant of smoking behaviour.

In a refreshingly different study, which examined smokers "Psychoticism" (P) (* see footnote on page which follows) scores (Eysenck and Eysenck, 1976) as well as their "E" and "N" scores,

McManus and Weeks (1982) found that smoking did not relate to extraversion, but to Psychoticism. They suggested that this relationship was the true one, previous findings having been due to a contaminated measure of "E". (Previous studies had used the Eysenck Personality Inventory (EPI) (Eysenck and Eysenck, 1964) as an assessment tool. This questionnaire does not have a "P" scale, but certain "P" items are inadvertently included in the "E" scale. McManus and Weeks used the modified version, the Eysenck Personality Questionnaire (EPQ) (Eysenck and Eysenck, 1975) and were thus able to establish, they suggested, that smoking was really related to "P" and not "E"). Eysenck himself, together with L.J. Eaves (Eysenck and Eaves, 1980) produced data supporting this contention, concluding that "most of what was said ... about E would now apply to P ". Finally, Spielberger and Jacobs (1982), using the EPQ and the State-Trait Personality Inventory (STPI) (Spielberger, 1979), found that smokers had significantly higher scores than non-smokers on the E, N, and P scales of the EPQ (and lower scores on the "Lie" scale), and that female smokers had higher STPI anxiety scores than female non-smokers, the converse being the case for male-smokers.

It is only too clear from the above review that the relationship

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*Psychoticism" (P) in Eysenck's model of personality, represents an individual's degree of "toughmindedness" and is orthogonally related to the dimensions of "Extraversion-Introversion" and "Neuroticism-stability". "Psychopathy" would, perhaps, have been a more appropriate term for the personality characteristics described by "P".

between smoking and personality factors is unclear and confusing. This is yet another area where further investigation is needed.

(iv) "Light" versus "Heavy" smoking (and the "Internal/External" dimension).

Another factor which may be regarded as a "non-specific" influence on treatment outcome is the individual's baseline rate of smoking. "Heavy" and "Light" smokers (terms which are necessarily arbitrarily defined) are supposed, by a number of authors (Russell et al, 1974; Schachter, 1977) to smoke for different reasons and, therefore, to be different types of smokers. Closely related to this differentiation is the distinction between "internal" smokers (who smoke largely as a result of internal, physiological stimuli) and "external" smokers (who smoke in response to environmental cues). Schachter (1977) argued that nicotine regulation was the primary aim of heavy smokers, whereas the use of smoking for the regulation of emotional states induced by multiple sources applied to light-smokers (Leventhal and Cleary, 1980). In support of this, Herman (1974) had already found light smokers to be more responsive to external cues than were heavy smokers. However, Herman did find light smokers to be responsive to changes in nicotine level; Leventhal and Cleary also make the point that many light-smokers have great difficulty in stopping smoking.

Russell (1974) divided smokers' stated reasons for smoking into two major orthogonal dimensions, which can be broadly described as pharmacological and socio-psychological. These two types may

be related respectively, to the "heavy" or "internal" physiologically-dependent smokers and the "light" or "external" smokers, who are more influenced by the surrounding environment.

Experimental evidence supporting this typology has been provided by Glad and Adesso (1976). They observed the behaviour of 140 subjects in a waiting room, where confederates of the experimenters either smoked or did not smoke. In the first condition, the subjects smoked more, and this effect was most marked in those individuals who smoked less than ten cigarettes a day. The study by Herman (1974) has already been mentioned.

(v) Expectancy

Theories regarding the role of expectancy in behavioural treatments of psychological problems are widely accepted (eg. Bandura, 1976). However, few studies have been carried out which examine the influence of subjects' expectations, on their response to treatment for cigarette smoking.

Blittner, Goldberg and Merbaum (1978) matched three groups of (18) subjects on baseline rate of smoking, age and number of years smoking. Each group was administered what were purported to be "personality tests". The first group (labelled by Blittner et al the "cognitive self-control treatment set") were told that the results of their tests had indicated that "they had strong willpower and great potential to control and conquer their desires and behaviour"(p.555) and that they were almost certain to be able to stop smoking (Thus a "self-control belief system" was established and reinforced). The second group did

not receive such information. These two groups were then treated with a self-control package. The third group were put on a waiting-list.

The "cognitive set" group were smoking at 10% of baseline by the end of treatment and 33% at 14 month follow-up. In contrast, the self-control package alone groups' rates were 32% and 65%, respectively, and the waiting-list control groups' rates were 93% and 92%. The groups' relative abstinence rates reflected these figures.

Blittner et al concluded that manipulation of expectancy had exerted a positive-effect on treatment outcome. However, this study can be criticised from a methodological point of view, in that the expectancy manipulation was not carried out "blind" by the therapist (and so experimenter bias and expectation may have played a part in the obtaining of the results) and, further, in that only self-report measures were used to calculate rate and abstinence data.

Despite these criticisms, and the fact that Weston (unpublished MSc dissertation, 1980) failed to confirm these findings, in a similar study using self-treatment manuals, expectancy would seem to play at least some part in smokers' response to treatment. It may well be that smokers who have a naturally high (rather than artificially increased) level of expectancy will benefit more from treatment than those who are pessimistic about their prospects. Once again, the conclusion is that further studies are required to examine this issue.

g) Conclusion

As was stated at the outset, this review of the literature has been cursory. Detailed descriptions of experiments conducted in the field of smoking research have only occasionally been given, as a number of more comprehensive reviews already exist (Bernstein and McAlister, 1976; Lichtenstein and Danaher, 1976; Raw, 1978; Frederiksen and Simon, 1979; Pechacek, 1979). The purpose has been to provide an overview of the work done to date, to delineate areas of research of especial current interest (viz. research on nicotine regulation) and to identify issues which warrant further investigation.

This latter aim formed the basis for the rationale of the present study, in which a number of pertinent issues were examined in detail in an attempt to further our knowledge in this important field of research. The rationale for this research endeavour will now be presented.

2. RATIONALE

It is clear from the foregoing review of the literature that, although rather more effective methods of treatment have recently been developed, tobacco smoking is still a behaviour which is exceptionally resistant to long-term modification. There is little doubt that the most effective psychological interventions are those which rely on the application of "packages" of treatment methods; it also seems likely that "multicomponent" packages, which include both self-control and therapist-administered techniques, will be superior in their effectiveness to less comprehensive packages. However, no systematic investigations have been conducted, to date, which specifically examine the relative efficacy of comprehensive self-control packages as compared to multi-element packages. This is an important practical issue, as, were it to be found that a sophisticated, broadly based self-control package is as effective in modifying smoking behaviour as a rather more complex package, which includes therapist-administered techniques, implications would arise as to the cost-effectiveness of the latter approach and, more specifically, with regard to the number of smokers who could be treated in a clinical setting. It is felt, however, that, in view of the tentative research findings to date, multicomponent packages hold more promise than any other psychological approach, as long as they are not over-complex (as was the case, for example, in the study by Lando (1978)). The present study acknowledges the statement by Bernstein and McAlister (1976) that "the multicomponent approach ... would seem to warrant further and more rigorous evaluation" (p.97) and Frederiksen and Simon's more recent (1979) assertion that "the area of multi-element treatment packages requires a great deal of study" (p.536-537).

The primary aim of this study then, is to assess the relative efficacy of a carefully designed self-control treatment package, as compared with a multicomponent package, which includes therapist-administered treatment methods, but is otherwise identical in form.

Where the decision was taken as to which individual elements should comprise the treatment packages used in this study, attention was paid to the sophisticated "behavioural contingency" model of smoking proposed by Frederiksen and Simon (1979). This model has been reviewed above (p.18). The packages to be employed thus address both overt and covert behaviours associated with smoking and also relate to concomitant environmental or situational events. Physiological processes are also taken into account, in that a method will be used which, it is hoped, will ameliorate the aversive effects of nicotine withdrawal (focussed relaxation training). As well as being consistent with this particular model of smoking behaviour, the treatment methods employed span the range of self-control techniques described above (p.76-83.) The "environmental planning" methods of stimulus control (hierarchical reduction and deprived response performance) and contingency contracting ("social" contracting and "therapeutic" contracting) are utilized; the "behavioural programming" method of self-punishment (monetary deprivation) is used; and the cognitive-control technique of coverant-control is employed. The two "therapist administered" methods which, it is believed, are likely to add to the effectiveness of a simple self-control package, are an imaginal aversive procedure (covert-sensitization, a technique which, according to Lichtenstein & Danaher (1976), " ..... deserves additional empirical study" (p.105)), and a relaxation procedure designed to help with the unpleasant symptoms of

nicotine withdrawal (focussed relaxation training). It is felt, therefore, that the treatment packages used in this study are soundly based, from a theoretical view-point, at the same time as deserving empirical evaluation.

A further issue, which the present study addresses, and one which the author considers to be of paramount importance from both a clinical and a theoretical perspective, is that of controlled, or reduced, smoking as a treatment goal, in contrast to the goal of complete abstinence from smoking. Although it may be argued that the occurrence of nicotine regulation invalidates the former goal, it is far from clear that smokers who reduce their rate of smoking will compensate by changing their smoking topography and therefore be at equal risk, from a health perspective, as formerly. It was concluded above, after a discussion of the research on nicotine regulation, that "there is not yet sufficient evidence available to preclude treatment efforts which aim to establish a lower level of smoking ....." (p.35) and, later, that " .... controlled smoking .... would seem to be a viable alternative goal, worthy of investigation" (p.93). In view of this, taking into account the fact that many smokers attempting to abstain reach a "stuck-point" (Foxx and Brown, 1979) and recognizing that a number of studies have provided support for the viability of reduced smoking as an alternative goal (see pages 94-96, above), the present author feels it worthwhile investigating the ability of the treatment packages to be utilized in this study to bring about a permanently reduced level of smoking in subjects, as well as assessing their efficacy in effecting total abstinence.

Little research has taken place regarding the concept of "light" versus "heavy" smoking. In the preceding review, this dimension was related to the "internal-external" smoking dimension; to re-iterate, it has been suggested that "heavy" smokers smoke in response to internal, physiological stimuli, whereas "light" smokers respond more to environmental, or external, cues. (Russell et al, 1974; Schachter, 1977). Thus, it would follow that "heavy", "internal" smokers are, perhaps, more addicted to nicotine than their "light" smoking counterparts and would therefore respond less well to a primarily psychologically oriented treatment programme. Leventhal and Cleary (1980) did comment, however, that many light-smokers have great difficulty in stopping. This, then, is an issue which, it is felt, warrants empirical study, hence its inclusion in the present investigation.

A final note with respect to the treatment rationale of this study: one of the clearest conclusions emerging from research in this field is that short-term changes in smoking behaviour are relatively easy to bring about, but that the long-term maintenance of non-smoking or reduced smoking is a far more elusive target. The majority of the techniques comprising the treatment packages in this study are included because, it is believed, they lend themselves to long-term usage or permanent application. The maintenance of behaviour change is the primary aim of treatment in this study.

3. METHOD

a) Experimental Design

The three variables investigated in this experiment, to retierate, were (i) a self-control treatment package versus this same package, combined with therapist-administered treatment techniques, (ii) abstinence versus reduction in smoking rate (i.e., 100% versus 75% reduction) and (iii) "heavy" versus "light" smoking (see below for definitions).

These variables were examined using a three-way factorial design, as illustrated in Fig. 3.1.

		Self-control package	Self-control package plus therapist-administered techniques.	
Heavy	100% reduction	GRP.1	GRP.5	GRP.9 Control Group (Heavy)
	75% reduction	GRP.2	GRP.6	
Light	100% reduction	GRP.3	GRP.7	GRP.10 Control Group (Light)
	75% reduction	GRP.4	GRP.8	

Table . 3.1. - The Experimental Design

For the purposes of conciseness, clarity and convenience, groups will generally be referred to, in the following sections, by number. Thus, restating this design using group numbers as the primary descriptor :-

Treatment Group 1

Received the self-control (SC) package, were heavy smokers (H) and aimed at total abstinence from smoking (100%). (SC/H/100)

Treatment Group 2

Received the self-control package, were heavy smokers and aimed at reducing their rate of smoking by 75% (SC/H/75).

Treatment Group 3

Received the self-control package, were light smokers (L) and aimed at total abstinence. (SC/L/100).

Treatment Group 4

Received the self-control package, were light smokers and aimed at 75% reduction. (SC/L/75).

Treatment Group 5

Received the self-control package plus therapist-administered techniques (SC+), were heavy smokers and aimed at total abstinence (SC+/H/100).

Treatment Group 6

Received the self-control package plus therapist-administered techniques, were heavy smokers and aimed at 75% reduction (SC+/H/75).

Treatment Group 7

Received the self-control package plus therapist administered techniques, were light smokers and aimed at total abstinence (SC+/L/100).

Treatment Group 8

Received the self-control package plus therapist-administered techniques, were light smokers and aimed at 75% reduction (SC+/L/75).

Control Group 1

Were heavy smokers (C/H).

Control Group 2

Were light smokers (C/L).

In summary, using the above abbreviations, this information is presented in condensed form in Table 3.2.

GROUP	DESCRIPTION
1	SC/H/100
2	SC/H/75
3	SC/L/100
4	SC/L/75
5	SC+/H/100
6	SC+/H/75
7	SC+/L/100
8	SC+/L/75
CONTROL 1	CH
CONTROL 2	CL

KEY

C = control group

SC = self-control package

SC+ = self-control package plus therapist administered techniques.

H = heavy smokers

L = light smokers

100 = total abstinence target

75 = 75% reduction target

Table 3.2 - Group Descriptions

b) Subject recruitment and selection

An article was published in a local newspaper, describing the service to be offered by the Clinical Psychology Department at Birch Hill Hospital, Rochdale, designed to help people stop or reduce their cigarette smoking.

Almost 500 enquiries were received in response to this article and all respondents were forwarded a two-part questionnaire, being told that, upon their returning this questionnaire, their names would be placed on a waiting list for treatment and they would be contacted in due course. Form A of this questionnaire provided personal data and information concerning the individual's smoking history and aspects of smoking behaviour. Form B, the second part, was designed to yield a rough measure of the individual's motivation to stop/reduce smoking (see below).

Three hundred and ten correctly completed questionnaires were returned (a return rate of approximately 60%). As this sample was of substantial size, the opportunity was taken to conduct a demographic survey, the results of which are presented in Appendix I of this thesis, along with a copy of the questionnaire. (My thanks are due to Dr. Martin Raw, for permission to use his ARU Smoking Questionnaire as the basis for the one employed in this experiment).

Questionnaires were numbered, consecutively, as they were returned, and subjects were allocated to treatment and control groups by using random-number tables. (Subjects were asked to indicate, on their questionnaires, whether they wished to abstain from smoking or to reduce their rate of smoking. A preponderance of hopeful "abstainers" was evident, so, when subjects were being allocated to groups, those whose stated target was contrary to the design of that particular group were returned to the "pool" and further subjects randomly selected, until the required number was obtained).

Eight subjects were allocated to each group. This was felt to be the optimum number of subjects per group, as any less would cause problems with respect to statistical analysis, in the event of more than minimal subject attrition, and any more would introduce an element of cumbersomeness into the data-collection procedure and group treatment, per se. (In the event, an unexpectedly high rate of subject attrition occurred and, in retrospect, a higher "n" per group would have been more appropriate; please see the Discussion for an elaboration of this point). Thus a total of sixty-four subjects commenced treatment, and a further sixteen subjects acted as controls.

In view of the fact that one variable under investigation was the response of "heavy" and "light" smokers to treatment, it was necessary

to obtain baseline-rate data on subjects, before allocating them to a "heavy" or "light" (but otherwise identical) group (see below); in this one respect then, random allocation to groups was not appropriate.

During the period between pre-treatment assessment and the commencement of therapy, in the case of five of the eight treatment groups, two subjects dropped-out (failing to attend for the first group treatment session). As it was not possible to replace these subjects at this time, thus restoring the original number in the group, it was decided to randomly eliminate two subjects from the three remaining experimental groups, in order to equalise the groups and therefore facilitate statistical analysis. The control groups were also comparibly reduced in size. (The eliminated subjects were offered individual treatment, independently of this research study). Thus, a final "N" of forty-eight subjects was used in the study, with twelve additional control subjects.

The pre-treatment characteristics of the total subject group are presented at the beginning of the Results section.

c) Therapists and location of therapy and assessment

The author (at that time holding the post of Senior Clinical Psychologist with Rochdale Health Authority) conducted all group-treatment sessions. These were held in the evenings at the main Psychology Department and had a duration of 1½ hours.

Initial individual treatment sessions were conducted by the author in all cases; subsequently, a proportion of individuals were treated by the author's assistant, a trained nurse-therapist, because of time constraints. This was not a confounding factor in treatment, as individual sessions were standardized, being recorded on audio-tape by the author.

Assessment sessions were conducted at Rochdale Infirmary, where blood-sampling and weighing facilities were available. Lung-function assessment was conducted by appointments arranged with the technician, these appointments taking place over a period of up to five days following the assessment session, per se. All assessment sessions were held in the evening (to maximise attendance); defaulters were, when necessary, seen individually, during the daytime, as soon as possible after the assessment-date.

d) Assessment

(i) Schedule

Experimental subjects were assessed at six points in time :-

(a) Pre-treatment (ten days before the commencement of treatment).

(b) Mid-treatment (three weeks after the commencement of treatment).

(c) Post-treatment (ten days after the final treatment session).

(d) 3-month follow-up.

(e) 6-month follow-up.

(f)12-month follow-up.

Control groups were assessed at two points in time, these being

(a) Pre-treatment equivalent

and (b) Post-treatment equivalent (8 weeks later)

(The assessment and treatment schedule is tabulated in detail in Appendix II).

(ii) Measures and rationale

The measures used in this experiment may be conveniently classified under these headings :-

a) Self-report measures

b) Physiological measures

and c) Personality measures

The specific measures used are presented in Table 3.3 and described below.

<table>
<tr><th>SELF-REPORT MEASURES</th><th>PHYSIOLOGICAL MEASURES</th><th>PERSONALITY</th></tr>
<tr><td>Smoking rate (and tar and nicotine intake estimates).</td><td rowspan="2">Serum thiocyanate (SCN)</td><td rowspan="2">Eysenck Personality Questionnaire (EPQ)</td></tr>
<tr><td rowspan="2">"Degree of use" and "Degree of benefit" ratings - weekly and overall.</td></tr>
<tr><td rowspan="2">Gross Body Weight</td><td rowspan="2">Symptom Check List (SCL) 90 Questionnaire</td></tr>
<tr><td>Situational anxiety ratings</td><td rowspan="5">Respiratory-functioning :
(i) Forced Expiratory Volume (FEV)
(ii) Forced Vital Capacity (FVC)
(iii) FEV/FVC Ratio
(iv) CO Transfer Factor</td></tr>
<tr><td>Craving intensity ratings</td><td rowspan="4">Cattell 16 P.F. Questionnaire</td></tr>
<tr><td>"Internal" vs. "External" smoking.</td></tr>
<tr><td>Expectancy rating</td></tr>
<tr><td>Motivation score</td></tr>
</table>

Table 3.3 - Assessment Measures

a) Self-Report Measures

(i) Smoking Rate

This was considered to be the primary outcome measure of this experiment (given its validation by serum thiocyanate measurement). As abstinence from smoking was not always the goal of treatment (see experimental design) abstinence rate was not the outcome measure of choice. Although, as discussed earlier in the review of the literature (see page 36), rate data is rather more susceptible to the reactivity effects of self-monitoring than is abstinence data (Kazdin, 1974) and abstinence is more easily detected biochemically, more powerful statistical analyses may be used with rate data. Abstinence is a less sensitive indicator of differential treatment effects. The present author regards rate as a superior and more meaningful measurement but

fully agrees with Colletti and Stern (1980) that, wherever possible, both abstinence and rate data should be reported; this practice will be adopted, where appropriate, in this study. Daily tar and nicotine intake levels were computed directly from self-reported daily smoking rate, using current H.D.U.K. tables. (The questionable validity of such unobjective estimates is fully recognized by the author and this issue is addressed in the Discussion).

(ii) "Use" and "Benefit" ratings

All subjects were required to rate, on a five-point scale, the degree to which they had used each method of treatment (i.e. each component of the treatment package), over the previous week. It was hoped that such "use" ratings provided a measure of subjects' adherence to therapeutic instructions, but it is recognized that this measure lacked objectivity and could not be reliably corroborated; the only way of doing so would have been to employ observers in the subjects' environment and this was not practical. It was, on the whole, however, felt that subjects' responses on the rating scales were honest and therefore relatively reliable.

In addition, subjects were asked to indicate, again on a five-point scale, the degree of benefit obtained from the use of each technique, over the previous week (i.e. the extent to which that particular method had helped to reduce the rate of smoking). Subjects were also required to indicate, on a finer, 10-point scale, the benefit obtained from treatment over the previous week, at each meeting. Overall benefit ratings were taken at all assessment sessions (excluding the first).

(Copies of the rating-scales used are included in Appendix III).

(iii) Situational anxiety ratings

During baseline and all subsequent assessments, subjects were

required to indicate, on a five-point scale, their level of anxiety immediately before lighting each cigarette. This was done over a period of ten days and it was thus possible to compute a "mean anxiety rating" for each period of assessment, for each subject (see Daily Record Card, Appendix III). It was believed that an "inverted U" shaped change over time would be apparent in the anxiety ratings of successful subjects and that certain conclusions relating to future research in this field may be drawn.

(iv) Craving intensity ratings

These ratings were obtained in precisely the same way as the situational anxiety ratings (see (iii) above). Again, a five-point scale was used(see Daily Record Card, Appendix III), and an "inverted U" shaped pattern was predicted.

(v) "Internal" versus "External" smoking

Subjects were asked to indicate whether cigarettes smoked throughout the ten day assessment periods were in response to internal or external stimuli. These terms were clearly defined for subjects. (See Daily Record Card, Appendix III). It was expected that "light" smokers would indicate more "external" cigarettes and that "heavy" smokers would smoke more as a result of internal stimuli; further, it was believed that smoking would become more "internal", as rate was reduced, and that important information with regard to future research may be obtained, if this proved to be the case.

(vi) "Expectancy" ratings

All subjects indicated, prior to treatment (but following the standardized introductory talk - see "procedure"), their level of "expectancy", on a 10-point rating scale. This rating was considered

to reflect how much benefit each subject expected to receive from the treatment programme (see Appendix III). In view of previous, but tentative, research findings, it was felt that a high level of expectancy would be related to successful response to treatment (Blittner, Goldberg and Merbaum, 1978).

(vii) "Motivation" scores

A score which was considered to reflect each subject's motivation to stop/reduce smoking was obtained, prior to treatment, by analyzing responses to the self-statements and questions presented in Form B of the questionnaire which was completed by all subjects, when responding to the offer of treatment (see "Subject recruitment and selection", above). This questionnaire is reproduced, in full, in Appendix I, but the motivation-related items are further reproduced in Appendix I. The author's considered opinion, and that of his colleagues, was used in ascribing weights to the alternative responses available for each item and these weights are indicated on the form in this Appendix. (As the "Motivation Score" was intended only to be a rough assessment and was considered to be an ancillary measure of purely incidental and anecdotal interest (at least in the present study), no attempt was made to validate the questionnaire or to assess its reliability. This matter will be discussed at a later point in this thesis).

(As with Form A of the questionnaire, my thanks are due to Dr. Martin Raw for permission to use his materials as the basis for part of the "Motivation" questionnaire).

b) Physiological Measures

(i) Serum Thiocyanate (SCN-)

This "molecular" measure was used as an objective check of subjects' self-reported rate of smoking. As blood SCN- levels have

been found to reliably distinguish smokers from non-smokers (Butts, Kuehnemann and Widdowson, 1974; Vogt, 1977) and as blood-sample analysis is relatively easy, compared to saliva or urine analysis, this was considered to be the SCN- measure of choice. (These alternative sampling methods, as mentioned earlier - p.48 - are, moreover, prone to contamination by the consumption of certain foods). From a broader perspective, SCN measurement was felt to be preferable to carbon-monoxide (CO) or carboxyhaemoglobin (COHb) measurement, as the half-life of these latter substances is far shorter than that of SCN-, thus increasing the probability of obtaining "false-negatives" (i.e. smokers who appear to be abstinent or smoking at a significantly lower level) when assessing subjects, (Densen et al, 1967).

(ii) Gross Body Weight

As discussed in the review of the literature (p.48-52), gross body weight has been found to reliably correlate with smoking behaviour and, moreover, to be influenced by changes in smoking behaviour; more specifically, reduced smoking has been identified with increased weight (e.g. Brožek and Keys, 1957; Glauser et al, 1970; Gordon et al, 1975; Blitzer, Rimm and Giefer, 1977). It was decided to use this particular "molar" measure, then, as a secondary physiological check on self-report (SCN- being the primary measure), but also because the weight-gain phenomenon continues to be one of interest, in its own right. This phenomenon and the findings of this study will be covered at some length in the Discussion.

(iii) Respiratory Functioning

As in the case of body weight, lung-functioning has been shown to correlate with cigarette smoking and, specifically, to improve with reduced or estimated smoking (e.g. McCarthy, Craig and Cherniak, 1976;

Paxton and Scott, 1981). Apart from providing a further, objective check on self-reports, this additional molar measure was used, in this study, because of the clear association between improved respiratory functioning and improved physical health (e.g. U.S.D.H.E.W., 1979) and, therefore, the measure's inherent meaningfulness as an indication of the subjects' having benefitted from treatment.

Four separate lung-function measures were, in fact, used. These were a) FEV, (Forced Expiratory Volume ... the volume of air which can be expelled from the lungs in the first second of exhalation, after inhaling fully) b) FVC (Forced Vital Capacity ... the volume of air which can be contained in the lungs, at full inhalation) c) FEV/FVC (the ratio between a) and b)) and, d) T.F. (Carbon monoxide transfer factor ... the rate at which carbon monoxide combines with haemoglobin, in the alveolar capillaries, to produce carboxyhaemoglobin (COHb), this being an index of the diffusing capacity of the alveolar capillary membrane). The first three of these measures were obtained using a Wet Spirometer, similar to the Vitalograph Spirometer described by Drew and Hughes (1969) and the last by a standard infra-red CO analyzer, using the Single Breath technique.

c) Personality Measures

As was concluded in an earlier section, regarding the non-specific factor of "personality" in smoking-cessation research, "the relationship between smoking and personality factors is unclear and confusing. This is ... (an) area where further investigation is needed" (p.101-102).

It was decided to administer, at each assessment point, a battery of personality questionnaires, in the hope that, firstly, certain personality characteristics would be found to predict a positive

response to treatment (or otherwise) and, secondly, that certain changes over time may be apparent in certain "personality" characteristics (or, more accurately, characteristic patterns of behaviour) as a result of modified smoking behaviour. As so many conflicting results have been obtained by past studies, no firm hypotheses were forwarded, as to what findings may emerge.

The specific measures used were :-

(i) The Eysenck Personality Questionnaire (EPQ) (Eysenck and Eysenck, 1975), which yields scores on the dimensions of Extraversion-Introversion, Neuroticism and "Psychoticism" and also provides a "Lie" score.

(ii) The Symptom Check List (SCL) 90 (Derogatis, Lipman and Covi, 1973) a prototype of the SCL-90-R (Derogatis, 1975). This questionnaire yields symptom scores on nine scales (somatic anxiety, obsessive compulsiveness, general anxiety, depression, interpersonal sensitivity, phobic anxiety, hostility, paranoid ideation and psychoticism) and also provides a general index of emotional disturbance (General Symptomatic Index).

(iii) Cattell's 16PF (16 Personality Factor) Questionnaire (IPAT, 1970) (1979 form), which yields scores on 16 personality dimensions (see Appendix III), including intelligence (Factor B), self-sufficiency (Factor Q_2) and level of tension (anxiety) (Factor Q_4).

e) Treatment Techniques

(i) The Self Control Package

As stated in the Review of the Literature (p.76), Lichtenstein and Danaher (1976) identified three types of self-control strategies: environmental planning, behavioural programming and cognitive control. The self-control treatment package used in

this study was comprised, in an effort to be comprehensive, of methods relating to each of these strategies. These methods are listed in Table 3:4 and are described below.

Further, an effort was made to achieve consistency between the methods employed and the "behavioural contingency" model of smoking, proposed by Frederiksen and Simon (1979); this model has been discussed in detail (p.18-21). Figure 3:1 shows how the techniques utilized relate to this model (along with the "therapist-administered" techniques used in the more elaborate, multicomponent package).

ENVIRONMENTAL PLANNING	BEHAVIOURAL PROGRAMMING	COGNITIVE CONTROL
Hierarchical reduction Deprived response performance Contingency contracting ("Social" plus "Therapeutic")	Self-punishment (Monetary deprivation)	Coverant control

Table 3:4

Self-Control Treatment Methods and their relation to different types of Self-Control Strategies

It can be seen from Figure 3:1 that the self-control package addresses not only the smoking event itself, but also its antecedents and consequences, at an overt and covert behavioural level. (The more comprehensive package also deals, it will be noted, with the physiological antecedent of tension/anxiety/craving, as well as adding a further cognitive technique).

BEHAVIOUR	ANTECEDENTS		SMOKING EVENT		CONSEQUENCES
OVERT	Hierarchical Reduction	→	Deprived Response Performance	→	Monetary Deprivation. Contingency-contracting ("Social" plus "Therapeutic")
COVERT	Coverant Control Covert sensitization*	→	-	→	(Coverant Control)
PHYSIOLOGICAL	Focussed Relaxation Training*	→	-	→	-

Figure 3:1

Relationship of treatment methods used to the "behavioural contingency" model of smoking (Frederiksen and Simon, 1979).

(Methods with an asterisk are "therapist-administered")

The individual self-control methods used were :-

a) Hierarchical Reduction

Support for this method, as part of a package treatment, was found by Brockway et al (1977) and, to some extent, by Gutmann and Marston (1967). The technique entails the individual's eliminating smoking in a systematic fashion, in situations where smoking is a high-probability behaviour. Thus, the associational bonds which have developed, over time, between certain environmental stimuli or events and the response of smoking, are weakened. It was believed that the

elimination of smoking in "hard" situations before "easy" would maximise the effectiveness of this method of self-control, as, in the early stages of the treatment programme, subjects' ability to reduce their smoking rate was expected to be higher than in the later, more stressful stages. (Hills (1983) has since provided evidence supporting this particular way of implementing hierarchical reduction as a therapeutic technique). A five-step hierarchy was employed, the details of which are described in "Procedure", below.

b) Deprived Response Performance

This method requires smoking behaviour to be limited to situations which are devoid of inherently stimulating properties, being analagous to the "time-out (from positive reinforcement)" procedure often used in other fields of behaviour modification (e.g. Blackham and Silberman, 1975). Smoking thus comes to be associated with the removal of positive, reinforcing environmental stimuli and becomes, in effect, a behaviour which is thereby punished. As with the method of Hierarchical Reduction, Deprived Response Performance was utilized in an additive fashion in this experiment, the circumstances under which smoking was permitted becoming increasingly limited and free from positive stimulation, as the treatment programme progressed. Again, the implementation of this technique (which has found some support in the literature (Greenberg and Altman, 1976) is described, in detail, under "Procedure".

c) Contingency Contracting

Some support has been found for the usefulness of this technique (see Review of the Literature - p.79). Two types of contracting were used in this experiment. First, whereby subjects were required to sign

a written contract with the therapist, at the initial assessment session, undertaking to attend all assessment and treatment sessions and to comply with all instructions given during treatment, "to the best of their ability". The commitment, on the part of the therapist, was to offer what was believed to be an effective treatment programme designed to help the individual attain the desired smoking target.

Secondly, "social contracting" required each subject to make explicit to friends, relatives and working associates that the intention was to reduce smoking over the coming five weeks (six treatment sessions), with the aim of either attaining total abstinence or reduced smoking (whichever was the individual case) at the end of this period.

The essence of these contracting techniques is that negative consequences are contingent on failure to work towards and to achieve the behavioural target set; in the case of the therapeutic contract, the negative consequences would be the termination of therapy and/or the disapproval of the therapist; in the case of the social contract, embarrassment, shame, peer disapproval, and other aversive consequences would be the outcome.

(A copy of the "therapeutic contract" is included in Appendix III).

d) Monetary Deprivation

This method is firmly based on the operant learning principle that, if a behaviour is consistently punished, that behaviour will, over time, decrease in its frequency/strength. Thus subjects in this study were required to "fine" themselves a pre-determined amount of money, upon smoking each cigarette. The "severity" of the fines increased from week to week, so this technique was designed to become increasingly powerful as the treatment programme progressed. The money accumulated by individuals was, at the end of each week, donated to a

charity of their choice.* Axelrod et al (1974) found support for this technique.

e) Coverant (covert operant) control

This cognitive behavioural technique (based on Premack's differential probability hypothesis - Premack (1965) - and initially described by Homme (1965)) aimed at reducing the frequency of subjects' smoking behaviour essentially by modifying their attitude towards smoking. The method involves the identification of (cognitive) behaviours which are incompatible with the response of smoking, and the systematic strengthening of these behaviours, by making high-probability behaviours contingent on their occurrence. More specifically, subjects were required to use one of a number of pre-determined "anti-smoking" coverants (ideas perceived as aversive in relation to smoking) in response to the urge to smoke, and to immediately follow this with a "pro-non-smoking" coverant (a positive idea related to not smoking); given that the desire to smoke was resisted, these cognitions were then to be positively reinforced by a positive self-statement (such as "I am controlling my smoking well", "I am succeeding with this", etc.), this being a high probability behaviour (increasing in probability as control was achieved) and therefore a suitable reinforcer. Coverant statements were determined by the subjects, not by the experimenter, and were individualized. Figure 3.2 illustrates the coverant control procedure. The successful use of coverant control, as a package component, has been reported by Tooley and Pratt (1967) and Keutzer (1968).

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* It is appreciated that donating this money to a chosen charity would detract from the aversiveness of this technique; however, it was felt to be unethical to instruct subjects to give the money to a charity or organization towards which they felt some antipathy. The fining procedure was nevertheless reported as having aversive properties. (see Watson & Tharp, 1972).

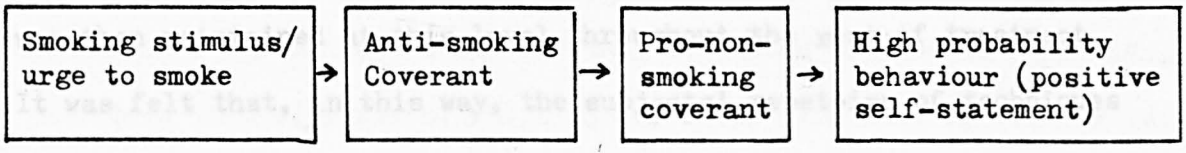

Figure 3.2

The technique of coverant control

(ii) Therapist Administered Techniques

a) Covert Sensitization

This is a cognitive, aversive behaviour modification technique, pioneered by Cautela (Cautela, 1967, 1970, 1971). It entails the imaginal pairing of a noxious stimulus with "approach" behaviours and the pairing of a pleasant stimulus, again imaginally, with "avoidance" behaviours. Support for covert sensitization in the modification of cigarette smoking has been equivocal (positive findings, for example, being reported by Wagner and Bragg (1970), Sachs, Bean and Morrow (1970) and Sipich, Russell and Tobias (1974), but little support being provided by Weiss (1974) or by Wisocki and Rooney (1974)). Lichtenstein and Danaher (1976) remarked, however, that "(although the support for covert sensitization) appears to be relatively weak ... the economy and portability of the procedure suggest ... that it deserves additional empirical study" (p.104-105). More recently, Frederiksen and Simon (1979) suggested that covert sensitization combined with other procedures, in a package, may yield more favourable results; hence the method's use in this study.

This technique was employed here by seeing subjects individually for weekly sessions, the first session being conducted by the experimenter, and subsequent sessions using tape recorded instructions, thus ensuring a standardized treatment format. The "noxiousness" of the aversive

imagery was increased gradually until the third treatment session and was then maintained at this level throughout the rest of treatment. It was felt that, in this way, the subjects' repetoire of techniques to use would be strengthened as treatment progressed and also that this cumulative approach would obviate subjects' satiation to the noxious stimulus presented. A transcript of the covert-sensitization procedure used is reproduced in Appendix IV.

b) Focussed Relaxation Training

This method was used by Ravensborg (1976) with reported success. It was expected that withdrawal from cigarettes would result in an increased level of physiological arousal in subjects (anxiety); a method of relaxation designed to reduce muscular tension was, therefore, believed to be of potential benefit. The technique can be compared to that of Autogenic Relaxation (Schultz, 1959), but differs in that subjects are required to focus specifically on those areas where tension is perceived, as a result of craving for tobacco.

In the present experiment, this technique was used in combination with Progressive Muscular Relaxation (Jacobson, 1938). The initial session (this and all subsequent sessions were conducted individually) entailed the experimenter's instructing the subjects verbally on how to proceed; later sessions, as was the case with covert sensitization, employed tape-recorded instruction, to ensure standardized presentation. (A transcript of the relaxation procedure is reproduced in Appendix IV).

It was concluded, at the end of the Rationale for this study, that the long-term modification of smoking was the primary aim of treatment and that, with this in mind, many of the self-control package elements used were intended to be applicable after the desired target had been

attained. Thus, not only were subjects encouraged to use the methods continually during treatment, but they were also advised to continue to employ them afterwards, where appropriate. The same applied to the "therapist-administered" techniques, in that it was hoped that, as a result of practice - subjects were asked to use these two methods daily, between treatment sessions, and were given written aids for this purpose (see Appendix IV) - the methods could easily be utilized after the formal conclusion of treatment.

f) Procedure

Eight treatment groups comprised this experiment. In order to examine the differential responses of heavy and light smokers to treatment, groups were run concurrently, two at a time, these groups being identical in form (i.e. receiving the same treatment package and having the same treatment goal) but differing in their baseline rates of smoking.

Thus sixteen randomly selected (see section b), above) subjects attended for pre-treatment assessment. The experimenter presented a half-hour standardized talk to this group, based on handouts giving an overview and a cursory description of the treatment programme and its aims. (See Appendix III). Opportunity was then offered for questions to be asked, concerning treatment and assessment. Subjects then gave blood samples, were weighed, and individual appointments were arranged, for some convenient time over the following few days, for respiratory functioning measurement. The personality questionnaires were completed during this session, as were the "expectancy" ratings. Subjects were then seen, individually, by the experimenter and were each given a set of daily record cards (see Appendix III), to be filled in over the next ten days and returned then in a stamped addressed envelope to the

experimenter. These record cards were explained in detail and further opportunity was given for any questions to be asked about the treatment programme. Subjects were urged to attempt to smoke at their usual rate, whilst keeping the record cards; thus it was hoped that the reactivity effects of self-monitoring would be avoided, or, at least, minimized. Subjects read and signed the "Therapeutic Contract" document (see Appendix III).

Addresses and telephone numbers having been obtained, subjects were told that they would be asked to attend for the first group treatment session in approximately twelve days time.

Once the record cards had been returned by all sixteen subjects, this group was divided into two groups of eight, according to baseline smoking frequency (the eight heaviest smokers comprising one group and the eight lightest the other). Subjects were then contacted and asked to attend on either that or the following evening.

(i) Group Treatment. At the first group session, subjects (by then, six per group - see section b), above) were asked to complete the "hierarchy" and "covert statements" lists (for use with the Hierarchical Reduction and Coverant Control treatment techniques, respectively); the elements used here were chosen by each individual subject, with non-directive guidance being given, if necessary, by the experimenter. All self-control techniques were introduced at this stage: coverant control and "social-contracting" were used to their full extent from this first session onwards; hierarchical reduction, deprived response performance and monetary deprivation were commenced at their first level.

At subsequent group meetings, the level of these latter three techniques was increased, as described in "Treatment Techniques" (section e), above), and as detailed in Table 3.5, below.

WEEK	Social Contracting and Therapeutic Contracting	Coverant Control	Hierarchical Reduction	Deprived Response Performance	Monetary Deprivation
1	constant	constant	Eliminate first ("hardest") hierarchy item	No smoking whilst watching T.V., reading, listening to radio, records.	Fine of 1p per cigarette smoked
2	constant	constant	Eliminate second hierarchy item	As above, plus: no smoking in company of other people	Fine of 3p per cigarette smoked
3	constant	constant	Eliminate third hierarchy item	As above, plus: smoking limited to one chosen room (at work and at home.	Fine of 5p per cigarette smoked
4	constant	constant	Eliminate fourth hierarchy item	As above, plus: smoking must be limited to the W.C. (at work & at home)	Fine of 7p per cigarette smoked
5	constant	constant	Eliminate fifth hierarchy item	As above, plus : smoking limited to standing up in the W.C. (at work and at home)	Fine of 10p per cigarette smoked
6	constant	constant	Continue as fifth week	Continue as fifth week	Continue as fifth week

Table 3.5

Weekly phases of self-control treatment

Group meetings always followed the same format. Sessions commenced with subjects completing the weekly "use" and "benefit" ratings and the ratings of benefit obtained, generally, since the preceding meeting. Each subject then informed the group of the progress made over the previous week, with specific reference to the treatment techniques being used. Any difficulties experienced in following instructions were discussed and further advice and guidance was given, when necessary, by the therapist. Successful reduction of smoking and/or adherence to therapeutic instructions was positively reinforced (socially) by the therapist and similar reinforcement was encouraged to be directed towards the individual in question, by other group members.

Therapeutic instructions were repeated, after this feedback phase of treatment, and new instructions given accordingly (i.e. appropriate to the next "level" of self-control). All instructions were reinforced by providing subjects with weekly handouts, detailing their exact programme for the coming week.(See Appendix III). Group sessions lasted between 1½ and 2 hours.

(ii) Individual Treatment. Each subject was seen individually, during the daytime when possible, but during the evening if not, once a week. Those subjects using all treatment methods, (i.e. including the "therapist-administered" techniques) spent this one-hour session receiving direct instruction and training in the use of covert-sensitization and focussed relaxation. As described in section e), above, after the first, introductory session, tape-recorded instruction was utilized, in order to ensure standardized presentation of instructions. (As with the group treatment self-control methods, supplementary handouts were used to reinforce the use of these techniques). The

remaining 15/20 minutes of the individual treatment session was spent in allowing the subject to report on his/her progress in treatment, but an effort was made, on the part of the experimenter, to be non-directive and as passive as possible, during this period of discussion.

N.B. In order to control for the amount of time spent receiving treatment by each subject, those subjects not using the therapist-administered methods were also seen, individually, once a week, for a one-hour session. This period was spent discussing the subject's progress but, again, the experimenter took care to be non-directive and to be passive, as far as this was possible. When appropriate, the subject was encouraged to talk about his/her smoking history and experiences, discussing these issues being seen as unrelated to response to treatment or treatment outcome.

At the end of the five week treatment period (which was comprised of six group and six individual treatment sessions for all subjects), subjects were instructed to attempt to adhere to their individual target levels of smoking. In the case of those subjects reducing their smoking rate by 100%, this target was zero. In the case of those aiming at reduced (controlled) smoking, the target was 25% of their pre-treatment rate; this post-treatment rate differed, of course, between subjects, as did their pre-treatment rates. Subjects were advised to continue to utilize the self-control skills now learned, where necessary or appropriate.

A further set of 10-days record cards were handed out to subjects (as was the case for pre-treatment and mid-treatment assessment) and a time set for attending for post-treatment assessment, when these cards were to be returned to the experimenter for analysis.

(The schedule for assessment and for both group and individual treatment sessions is presented, in detail, in Appendix II).

g) The Control Groups

As illustrated in section a), above, and in Fig. 3.1, "The Experimental Design", two control groups were used in this study. As was the procedure with the experimental groups, subjects were randomly selected from the pool of subjects; it was known, at this time, that each experimental group had consisted of six subjects, so the total number of control subjects selected was twelve.

This group was assessed on all measures, using a procedure identical to that employed with experimental subjects, and the same standardized introductory talk was given. Therapeutic contracts were signed and each subject was given a set of record cards to be filled in over the next ten days, to be then returned to the experimenter. As with the experimental groups, subjects were urged to attempt to smoke at their usual rate during this baseline period.

(For the purposes of comparison and statistical analysis, this group was divided into two equal-sized sub-groups, according to the baseline rate of smoking, in the same way as were the experimental groups, once the daily record-cards had been returned; hence the use of both a "heavy" and a "light" smoking control group in this experiment).

It was explained to the twelve control subjects (individually), that, because it was essential, from an experimental research point of view, to obtain accurate baseline data, the mean of two separate measures would be used for each parameter examined, and so a second assessment session would be held "seven or eight weeks", after the first batch of record cards had been returned. The second set of 10 days' cards were posted to control subjects five weeks after the end

of their baseline data collection period, this being the exact duration of treatment, for the experimental groups. The length of time between "pre-treatment" and "post-treatment" assessments was, for all groups in this study, between 55 and 60 days (this slight variation being a result of accommodating subjects' occasional difficulties in attending for assessment on certain evenings).

It was not considered to be justifiable, ethically, nor, for that matter, particularly necessary, to conduct the equivalent of "mid-treatment" assessment with control subjects. Analysis was thus based on "pre- and post-treatment" equivalent assessments.

Following the two assessment sessions, all control subjects were then treated, outside the context of this research project. For this reason, it was not possible to obtain short or long-term follow-up data on the control groups; witholding treatment for any longer than the period actually employed would have been ethically unacceptable and it is believed that subjects would have had difficulty in accepting such a delay.

Control group characteristics are presented in the Results section of this thesis.

4. HYPOTHESES

All hypotheses are presented in the form of experimental hypotheses, unless otherwise indicated. Where the null hypothesis(es) applies, this is indicated by (N).

(i) Type of treatment

It was hypothesized that those groups receiving treatment consisting of the "self-control" package plus "therapist-administered" methods would obtain greater benefit from treatment than those groups receiving the "self-control" package alone, but that all treatment groups would significantly reduce their rate of smoking, regardless of treatment condition.

(ii) Goals of treatment

It was hypothesized that those groups having a goal of reduced or controlled smoking would be as successful in achieving their goal as would the groups whose desire was to abstain totally from cigarette smoking.

(iii) "Heavy" versus "Light" smokers

It was hypothesized that "heavy" smokers would be less successful in achieving their goal than would "light" smokers.

(iv) Physiological correlates

It was hypothesized that: a) serum thiocyanate (SCN-) levels would be significantly reduced, as a result of treatment, in all groups, but that this reduction would be more pronounced in the groups receiving the "additional" treatment techniques and less pronounced in groups aiming at "controlled" smoking. b) respiratory functioning would significantly improve in all groups, but that this improvement would be more pronounced in those groups receiving the "additional" treatment techniques and less pronounced in those groups aiming at "controlled"

smoking, and, c) all groups would significantly increase in weight as a result of treatment, but that this increase would be more pronounced in those groups receiving the "additional" treatment techniques and less pronounced in those groups aiming at "controlled" smoking.

(v) Personality measures

It was hypothesized that no significant changes would take place over time, as a result of treatment, on any of the personality factors measured. (N).

(vi) Predictive factors

It was hypothesized that, of the measures taken, pre-treatment level of motivation and level of expectancy would significantly and positively correlate with degree of reduction in smoking rate.

It was hypothesized that none of the remaining pre-treatment measures would predict outcome. (N).

(vii) Additional correlates

It was hypothesized that: a) self-reported rate of smoking and serum thiocyanate level measurements would correlate significantly and positively, and, b) baseline (pre-treatment) smoking rate and extent of "internal" smoking (cigarettes smoked as a response to internal rather than external cues) would correlate significantly and positively.

(viii) "Use" and "Benefit" ratings

It was hypothesized that all treatment techniques would be used by subjects to the same degree and that subjects would obtain equal benefit from all treatment techniques (N).

It was further hypothesized that degree of use of and degree of benefit obtained from treatment techniques would be significantly and positively correlated and that rated degree of overall benefit obtained

from treatment would correlate significantly and positively with self-reported reductions in smoking-rate.

(ix) Maintenance of change

It was hypothesized that the benefits obtained from treatment would still be evident at 3 month, 6 month and 1 year follow-up assessment.

(x) Control groups

It was hypothesized that no significant changes would occur within the control groups, on any of the measures taken, between their first and second assessments (N).

5. RESULTS

In this section, groups will often be referred to by number. For definitions, please refer to Table 3.2, which appears in the section "Experimental Design" (p.112).

a) Pre-treatment characteristics of the experimental subjects

The pre-treatment characteristics of the total experimental subject group (N = 48), expressed as means and standard deviations on the various measures taken, are presented in Table 5.1. In keeping with the three types of outcome measure used, the Table is subdivided into three sections - (i) self-report data, (ii) physiological data and (iii) personality data.

Variable	Mean	S.D.
Smoking rate (mean no.cigs/day)	23.60	8.36
Tar intake (mean mg./day)	334.02	142.74
Nicotine intake (mean mg./day)	28.88	11.27
Anxiety rating (mean, 0-5 scale)	1.63	0.72
Craving intensity (mean, 0-5 scale)	2.39	0.68
"Internal" smoking (% tot.cigs.smoked)	72.85	22.86
Expectancy rating (o-10 scale)	8.69	1.75
Motivation score (max. 158) (min. 42)	117.92	9.24

Table 5.1 (i)

Pre-treatment characteristics of the experimental subjects - self-report data (N = 48)

Some cursory comments on Table 5.1 (i) are appropriate here.

As previously mentioned, tar and nicotine intake figures were not measured independently, but were computed from smoking rate and brand of cigarette smoked, using H.D.U.K. tables; these figures are, therefore, only a rough index of intake.

The mean anxiety rating of 1.63 falls between the "totally free from anxiety" and "slightly anxious" points on the five-point rating scale, this being a relatively low mean rating.

The mean "craving-intensity" rating of 2.39 reflects a "slight" to "moderate" degree of craving, according to the scale-point descriptions used. (For "anxiety" and "craving" scale-point descriptions, see Daily Record Card, Appendix III).

The mean expectancy rating of 8.69 is at the upper end of the ten-point rating scale, reflecting a high degree of expectancy in the subject-group.

Variable	Mean	S.D.
SCN-	152.94	37.98
Gross body weight (kg.)	67.51	10.23
Lung function		
(i) FEV_1	89.25	15.08
(ii) FVC	100.02	15.70
(iii) FEV/FVC	71.69	9.88
(iv) TF	87.58	14.55
Age (years)	42.35	10.35

Table 5.1 (ii)

Pre-treatment characteristics of the experimental subjects - physiological data (N = 48)

Commenting briefly on Table 5.1 (ii): the mean serum thiocyanate level of 152.94μmol/litre is comparable with mean levels reported in other studies, for this mean rate of smoking. Butts, Kuehnemannand Widdowson (1974) reported a mean of approximately 152μmol/litre (N = 12) for 20 a day smokers and Vogt et al (1977) a mean of approximately 175μmol/litre for 20+ a day smokers (N not reported). Non-smoker SCN^- levels were reported as being approximately 44μmol/litre (N = 167) and 65μmol/litre (N not reported), respectively.

The mean gross body weight (67.51 kg.) for this age group (mean 42.35 years) conforms closely to the expected average weight for adults (64.4 kg), sexes being equally represented (as they were in this experimental group), (Palmer, 1980).

With regard to the Lung Function measures taken, the FEV_1 mean of 89.25 compares poorly with the expected (healthy) mean of 100; the same is not true with the mean FVC figure of 100.02 (again, the expected mean being 100). The mean FEV/FVC ratio of 71.69, although not "pathological" (70.00 being the commonly accepted cut-off point (Bass, 1974)), is nevertheless relatively low. Cotes (1975) quotes data to show that this ratio is typically 79.00 for smokers and 82.00 for non- smokers (p.368). The mean Co Transfer Factor of 87.58 is rather lower than the expected mean of 100.

Regarding Table 5.1 (iii) overleaf, the mean scores on all EPQ factors are within normal limits, as are the mean (T) scores on all SCL-90 factors (although the mean "somatic anxiety" score of 46.35 on the SCL-90 does approach the T score of 50 - this

Variable	Mean	S.D.
EPQ		
Psychoticism	3.25	1.97
Extraversion	13.08	4.99
Neuroticism	13.19	5.06
Lie Scale	7.06	3.83
SCL-90		
Somatic anxiety	46.35	14.58
Obsessive compulsiveness	40.65	11.69
Interpersonal sensitivity	39.56	10.77
Depression	36.65	10.53
Anxiety	38.71	12.67
Hostility	39.29	13.78
Phobic anxiety	22.81	25.08
Paranoid ideation	33.56	20.75
Psychoticism	34.00	18.30
General symptomatic index	38.40	8.70
Positive symptom distress level	39.75	8.85
Positive symptom total	39.71	9.71
16PF		
Factor A (Reserved/outgoing)	5.5	2.04
Factor B (Intelligence)	7.52	1.64
Factor C (Emotional stability)	4.88	2.13
Factor E (Assertiveness)	6.10	1.95

Table 5.1 (iii)

Pre-treatment characteristics of the experimental subjects - personality data (N = 48)

Variable	Mean	S.D.
Factor F (Sobriety)	5.60	1.88
Factor G (Conscientiousness)	5.23	1.90
Factor H (Shyness)	5.90	2.07
Factor I (Tough mindedness)	5.75	1.90
Factor L (Suspiciousness)	5.63	2.20
Factor M (Imaginativeness)	6.33	1.74
Factor N (Forthrightness)	5.29	1.30
Factor O (Apprehensiveness)	5.56	2.09
Factor Q_1 (Conservatism)	5.69	1.90
Factor Q_2 (Self-sufficiency)	5.04	1.96
Factor Q_3 (Self-control)	4.94	2.03
Factor Q_4 (Tenseness)	6.00	2.32

Table 5.1 (iii) (Contin.)

Pre-treatment characteristics of the experimental subjects personality data (N = 48).

being a 0-100 scale - which is regarded as clinically significant). All 16PF mean sten scores are within normal limits, with the exception of the mean score of 7.52 on Factor B (Intelligence). This shows that the experimental group was, on average, rather more intelligent than the general population.

Sex. The sexes were equally represented in the group of experimental subjects, 47.09% of the subjects being male and 52.91% female. (Chi-square (X^2) = 0.08, df = 1, N.S*).

* Here, and throughout this section, a "significant" level of probability will be considered as being $p \leq 0.05$. Where $p > 0.05$ the term "N.S." will be used to denote non-significance.

b) Pre-treatment comparison of experimental groups on the measures employed

A one-way analysis of variance was performed to examine the equality of the experimental groups on each measure taken pre-treatment.

As the eight treatment groups were derived from four larger groups (see Procedure, Section 3 f), above), these four groups later being divided into "heavy" versus "light" smokers, analysis was performed on the four groups of 12 subjects, for the variables of base-rate smoking, tar intake and nicotine intake. All eight groups were compared in the case of the remaining measures.

Where significant differences between group means were found, the Tukey (a) Test, a multiple comparison test, was employed, in order to ascertain which group means were reliably different.

(i) Self-report measures

(a) Smoking rate

The four treatment groups did not differ significantly on pre-treatment smoking rate ($F\ (3,44) = 0.73$ N.S.). (See Table 5.2) (Anova Table 1; Appx. VI).

Group	1 (subgroups 1 & 3)	2 (subgroups 2 & 4)	3 (subgroups 5 & 7)	4 (subgroups 6 & 8)
Mean daily no. smoked	24.17	25.92	20.92	23.42
S.D.	9.69	8.36	8.96	6.35

Table 5.2

Comparison of the four main treatment groups on pre-treatment smoking rate

(b) Tar intake

The four treatment groups did not differ significantly on estimated pre-treatment tar-intake ($F(3,44) = 0.09$ N.S.). (See Table 5.3) (ANOVA Table 2; Appx. VI)

Group	1 (subgroups 1 & 3)	2 (subgroups 2 & 4)	3 (subgroups 5 & 7)	4 (subgroups 6 & 8)
Mean daily tar intake (mg.)	342.83	347.17	319.75	326.33
S.D.	132.52	142.07	192.90	107.41

Table 5.3

Comparison of the four main treatment groups on estimated pre-treatment tar intake

(c) Nicotine intake

The four treatment groups did not differ significantly on estimated pre-treatment nicotine intake ($F(3,44) = 0.57$ N.S.). (See Table 5.4) (ANOVA Table 3; Appx. VI)

Group	1 (subgroups 1 & 3)	2 (subgroups 2 & 4)	3 (subgroups 5 & 7)	4 (subgroups 6 & 8)
Mean daily nicotine intake (mg.)	29.58	29.75	25.75	30.92
S.D.	11.27	11.51	13.34	9.23

Table 5.4

Comparison of the four main treatment groups on estimated pre-treatment nicotine intake

(d) Anxiety ratings

The eight treatment groups differed significantly on pre-treatment anxiety ratings ($F(7,40) = 2.45$. $p < 0.05$). Further analysis, using the Tukey (a) Test, showed Group 6 to be reliably higher on anxiety than Groups 3 and 5 (HSD = 119.23 $p < 0.05$) (See Table 5.5) (ANOVA Table 4; Appx.VI)

Group	1	2	3	4	5	6	7	8
Mean Anxiety Rating (1-5 scale)	1.38	1.91	1.18	1.50	1.30	2.48	1.54	1.72
S.D.	0.29	1.06	0.19	0.36	0.41	0.99	0.51	0.76

Table 5.5

Comparison of the treatment groups on pre-treatment anxiety ratings

(e) Craving Intensity

The eight treatment groups did not differ significantly on pre-treatment craving intensity ($F(7,40) = 1.02$ N.S.). (See Table 5.6) (ANOVA Table 5; Appx. VI)

Group	1	2	3	4	5	6	7	8
Mean craving intensity (1-5 scale)	2.20	2.58	2.13	2.18	2.23	2.97	2.42	2.45
S.D.	0.70	0.85	0.59	0.64	0.35	0.91	0.75	0.51

Table 5.6

Comparison of the treatment groups on pre-treatment craving intensity

(f) "Internal/External" Smoking

The eight treatment groups did not differ significantly on the variable of "Internal" vs "External" smoking, at pre-treatment. ($F(7,40) = 0.50$ N.S.). (See Table 5.7) (ANOVA Table 6; Appx. VI)

Group	1	2	3	4	5	6	7	8
Mean % "internal" smoking	64.33	81.17	76.33	62.83	69.83	80.33	72.17	75.33
S.D.	31.51	19.45	26.63	25.81	7.96	30.74	18.15	20.15

Table 5.7

Comparison of the treatment groups on pre-treatment "internal" vs. "external" smoking

(g) Expectancy

The eight treatment groups did not differ significantly on level of expectancy at pre-treatment ($F(7,40) = 1.13$ N.S.) (See Table 5.8) (ANOVA Table 7; Appx. VI)

Group	1	2	3	4	5	6	7	8
Mean expectancy (0-10 scale)	9.33	9.00	9.50	8.67	9.50	7.83	8.50	7.50
S.D.	1.03	1.26	0.84	1.86	0.84	1.94	2.51	2.54

Table 5.8

Comparison of treatment groups on pre-treatment level of expectancy

(h) Motivation

The eight treatment groups did not differ significantly on level of motivation at pre-treatment ($F(7,40) = 1.46$ N.S.) (See Table 5.9) (ANOVA Table 8; Appx. VI)

Group	1	2	3	4	5	6	7	8
Mean motivation score	117.83	125.00	111.83	115.83	115.67	118.17	126.83	112.17
S.D.	4.83	6.87	9.22	7.22	10.98	6.79	4.54	12.50

Table 5.9

Comparison of treatment groups on pre-treatment level of motivation

(ii) Physiological Measures

(a) Serum thiocyanate (SCN^-)

The eight treatment groups did not differ significantly on SCN^- levels at pre-treatment ($F(7,40) = 0.65$ N.S.) (See Table 5.10) (ANOVA Table 9; Appx. VI)

Group	1	2	3	4	5	6	7	8
Mean SCN^- (μmol/litre)	132.00	142.00	149.33	131.50	147.50	125.33	124.33	111.50
S.D.	22.93	45.46	34.15	69.38	35.45	41.53	15.37	19.53

Table 5.10

Comparison of treatment groups on pre-treatment SCN^- levels

(b) Gross body-weight

The eight treatment groups did not differ significantly on the measure of gross body-weight at pre-treatment ($F(7,40) = 1.88$ N.S.) (See Table 5.11) (ANOVA Table 10; Appx. VI).

Group	1	2	3	4	5	6	7	8
Mean weight (Kg.)	64.62	77.88	61.52	72.42	67.48	65.22	62.88	68.08
S.D.	10.66	12.88	4.50	10.04	8.98	7.37	11.19	8.88

Table 5.11

Comparison of treatment groups at pre-treatment on gross body-weight

(c) Lung function

(ii) FEV_1

The eight treatment groups did not differ significantly on the measure of FEV_1 at pre-treatment ($F(7,40) = 1.05$ N.S.). (See Table 5.12) (ANOVA Table 11; Appx. VI)

Group	1	2	3	4	5	6	7	8
Mean FEV_1	81.83	83.67	84.33	94.67	86.33	98.33	96.00	88.83
S.D.	14.93	15.60	19.84	9.37	21.29	9.93	12.43	12.32

Table 5.12

Comparison of the treatment groups at pre-treatment on FEV_1.

(ii) FVC

The eight treatment groups did not differ significantly on the measure of FVC at pre-treatment ($F(7,40) = 0.79$ N.S.) (See Table 5.13) (ANOVA Table 12; Appx. VI)

Group	1	2	3	4	5	6	7	8
Mean FVC	97.50	97.17	97.83	100.50	91.83	109.33	107.67	98.33
S.D.	20.20	8.13	23.73	9.35	19.83	13.24	8.33	16.45

Table 5.13

Comparison of treatment groups at pre-treatment on FVC

(iii) FEV/FVC

The eight treatment groups did not differ significantly at pre-treatment on the measure of FEV/FVC ($F(7,40) = 0.91$ N.S.) (See Table 5.14).(ANOVA Table 13; Appx. VI)

Group	1	2	3	4	5	6	7	8
Mean FEV/FVC	65.50	69.17	74.67	79.00	73.00	74.00	71.00	70.50
S.D.	13.08	9.09	16.95	7.18	7.07	7.80	7.27	10.21

Table 5.14

Comparison of treatment groups at pre-treatment on FEV/FVC

(iv) CO Transfer Factor

The eight treatment groups did not differ significantly at pre-treatment on the measure of CO Transfer Factor (F (7,40) = 0.87 N.S.) (See Table 5.15) (ANOVA Table 14; Appx.VI).

Group	1	2	3	4	5	6	7	8
Mean CO Transfer Factor	78.50	92.17	83.17	97.00	87.17	87.83	89.17	85.67
S.D.	13.90	12.22	19.17	11.26	9.70	11.62	17.07	19.25

Table 5.15

Comparison of treatment groups at pre-treatment on CO Transfer Factor

(iii) Personality measures

(a) Eysenck Personality Questionnaire (EPQ)

Pre-treatment between group comparisons are detailed in Table 5.16 (i) (p.153).

It can be seen from this table that the treatment groups did not differ significantly on any of the EPQ personality factors at pre-treatment. (ANOVA Tables 15-18; Appx. VI)

(b) SCL-90

Pre-treatment between group comparisons are detailed in Table 5.16 (ii). (p.154)

It can be seen that the treatment groups did not differ significantly on any of the SCL-90 personality factors at pre-treatment (ANOVA Tables 19-30; Appx. VI).

Variable	GRP.1		GRP.2		GRP.3		GRP.4		GRP.5		GRP.6		GRP.7		GRP.8		
	Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.	Comparison
Psychoticism	3.17	2.93	4.33	2.50	3.83	2.04	2.67	1.63	2.83	0.75	3.50	2.07	2.67	1.75	3.00	2.00	F (7,40) = 0.51 N.S.
Extraversion	13.50	7.01	15.83	3.19	11.83	5.71	12.67	5.39	14.50	4.04	12.67	4.97	14.00	3.85	9.67	5.32	F (7,40) = 0.81 N.S.
Neuroticism	13.67	5.89	13.33	2.58	9.83	4.62	13.33	4.80	11.00	6.10	16.33	4.50	15.00	6.16	13.00	4.86	F (7,40) = 0.99 N.S.
Lie Score	7.50	2.17	3.17	2.04	8.00	2.83	7.67	5.01	7.33	4.37	7.17	3.55	6.67	5.05	9.00	3.74	F (7,40) = 1.25 N.S.

Table 5.16 (i)

Comparison of treatment groups at pre-treatment on the EPQ factors

Variable	GRP.1		GRP.2		GRP.3		GRP.4		GRP.5		GRP.6		GRP.7		GRP.8		
	Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.	Comparison
Somatic Anxiety	52.50	9.14	54.17	10.98	35.67	18.92	48.00	8.92	45.83	6.49	43.50	22.12	45.50	4.18	45.67	23.15	F (7,40) = 0.90 N.S.
Obsessive Compulsiveness	41.00	9.86	45.83	8.38	34.67	5.82	43.00	4.47	38.17	19.95	36.67	18.07	40.33	3.83	45.50	13.67	F (7,40) = 0.68 N.S.
Interpersonal Sensitivity	43.17	8.04	43.50	5.32	35.83	4.07	41.17	8.40	35.17	17.76	40.33	5.54	40.00	6.36	37.33	20.95	F (7,40) = 0.48 N.S.
Depression	41.83	9.95	42.67	8.31	31.00	4.56	35.83	7.41	33.00	9.14	41.17	11.30	28.67	14.18	39.00	12.17	F (7,40) = 1.69 N.S.
Anxiety	42.00	9.80	42.67	6.47	31.00	16.30	41.33	7.94	35.50	19.22	41.83	3.65	38.00	6.75	37.33	21.83	F (7,40) = 0.58 N.S.
Hostility	41.83	8.75	45.83	7.39	24.67	20.44	44.17	6.05	36.17	18.79	43.67	4.55	40.67	5.61	37.33	20.19	F (7,40) = 1.58 N.S.
Phobic Anxiety	19.50	30.80	24.17	26.51	17.00	20.84	24.50	27.51	21.67	23.75	20.17	31.24	21.67	23.75	33.83	27.36	F (7,40) = 0.22 N.S.
Paranoid Ideation	47.50	8.57	28.67	23.75	32.33	16.22	37.50	18.93	31.00	24.81	42.50	5.21	20.83	22.84	28.17	31.78	F (7,40) = 1.02 N.S.
Psychoticism	39.17	21.33	41.50	5.86	22.83	19.13	30.67	16.69	27.67	22.43	35.50	18.58	42.00	2.83	32.67	27.68	F (7,40) = 0.81 N.S.
General Symptomatic Index	42.17	10.07	42.83	7.31	32.00	4.24	39.00	5.29	34.17	10.61	40.67	5.47	35.00	3.90	41.33	14.56	F (7,40) = 1.42 N.S.
Positive symptom distress level	43.00	9.23	45.67	12.13	35.00	5.33	37.83	5.19	38.50	6.22	44.00	9.49	32.33	5.47	41.67	10.52	F (7,40) = 1.85 N.S.
Positive symptom total	43.83	9.13	42.17	3.82	33.33	5.39	39.67	8.80	35.67	13.89	40.67	3.01	38.83	7.00	43.50	17.77	F (7,40) = 0.85 N.S.

Table 5.16 (ii)

Comparison of treatment groups at pre-treatment on SCL-90 factors

Variable	GRP.1		GRP.2		GRP.3		GRP.4		GRP.5		GRP.6		GRP.7		GRP.8		
	Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.	Comparison
Factor A	4.33	2.07	5.17	0.75	3.50	2.59	3.50	2.17	5.17	2.14	6.17	2.32	4.83	1.47	3.33	1.51	F (7,40) = 1.61 N.S.
Factor B	6.00	1.79	7.33	1.51	6.00	2.10	5.83	2.14	7.17	1.17	6.50	1.38	7.17	0.75	6.17	1.94	F (7,40) = 0.82 N.S.
Factor C	3.17	1.17	3.33	1.37	6.50	1.76	3.00	0.89	4.50	1.87	2.33	1.21	3.67	2.58	4.50	3.15	F (7,40) = 2.79 p<0.025
Factor E	6.33	1.75	6.33	2.25	4.50	2.07	4.33	1.86	4.67	1.97	4.50	2.07	5.17	1.47	5.00	2.00	F (7,40) = 1.03 N.S.
Factor F	4.17	2.32	5.83	1.47	4.00	2.00	3.83	1.60	5.17	1.17	4.83	1.60	5.00	2.53	4.00	2.10	F (7,40) = 0.84 N.S.
Factor G	5.17	1.83	3.33	1.86	3.33	2.25	5.17	2.40	4.00	1.90	4.50	1.52	4.50	1.52	3.83	1.94	F (7,40) = 0.86 N.S.
Factor H	5.00	2.53	5.83	2.48	4.17	1.47	5.00	2.28	5.67	0.82	4.17	2.14	5.50	2.26	3.83	2.23	F (7,40) = 0.79 N.S.
Factor I	5.00	1.79	4.00	1.79	5.67	1.86	5.50	1.64	4.67	2.25	3.17	2.32	4.83	2.23	5.00	0.63	F (7,40) = 1.11 N.S.
Factor L	5.67	0.82	6.00	1.67	3.17	1.72	4.00	3.03	4.67	2.34	3.50	1.76	4.67	2.58	5.33	2.42	F (7,40) = 1.36 N.S.
Factor M	4.67	1.37	5.67	1.75	6.33	2.34	5.00	1.10	5.83	1.72	5.67	1.63	5.00	1.67	4.50	2.26	F (7,40) = 0.77 N.S.
Factor N	4.33	0.82	4.17	1.72	4.33	1.03	5.00	1.10	4.00	1.26	3.83	1.60	4.00	1.41	4.67	1.63	F (7,40) = 0.48 N.S.
Factor O	4.50	3.21	5.00	1.67	3.83	2.14	4.83	0.75	4.83	2.48	4.67	2.07	4.00	2.37	4.83	2.32	F (7,40) = 0.22 N.S.
Factor Q_1	4.83	0.75	5.83	2.14	5.33	1.51	4.17	1.72	3.67	2.42	5.83	0.75	4.33	2.25	3.50	2.26	F (7,40) = 1.50 N.S.
Factor Q_2	4.33	0.82	2.00	1.41	5.17	1.60	3.83	1.72	3.67	1.51	3.83	3.19	3.83	1.47	5.67	1.75	F (7,40) = 2.23 N.S.
Factor Q_3	4.33	2.50	2.33	1.03	4.50	1.97	3.67	2.07	4.83	2.23	3.50	2.07	3.50	2.17	4.83	1.72	F (7,40) = 1.08 N.S.
Factor Q_4	5.67	1.21	7.33	1.21	3.50	2.26	5.33	1.97	4.33	1.97	4.83	2.14	5.00	3.10	4.00	2.97	F (7,40) = 1.72 N.S.

Table 5.16 (iii)

Comparison of treatment groups at pre-treatment on the 16PF factors

(c) 16PF

Pre-treatment between group comparisons are detailed in Table 5.16 (iii) (p.155).

It can be seen from this table that the eight treatment groups differed significantly on only one of the 16PF factors - Factor C (Emotional Stability). Further analysis, using the Tukey (a) Test, ascertained that the mean Factor C score of Group 3 was reliably higher than all other treatment groups means ($p < 0.05$), this group thus being more "emotionally stable". (ANOVA Tables 31-46; Appx.VI).

Age The eight treatment groups did not differ significantly on the variable of age ($F(7,40) = 0.58$ N.S.) (See Table 5.17) (ANOVA Table 47; Appx VI)

Group	1	2	3	4	5	6	7	8
Mean Age (years)	45.67	38.67	40.33	39.83	46.83	39.17	41.33	45.33
S.D.	10.31	3.50	12.56	7.73	10.32	8.68	13.50	13.47

Table 5.17

Comparison of treatment groups on the variable of age

c) Pre-treatment comparison of experimental and control groups

A one-way analysis of variance was performed to examine the equality of treatment groups with control groups at pre-treatment.

As two control groups were used, a "heavy" and a "light" group, the former was compared to the "heavy" smoking experimental groups and the latter to the "light" smoking experimental groups.

Where statistically significant differences between group means were found, further analysis was conducted, using the Tukey (a) Test, to ascertain which group means were reliably different.

Table 5.18, below, shows the F ratios and significance levels obtained for each variable measured. This table is divided into three sections ((i), (ii) and (iii)) according to the type of measure taken (self report, physiological and personality).

	"Light" smokers. Control Grp.1 & Grps.1, 2, 5, 6.		"Heavy" smokers. Control Grp.2 & Grps.3, 4, 7, 8.	
Variable	F(4,25)	Sig.	F(4,25)	Sig.
Smoking rate	1.42	N.S.	1.72	N.S.
Tar intake	0.32	N.S.	0.51	N.S.
Nicotine intake	1.05	N.S.	0.50	N.S.
Anxiety rating	3.68	$p<0.025$	1.96	N.S.
Craving intensity	0.93	N.S.	0.41	N.S.
Internal/External	0.52	N.S.	0.49	N.S.
Expectancy	1.71	N.S.	0.80	N.S.
Motivation	2.75	N.S.	3.75	$p<0.025$

Table 5.18 (i)
Comparison of experimental and control groups at pre-treatment on self-report measures

It can be seen from Table 5.18 (i) that the experimental and control groups differed significantly at pre-treatment on only two self-reported measures, rated anxiety and motivation.

Tukey's (a) Test showed that Groups 2 and 6 scored reliably higher on the measure of anxiety than did Groups 1 and 5 and Control Group 1 ($p < 0.05$) and that Group 7 obtained a higher "motivation" score than did all other "heavy" smoking groups, including Control Group 2 ($p < 0.05$).

	"Light" smokers. Control Grp.1 & Grps.1, 2, 5, 6.		"Heavy" smokers Control Grp. 2 & Grps.3, 4, 7, 8.	
Variable	F (4,25)	Sig.	F (4,25)	Sig.
SCN^-	0.54	N.S.	0.74	N.S.
Gross body weight	2.13	N.S.	1.30	N.S.
FEV_t	1.09	N.S.	0.74	N.S.
FVC	1.63	N.S.	0.49	N.S.
FEV/FVC	1.39	N.S.	1.11	N.S.
TF	1.12	N.S.	1.18	N.S.
Age	1.23	N.S.	0.38	N.S.

Table 5.18 (ii)

Comparison of experimental and control groups at pre-treatment on physiological measures

It can be seen from Table 5.18 (ii) that the experimental and control groups did not significantly differ on any of the physiological measures taken at pre-treatment.

	"Light" smokers Control Grp.1 & Grps.1, 2, 5, 6		"Heavy" smokers Control Grp.2 & Grps.3, 4, 7, 8	
Variable	F (4,25)	Sig.	F (4,25)	Sig.
EPQ				
Psychoticism	2.75	N.S.	0.39	N.S.
Extraversion	0.92	N.S.	0.63	N.S.
Neuroticism	2.31	N.S.	1.45	N.S.
Lie Score	2.18	N.S.	0.44	N.S.
SCL-90				
Somatic anx.	1.00	N.S.	0.81	N.S.
Obs.Comp.	0.46	N.S.	1.71	N.S.
Interp.Sens.	1.31	N.S.	0.35	N.S.
Depression	1.50	N.S.	1.55	N.S.
Anxiety	0.52	N.S.	0.60	N.S.
Hostility	1.42	N.S.	1.31	N.S.
Phobic anxiety	0.89	N.S.	0.46	N.S.
Paranoid ideation	1.41	N.S.	1.26	N.S.
Psychoticism	1.17	N.S.	0.86	N.S.
Gen.Symp.Index	1.47	N.S.	1.44	N.S.
Pos.Symp Dist. Level	1.03	N.S.	2.62	N.S.
Pos.Symp.Total	1.24	N.S.	0.83	N.S.

Table 5.18 (iii)

Comparison of experimental and control groups at pre-treatment on personality measures

	"Light" smokers Control Grp.1 & Grps.1, 2, 5, 6		"Heavy" smokers Control Grp.2 & Grps.3, 4, 7, 8	
Variable	F (4,25)	Sig.	F (4,25)	Sig.
16PF				
Factor A	1.22	N.S.	1.30	N.S.
Factor B	1.88	N.S.	0.53	N.S.
Factor C	2.22	N.S.	2.75	N.S.
Factor E	1.42	N.S.	0.37	N.S.
Factor F	1.94	N.S.	0.32	N.S.
Factor G	1.30	N.S.	0.81	N.S.
Factor H	0.88	N.S.	0.65	N.S.
Factor I	0.88	N.S.	0.27	N.S.
Factor L	2.03	N.S.	0.80	N.S.
Factor M	0.49	N.S.	1.11	N.S.
Factor N	0.11	N.S.	0.45	N.S.
Factor O	0.31	N.S.	0.40	N.S.
Factor Q_1	2.41	N.S.	0.75	N.S.
Factor Q_2	4.38	$p<0.01$	1.65	N.S.
Factor Q_3	1.59	N.S.	0.56	N.S.
Factor Q_4	2.26	N.S.	0.67	N.S.

Table 5.18 (iii) (contin.).

Comparison of experimental and control groups at pre-treatment on personality measures

It can be seen from Table 5.18 (iii) that the experimental and control groups differed significantly on only one of the personality measures taken at pre-treatment, 16PF Factor Q_2 - "self-sufficiency". Analysis using the Tukey (a) Test showed

that the mean Q_2 score for Control Group 1 was reliably higher than the mean score of Group 2 ($p < 0.05$).

d) Outcome Measures*

Initially, a two-way analysis of variance with repeated measures (ANOVA R-II) (Meyers and Grossen, 1974) was used to assess within-group changes over time on the measures taken and to examine inter-group differences.

(In the case of significant group differences emerging from the above analysis, suggesting that the three main factors being examined - type of treatment, baseline smoking rate and treatment goal - may be responsible for differential treatment effects, it was intended that more detailed analyses (further analysis of variance performed on collapsed group means) be conducted, to clarify this relationship. In the event, on none of the post-baseline measures were any salient significant group differences found, and so additional analysis proved to be unnecessary).

As the analysis was performed by computer, it was possible to employ an exact analysis of variance by creating dummy covariates to represent missing values (Bartlett, 1937); this technique yields more reliable data than does the "inexact" method of filling missing value cells by reference to the means of the data available.

Where significant differences between means were found, further analysis was conducted using the Tukey (a) Test, in order to establish which means were reliably different.

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*The results pertaining to the outcome measures are presented in summarized form at the end of this sub-section (Table 5.24.).

In all cases, measures were taken on six occasions (pre-treatment, mid-treatment, post treatment and at 3, 6 and 12 month follow-up).

(i) Self-Report Measures

(a) Smoking rate

(As four of the eight treatment groups in this study were attempting to reduce their smoking rate to 25% of baseline rather than aiming to abstain completely from smoking , to use the "raw" smoking rate (i.e. number of cigarettes smoked) as the datum for analysis would have been inappropriate; comparisons between "abstainers" and "reducers" would have been invalid.

Instead, the datum used for each subject, at each assessment point, was the degree of movement in the direction of the goal of that particular subject, expressed as a percentage. Thus, a "score" of 100(%) for a subject in an "abstaining" group would indicate that that subject had stopped smoking completely (i.e. a rate of zero), whereas a score of 100(%) for a subject in a "reducing" group would indicate that the subject was smoking at a rate of 25% of his initial baseline level; as "reducing" individuals' baselines differed, so did their target levels of smoking. "Scores" obtained by subjects could be either positive or negative, a negative value representing the subject's smoking at a rate greater than that reported at pre-treatment assessment.*).

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* To illustrate this method of obtaining rate data: if Subject "A", for example, were aiming at abstaining from smoking and had a pre-treatment smoking rate of 32 cigarettes a day and a 6 month follow-up rate of 18 a day, his obtained "score" would be: 14/32 X 100/1 = 43.75 (i.e. a 43.75% movement in the direction of his target). Subject "R", aiming at reducing smoking to 25% of his baseline rate,

There were no significant differences between groups, in the degree of movement towards target, over all assessment points ($F(7,28) = 1.08$ N.S.) (See Fig. 5.1 p. 164).

For all groups combined, a significant change in smoking rate occurred over time ($F(5,144) = 39.85$ $p < 0.001$). The Tukey (a) Test showed reliable differences between the pre-treatment and mid-treatment means (decrease in rate), the mid-treatment and post treatment means (decrease in rate) and the post-treatment and 3-month follow-up means (increase in rate), all these differences being significant at the $p < 0.01$ level. Non-significant increases in rate occurred from 3 to 6 month follow-up and from 6 to 12 month follow-up ($p > 0.05$), but the increase from 3 to 12 month follow-up was statistically significant (Tukey (a) $p < 0.01$). (See Figure 5.2 p. 164)

There was no significant groups x time interaction effect ($F(35,144) = 1.20$ N.S.). (ANOVA Table 48; Appx. VI).

(b) Abstinence rates for subjects attempting to abstain

Twenty-four subjects (four of the eight treatment groups) were attempting to abstain from smoking. Table 5.19 shows the number of subjects (and the percentage of the total number) abstaining at each of the six assessment points. Figure 5.3, below, further illustrates these abstinence rates. (See over). There were no significant differences, over all assessment points between groups, with regard to abstinence rate ($F(3,15) = 2.05$ N.S.)

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and smoking at these same rates, would obtain a "score" of: $14/(32 - (32/4)) \times 100/1 = 14/24 \times 100/1 = 58.33$ (i.e. a 58.33% movement in the direction of his target). For the convenience of analysis, figures thus obtained were rounded to the nearest whole per-cent.

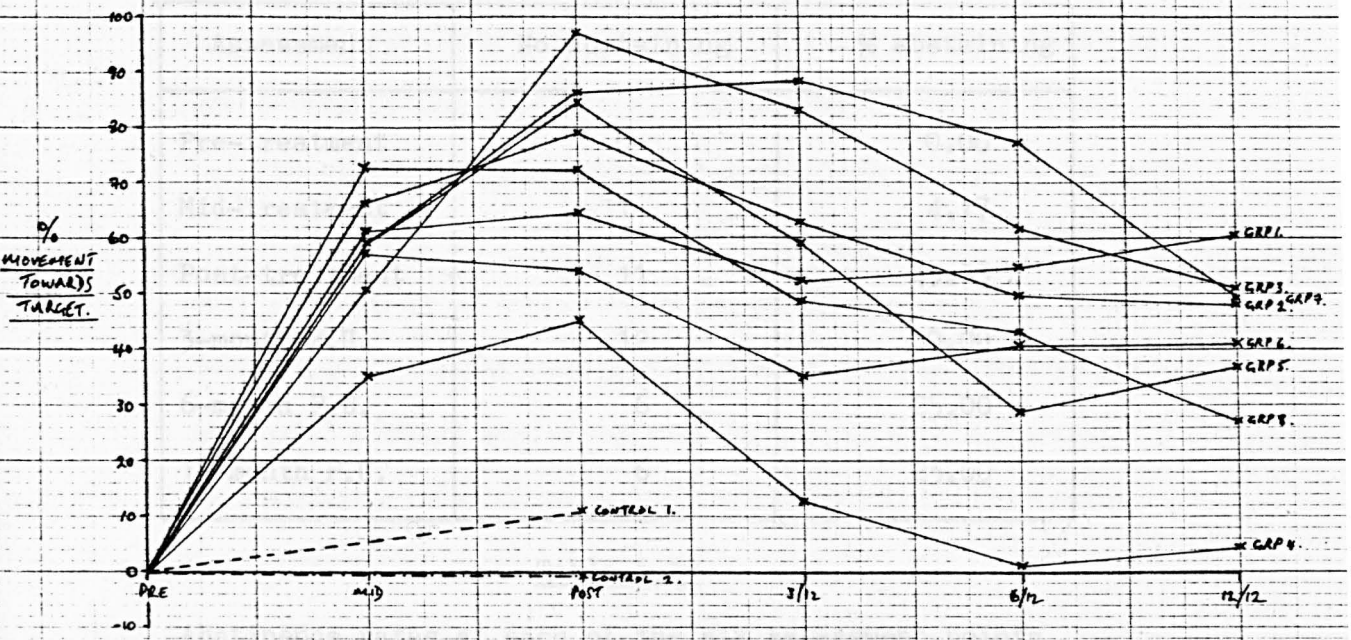


FIG. 5.1

INDIVIDUAL GROUPS' SMOKING RATES: DEGREE OF MOVEMENT TOWARDS TARGET-RATE.

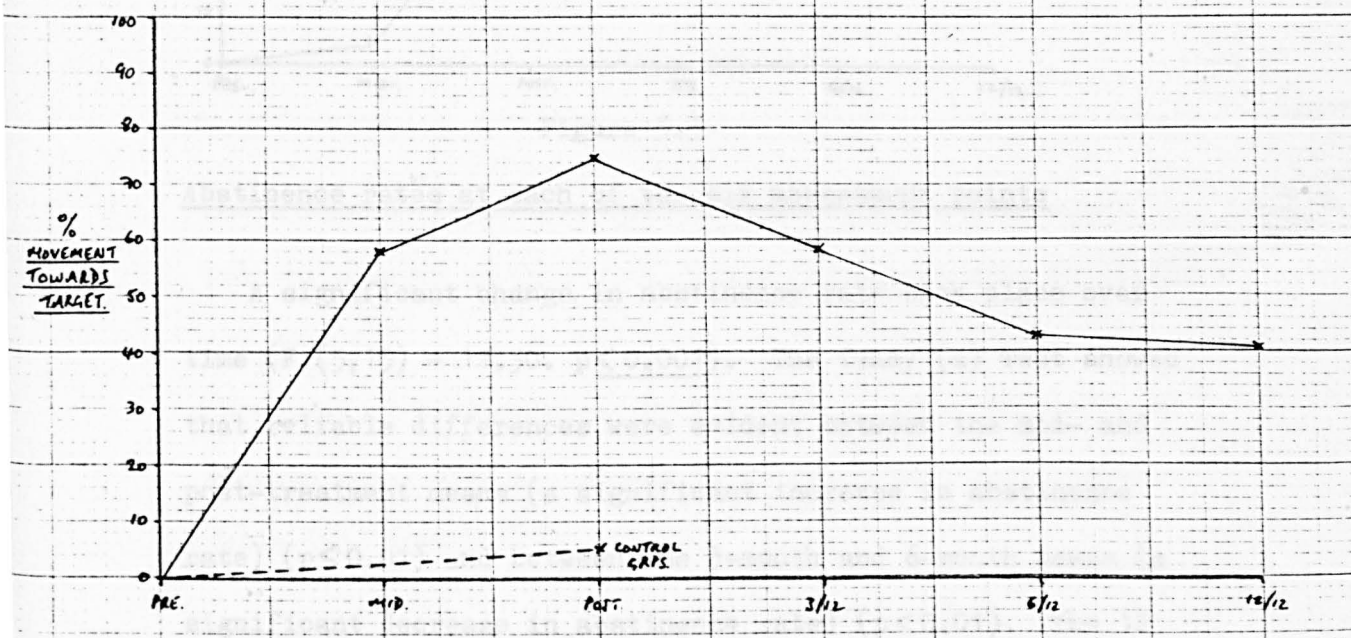


FIG. 5.2.

SMOKING RATE : DEGREE OF MOVEMENT TOWARDS TARGET-RATE.
(ALL GROUPS COMBINED.)

Assessment	No.abstaining	% abstaining
Pre-treatment	0	0.00
Mid-treatment	1	4.17
Post-treatment	11	45.83
3-month F.U.	12	50.00
6-month F.U.	6	25.00
12-month F.U.	6	25.00

Table 5.19

Abstinence rates at each of the six assessment points

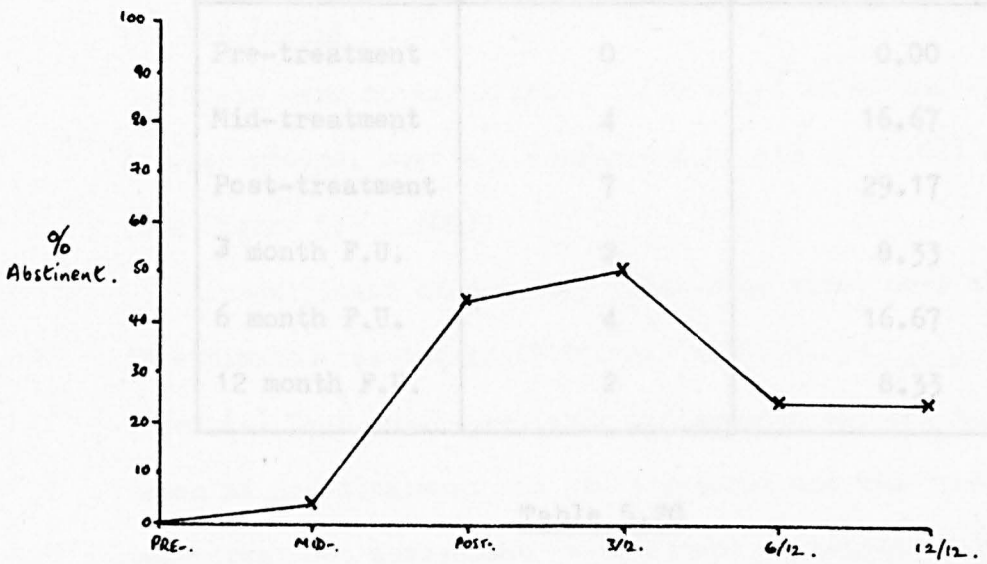


Figure 5.3

Abstinence rates at each of the six assessment points

A significant change in abstinence rate took place over time ($F(5,15) = 11.30$. $p \leq 0.001$). The Tukey (a) Test showed that reliable differences were evident between the mid- and post-treatment means (a significant increase in abstinence rate) ($p \leq 0.01$) and between the 3-month and 6-month means (a significant decrease in abstinence rate) ($p \leq 0.01$). The 12

month follow-up mean was still significantly higher than the pre-treatment mean ($p < 0.01$). (ANOVA Table 49; Appx. VI).

(c) "Success" rates for subjects attempting to reduce smoking.

Twenty-four subjects (four of the eight treatment groups) were attempting to reduce smoking to 25% (or less) of baseline. Table 5.20 shows the number of subjects (and the percentage of the total number) achieving this target at each of the six assessment points. Figure 5.4, below, further illustrates these "success" rates.

Assessment	No.Successful	% Successful
Pre-treatment	0	0.00
Mid-treatment	4	16.67
Post-treatment	7	29.17
3 month F.U.	2	8.33
6 month F.U.	4	16.67
12 month F.U.	2	8.33

Table 5.20

"Success" rates for reducers at each of the six assessment points

There were no significant differences, over all assessment points, between groups, with regard to success rate ($F(3,15) = 2.42$ N.S.)

There was no statistically significant change, over time, in success rate, over all groups ($F(5,15) = 2.53$ N.S.) (ANOVA Table 50; Appx.VI).

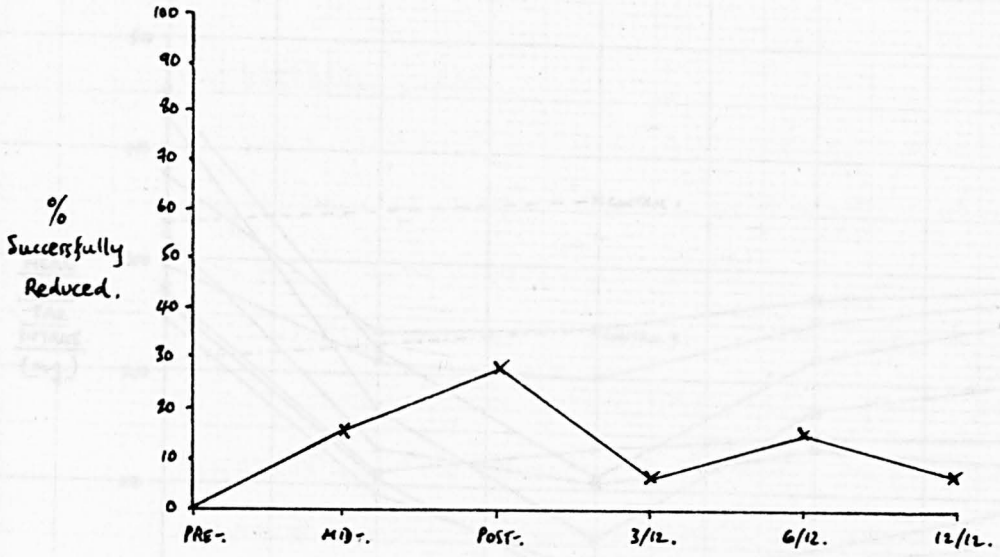


Figure 5.4

"Success" rates for reducers at each of the six assessment points

(d) Tar Intake

There were no significant differences in estimated tar-intake between groups, over all assessment points ($F(7,28) = 1.65$ N.S.) (See Figure 5.5 p.168).

A significant change took place over time, over all groups, in estimated tar-intake ($F(5,144) = 30.96$. $p \leq 0.001$). The Tukey (a) Test showed reliable differences between tar-intake means at pre-treatment and mid-treatment and mid-treatment and post-treatment assessment (significant decreases in intake) ($p < 0.01$), and between post-treatment and 6-month follow-up assessment (a significant increase) ($p < 0.01$). The increase from 6 month to 12 month follow-up was non-significant. (See Figure 5.6, p.168).

There was no significant groups x time interaction effect ($F(35,144) = 1.21$ N.S.) (ANOVA Table 51; Appx. VI).

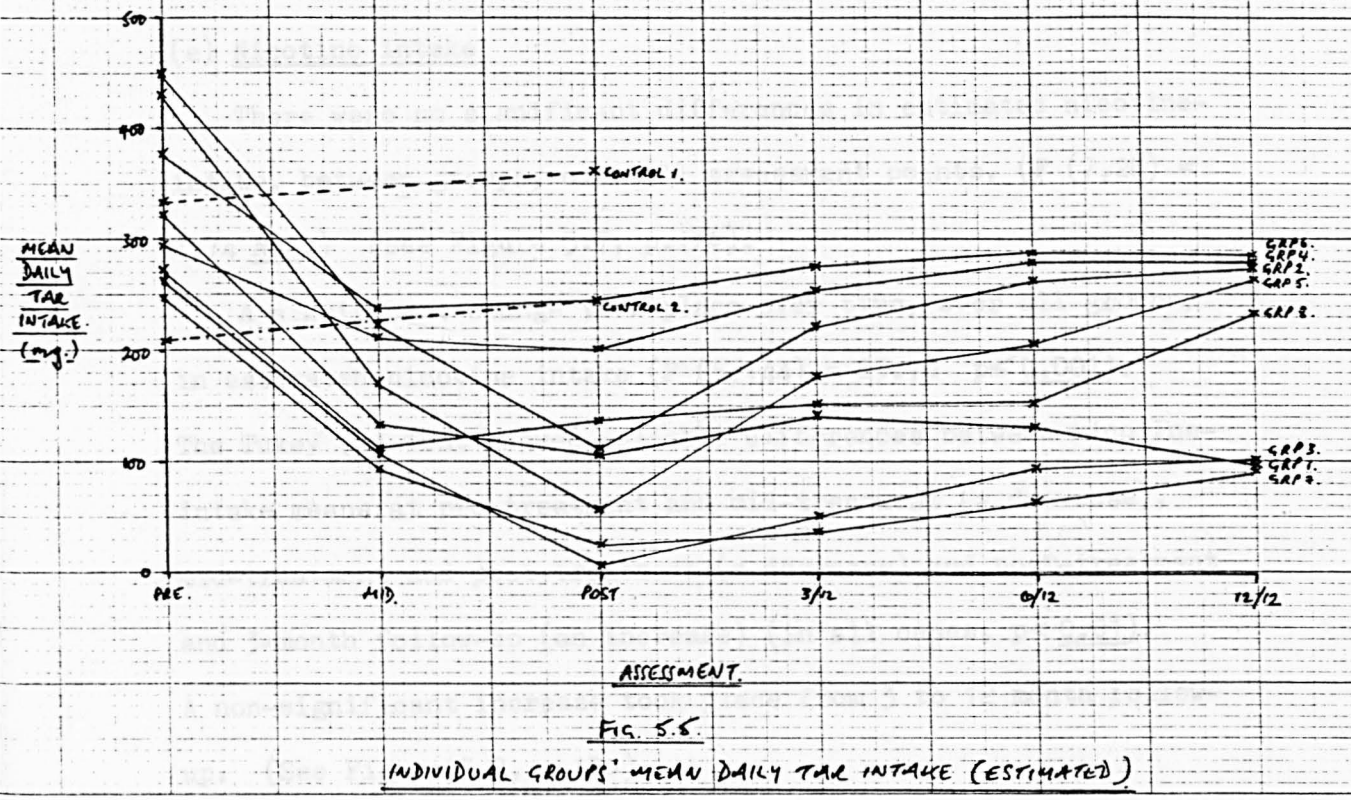


FIG. 5.5.

INDIVIDUAL GROUPS' MEAN DAILY TAR INTAKE (ESTIMATED)

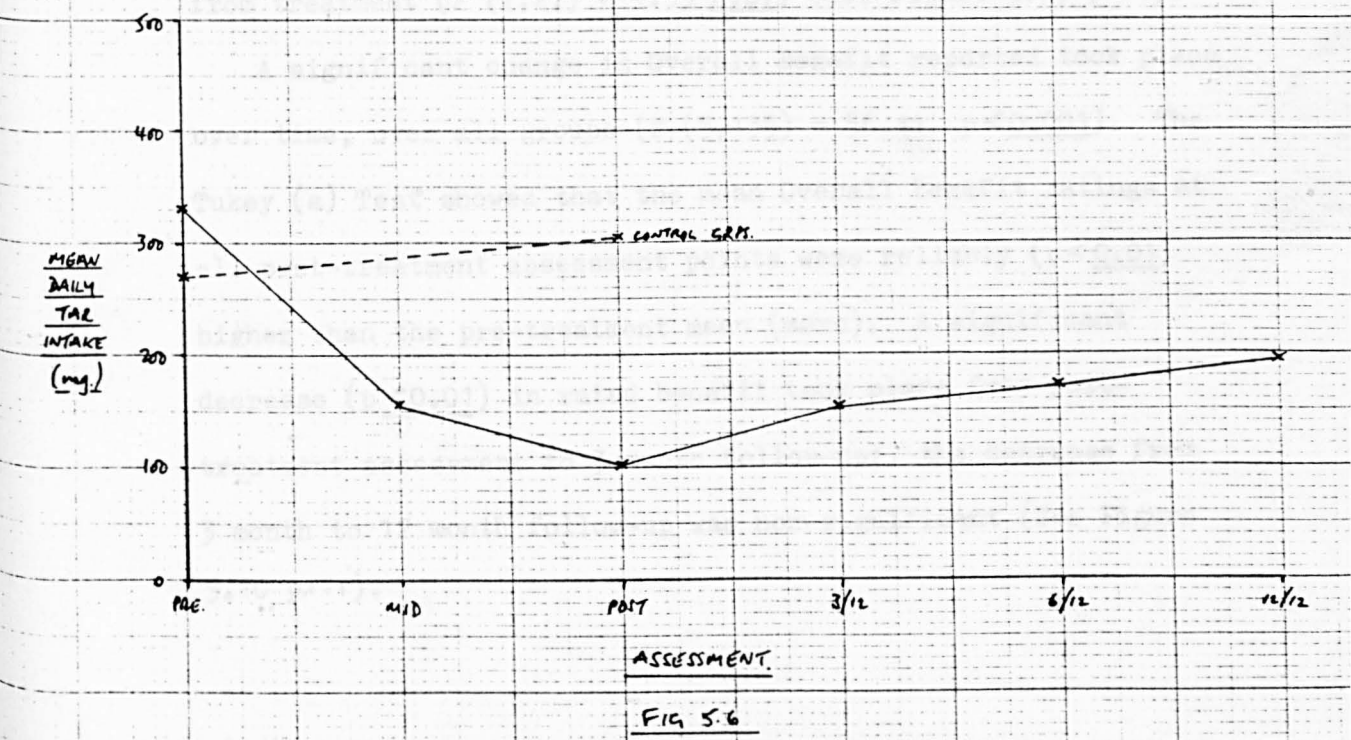


FIG 5.6

MEAN DAILY TAR INTAKE (ESTIMATED)

(ALL GROUPS COMBINED)

(e) Nicotine intake

There were no significant differences in estimated nicotine-intake, between groups, over all assessment points, ($F(7,28) = 1.74$ N.S.) (See Figure 5.7, p.170).

A significant change took place over time, over all groups, in estimated nicotine intake ($F(5,144) = 29.70$ $p < 0.001$). The Tukey (a) Test showed reliable differences between nicotine-intake means at pre-treatment and mid-treatment (a decrease), mid-treatment and post-treatment (a decrease) and post-treatment and 3-month follow-up (an increase) (in all cases, $p < 0.01$). A non-significant increase took place from 3 to 12 month follow-up. (See Figure 5.8, p.170).

There was no significant groups x time interaction effect ($F(35,144) = 1.06$ N.S.) (ANOVA Table 52; Appx. VI).

(f) Overall Benefit ratings

There were no significant differences between groups, over all assessment points, in reported Overall Benefit obtained from treatment ($F(7,27) = 1.33$ N.S.) (See Figure 5.9, p.171).

A significant change in Overall Benefit reported took place over time, over all groups ($F(5,135) = 56.41$ $p < 0.001$). The Tukey (a) Test showed that the mean Overall Benefit ratings at all post-treatment assessment points were reliably ($p < 0.01$) higher than the pre-treatment mean (zero). A significant decrease ($p < 0.01$) in rated benefit took place from post-treatment assessment to 3 month follow-up; the decrease from 3 month to 12 month follow-up was non-significant (See Figure 5.10 p.171).

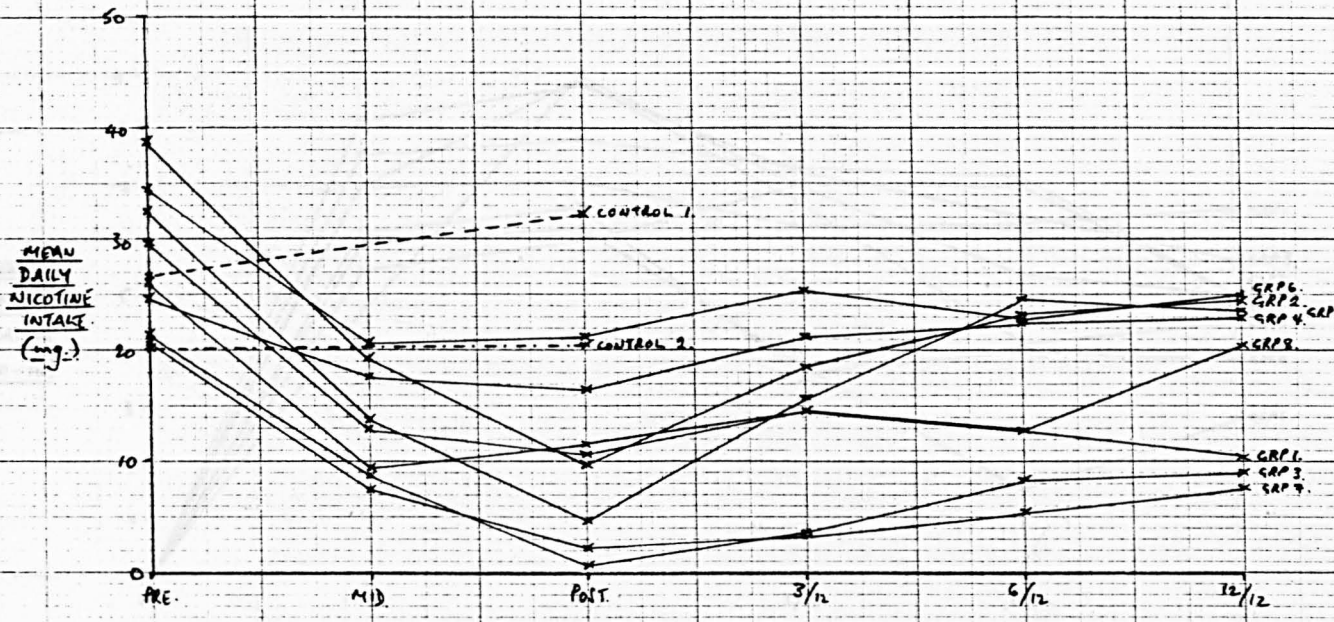


FIG 5.9.

INDIVIDUAL GROUPS' MEAN DAILY NICOTINE INTAKE (ESTIMATED).

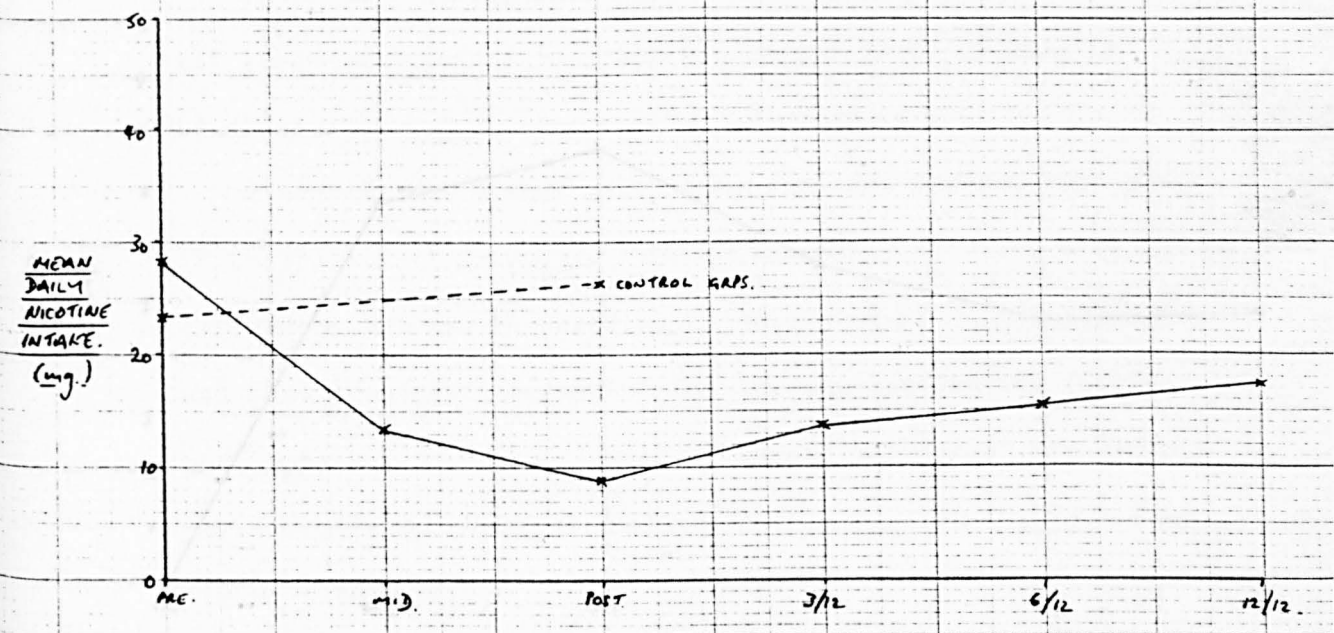


FIG 5.8

MEAN DAILY NICOTINE INTAKE (ESTIMATED)

(ALL GROUPS COMBINED.)

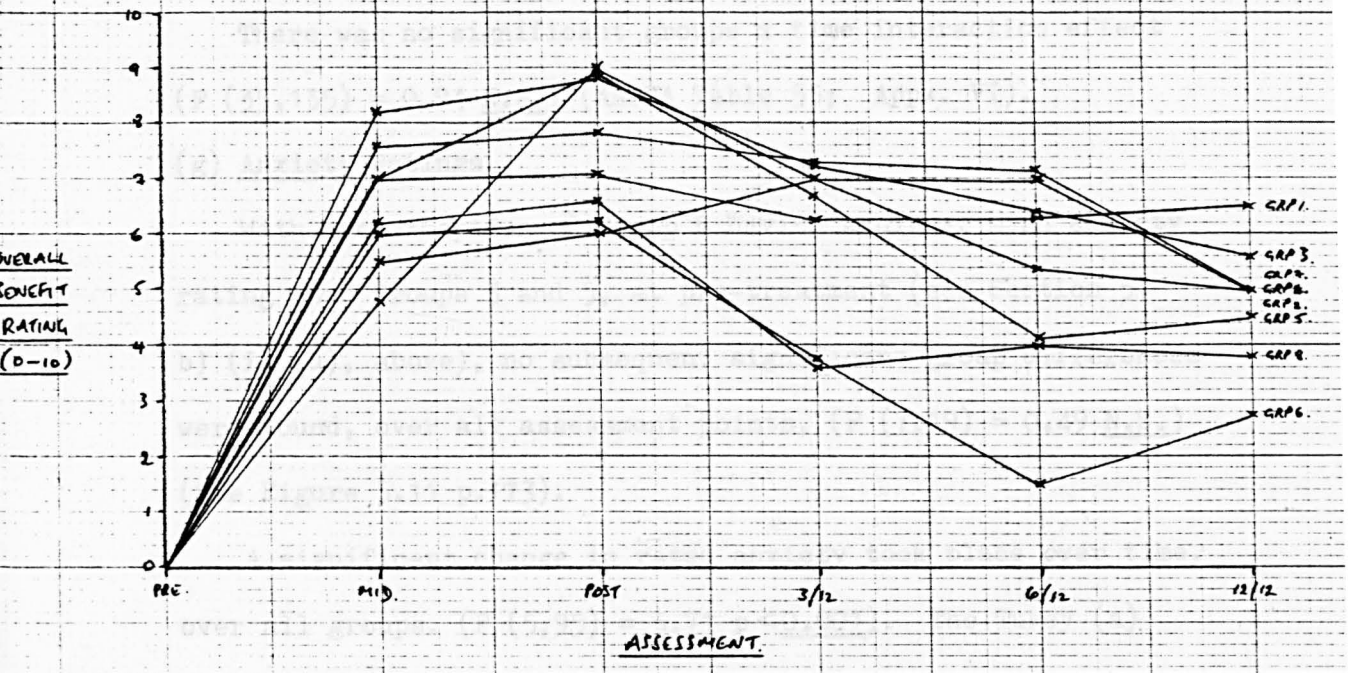


FIG. 5.9.
INDIVIDUAL GROUPS' BENEFIT RATINGS

FIG. 5.10.
BENEFIT RATINGS
(ALL GROUPS COMBINED.)

There was no significant groups x time interaction effect ($F(35,135) = 0.94$ N.S.) (ANOVA Table 53; Appx. VI).

(g) Anxiety Ratings

With the exception of Group 6 having a higher mean anxiety rating than Groups 3 and 5, at pre-treatment (see Section 5 b) (i) (d), above), no subsequent significant group differences were found, over all assessment points, ($F(7,19) = 0.49$ N.S.) (See Figure 5.11 p.173).

A significant change in rated anxiety took place over time, over all groups. ($F(5,95) = 5.71$ $p < 0.001$). The Tukey (a) Test showed that the mean anxiety rating increased significantly ($p < 0.01$) from mid-treatment to post-treatment assessment and decreased significantly from post-treatment to 3-month follow-up ($p < 0.01$) (See Figure 5.12 p.173).

There was no significant groups x time interaction effect ($F(35,95) = 1.56$ N.S.) (ANOVA Table 54; Appx. VI).

(h) Craving Intensity

There were no significant differences between groups, over all assessment points, on reported intensity of craving ($F(7,19) = 1.40$ N.S.) (See Figure 5.13 p.174).

A significant change over time, over all groups, took place ($F(5,95) = 2.71$ $p < 0.05$). The Tukey (a) Test showed a reliable difference between the craving intensity means at mid-treatment and post-treatment assessment (an increase in craving intensity) ($p < 0.05$) and the means at post-treatment assessment and 3-month follow-up (a decrease in craving intensity), ($p < 0.05$). (See Figure 5.14, p.174).

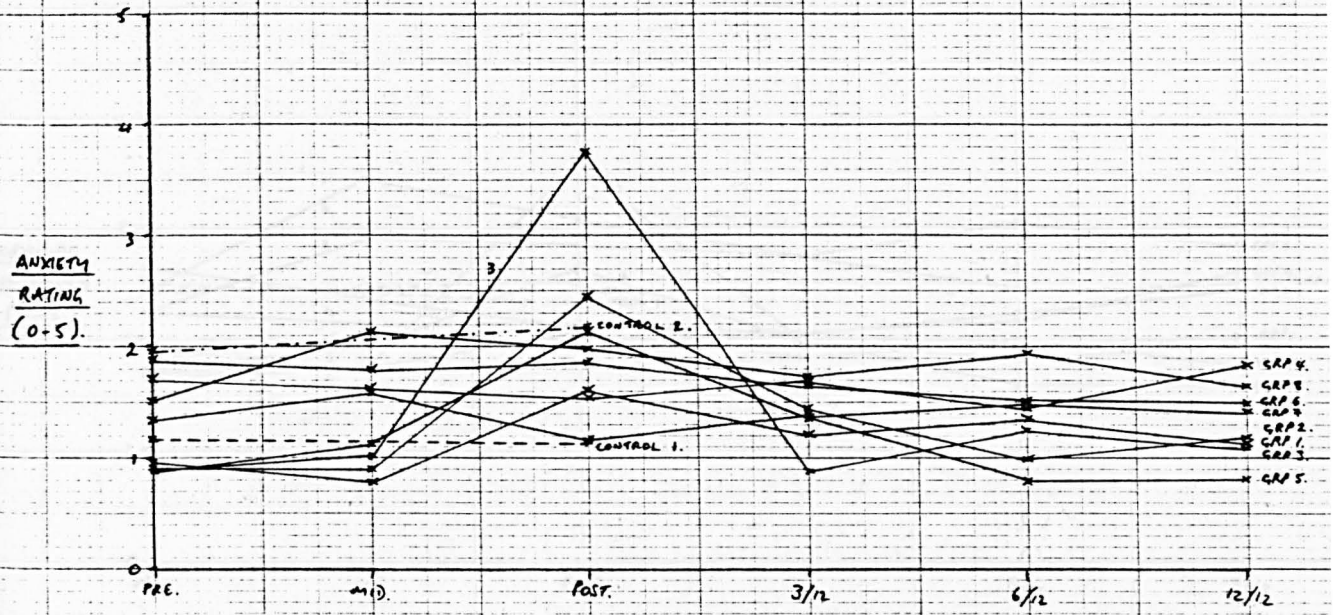


FIG. 5.11

INDIVIDUAL GROUPS' ANXIETY RATINGS.

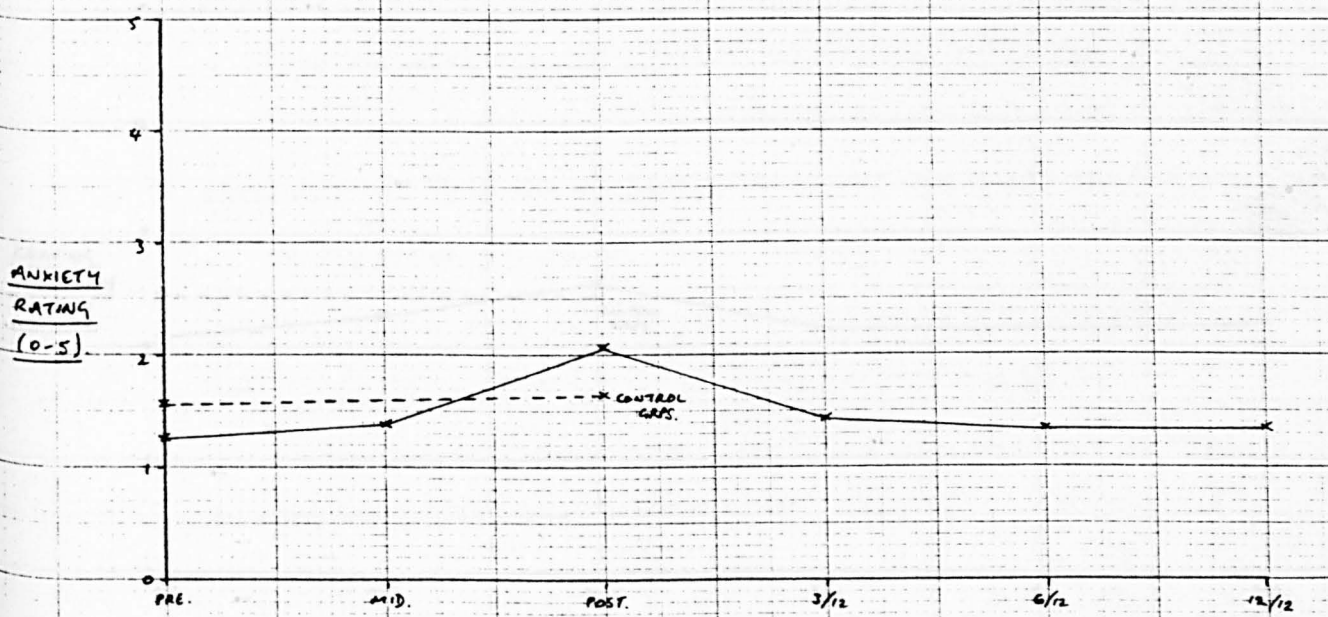
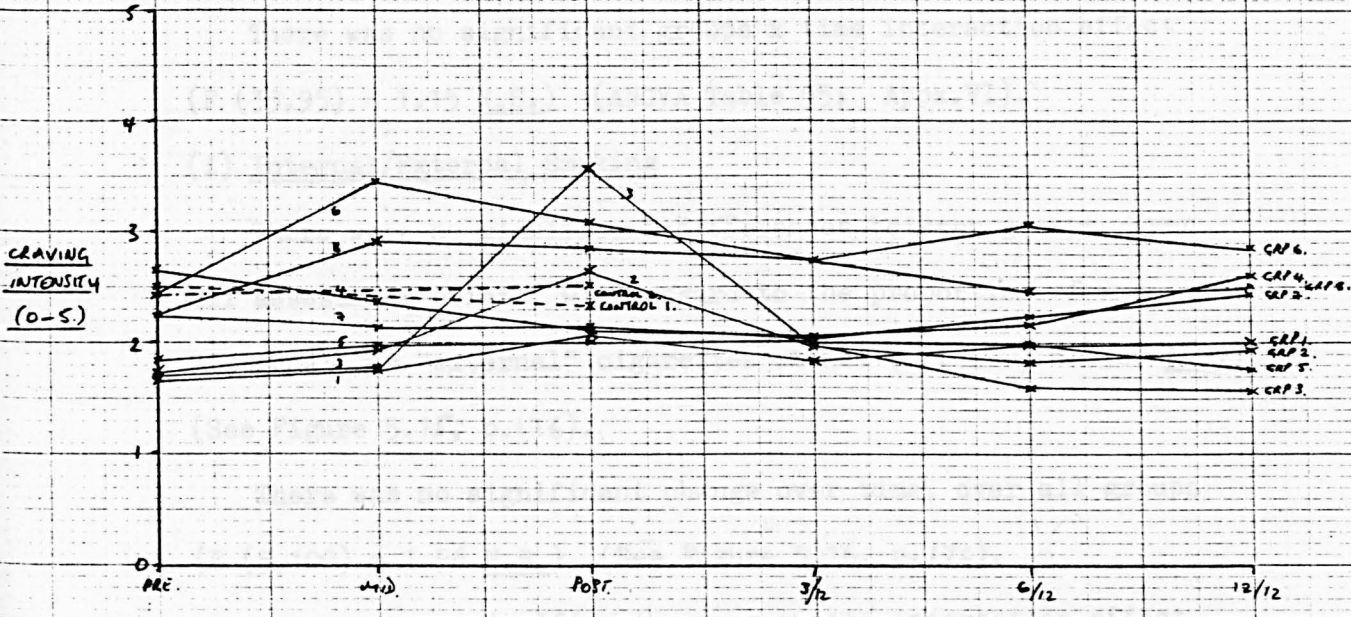


FIG. 5.12.

ANXIETY RATINGS

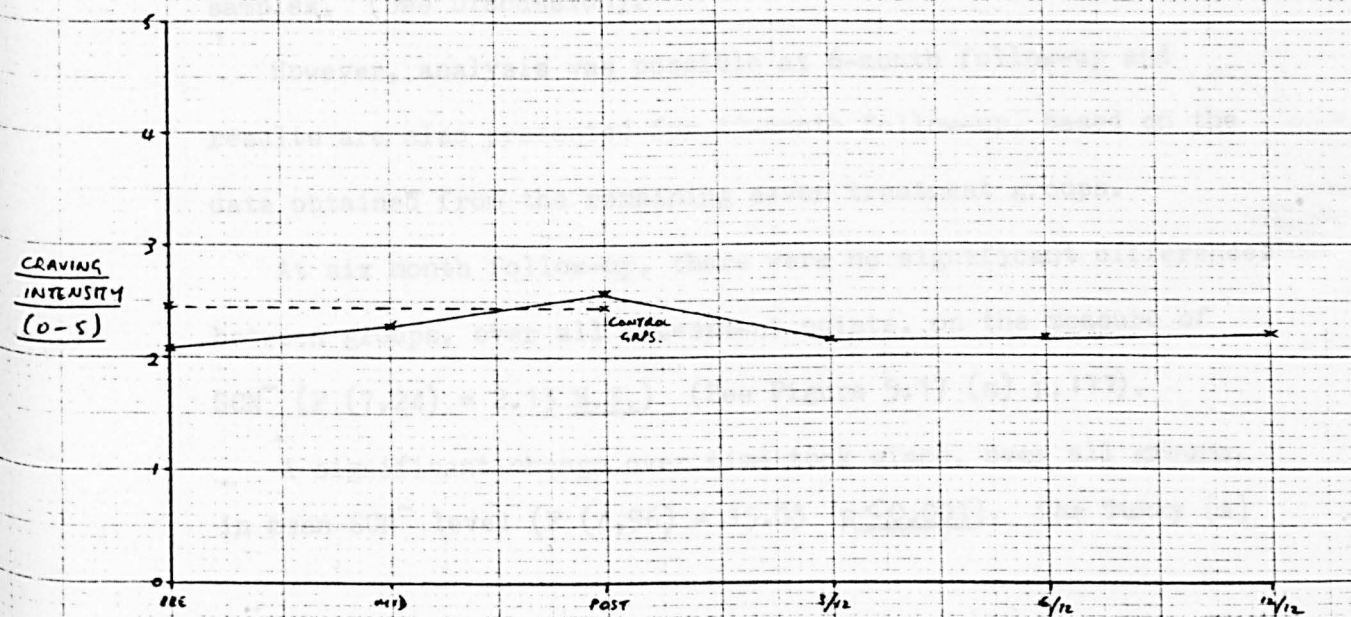
(ALL GROUPS COMBINED.)



ASSESSMENT

FIG. 5.13.

INDIVIDUAL GROUPS' CRAVING INTENSITY



ASSESSMENT.

FIG. 5.14.

CRAVING INTENSITY (ALL GROUPS COMBINED)

There was no significant groups x time interaction effect (F (35,95) = 1.15 N.S.) (ANOVA Table 55; Appx.VI).

(i) Internal/External Smoking

There were no significant differences between groups, over all assessment points, with regard to the proportion of "internal" vs "external" cigarettes smoked (F (7,20) = 1.00 N.S.) (See Figure 5.15, p.176).

There was no significant change over time, over all groups (F (5,100) = 1.96 N.S.) (See Figure 5.16, p.176).

There was no significant groups x time interaction effect (F (35,100) = 0.99 N.S.) (ANOVA Table 56; Appx. VI).

(ii) Physiological measures

(a) Serum Thiocyanate (SCN^-)

Insufficient data was available for a full analysis to be performed on SCN^- outcome data, despite the ability of the analysis to handle missing data, at the 12-month follow-up point: several subjects in Group 2 failed to supply blood-samples. (See Discussion).

However, analysis was possible at 6-month follow-up and results are also presented for 12-month follow-up, based on the data obtained from the remaining seven treatment groups.

At six month follow-up, there were no significant differences between groups, over all assessment points, on the measure of SCN^- (F (7,24) = 2.13 N.S.) (See Figure 5.17 (a) p.177).

A significant change over time took place, over all groups, in mean SCN^- level (F (4,96) = 15.03 $p<0.001$). The Tukey (a)

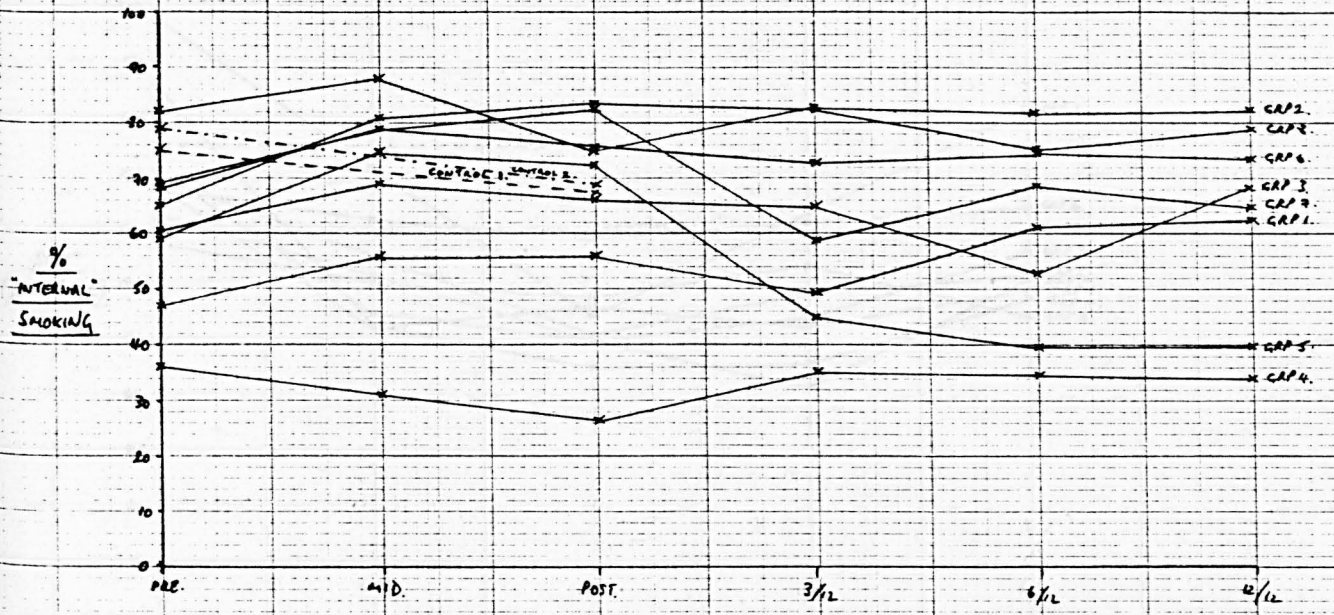


FIG 5.15.

INDIVIDUAL GROUPS' % "INTERNAL" SMOKING.

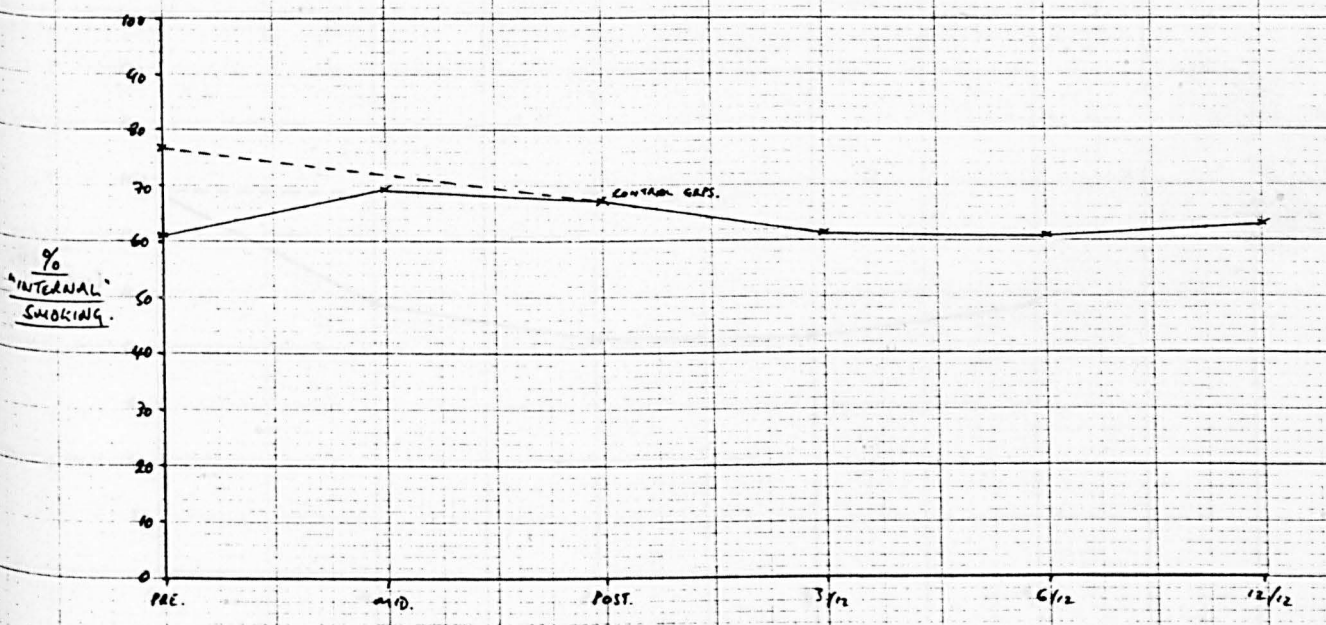


FIG. 5.16.

% "INTERNAL" SMOKING

(ALL GROUPS COMBINED).

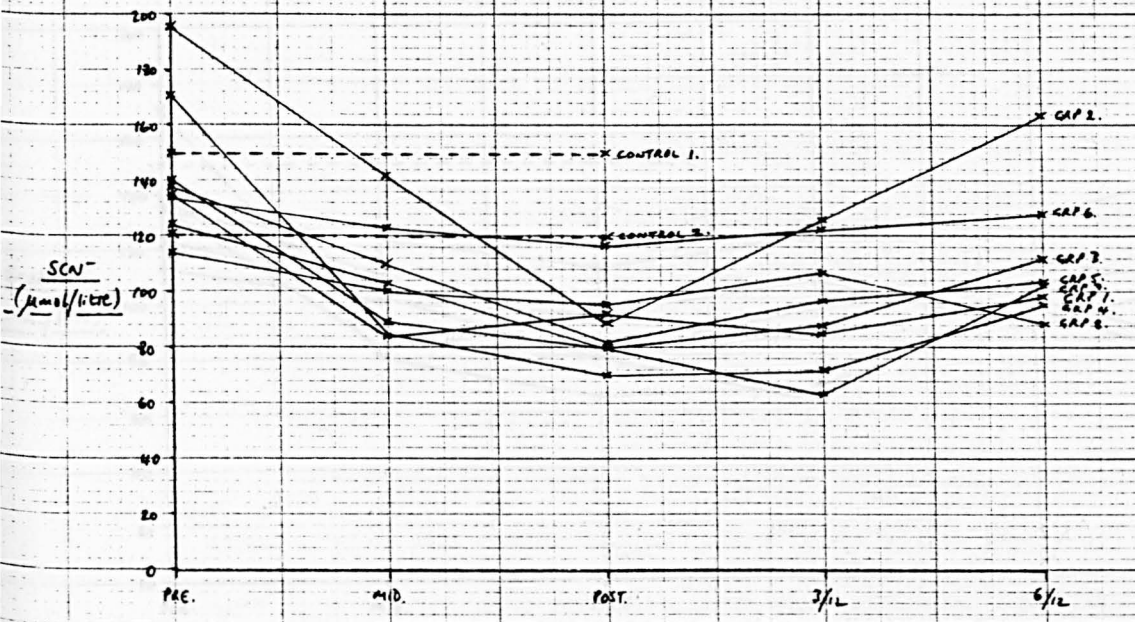


FIG 5.17(a)

INDIVIDUAL GROUPS' SCN⁻ LEVELS: (TO SIX-MONTH F.U.)

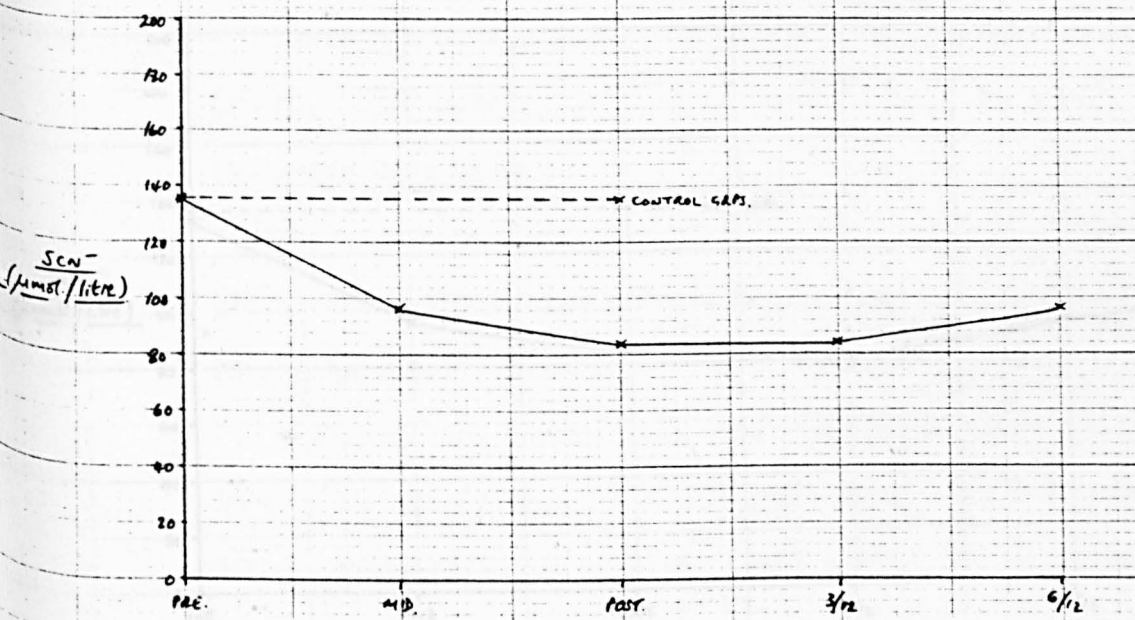


FIG 5.18(a)

SCN⁻ LEVELS: (TO SIX-MONTH F.U.)

(ALL GROUPS COMBINED.)

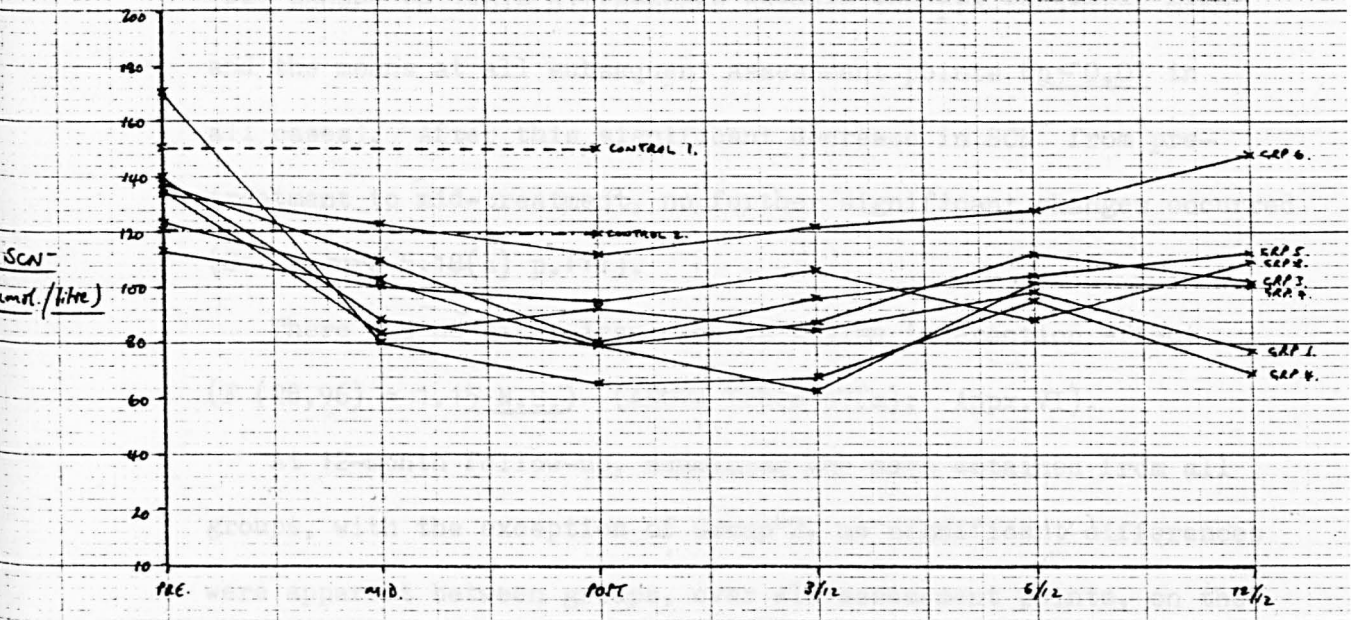


FIG 5.17 (6)

INDIVIDUAL GROUPS' SCN⁻ LEVELS : (TO 1YR F.U. - EXCLUDING GRP 2.)

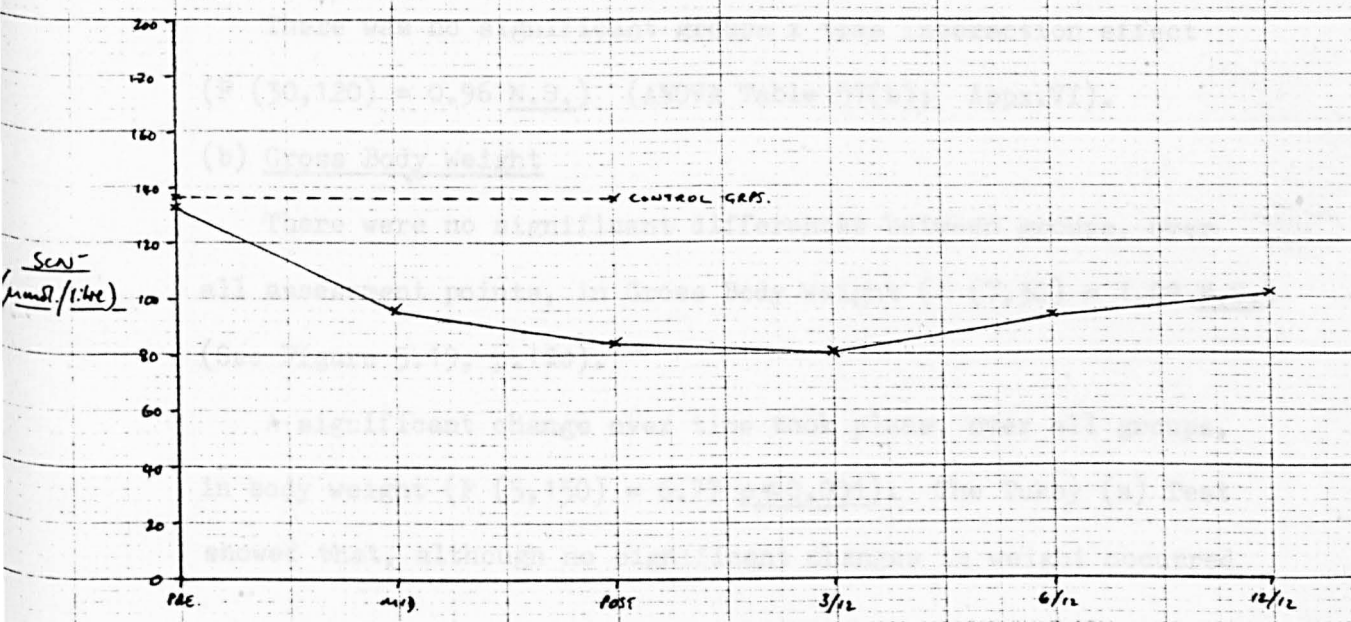


FIG. 5.18(6).

SCN⁻ LEVELS : (TO 1YR F.U. - EXCLUDING GRP 2.)

(ALL GROUPS COMBINED).

Test showed reliable differences between the pre-treatment mean and the means at all subsequent assessment points ($p<0.01$ in all cases). After this significant decrease in SCN^- from pre-treatment to mid-treatment, no further significant changes occurred (See Figure 5.18(a) p.177).

There was no significant groups x time interaction effect ($F(28,96) = 1.15$ N.S.) (ANOVA Table 57(a); Appx.VI).

At 12-month follow-up, examining the data obtained from all groups, with the exception of Group 2, no significant differences were apparent between groups, over all assessment points, on the measure of SCN^- ($F(6,24) = 2.34$ N.S.) (See Figure 5.17(b) p.178).

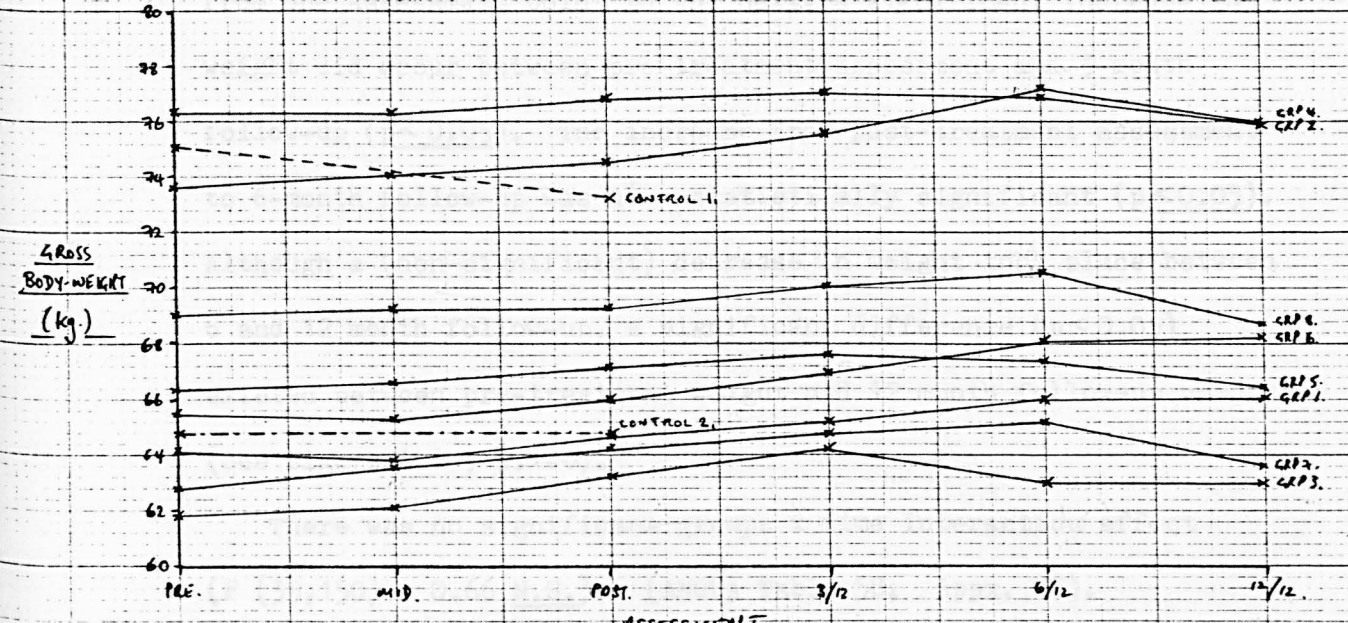
Again, a significant change over time took place, over all groups ($F(5,120) = 9.10$ $p<0.001$) and, as at 6-month follow-up assessment, reliable differences between the pre-treatment mean and all other means were shown by the Tukey (a) Test ($p<0.01$ in all cases). A non-significant increase in mean SCN^- level had taken place from six- to twelve-month follow-up ($p>0.05$). (See Figure 5.18(b), p.178).

There was no significant groups x time interaction effect ($F(30,120) = 0.96$ N.S.) (ANOVA Table 57(b); Appx.VI).

(b) Gross Body Weight

There were no significant differences between groups, over all assessment points, in Gross Body Weight ($F(7,30) = 1.29$ N.S.) (See Figure 5.19, p.180).

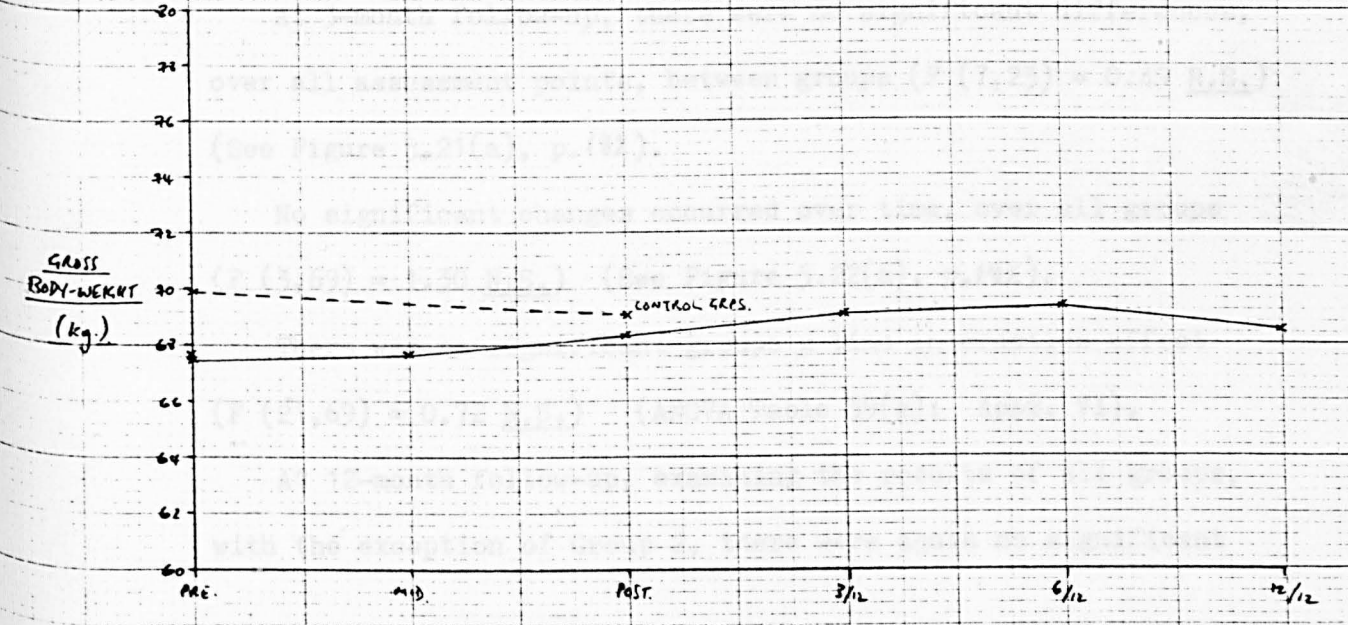
A significant change over time took place, over all groups, in body weight ($F(5,150) = 6.77$ $p<0.001$). The Tukey (a) Test showed that, although no significant changes in weight occurred



ASSESSMENT.

FIG. 5.19.

INDIVIDUAL GROUPS' GROSS BODY-WEIGHT.



ASSESSMENT.

FIG 5.20.

GROSS BODY-WEIGHT.

(ALL GROUPS COMBINED).

from one assessment point to the next, a significant increase in weight did occur between pre-treatment assessment and 3-month follow-up ($p < 0.05$). The increase from post-treatment assessment to 6-month follow-up was also statistically significant ($p < 0.05$). Although a (non-significant) decrease in weight took place between 6 and 12 month follow-up, a significant difference ($p < 0.05$) existed between pre-treatment weight and 12 month follow-up weight. (See Figure 5.20, p.180).

There was no significant groups x time interaction effect ($F(35,150) = 0.66$ N.S.). (ANOVA Table 58; Appx. VI).

(c) Lung Function

As was the case with SCN^- measurement at long-term follow-up, insufficient Group 2 subjects attended for Lung-function testing. (See Discussion). Analysis on all eight treatment groups was only possible up to the 3-month follow-up assessment point; analysis was performed on the seven remaining groups, however, at 12-month follow-up.

(i) FEV_1

At 3-month follow-up, there were no significant differences, over all assessment points, between groups ($F(7,23) = 0.49$ N.S.) (See Figure 5.21(a), p.182).

No significant changes occurred over time, over all groups ($F(3,69) = 1.30$ N.S.) (See Figure 5.22(a), p.182).

There was no significant groups x time interaction effect ($F(21,69) = 0.72$ N.S.) (ANOVA Table 59(a); Appx. VI).

At 12-month follow-up, examining the results of all groups, with the exception of Group 2, there were again no significant

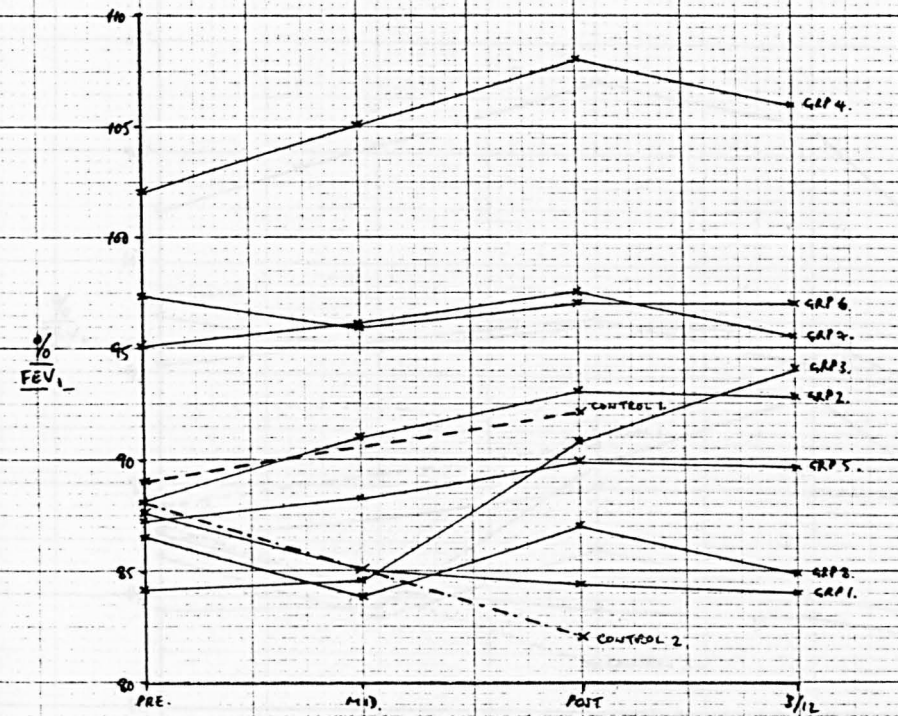


FIG. 5.21(a)

INDIVIDUAL GROUPS' % FEV$_1$: (TO 3-MONTH F.U.)

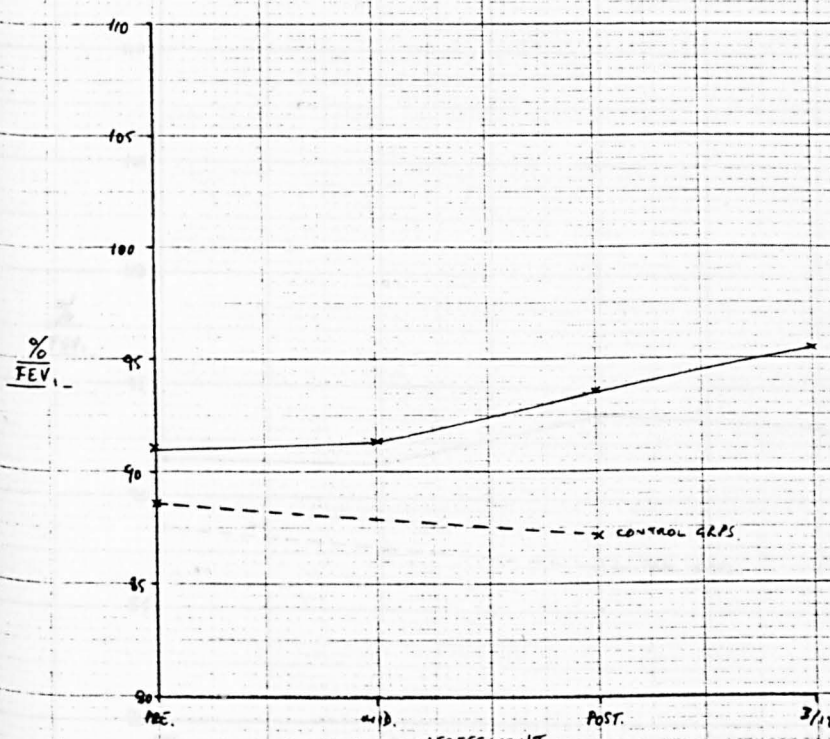


FIG. 5.22(a)

(ALL GROUPS COMBINED) % FEV$_1$: (TO 3-MONTH F.U.)

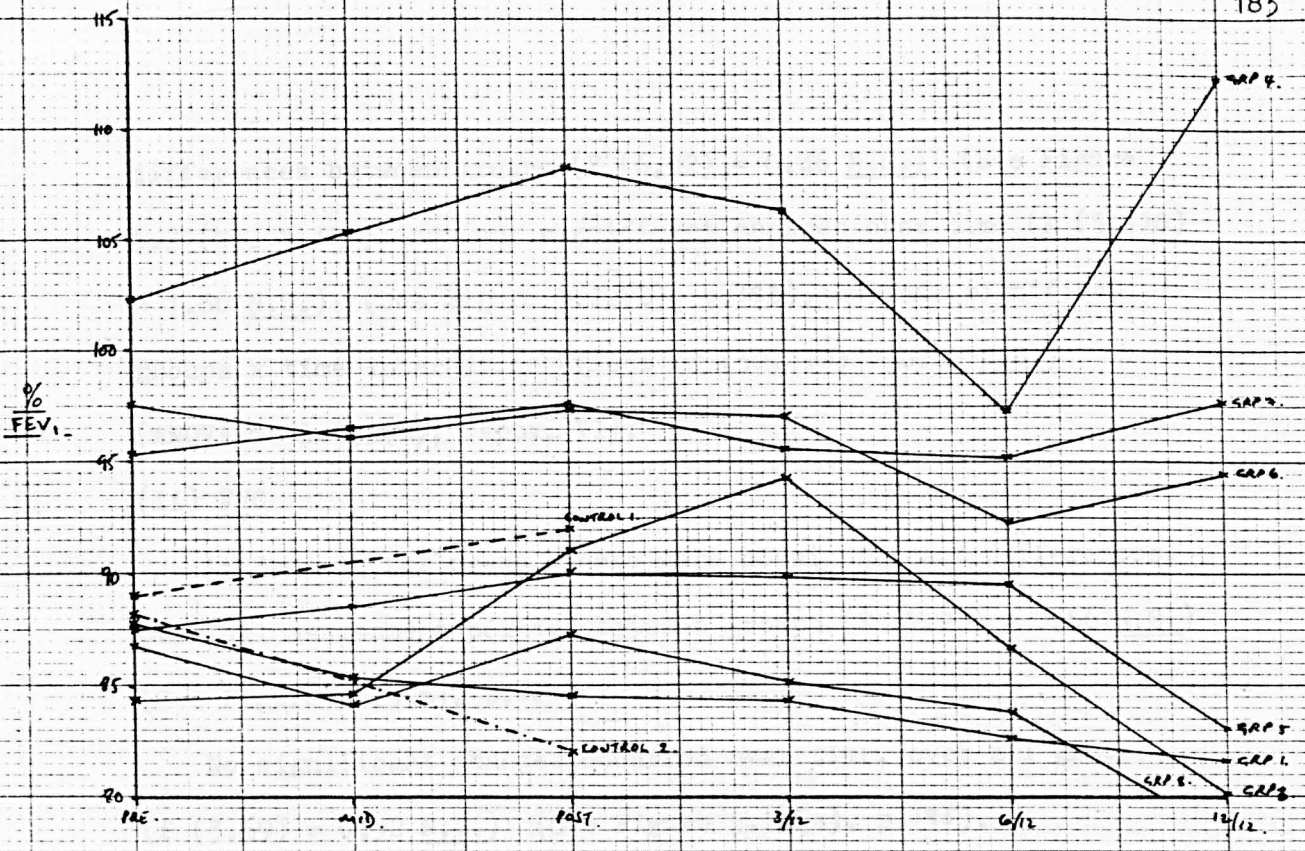


FIG 5.21(6)

INDIVIDUAL GROUPS' % FEV_1 : (TO 1YR F.U.; EXCLUDING GRP 2.)

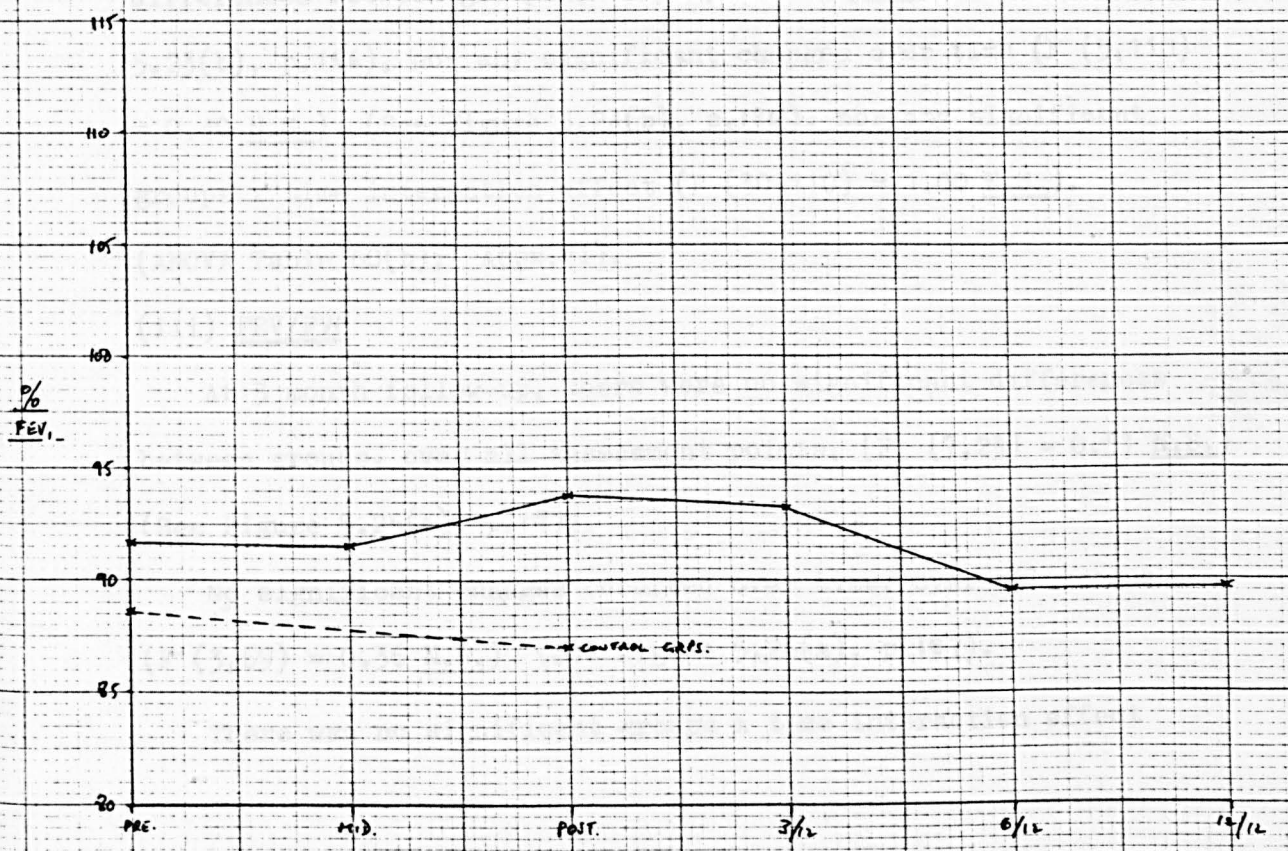


FIG 5.22(6)

% FEV_1 : (TO 1YR F.U.; EXCLUDING GRP. 2.)

(ALL GROUPS COMBINED)

differences between groups (F (6,22) = 0.46 N.S.) (See Figure 5.21(b), p.183), nor any significant changes over time (F (5,110) = 1.80 N.S.) (See Figure 5.22(b), p.183), nor any significant groups x time interaction effect (F (30,110) = 0.87 N.S.) (ANOVA Table 59(b); Appx. VI).

(ii) FVC

At 3 month follow-up, there were no significant differences, over all assessment points, between groups (F (7,23) = 0.66 N.S.) (See Figure 5.23(a), p.185).

No significant changes occurred over time, over all groups (f (3,69) = 0.92 N.S.) (See Figure 5.24(a), p.185).

There was no significant groups x time interaction effect (F (21,69) = 0.90 N.S.). (ANOVA Table 60(a); Appx. VI).

At 12 month follow-up, examining the results of all groups, with the exception of Group 2, there were again no significant differences between groups (F (6,22) = 0.63 N.S.) (See Figure 5.23(b), p.186), nor any significant changes over time (F (5,110) = 0.40 N.S.) (See Figure 5.24(b), p.186), nor any significant groups x time interaction effect (F (30,110) = 1.00 N.S.). (ANOVA Table 60(b); Appx.VI).

(iii) FEV/FVC

At 3 month follow-up, there were no significant differences between groups, over all assessment points, (F, (7,23) = 0.51 N.S.) (See Figure 5.25(a), p.187).

No significant changes occurred over time, over all groups (F (3,69) = 0.36 N.S.) (See Figure 5.26(a), p.187).

There was no significant groups x time interaction effect

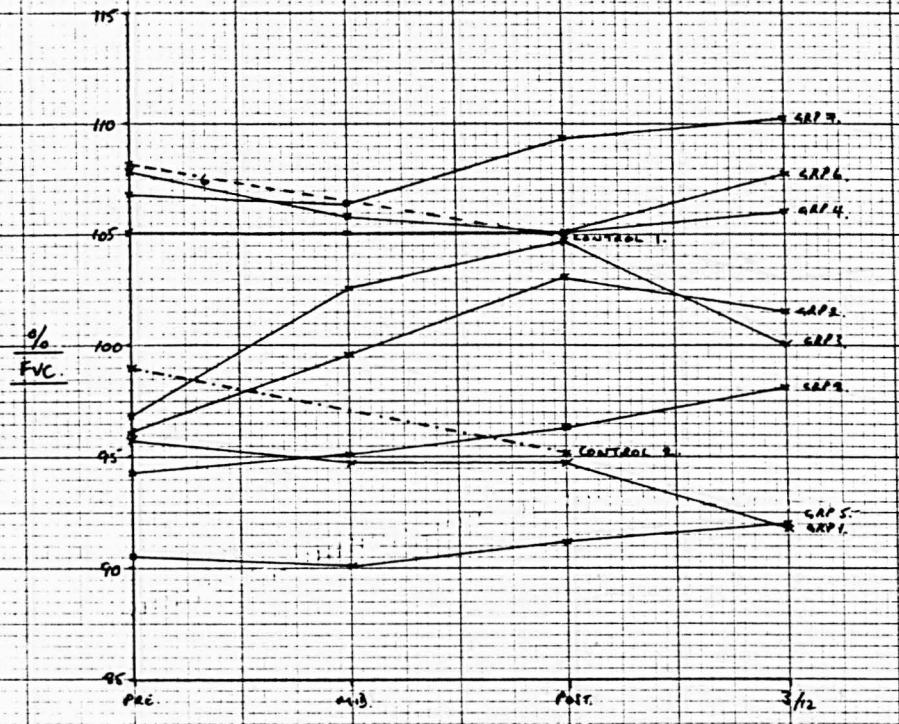


FIG 5.23 (c).

INDIVIDUAL GROUPS' % FVC : (TO 3-MONTH F.U.)

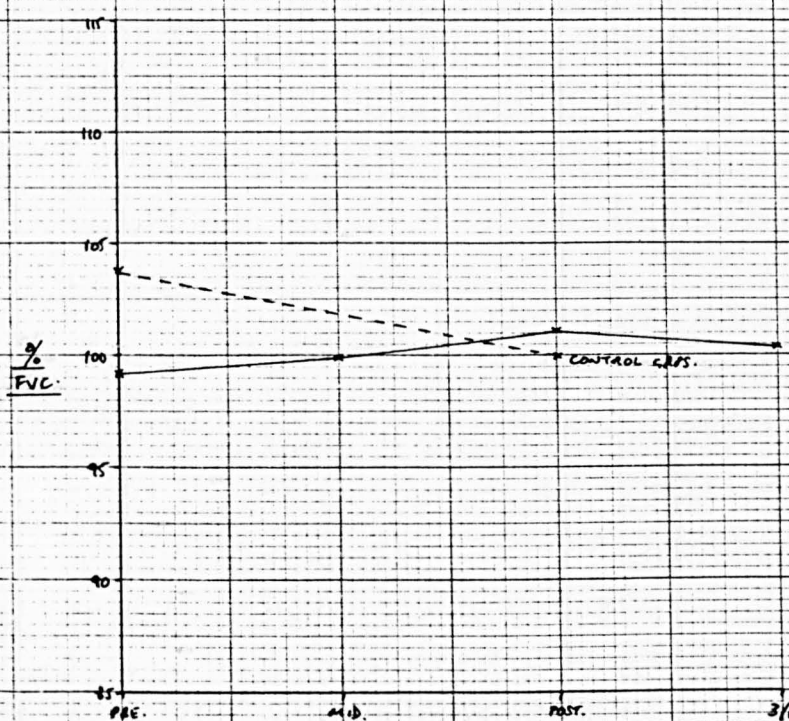


FIG 5.24 (a).

% FVC : (TO 3-MONTH F.U.)

(ALL GROUPS COMBINED)

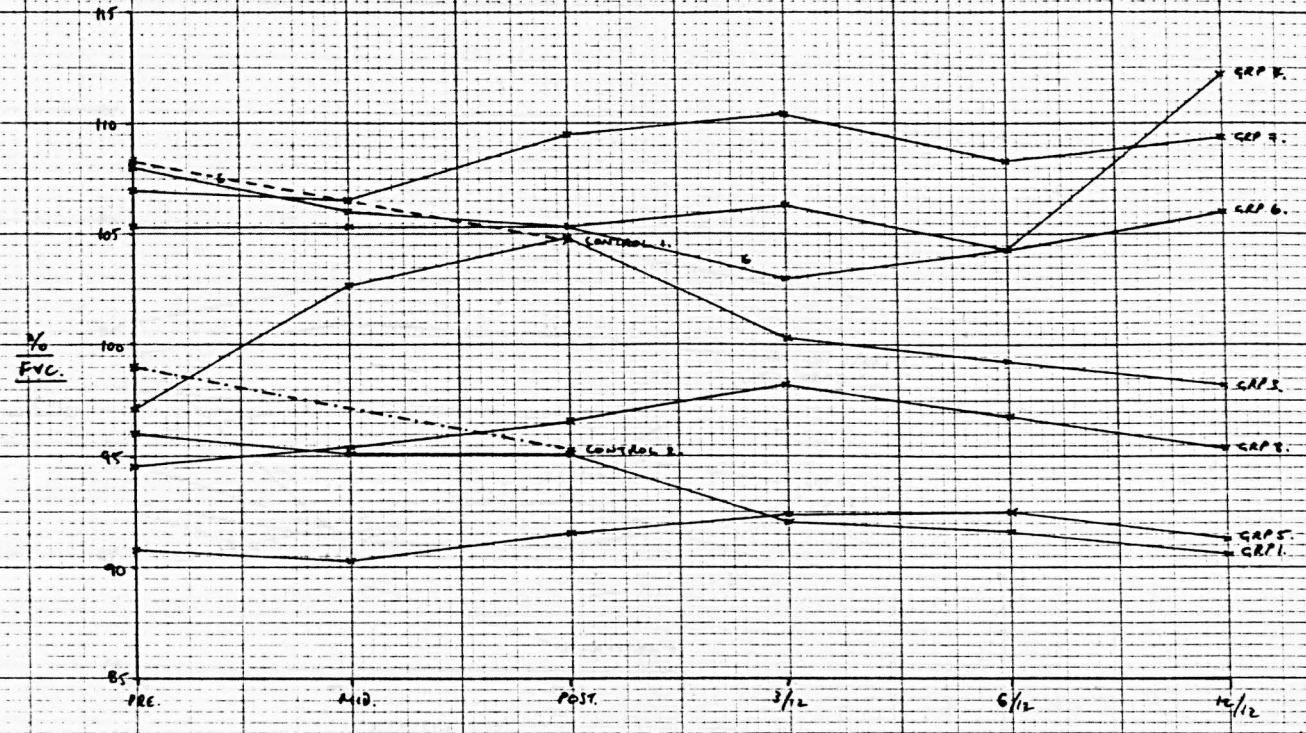


FIG 5.23(6)

INDIVIDUAL GROUPS' % FVC : (TO 1YR. F.U.; EXCLUDING GRP 2.)

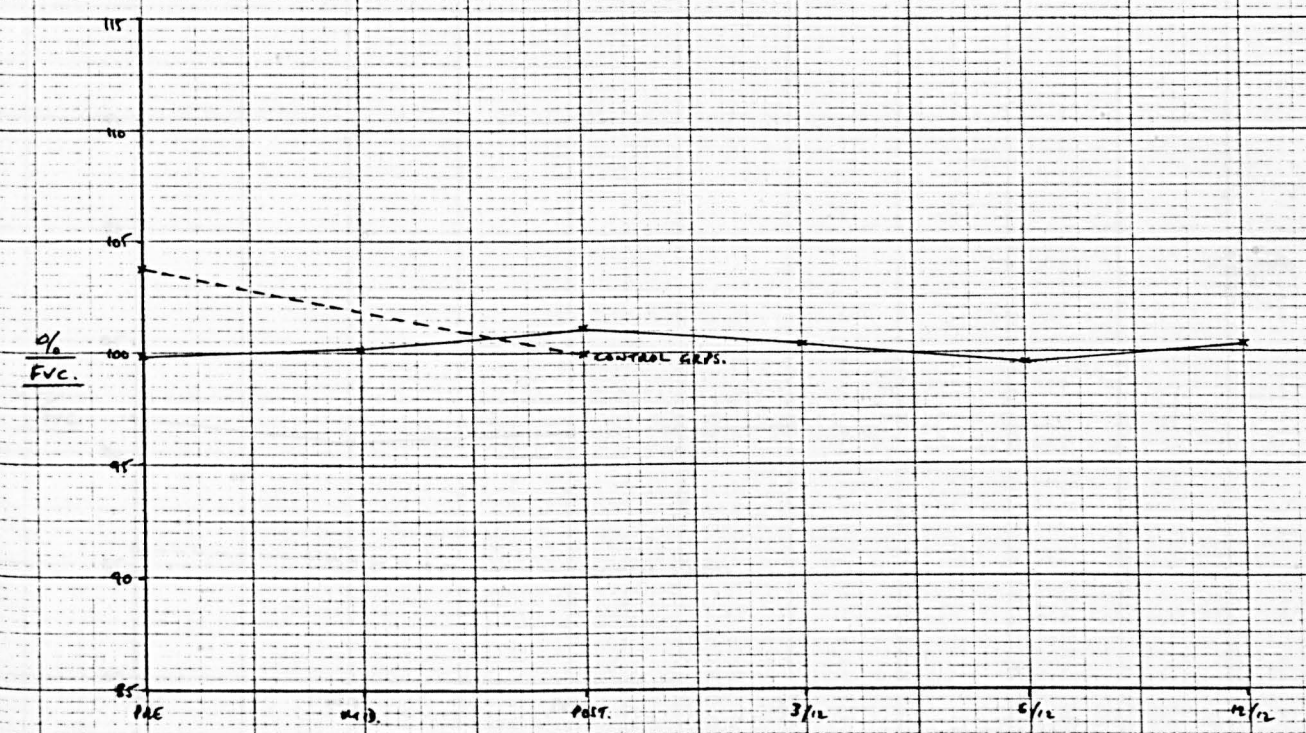


FIG 5.24(6)

% FVC : (TO 1YR. F.U.; EXCLUDING GRP 2.)

(ALL GROUPS COMBINED)

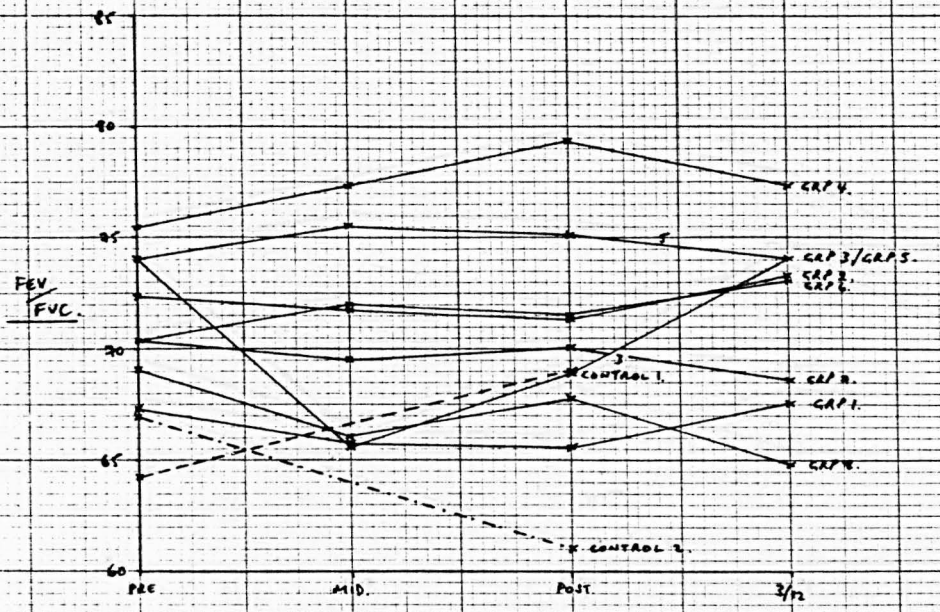


FIG 5.25(a)

INDIVIDUAL GROUPS' FEV/FVC RATIOS: (TO 3-MONTH F.U.)

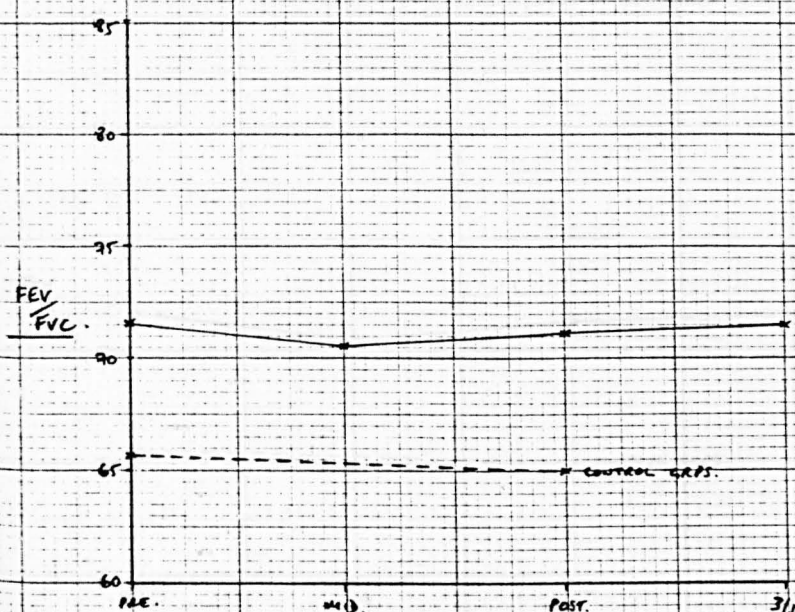
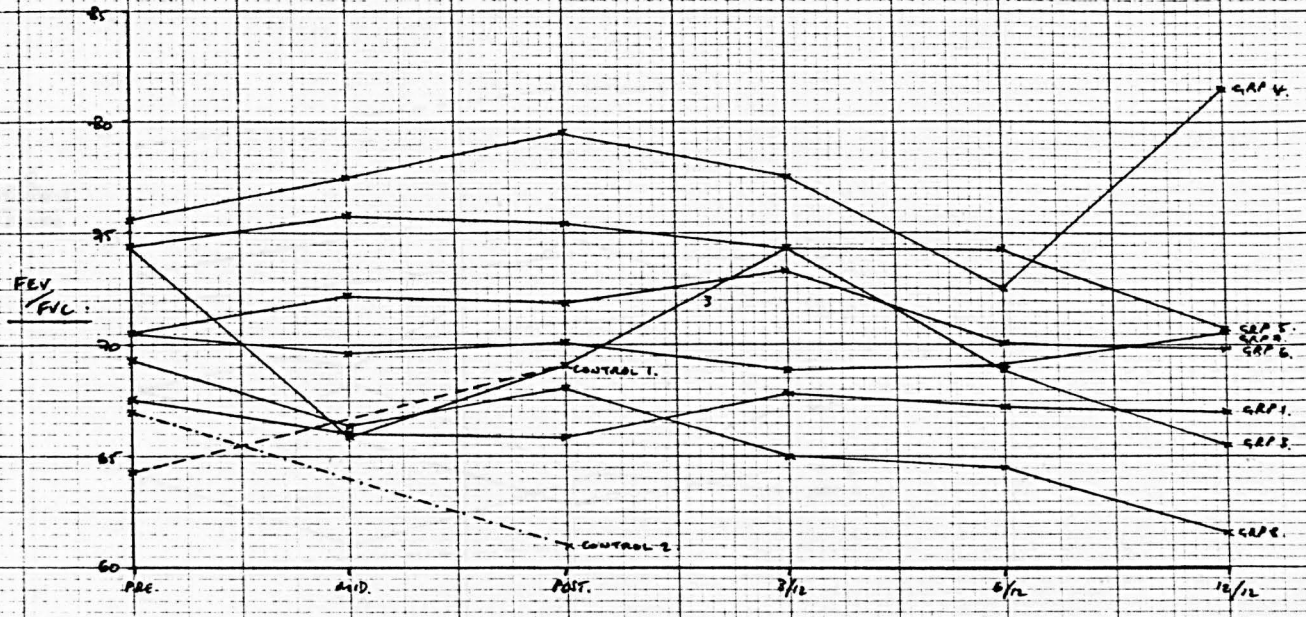


FIG 5.26(a)

FEV/FVC RATIO: (TO 3-MONTH F.U.)

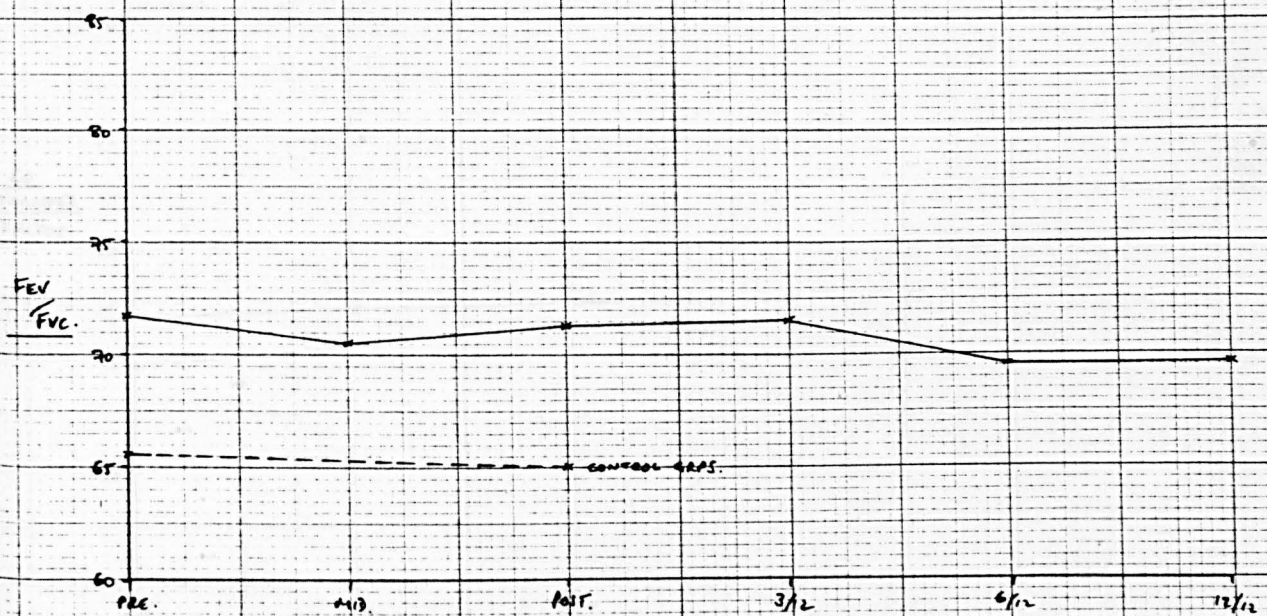
(ALL GROUPS COMBINED)



ASSESSMENT

FIG. 5.25(6)

INDIVIDUAL GROUPS' FEV/FVC RATIOS: (TO 1YR. F.U.; EXCLUDING GRP 2.)



ASSESSMENT.

FIG 5.26(6)

FEV/FVC RATIO: (TO 1YR F.U.; EXCLUDING GRP 2.)

(ALL GROUPS COMBINED.)

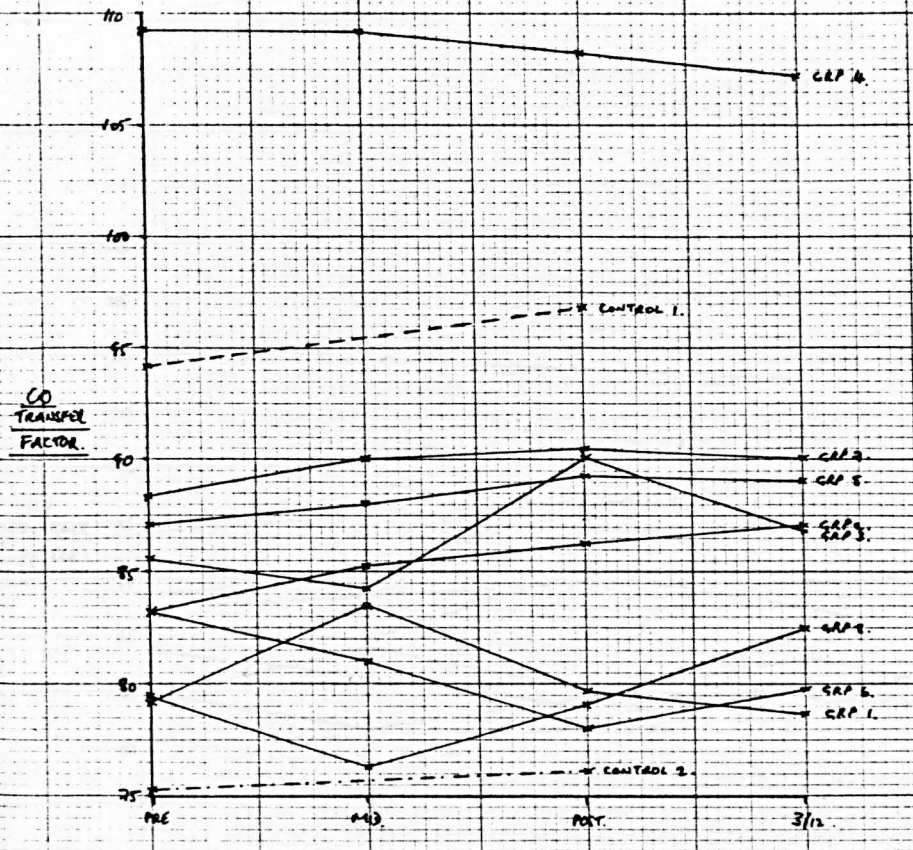


FIG 5.27(a)

INDIVIDUAL GROUPS' CO TRANSFER FACTORS : (TO 3-MONTH F.U.)

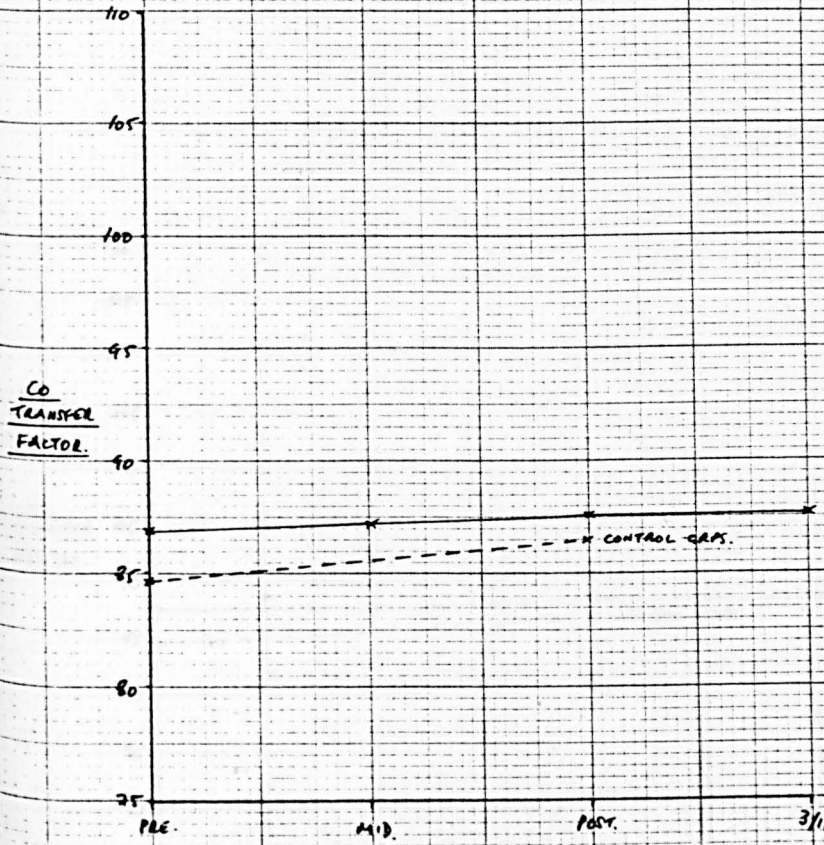


FIG 5.28(a)

CO TRANSFER FACTOR : (TO 3-MONTH F.U.)

(ALL GROUPS COMBINED).

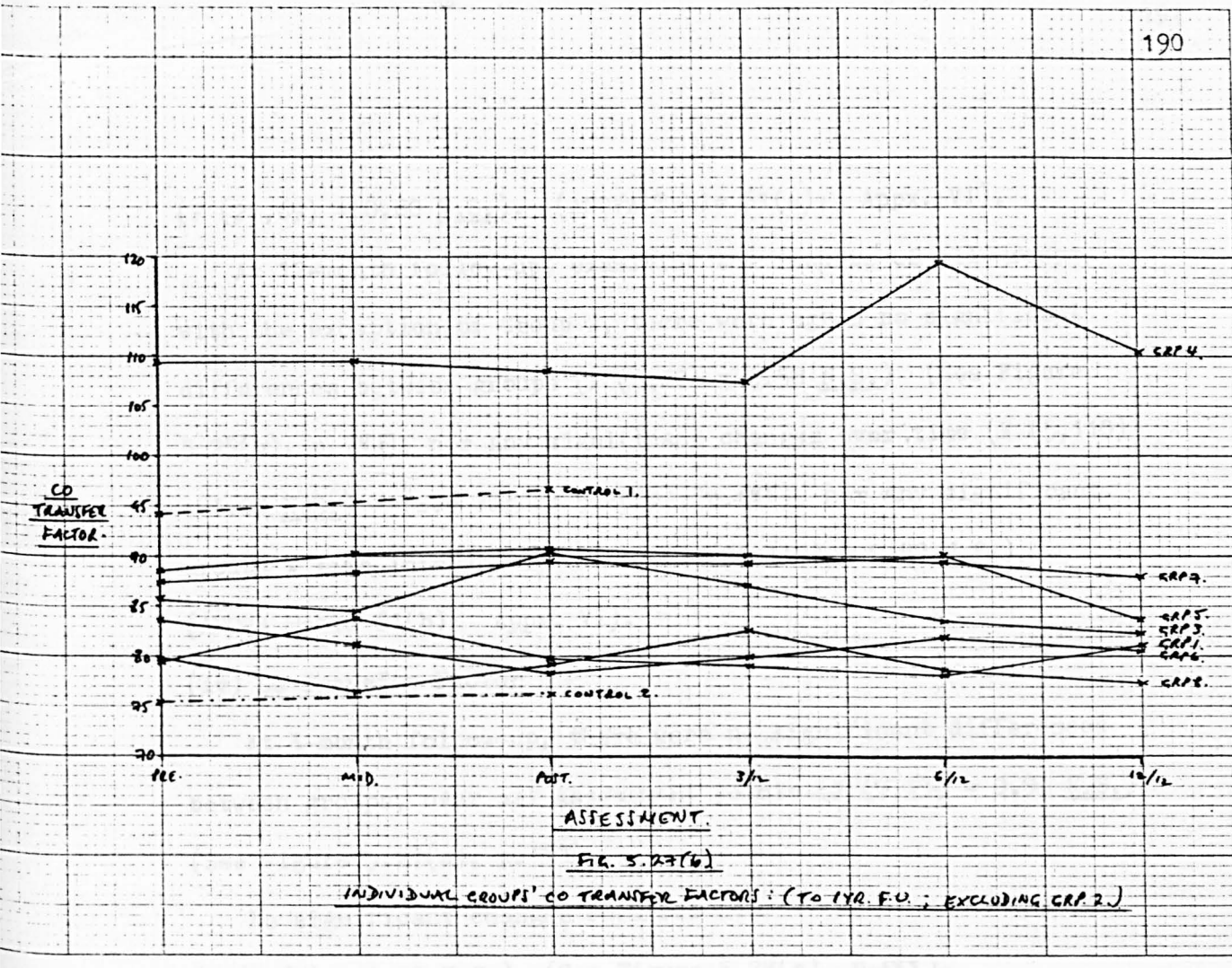


FIG. 5.27(6)

INDIVIDUAL GROUPS' CO TRANSFER FACTORS: (TO 1YR. F.U.; EXCLUDING GRP 2.)

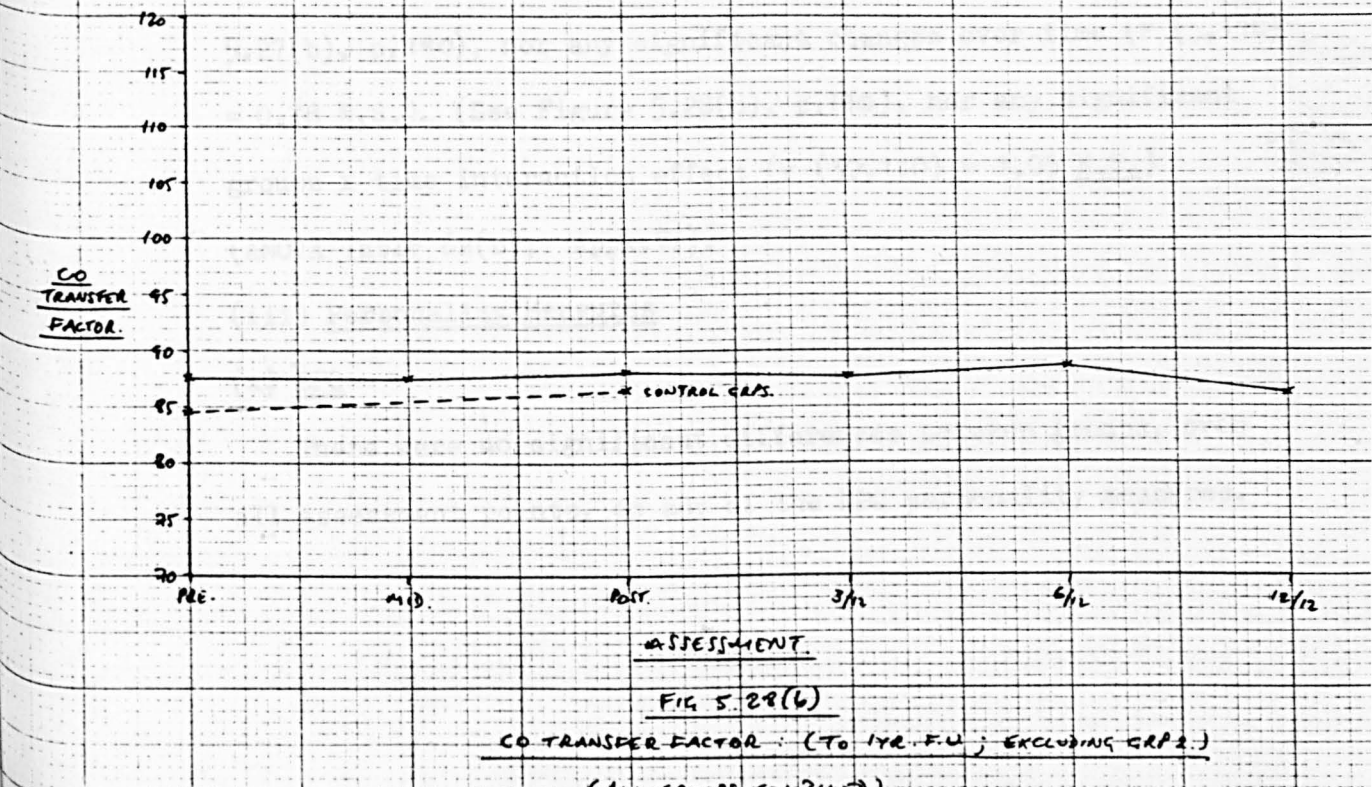


FIG 5.28(6)

CO TRANSFER FACTOR: (TO 1YR. F.U.; EXCLUDING GRP 2.)

(ALL GROUPS COMBINED)

(F (21,69) = 0.80 N.S.). (ANOVA Table 61(a); Appx. VI).

At 12-month follow-up, examining the results of all groups, with the exception of Group 2, there were again no significant differences between groups (F (6,22) = 0.58 N.S.) (See Figure 5.25(b), p.188), nor any significant changes over time (F (5,110) = 1.08 N.S.) (See Figure 5.26(b), p.188), nor any significant groups x time interaction effect (F (30,110) = 0.89 N.S.) (ANOVA Table 61(b); Appx. VI).

(iv) CO Transfer Factor

At 3-month follow-up, there were no significant differences between groups, over all assessment points (F (7,23) = 0.90 N.S.) (See Figure 5.27(a), p.189).

No significant changes occurred over time, over all groups (F (3,69) = 0.22 N.S.) (See Figure 5.28(a), p.189).

There was no significant groups x time interaction effect (F (21,69) = 1.28 N.S.) (ANOVA Table 62(a); Appx. VI).

At 12-month follow-up, examining the results of all groups, with the exception of Group 2, there were again no significant differences between groups (F (6,22) = 1.05 N.S.) (See Figure 5.27(b), p.190), nor any significant changes over time (F (5,110) = 0.78 N.S.) (See Figure 5.28(b), p.190), nor any significant groups x time interaction effect (F (30,110) = 1.05 N.S.) (ANOVA Table 62(b); Appx.VI).

(iii) Personality Measures

(a) EPQ

There were no significant differences between groups, over all assessment points, on any of the EPQ personality measures.

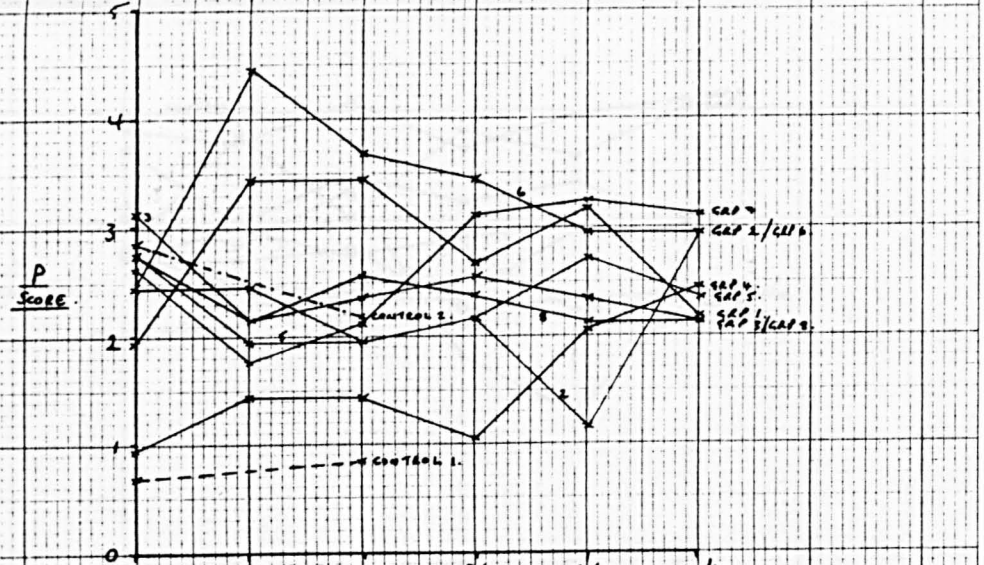


FIG. 5.29.

INDIVIDUAL GROUPS' EPQ (P) SCORES.

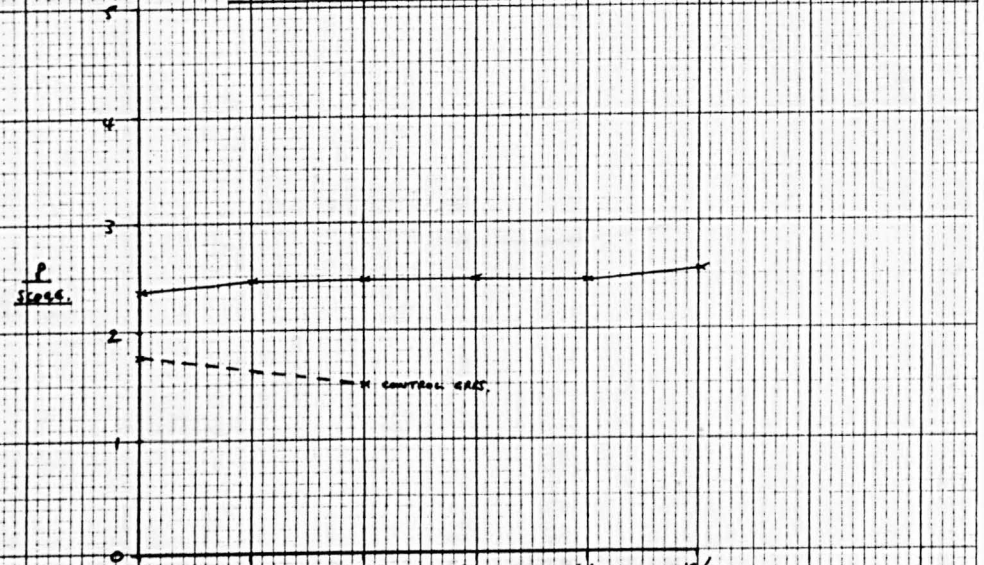


FIG. 5.30

EPQ (P) SCORES (ALL GROUPS COMBINED)

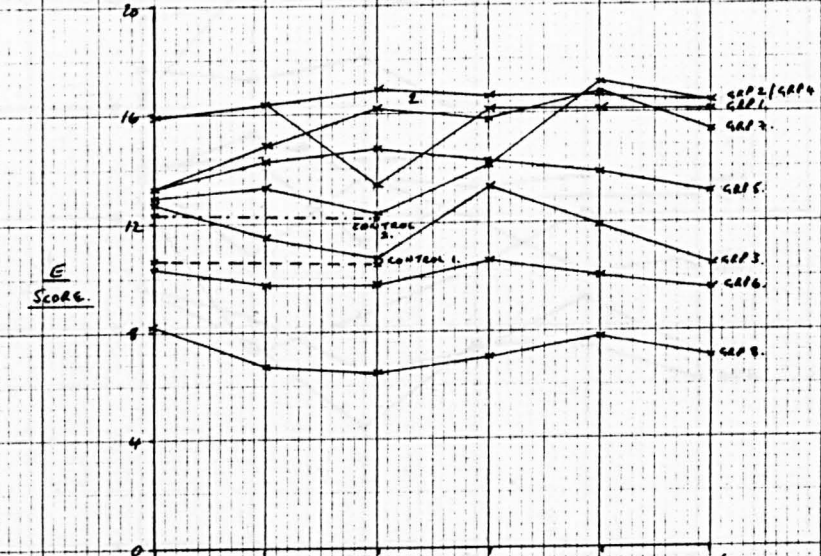


FIG. 5.31.

INDIVIDUAL GROUPS' EPQ (E) SCORES.

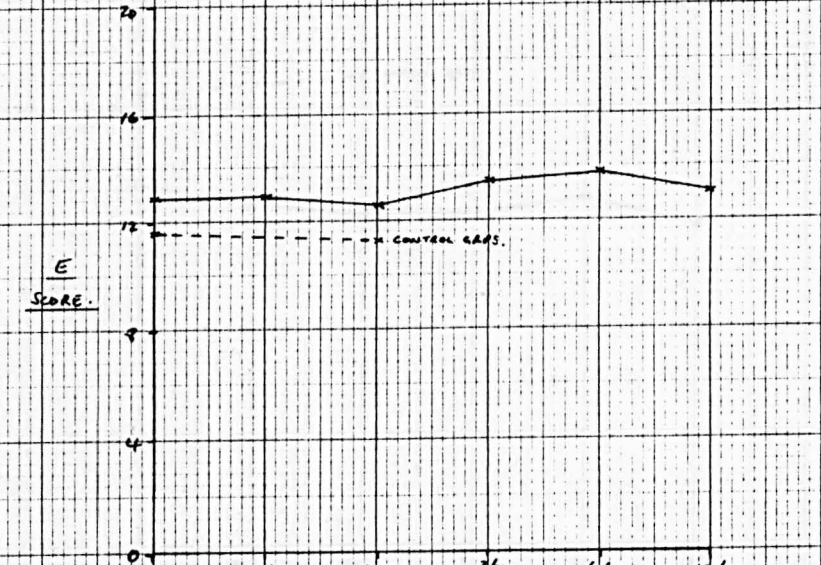


FIG. 5.32

EPQ (E) SCORES (ALL GROUPS COMBINED)

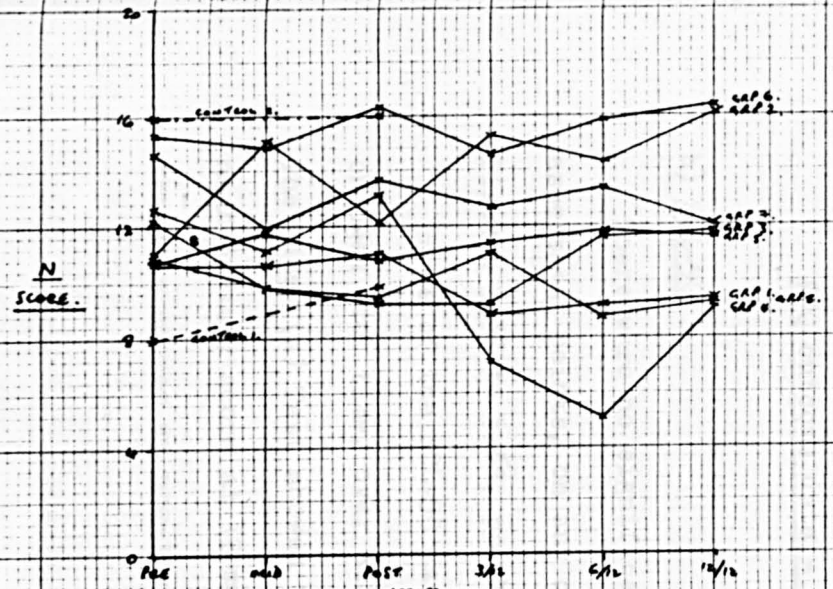


FIG. 5.33.

INDIVIDUAL GROUPS' EPQ(N) SCORES.

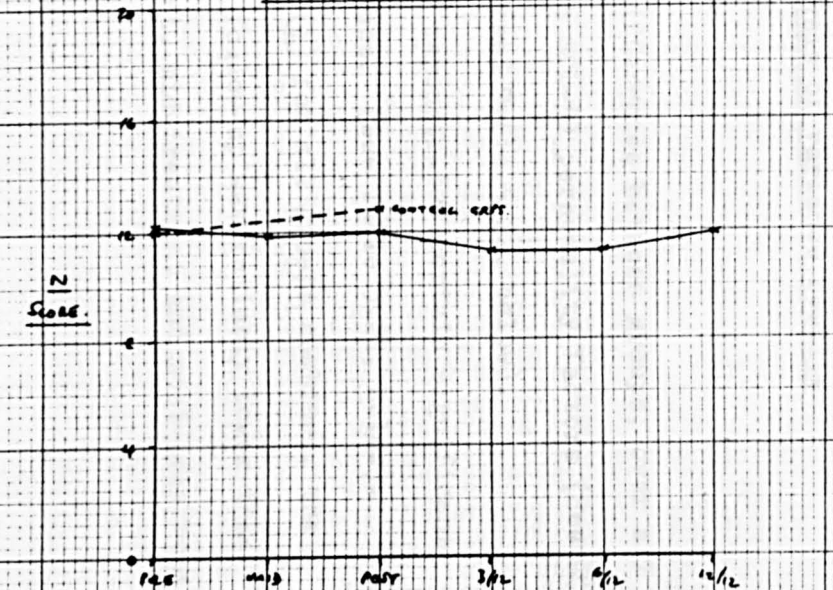


FIG. 5.34.

EPQ(N) SCORES (ALL GROUPS COMBINED)

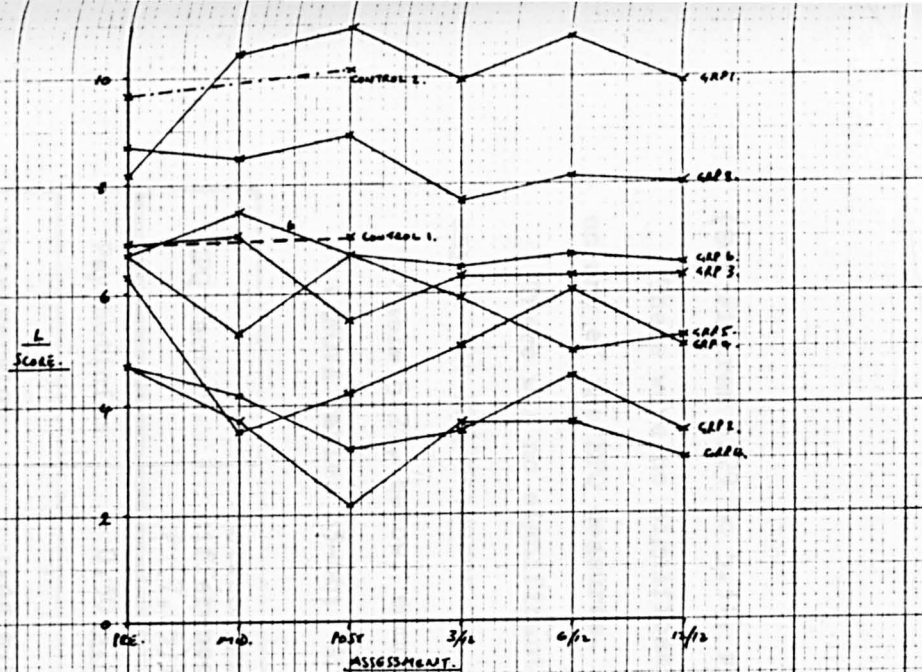


FIG. 5.35.

INDIVIDUAL GROUPS' EPQ(L) SCORES.

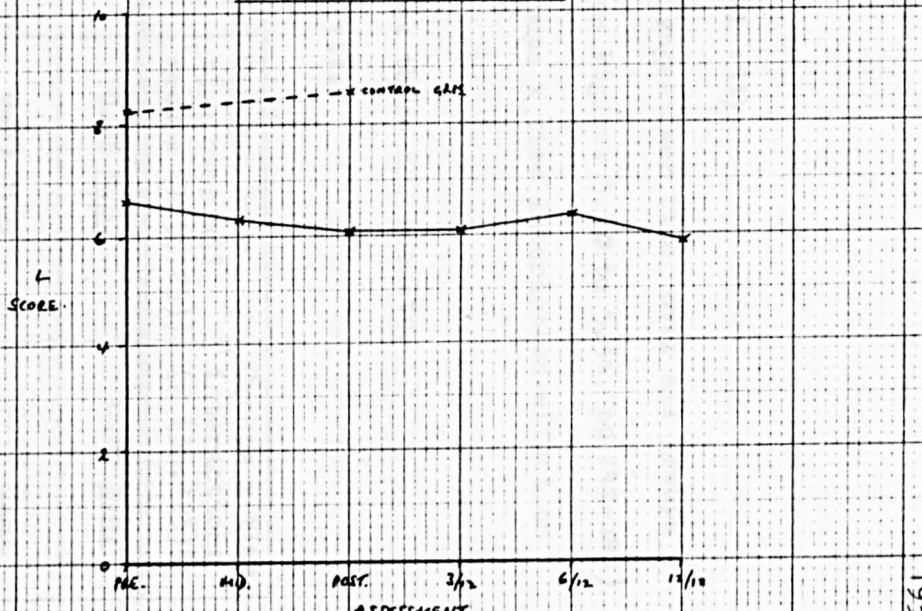


FIG. 5.36.

EPQ(L) SCORES (ALL GROUPS COMBINED).

(See Figures 5.29 to 5.35, pp. 192-193).

There were no significant changes over time, over all groups on any of the EPQ measures. (See Figures 5.30 to 5.36, pp. 192-193).

There was no significant groups x time interaction effect on any of the EPQ measures.

Table 5.21, below, summarizes these EPQ results.

(ANOVA Tables 63-66; Appx. VI).

EPQ Factor	Between groups differences (F (7,25)).	Changes over time over all groups (F(5,125))	Groups x time interactions (F (35,125))
Psychoticism	0.30 N.S.	0.05 N.S.	0.86 N.S.
Extraversion	1.44 N.S.	0.93 N.S.	0.87 N.S.
Neuroticism	0.77 N.S.	0.50 N.S.	0.89 N.S.
Lie Score	1.66 N.S.	1.47 N.S.	0.96 N.S.

Table 5.21

EPQ outcome data - analysis of variance results

b) SCL-90

There were no significant differences between groups, over all assessment points, on any of the SCL-90 personality factors (See Figures 5.37 to 5.59, pp. 196-201 and Table 5.22, below).

Three of the SCL-90 factors demonstrated certain significant changes over time, over all groups. (See Table 5.22, below). The Tukey (a) Test showed that (i) for all groups combined, the mean Anxiety score at 12-month follow-up was reliably lower than the mean scores at pre-treatment, mid-treatment and post-treatment

assessments ($p \leq 0.01$); (ii) for all groups combined, the mean Hostility score at 12-month follow-up was reliably lower than the mean scores at pre-treatment, mid-treatment and post-treatment assessments ($p \leq 0.025$); and that (iii) for all groups combined, the mean Psychoticism score at mid-treatment assessment was reliably lower than the mean score at pre-treatment ($p \leq 0.05$) and the 12-month follow-up mean reliably lower than the means at pre-treatment and post-treatment ($p \leq 0.05$). (See Figures 5.38 to 5.60, pp. 196-201).

There was no significant groups x time interaction effect on any of the SCL-90 measures.

Table 5.22, below, summarizes these SCL-90 results. (ANOVA Tables 67-78; Appx. VI).

SCL-90 Factor	Between group differences (F(7,25))	Changes over time over all groups (F(5,125))	Groups x time interactions (F(35,125)).
Somatic Anxiety	0.45 N.S.	1.01 N.S.	0.77 N.S.
Obsessive Compulsiveness	1.59 N.S.	0.86 N.S.	1.01 N.S.
Interpersonal Sensitivity	0.81 N.S.	1.22 N.S.	1.19 N.S.
Depression	1.68 N.S.	1.19 N.S.	1.01 N.S.
Anxiety	1.18 N.S.	3.29 $p<0.01$	1.16 N.S.
Hostility	0.58 N.S.	3.02 $p<0.025$	1.14 N.S.
Phobic Anxiety	0.55 N.S.	1.64 N.S.	0.94 N.S.
Paranoid Ideation	1.44 N.S.	0.43 N.S.	0.76 N.S.
Psychoticism	0.89 N.S.	2.71 $p<0.025$	0.74 N.S.
General Symptomatic Index	1.62 N.S.	1.29 N.S.	0.82 N.S.
Positive Symptom Distress Level	2.41 N.S.	1.63 N.S.	0.82 N.S.
Positive Symptom Total	0.92 N.S.	0.72 N.S.	0.81 N.S.

Table 5.22
SCL-90 outcome data - analysis of variance results

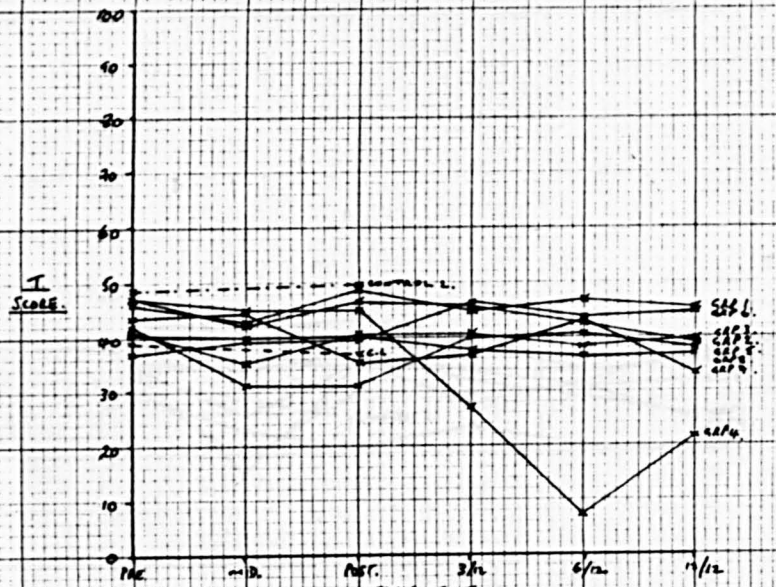


FIG. 5.37.

INDIVIDUAL GROUPS' SCL-90 "SOMATIC ANXIETY" SCORES.

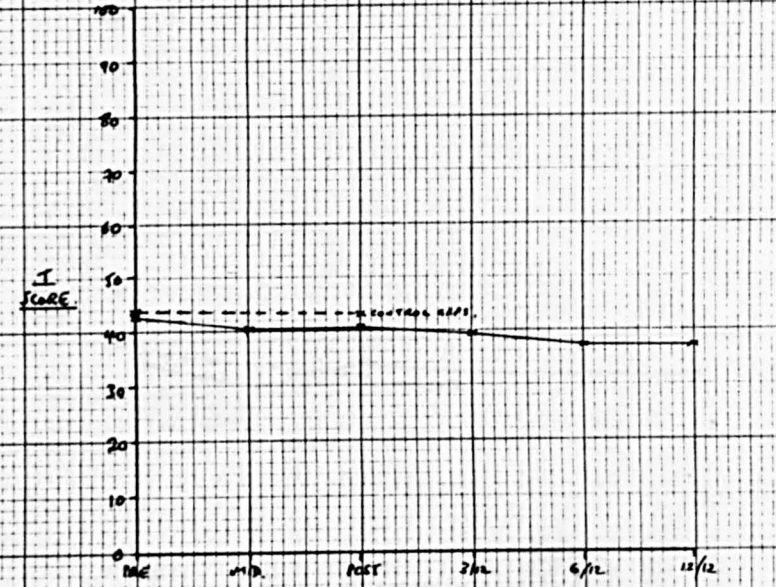


FIG 5.38.

SCL-90 "SOMATIC ANXIETY" SCORES (ALL GROUPS COMBINED).

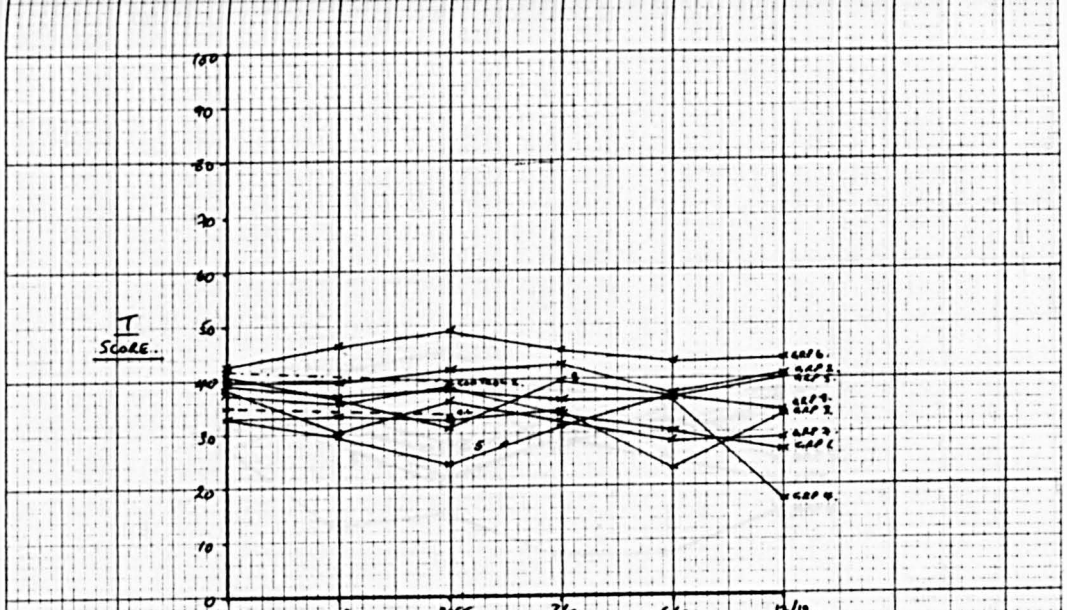


FIG 5.39.

INDIVIDUAL GROUPS' SCL-90 "OBSESSIVE-COMPULSIVENESS" SCORES.

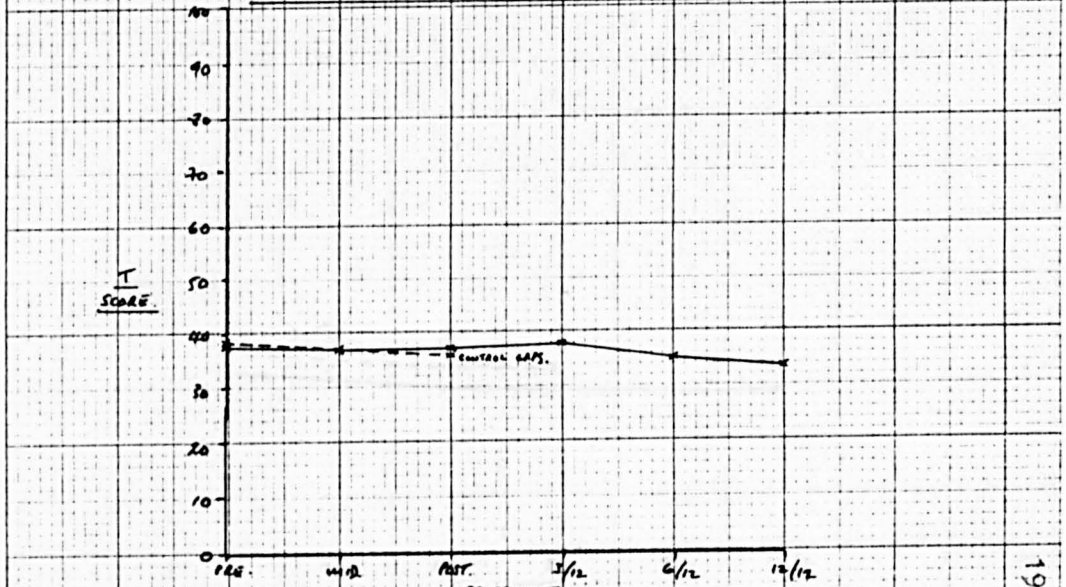


FIG 5.40.

SCL-90 "OBSESSIVE-COMPULSIVENESS" SCORES (ALL GROUPS COMBINED).

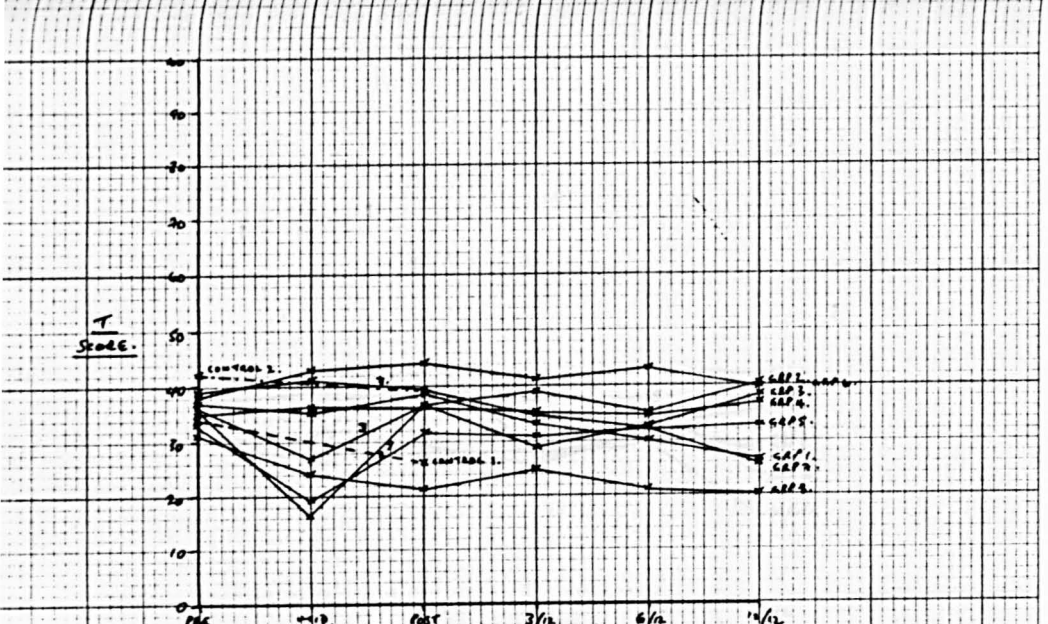


FIG 5.41.

INDIVIDUAL GROUPS' SCL-90 "INTERPERSONAL SENSITIVITY" SCORES.

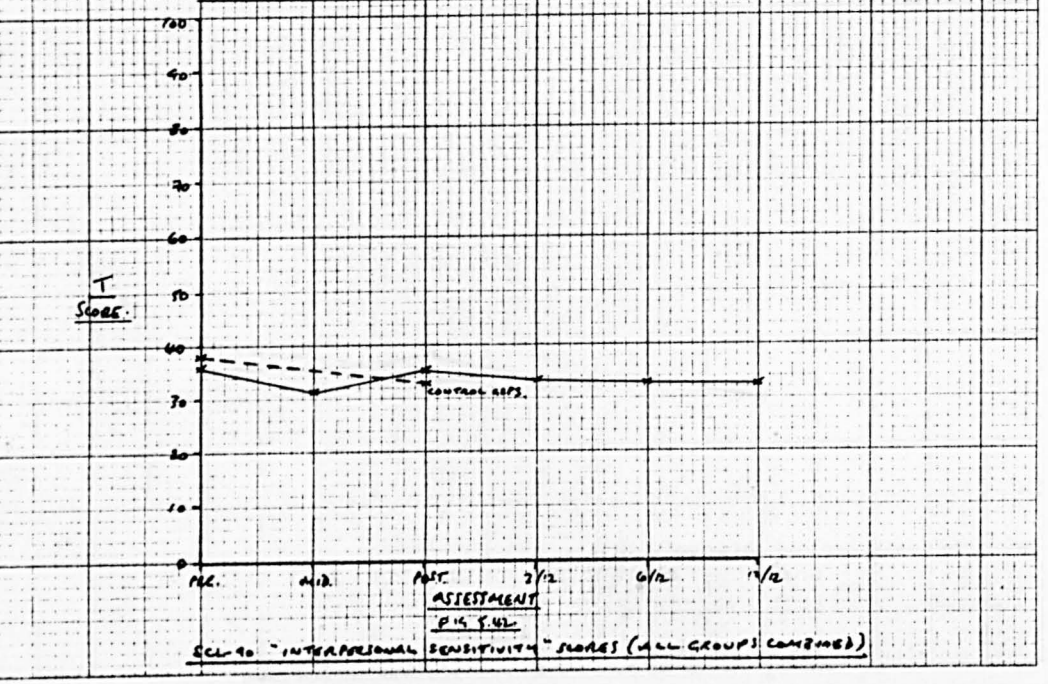


FIG 5.42.

SCL-90 "INTERPERSONAL SENSITIVITY" SCORES (ALL GROUPS COMBINED)

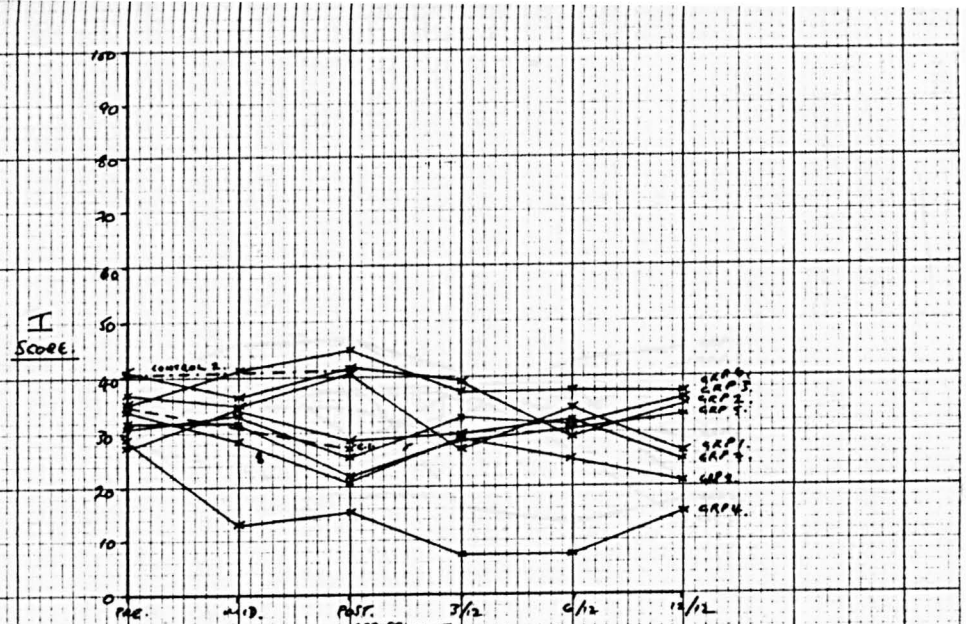


FIG 5.43.

INDIVIDUAL GROUPS' SCL-90 "DEPRESSION" SCORES

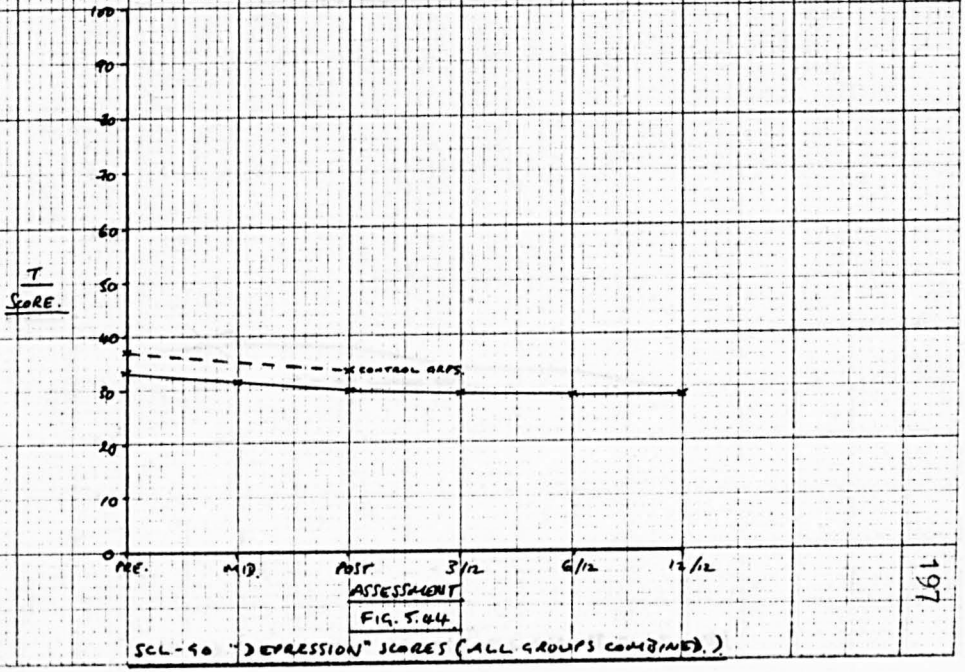
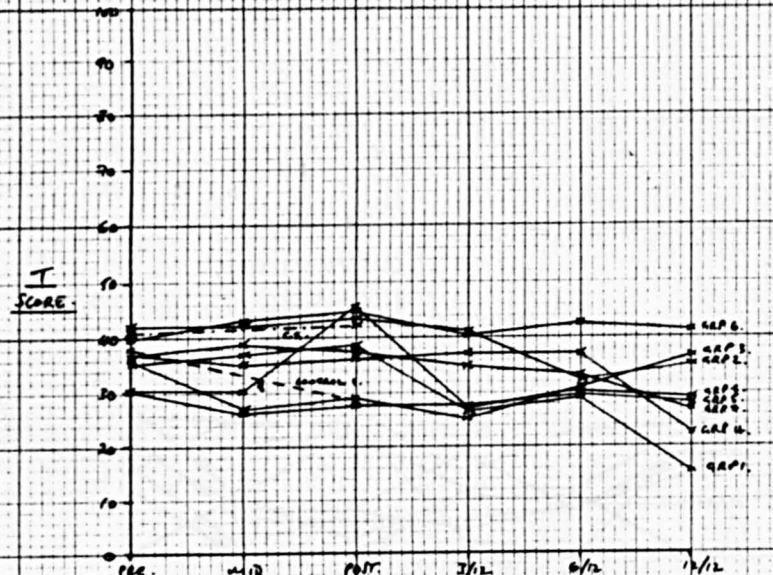


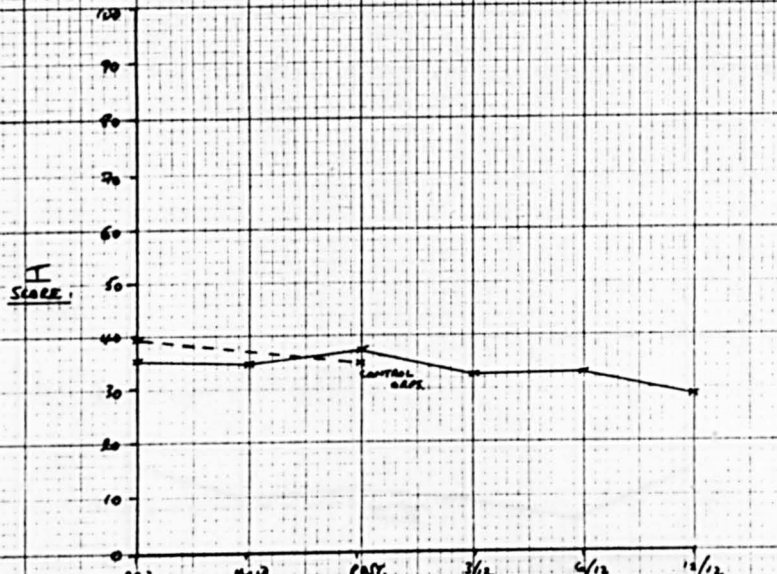
FIG. 5.44.

SCL-90 "DEPRESSION" SCORES (ALL GROUPS COMBINED.)



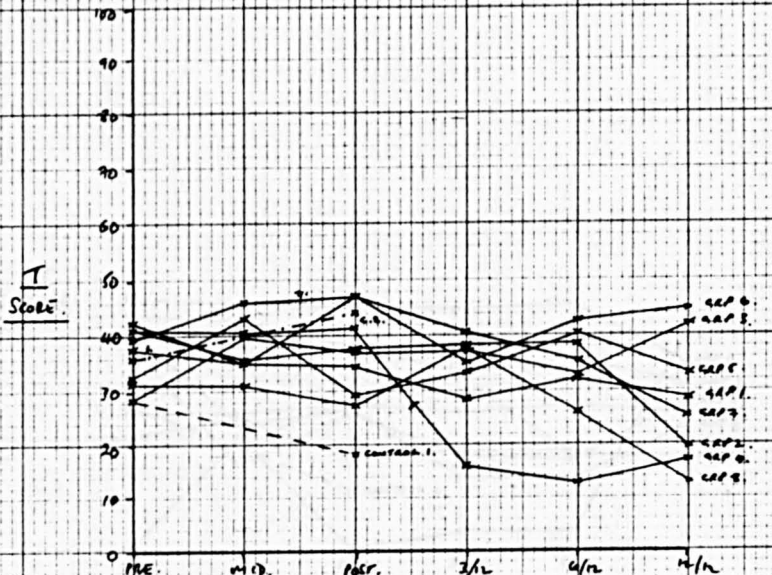
INDIVIDUAL GROUPS' SCL-90 "ANXIETY" SCORES.

FIG. 5.45.



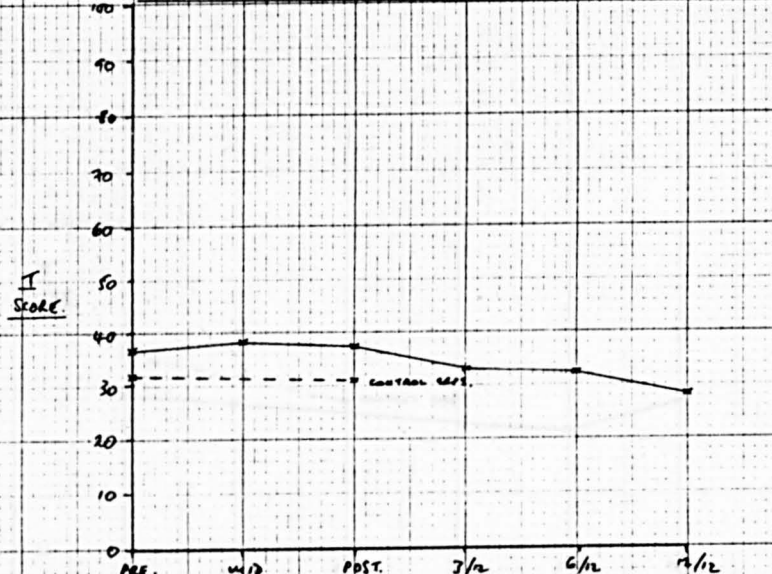
SCL-90 "ANXIETY" SCORES (ALL GROUPS COMBINED).

FIG. 5.46.



INDIVIDUAL GROUPS' SCL-90 "HOSTILITY" SCORES.

FIG. 5.47.



SCL-90 "HOSTILITY" SCORES (ALL GROUPS COMBINED).

FIG. 5.48.

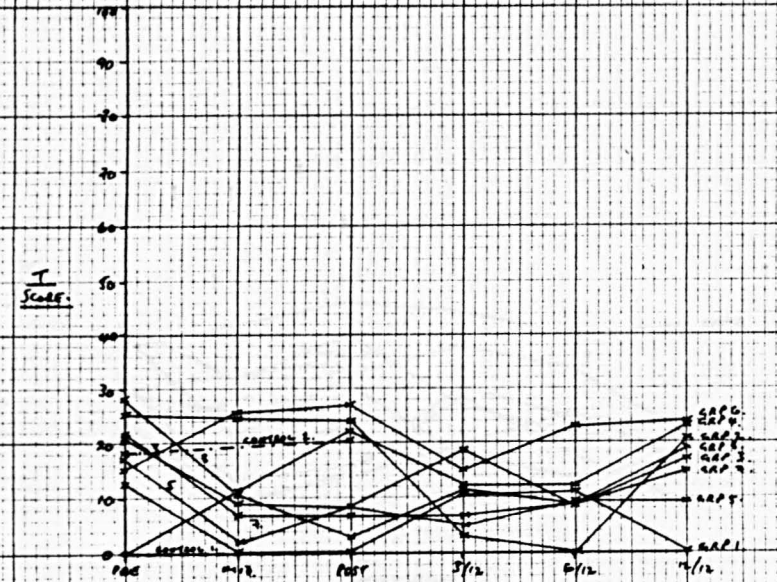


FIG. 5.49.

INDIVIDUAL GROUPS' SCL-90 "PHOBIC ANXIETY" SCORES.

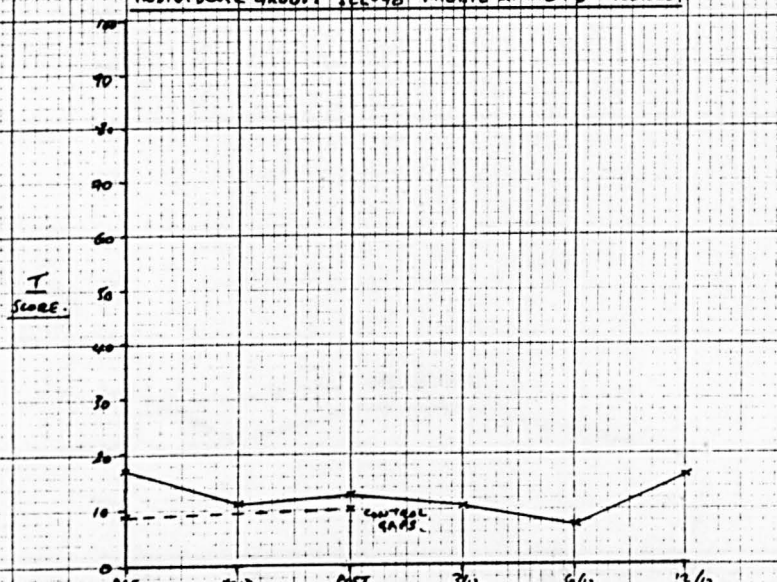


FIG. 5.50.

SCL-90 "PHOBIC ANXIETY" SCORES (ALL GROUPS COMBINED.)

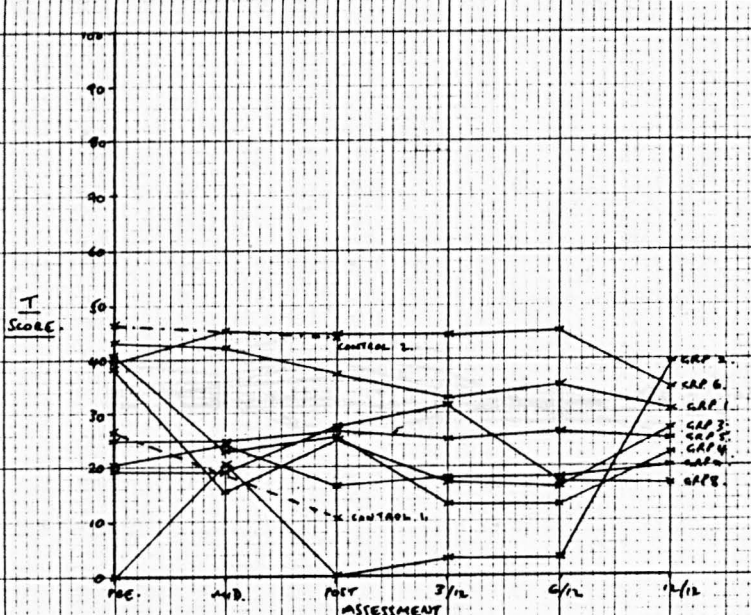


FIG. 5.51.

INDIVIDUAL GROUPS' SCL-90 "PARANOID IDEATION" SCORES

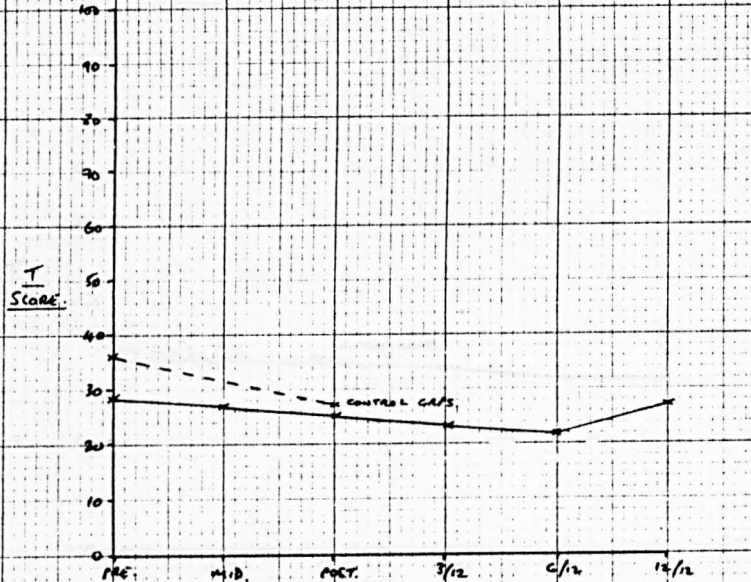


FIG. 5.52.

SCL-90 "PARANOID IDEATION" SCORES (ALL GROUPS COMBINED.)

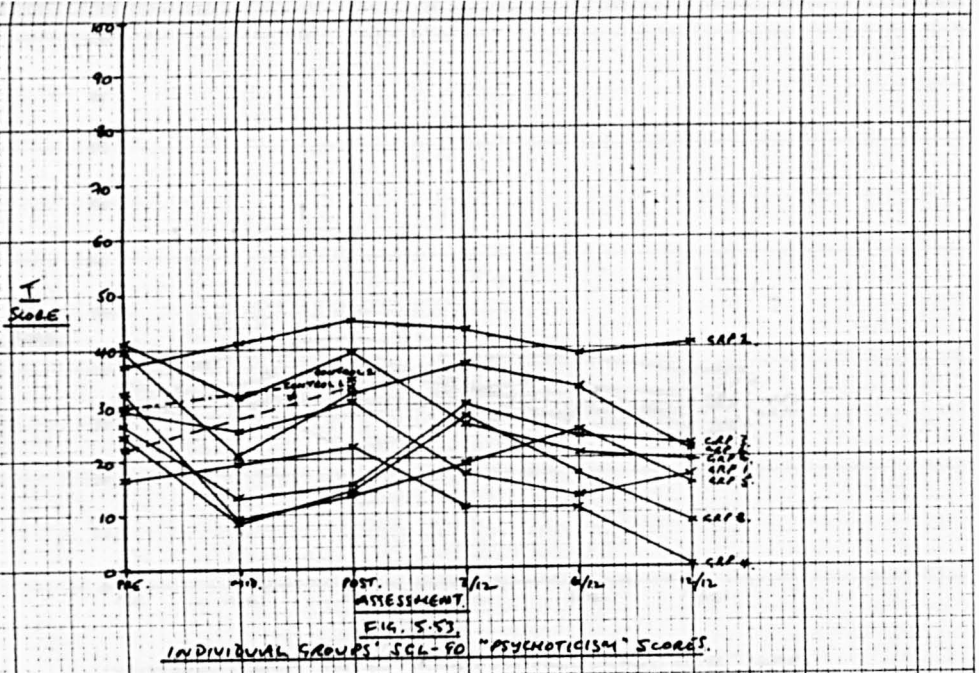


FIG. 5.53.

INDIVIDUAL GROUPS' SCL-90 "PSYCHOTICISM" SCORES.

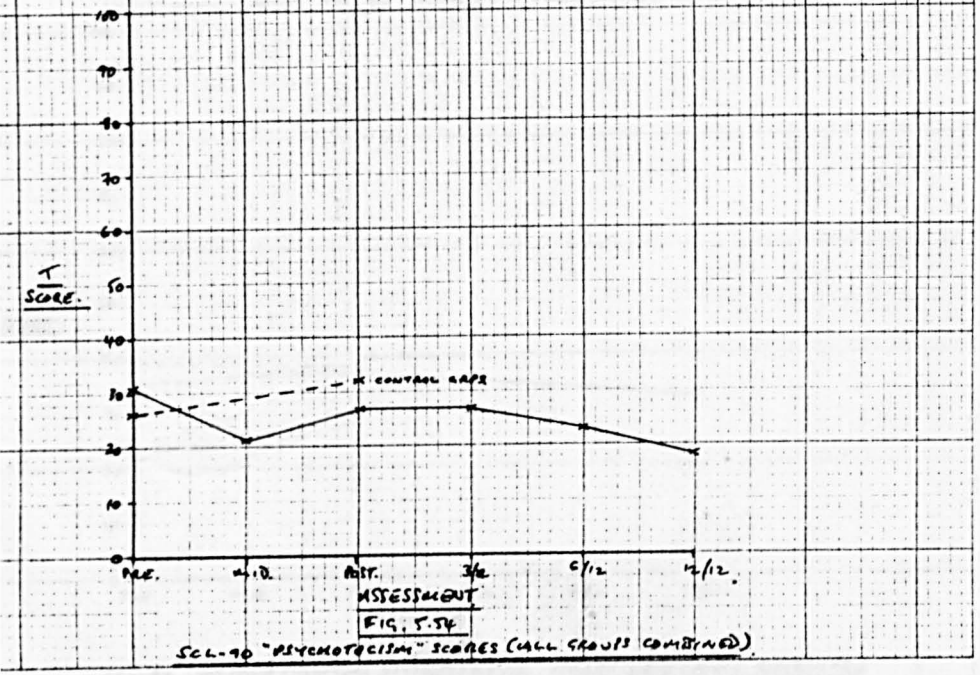


FIG. 5.54.

SCL-90 "PSYCHOTICISM" SCORES (ALL GROUPS COMBINED).

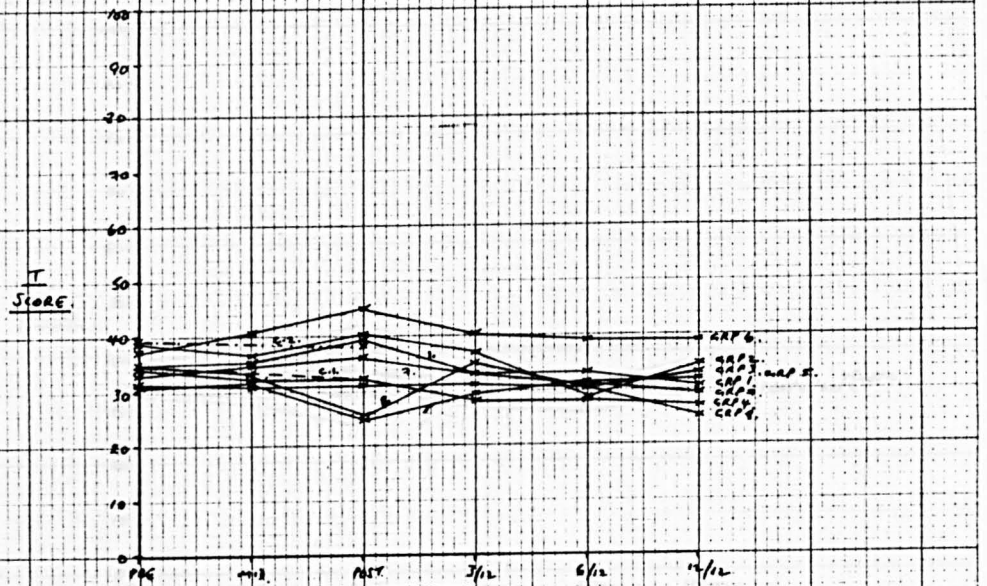


FIG 5.55.

(INDIVIDUAL GROUPS' SCL-90 "GLOBAL SYMPTOMATIC INDEX" SCORES.

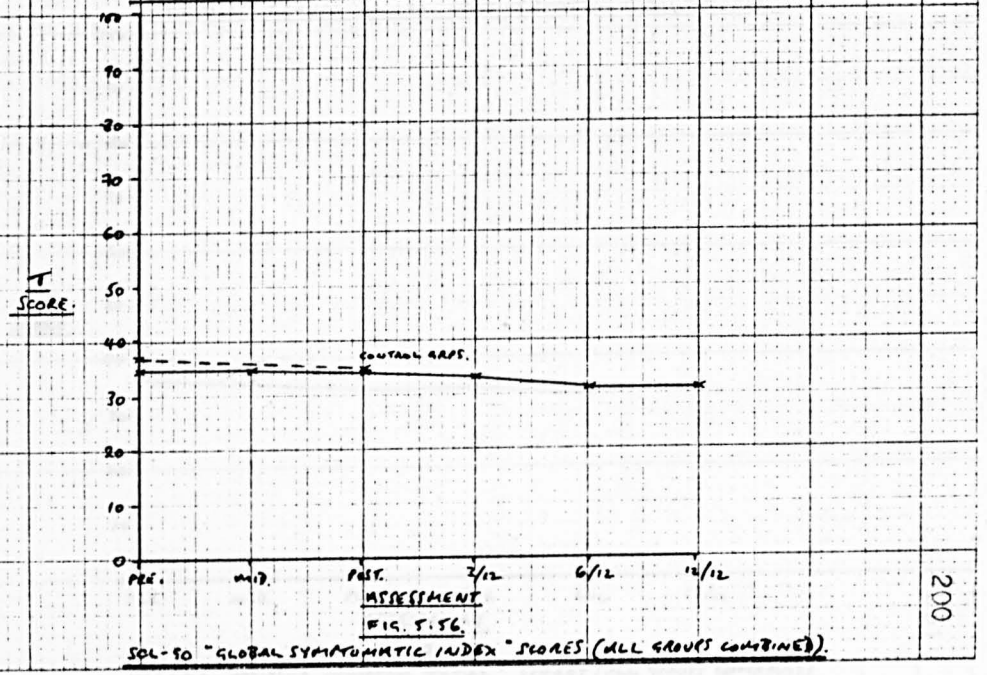


FIG. 5.56.

SCL-90 "GLOBAL SYMPTOMATIC INDEX" SCORES (ALL GROUPS COMBINED).

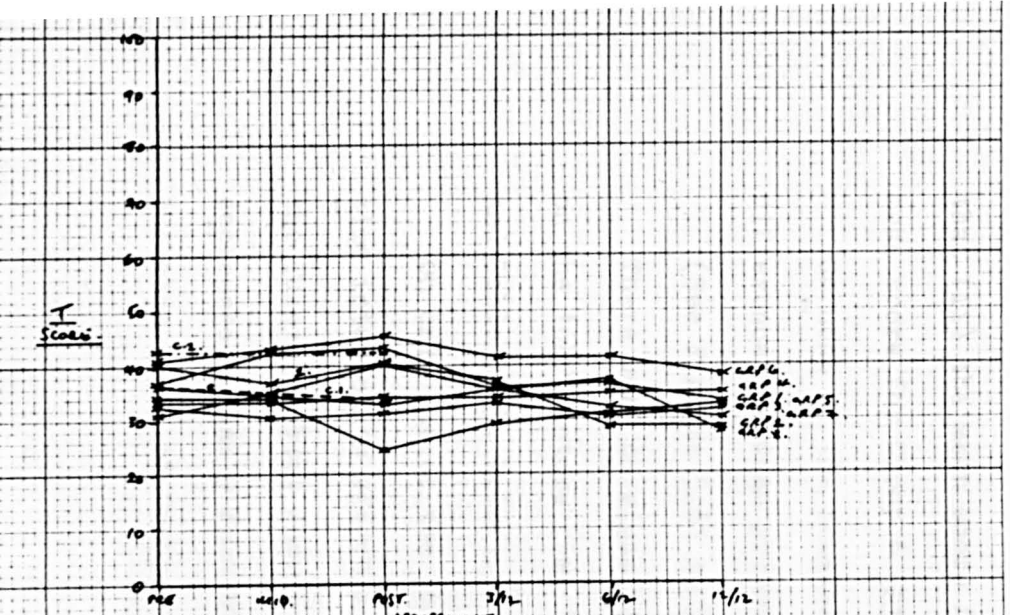


FIG. 5.57.

INDIVIDUAL GROUPS' SCL-90 "POSITIVE SYMPTOM DISTRESS LEVEL" SCORES.

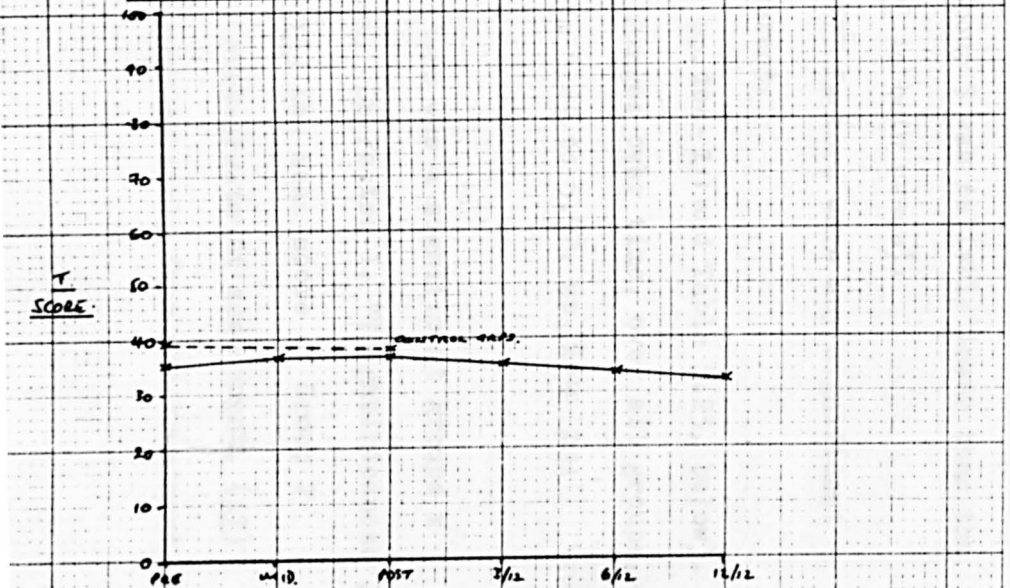


FIG. 5.58.

SCL-90 "POSITIVE SYMPTOM DISTRESS LEVEL" SCORES (ALL GROUPS COMBINED)

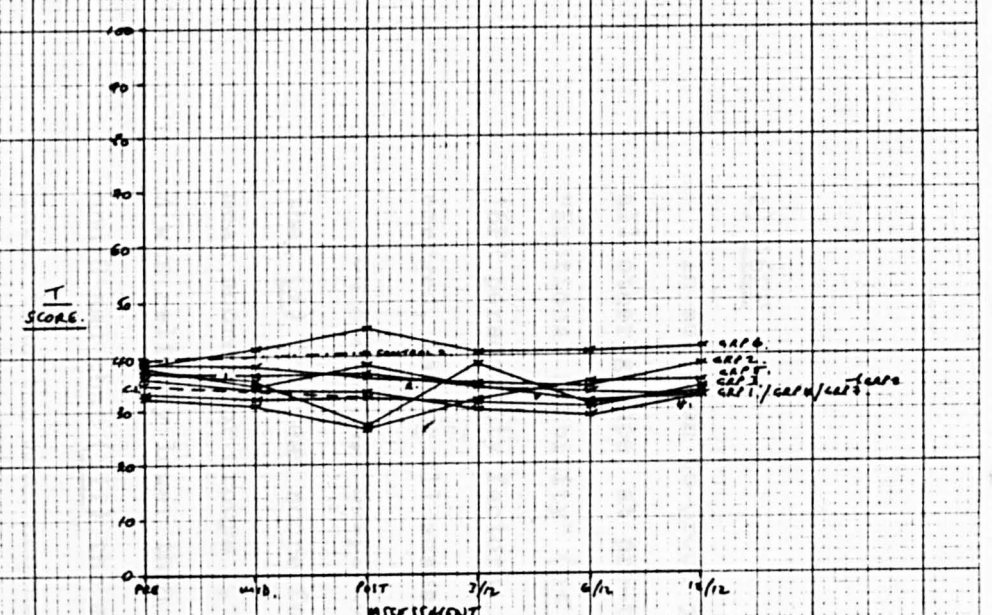


FIG. 5.59.

INDIVIDUAL GROUPS' SCL-90 "POSITIVE SYMPTOM TOTAL" SCORES.

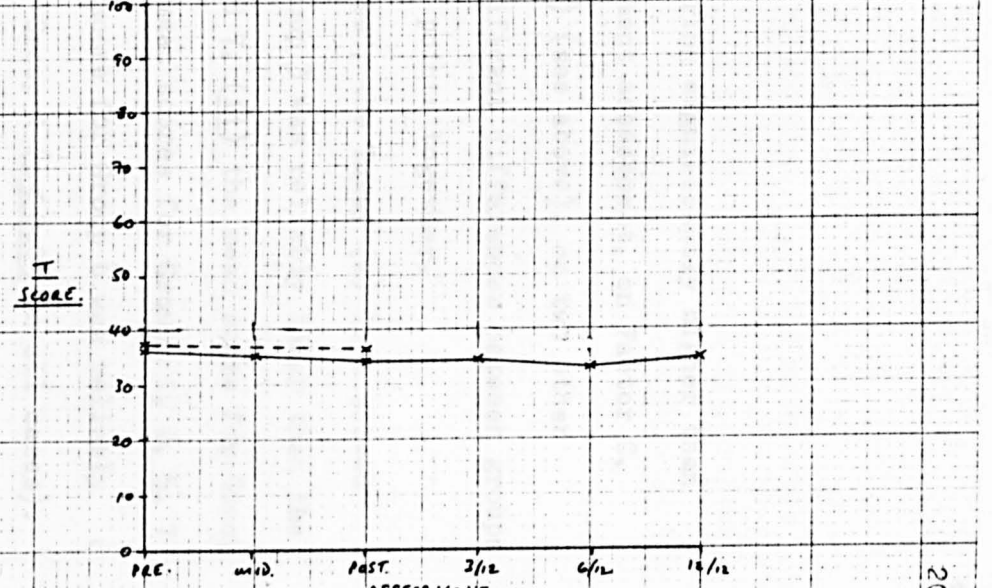


FIG. 5.60.

SCL-90 "POSITIVE SYMPTOM TOTAL" SCORES (ALL GROUPS COMBINED)

c) 16PF

In addition to Group 3 scoring significantly higher than all other groups, at pre-treatment assessment, on Factor C, "Emotional Stability" ($p \leq 0.05$) (see above), on two other 16PF personality factors, significant differences between groups emerged, taking all assessment points together.

On Factor L, "Suspiciousness", the Tukey (a) Test showed that (i) the mean score for Group 3 was reliably lower than the mean scores for Groups 1, 5 and 7, (ii) the mean score for Group 4 was reliably lower than the mean scores for Groups 1, 2, 5, 7 and 8 and that (iii) the mean score for Group 6 was reliably lower than the mean scores for Group 1 ($p \leq 0.05$ in all cases).

On Factor Q_1, "Conservatism", the Tukey (a) Test showed that (i) the mean score for Group 8 was reliably lower (ie. more "conservative") than the mean scores for Groups 2, 6 and 7 and that (ii) the mean score for Group 5 was reliably lower than the mean score for Group 6 ($p \leq 0.05$ in both cases).

On none of the remaining 16PF factors were any significant differences between groups apparent.

(See Figures 5.61 to 5.91, pp. 204-211 and Table 5.23, below).

There were no significant changes over time, over all groups, on the 16PF factors (See Figures 5.62 to 5.92, pp. 204-211 and Table 5.23, below).

On only one of the 16PF factors was a significant groups x time interaction effect apparent. On Factor I, "Toughmindedness", the Tukey (a) Test showed that (i) the mean score of Group 2 decreased reliably from mid- to post-treatment assessment, (ii) the mean score of Group 4 decreased reliably from mid-treatment

to 12-month follow-up assessment and that (iii) the mean score of Group 7 decreased reliably from pre-treatment to mid-treatment assessment (and was also reliably lower than pre-treatment at 12-month follow-up assessment) ($p \leq 0.05$ in all cases).

Table 5.23, below, summarizes these 16PF results.

(ANOVA Tables 79-94 ; Appx. VI).

16PF Factor	Between group differences (F(7,25))	Changes over time over all groups (F(5,125))	Groups x time interactions (F(35,125))
A	0.87 N.S.	0.75 N.S.	0.91 N.S.
B	0.79 N.S.	0.68 N.S.	0.86 N.S.
C	0.61 N.S.	1.84 N.S.	1.05 N.S.
E	0.83 N.S.	0.89 N.S.	1.08 N.S.
F	0.28 N.S.	0.39 N.S.	0.85 N.S.
G	0.73 N.S.	0.87 N.S.	1.27 N.S.
H	1.19 N.S.	1.79 N.S.	1.48 N.S.
I	1.65 N.S.	1.61 N.S.	1.64 $p<0.05$
L	3.31 $p<0.025$	1.00 N.S.	0.59 N.S.
M	0.48 N.S.	1.33 N.S.	0.98 N.S.
N	0.39 N.S.	0.53 N.S.	1.01 N.S.
O	1.27 N.S.	0.10 N.S.	0.73 N.S.
Q_1	2.76 $p<0.05$	0.80 N.S.	1.38 N.S.
Q_2	0.67 N.S.*	1.54 N.S.	1.03 N.S.
Q_3	0.89 N.S.	1.23 N.S.	0.93 N.S.
Q_4	0.81 N.S.	1.51 N.S.	1.25 N.S.

Table 5.23

16PF outcome data - analysis of variance results

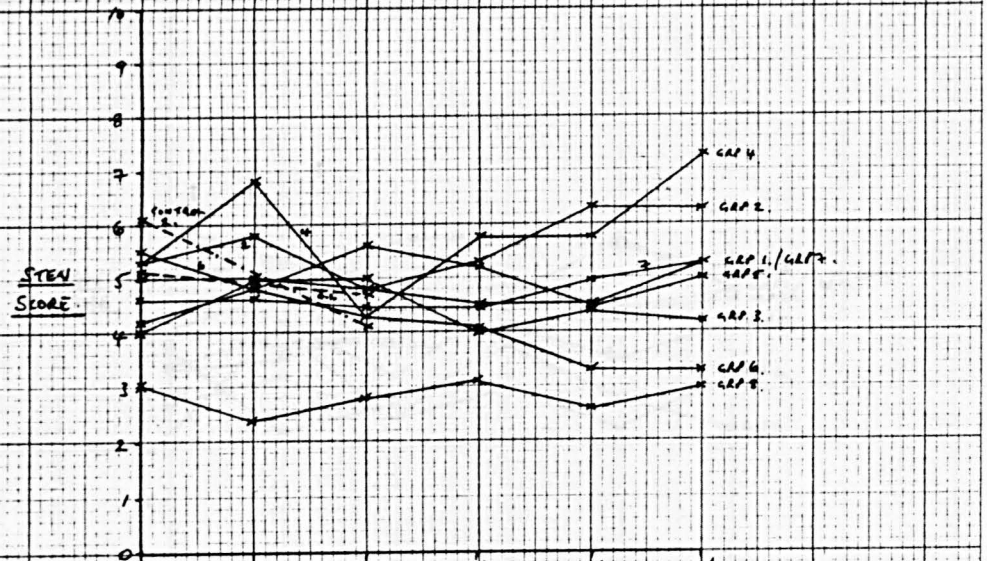


FIG 5.61

INDIVIDUAL GROUPS' 16PF "FACTOR A" SCORES.

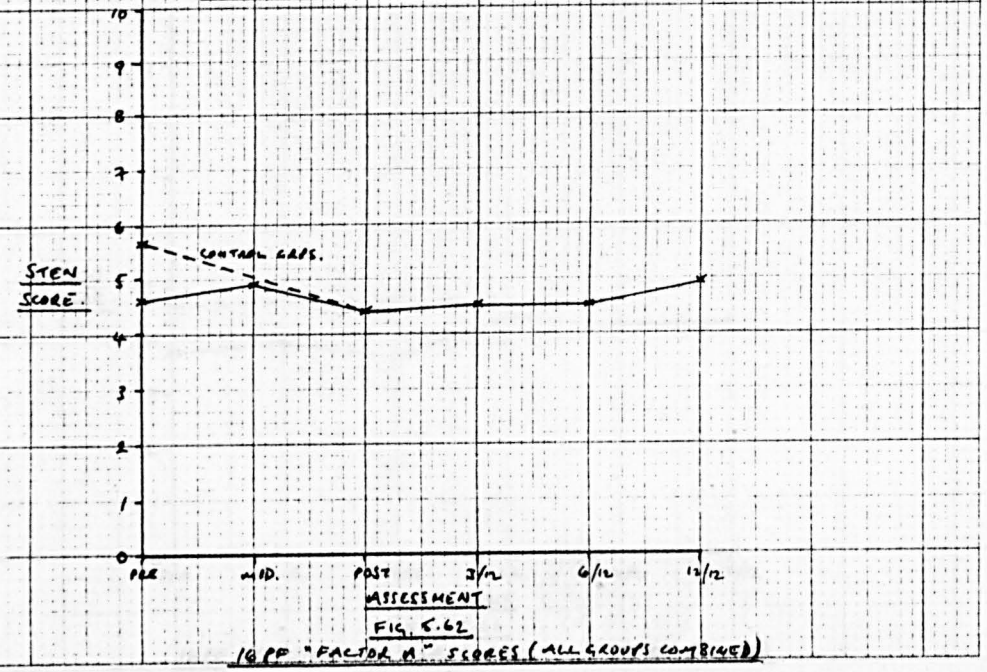


FIG. 5.62

16PF "FACTOR A" SCORES (ALL GROUPS COMBINED)

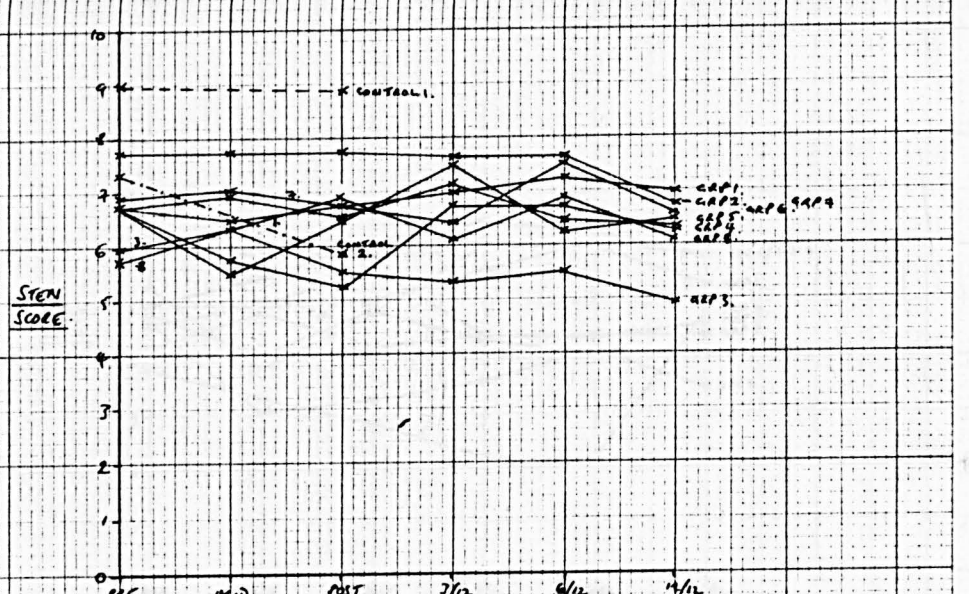


FIG. 5.63

INDIVIDUAL GROUPS' 16PF "FACTOR B" SCORES.

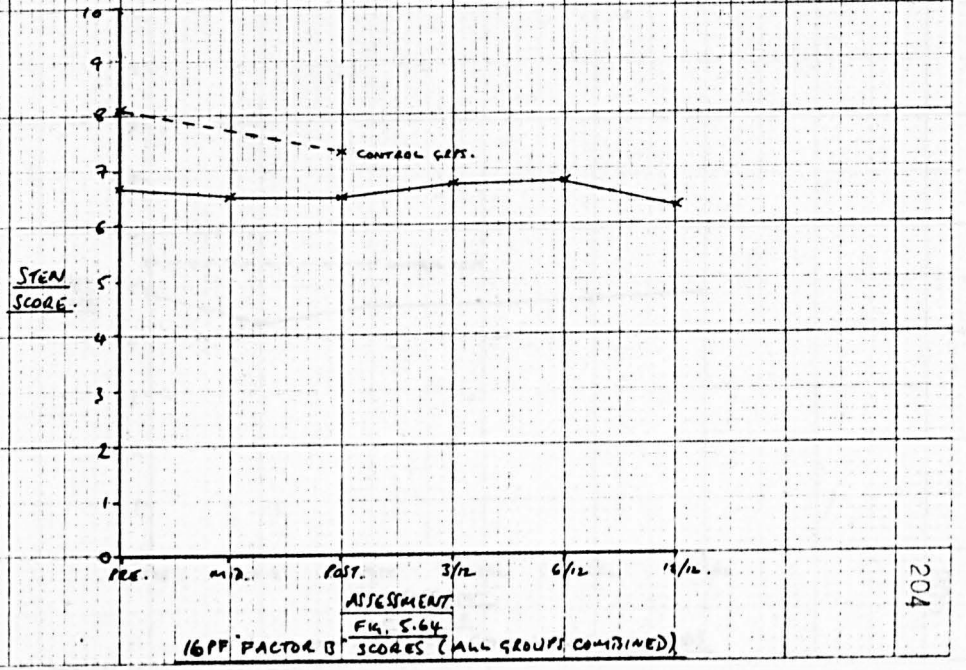


FIG. 5.64

16PF FACTOR B SCORES (ALL GROUPS COMBINED)

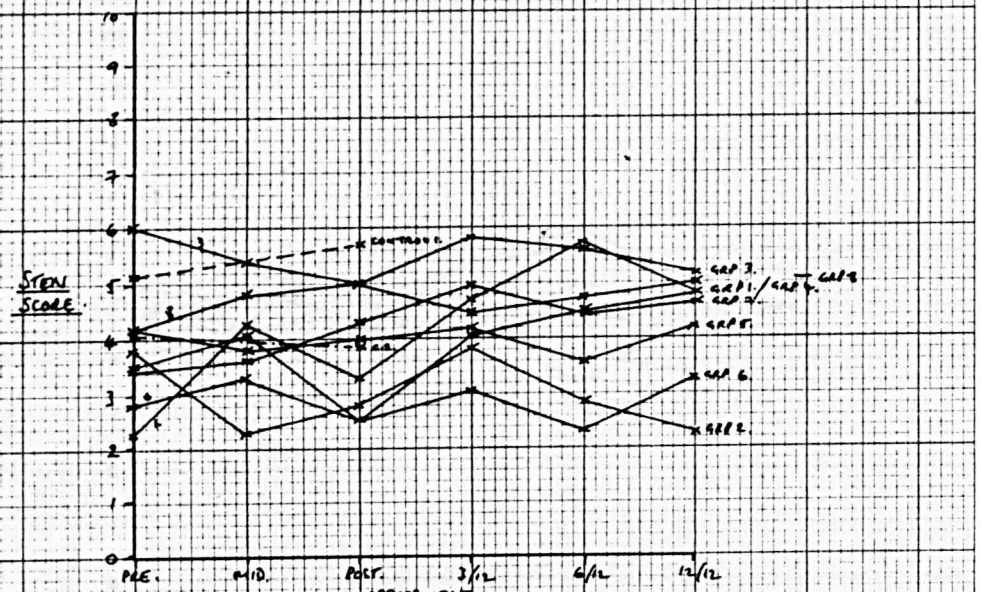


FIG. 5.65

INDIVIDUAL GROUPS' 16PF "FACTOR C" SCORES.

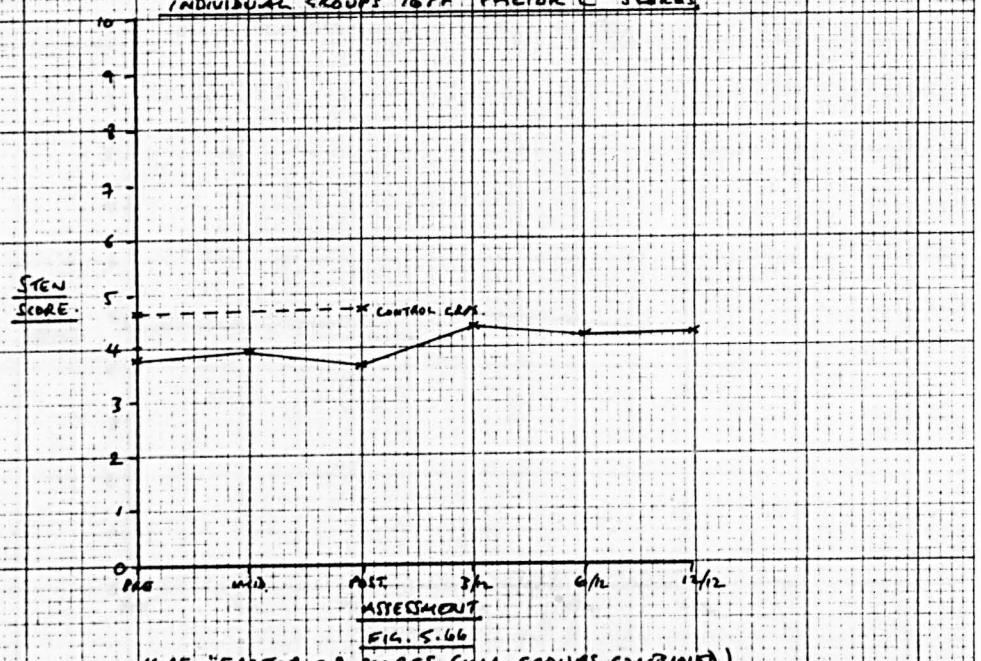


FIG. 5.66

16PF "FACTOR C" SCORES (ALL GROUPS COMBINED)

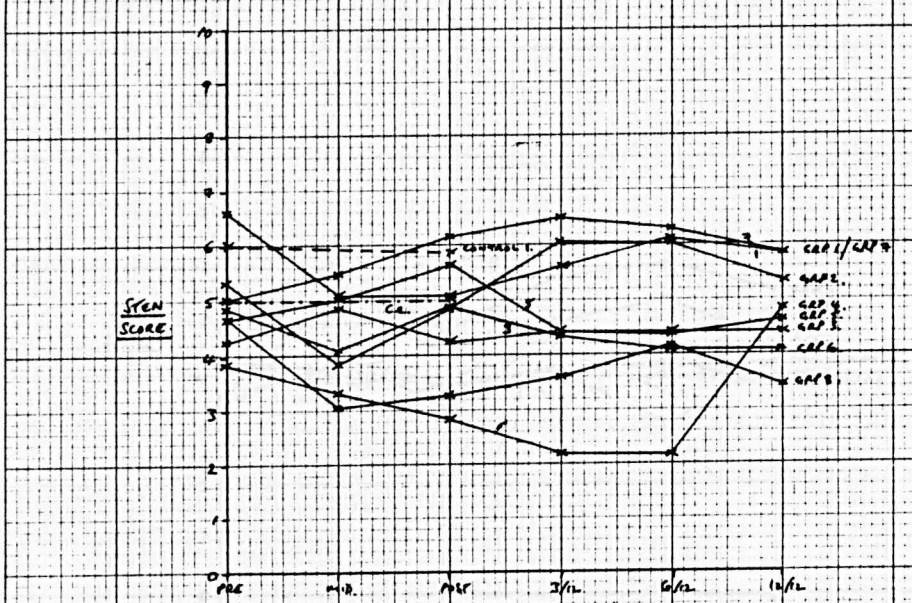


FIG 5.67

INDIVIDUAL GROUPS' 16PF "FACTOR E" SCORES.

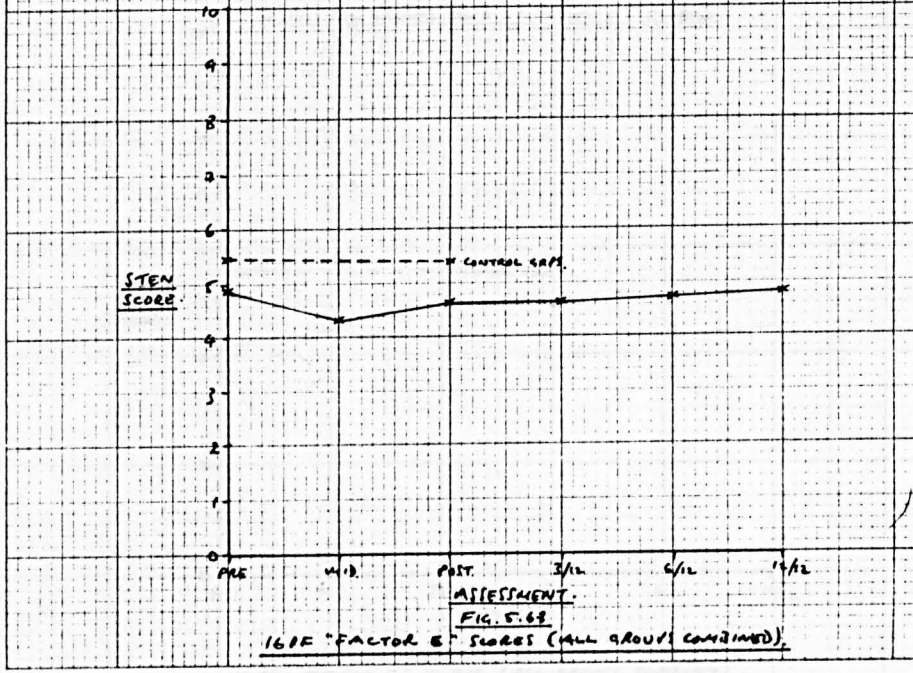
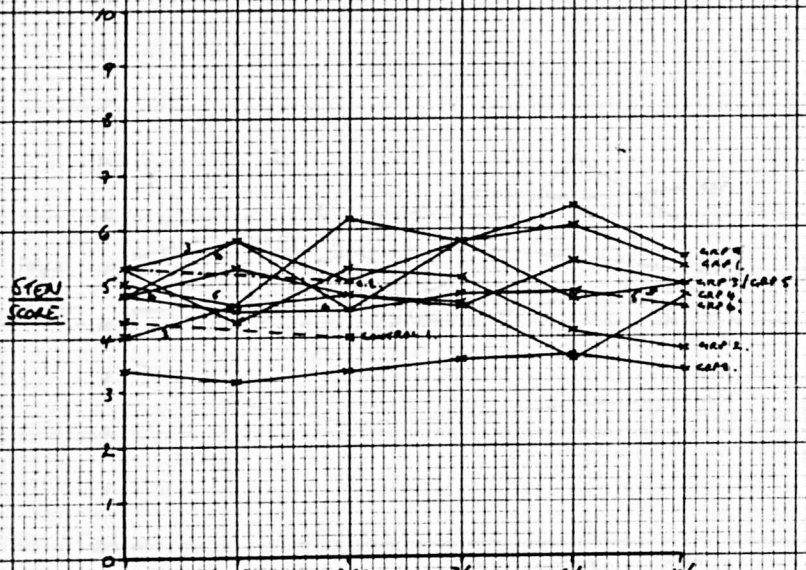


FIG. 5.68

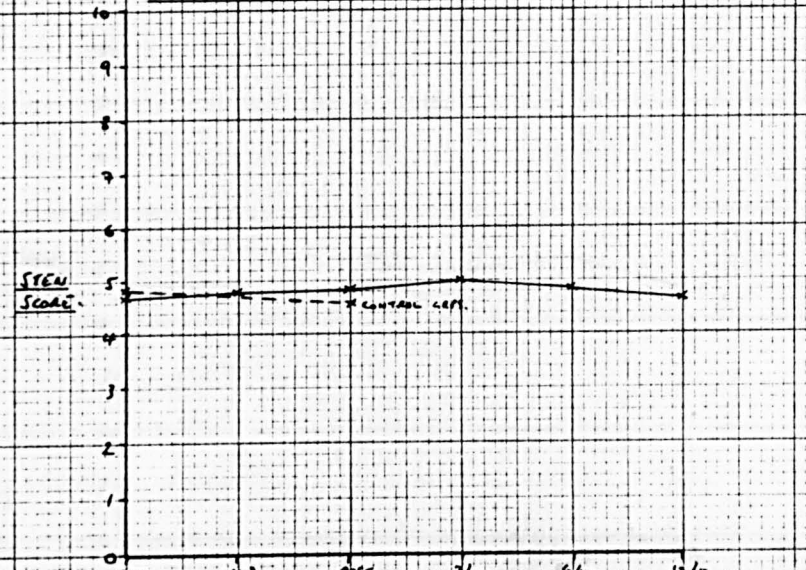
16PF "FACTOR E" SCORES (ALL GROUPS COMBINED).



ASSESSMENT.

FIG. 5.69

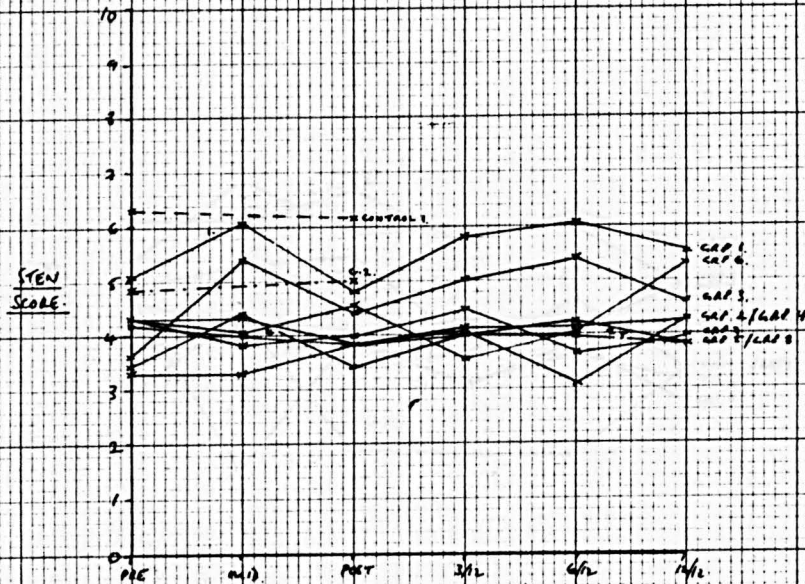
INDIVIDUAL GROUPS' 16PF "FACTOR F" SCORES.



ASSESSMENT.

FIG. 5.70.

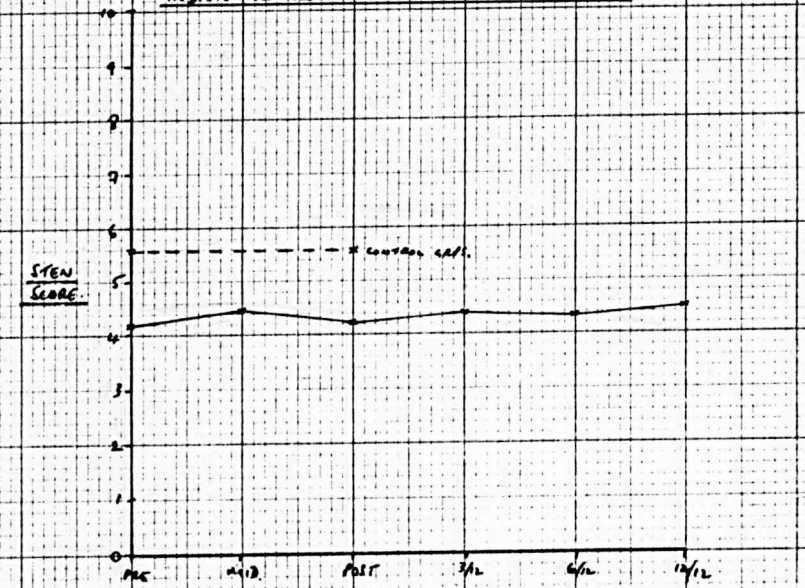
16PF "FACTOR F" SCORES (ALL GROUPS COMBINED)



ASSESSMENT.

FIG. 5.71.

INDIVIDUAL GROUPS' 16PF "FACTOR G" SCORES.



ASSESSMENT.

FIG. 5.72.

16PF "FACTOR G" SCORES (ALL GROUPS COMBINED)

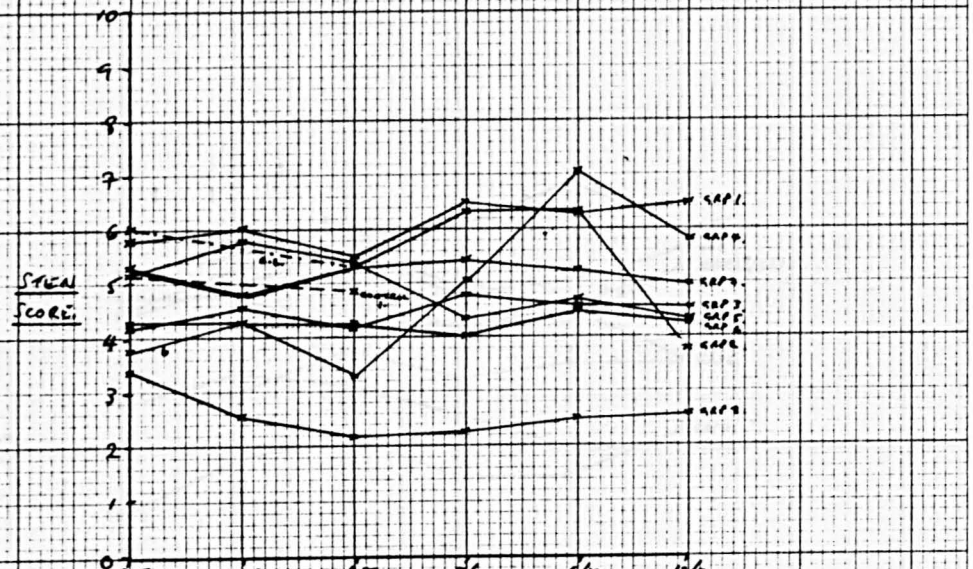


FIG. 5.73

INDIVIDUAL GROUPS' 16PF "FACTOR H" SCORES.

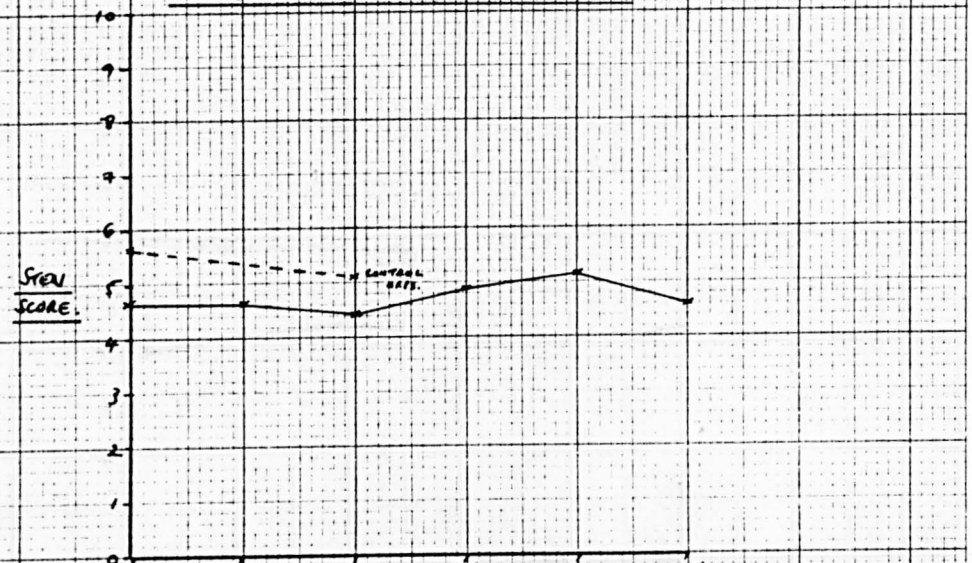


FIG. 5.74

16PF "FACTOR H" SCORES (ALL GROUPS COMBINED).

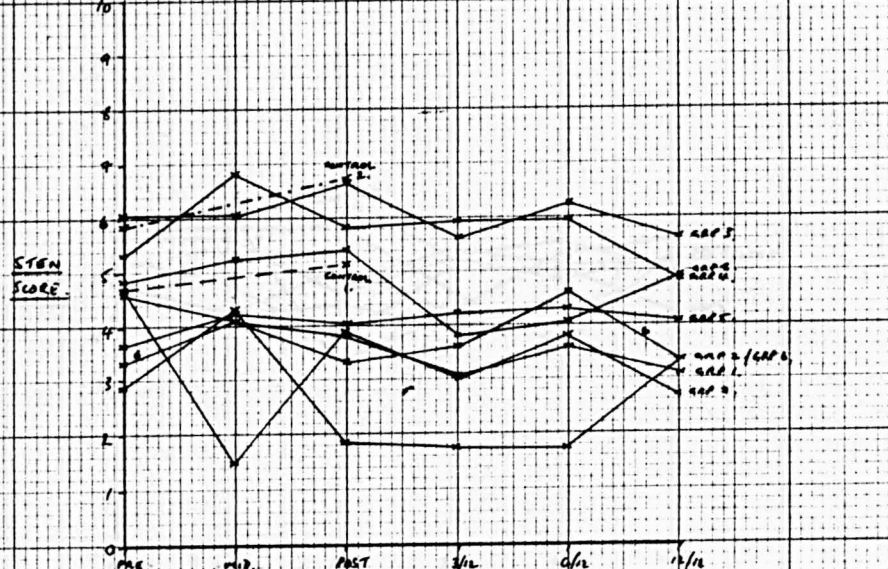


FIG. 5.75.

INDIVIDUAL GROUPS' 16PF "FACTOR I" SCORES.

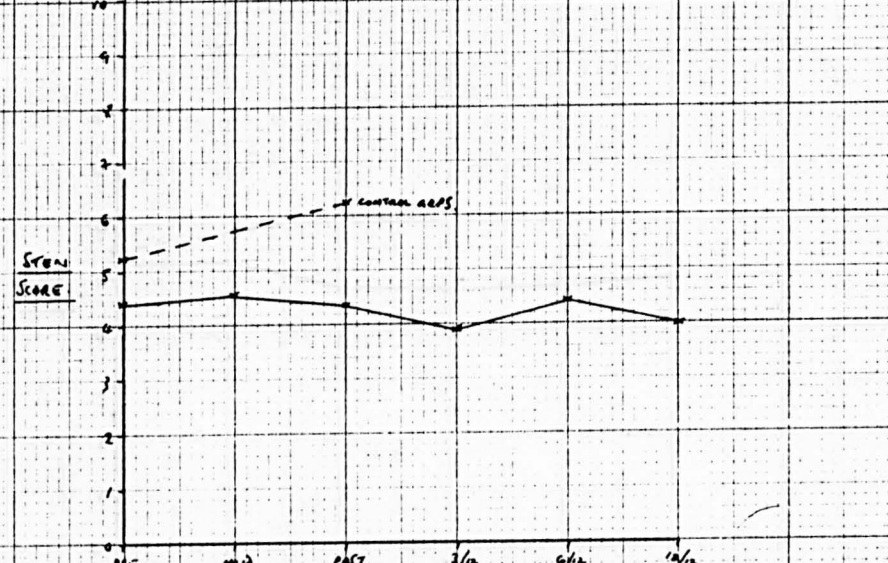


FIG. 5.76

16PF "FACTOR I" SCORES (ALL GROUPS COMBINED).

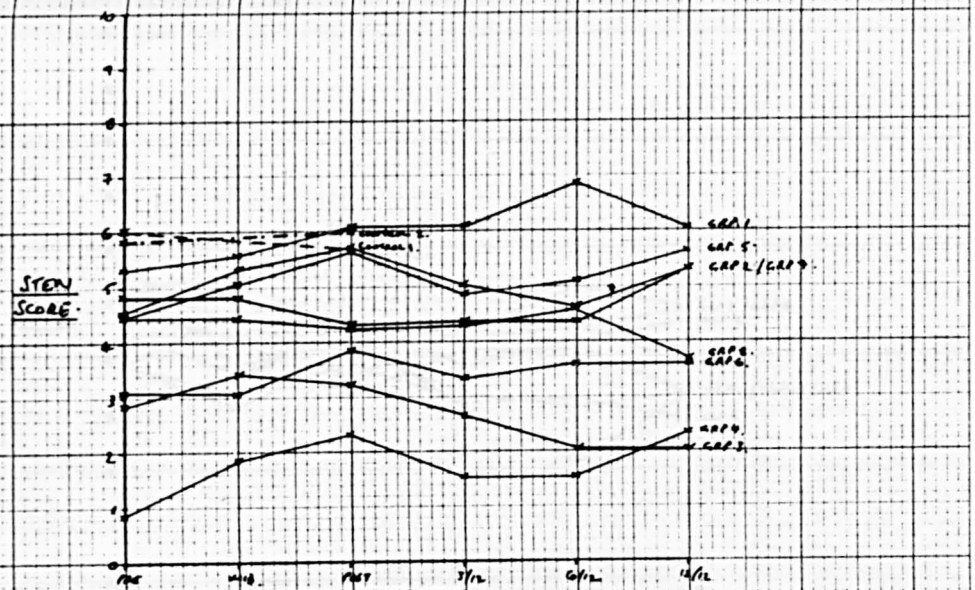


FIG. 5.77

INDIVIDUAL GROUPS' 16PF "FACTOR L" SCORES

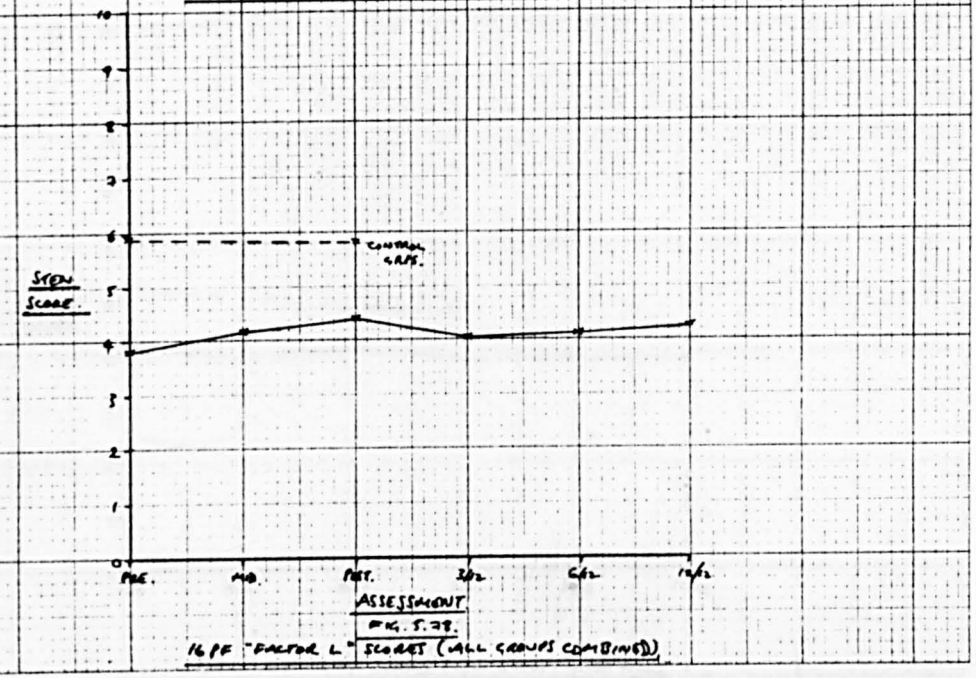


FIG. 5.78.

16PF "FACTOR L" SCORES (ALL GROUPS COMBINED)

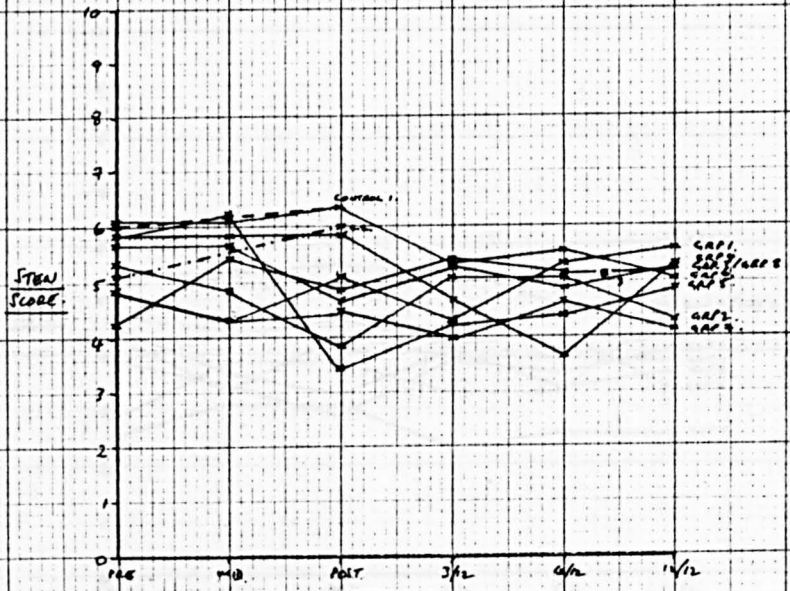


FIG. 5.79.

INDIVIDUAL GROUPS' 16PF "FACTOR M" SCORES

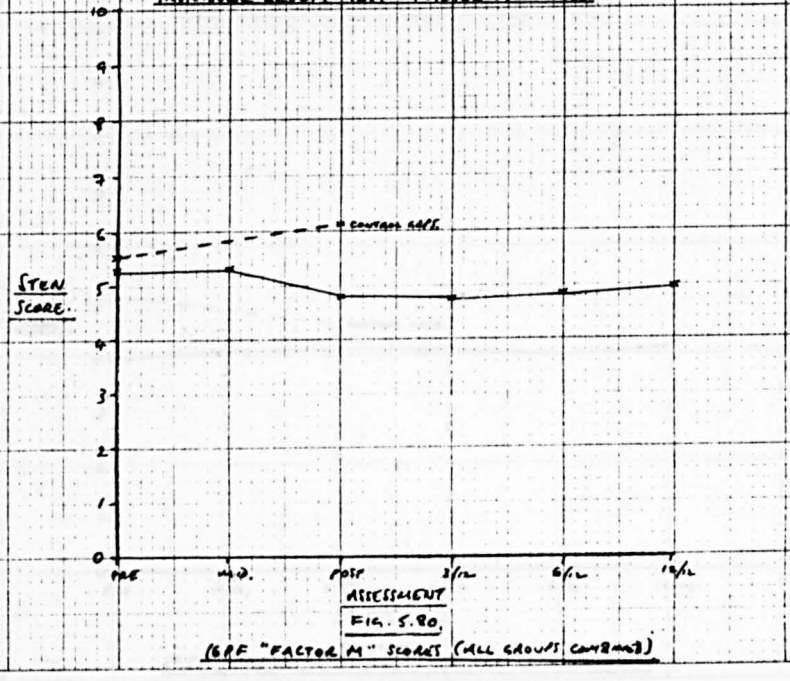


FIG. 5.80.

16PF "FACTOR M" SCORES (ALL GROUPS COMBINED)

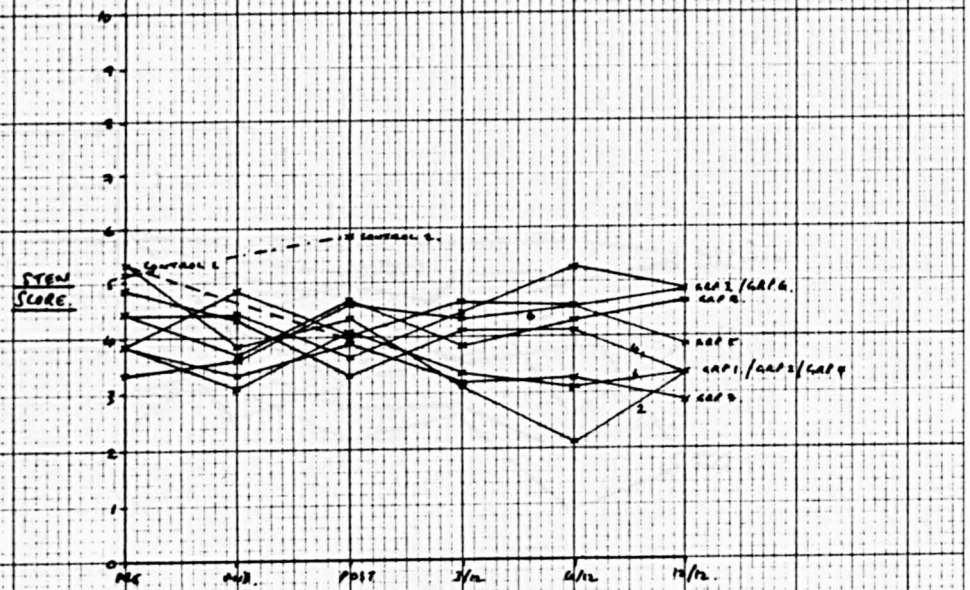


FIG. 5.81.

INDIVIDUAL GROUPS' 16PF "FACTOR N" SCORES.

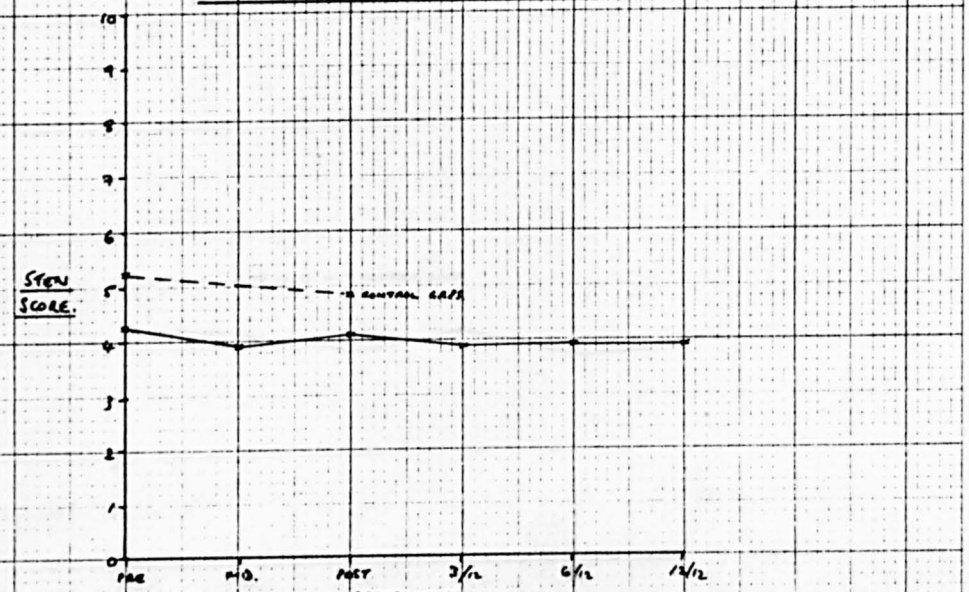


FIG. 5.82.

16PF "FACTOR N" SCORES (ALL GROUPS COMBINED)

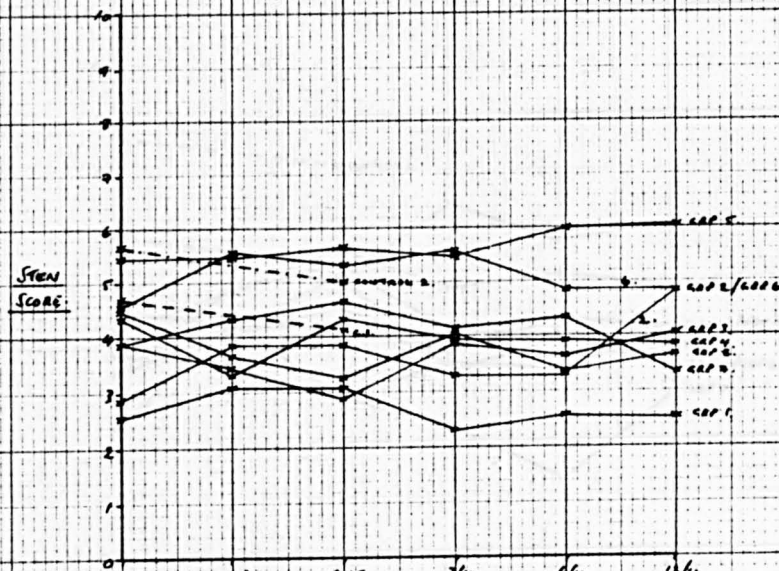


FIG. 5.83.

INDIVIDUAL GROUPS' 16PF "FACTOR O" SCORES.

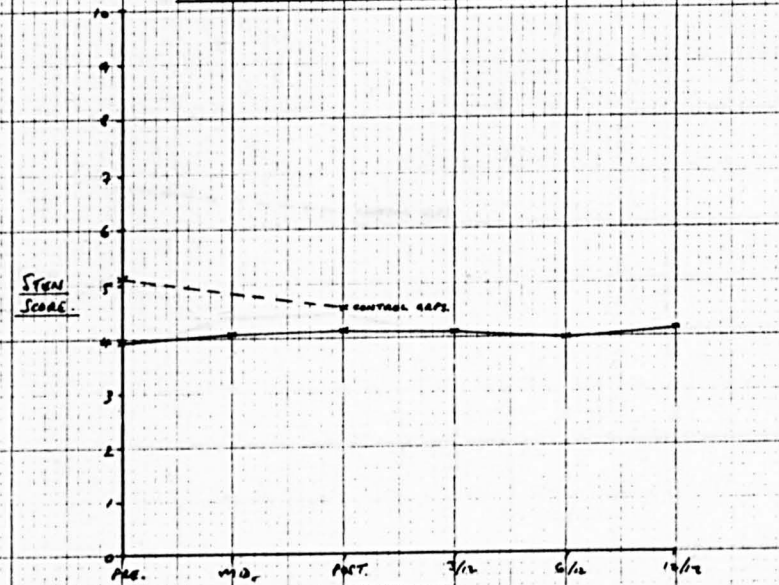


FIG. 5.84.

16PF "FACTOR O" SCORES (ALL GROUPS COMBINED)

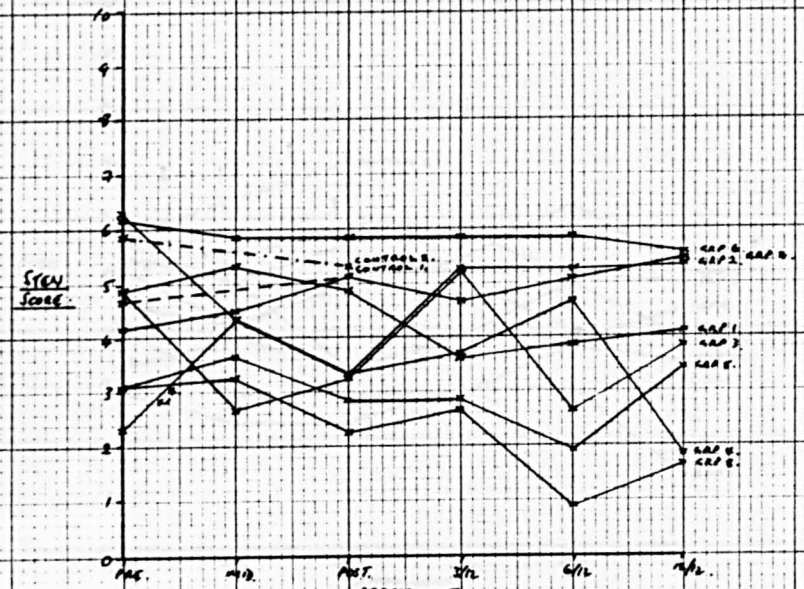


FIG. 5.85.

INDIVIDUAL GROUPS' 16PF 'FACTOR Q_1' SCORES.

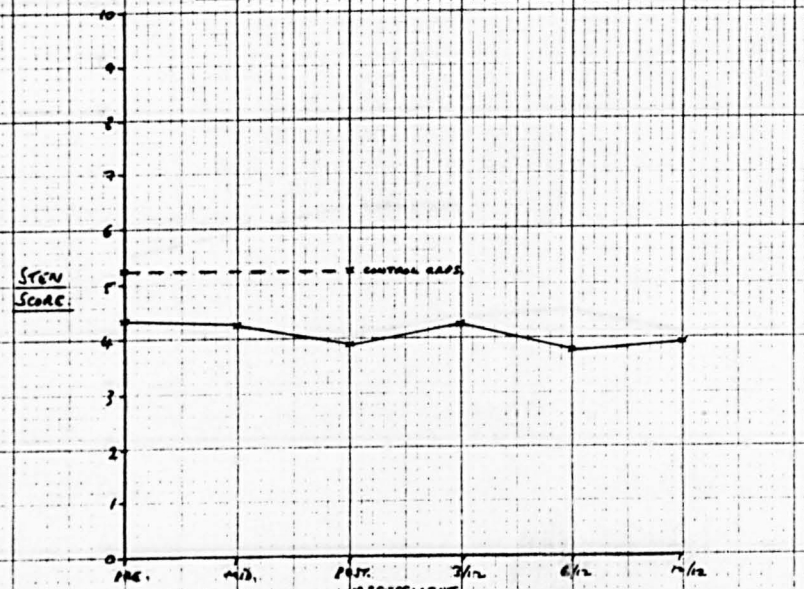


FIG. 5.86.

16PF "FACTOR Q_1" SCORES (ALL GROUPS COMBINED)

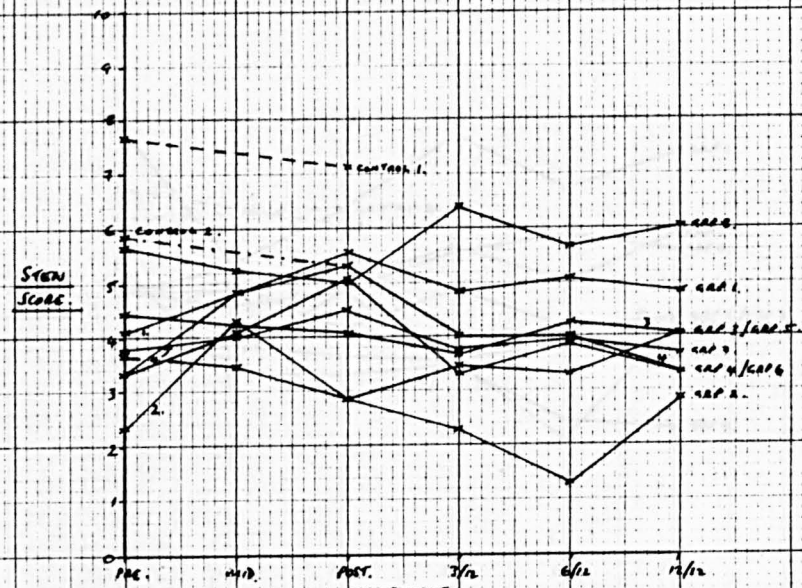


FIG. 5.87.

INDIVIDUAL GROUPS' 16PF "FACTOR Q_2" SCORES.

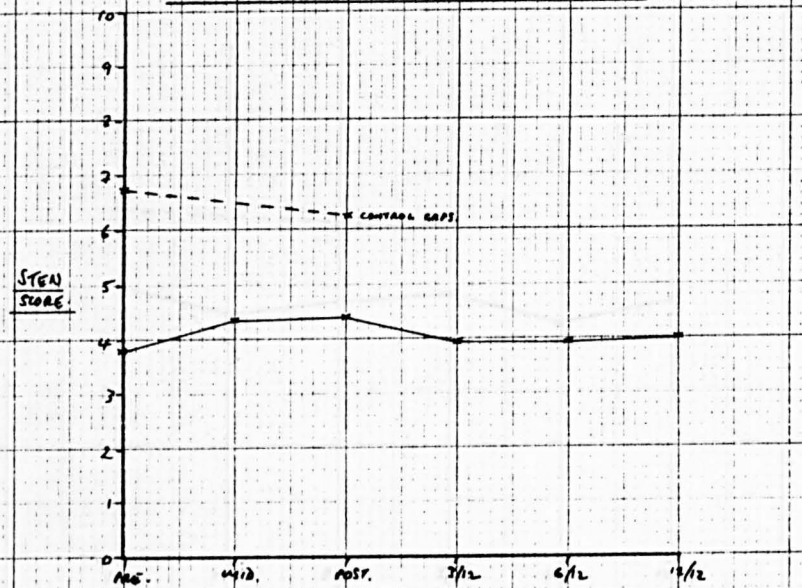


FIG. 5.88.

16PF "FACTOR Q_2" SCORES (ALL GROUPS COMBINED)

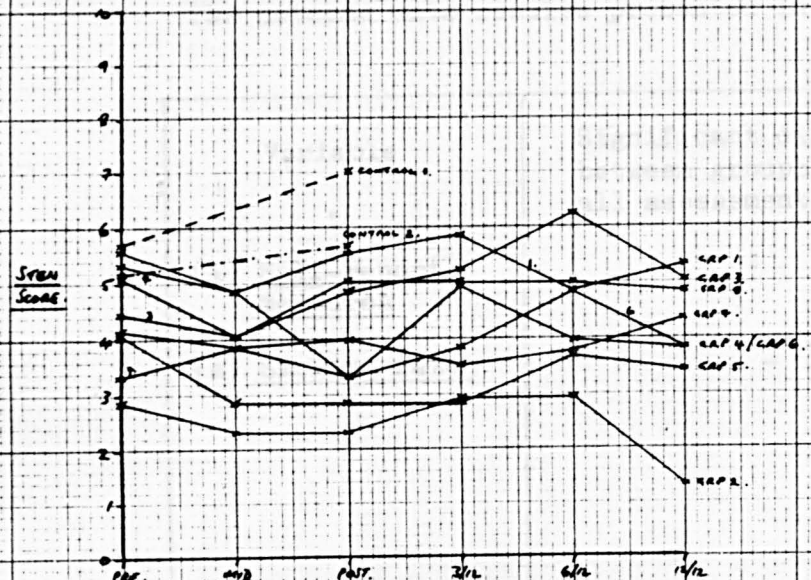


FIG. 5.89.

INDIVIDUAL GROUPS' 16PF "FACTOR Q_3" SCORES.

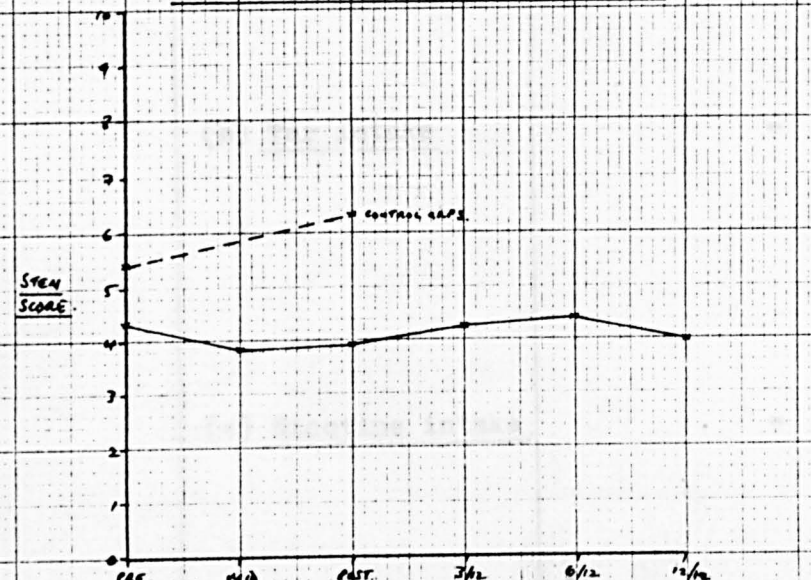


FIG. 5.90.

16PF "FACTOR Q_3" SCORES (ALL GROUPS COMBINED).

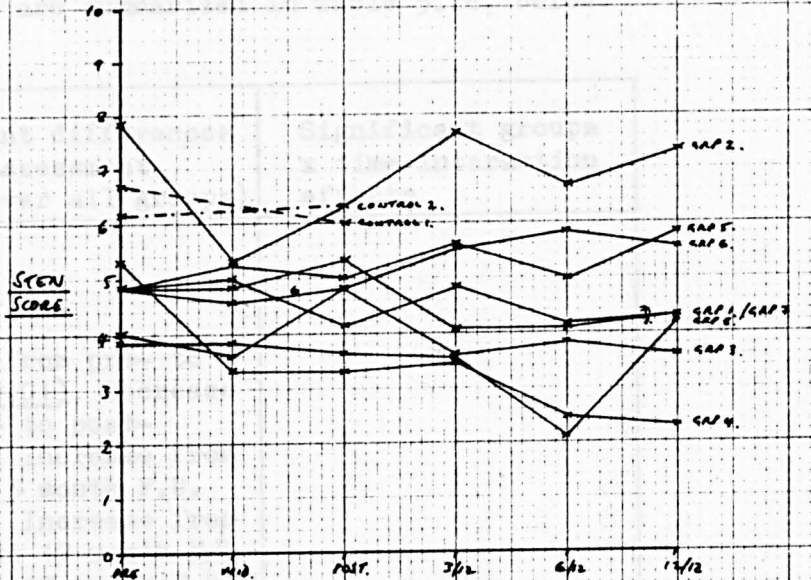


FIG. 5.91.

INDIVIDUAL GROUPS' 16PF "FACTOR Q_4" SCORES.

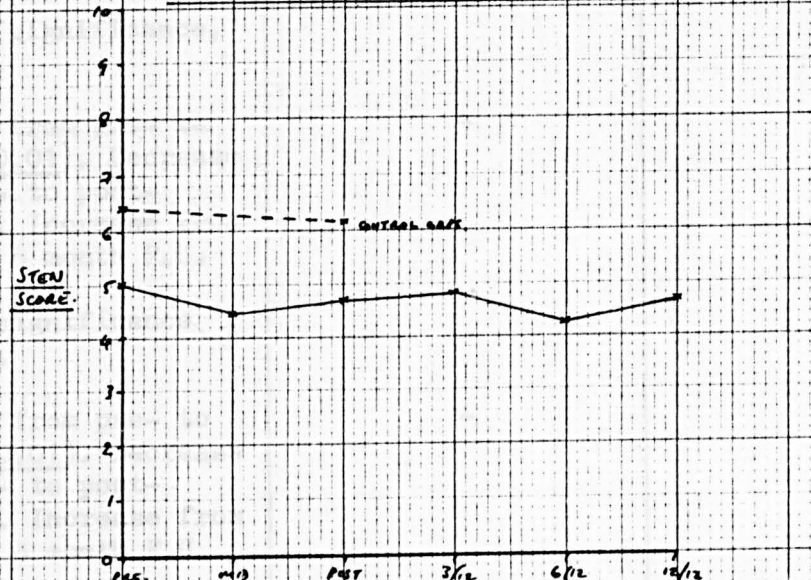


FIG. 5.92.

16PF "FACTOR Q_4" SCORES (ALL GROUPS COMBINED)

TABLE 5.28 OUTCOME DATA - SUMMARY OF RESULTS.

The outcome data results presented in the preceding section are summarized in Table 5.24, below.

Variable	Significant differences between groups (over all assessment points)	Significant differences between assessment points (over all groups)	Significant groups x time interaction effects
(i) Self-report measures			
(a) Smoking rate	-	Decrease from pre- to mid- ($p<0.01$). Decrease from mid- to post- ($p<0.01$). Increase from post- to 3 month F.U. ($p<0.01$). Increase from 3 month to 12 month F.U. ($p<0.01$). 12 month F.U. lower than pre-treatment ($p<0.01$) (Overall significance, $p<0.001$).	-
(b) Tar intake	-	Decrease from pre- to mid- ($p<0.01$). Decrease from mid- to post- ($p<0.01$). Increase from post- to 6 month F.U. ($p<0.01$) Overall significance $p<0.001$).	-
(c) Nicotine intake	-	Decrease from pre- to mid- ($p<0.01$). Decrease from mid- to post- ($p<0.01$). Increase from post- to 3 month F.U. ($p<0.01$). Overall significance $p<0.001$).	-

Table 5.24 Outcome data - summary of results.

Variable	Significant differences between groups (over all assessment points)	Significant differences between assessment points (over all groups)	Significant groups x time interaction effects
(d) Overall Benefit Rating	-	All assessment points significantly higher than pre-treatment ($p<0.01$). Increase from pre- to mid- ($p<0.01$) Decrease from post- to 3 month F.U. ($p<0.01$). Overall significance $p<0.001$).	-
(e) Anxiety ratings	(Group 6 higher than Groups 3 & 5 at pre-treatment. ($p<0.05$))	Increase from mid- to post- ($p<0.01$). Decrease from post- to 3 month F.U. ($p<0.01$). Overall significance $p<0.001$).	-
(f) Craving intensity	-	Increase from mid- to post- ($p<0.05$). Decrease from post- to 3 month F.U. ($p<0.05$). (Overall significance $p<0.05$).	-
(g) "Internal" vs "External" smoking	-	-	-

Table 5.24 (contin.)

Outcome data - summary of results

Variable	Significant differences between groups (over all assessment points)	Significant differences between assessment points (over all groups)	Significant groups x time interaction effects
(ii) Physiological measures			
(a) SCN^-	-	Decrease from pre- to mid- ($p<0.001$). (Non-significant increase by 12 month F.U.	-
(b) Gross body weight	-	Increase from pre- to 3 month F.U. ($p<0.05$). Increase from post- to 6 month F.U. ($p<0.05$). (12 month F.U. still significantly higher than pre-treatment ($p<0.001$) despite drop from 6 month to 12 month F.U. (N.S.)). (Overall significance $p<0.001$)	-
(c) Lung Function			
(i) FEV_1	-	-	-
(ii) FVC	-	-	-

Table 5.24 (contin.)

Outcome data - summary of results

Variable	Significant differences between groups (over all assessment points)	Significant differences between assessment points (over all groups)	Significant groups x time interaction effects
(iii) FEV/FVC	-	-	-
(iv) CO Transfer	-	-	-
(iii) Personality Measures			
(a) EPQ			
Psychoticism	-	-	-
Extraversion	-	-	-
Neuroticism	-	-	-
Lie Score	-	-	-
(b) SCL-90			
Somatic anxiety	-	-	-
Obsessive Compulsiveness	-	-	-
Interpersonal sensitivity	-	-	-
Depression	-	-	-

Table 5.24 (contin.)

Outcome data - summary of results

Variable	Significant differences between groups (over all assessment points)	Significant differences between assessment points (over all groups)	Significant groups x time interaction effects
Anxiety	-	12 month F.U. lower than pre-, mid- and post- ($p<0.01$).	-
Hostility	-	12 month F.U. lower than pre-, mid- and post- ($p<0.025$).	-
Phobic anxiety	-	-	-
Paranoid ideation	-	-	-
Psychoticism	-	Decrease from pre- to mid- ($p<0.05$). 12 month F.U. lower than pre- and post- ($p<0.05$). Overall significance $p<0.025$).	-
GSI	-	-	-
PSDL	-	-	-
PST	-	-	-
(c) 16PF			
Factor A	-	-	-
Factor B	-	-	-

Table 5.24 (contin.)

Outcome data - summary of results

Variable	Significant differences between groups (over all assessment points)	Significant differences between assessment points (over all groups)	Significant groups x time interaction effects
Factor C	(Group 3 higher than all other groups at pre-treatment ($p<0.05$)	-	-
Factor E	-	-	-
Factor F	-	-	-
Factor G	-	-	-
Factor H	-	-	-
Factor I	-	-	Group 7 decreases from pre- to mid- ($p<0.05$). Group 2 decreases from mid- to post- ($p<0.05$). Group 4 decreases from mid- to 12 month F.U. ($p<0.05$). Overall significance $p<0.05$).
Factor L	Group 3 lower than Groups 1,5 and 7 ($p<0.05$). Group 4 lower than Groups 1,2,5,7 & 8 ($p<0.05$) Group 6 lower than Group 1 ($p<0.05$). (Overall significance $p<0.025$).	-	-

Table 5.24 (contin.)

Outcome data - summary of results

Variable	Significant differences between groups (over all assessment points)	Significant differences between assessment points (over all groups)	Significant groups x time interaction effects
Factor M	-	-	-
Factor N	-	-	-
Factor O	-	-	-
Factor Q_1	Group 8 lower than Groups 2, 6 and 7 ($p<0.05$). Group 5 lower than Group 6 ($p<0.05$). Overall significance ($p<0.05$).	-	-
Factor Q_2	-	-	-
Factor Q_3	-	-	-
Factor Q_4	-	-	-

Table 5.24 (contin.)

Outcome data - summary of results

e) Control Group Outcome Measures

An analysis of variance with one repeated measure (ANOVA RI) (Meyers and Grossen, 1974) was used to identify any changes which took place within the two control groups over time (assessment took place at the "pre-treatment" and "post-treatment" equivalent points), on the various measures used.

The results of this analysis are presented in Tables 5.25 (i), (ii) and (iii), below.

Additionally, control group changes are represented graphically, where relevant, in Figures 5.1 to 5.92, relating to the treatment groups' outcome measures.

Variable	Comparison of means from "pre-" to "post-" assessment (F(1,5))	
	CONTROL GRP.1 ("Heavy")	CONTROL GRP.2 ("Light") (F(1,5))
Smoking rate (mean no.cigs/day)	2,76 (N.S.)	0.36 (N.S.)
Tar Intake (mean mg./day)	2.31 (N.S.)	1.80 (N.S.)
Nicotine intake (mean mg./day)	2.49 (N.S.)	0.02 (N.S.)
Anxiety rating (mean, 0-5 scale)	2.77 (N.S.)	1.77 (N.S.)
Craving Intensity (mean, 0-5 scale)	0.62 (N.S.)	1.49 (N.S.)
"Internal" smoking (% tot. cigs.smoked)	1.62 (N.S.)	2.13 (N.S.)

Table 5.25 (i)

Control group changes over time - self-report data

It can be seen from Table 5.25 (i) that no significant changes took place, on any of the self-report outcome measures, within either control group, from "pre-" to "post-" treatment equivalent points.

Variable	Comparison of means from "pre-" to "post-" assessment (F(1,5))	
	CONTROL GRP.1 ("Heavy")	CONTROL GRP.2 ("Light")
SCN^- (μmol/litre)	0.11 (N.S.)	0.69 (N.S.)
Gross body wt. (kg.)	3.83 (N.S.)	0.04 (N.S.)
Lung Function		
(i) FEV_1	2.17 (N.S.)	26.60 ($p<0.01$)
(ii) FVC	1.15 (N.S.)	2.20 (N.S.)
(iii) FEV/FVC	49.00 ($p<0.001$)	8.74 ($p<0.05$)
(iv) TF	0.93 (N.S.)	0.19 (N.S.)

Table 5.25 (ii)

Control group changes over time - physiological data

It can be seen from Table 5.25 (ii) that, although no significant changes took place, within either control group, over time, on the physiological measures of serum thiocyanate level or gross body weight, certain significant changes did occur with respect to lung-function measures.

Control group 1, the "heavy" smoking group, showed a highly significant increase in FEV/FVC ratio ($p<0.001$) from the first to the second assessment.

Control group 2, the "light" smoking group, demonstrated a significant ($p<0.01$) decrease in FEV, from the first to the second assessment and also a significant decrease ($p<0.05$) in FEV/FVC ratio.

These significant changes are illustrated in Figures 5.25, 5.21 and 5.25 respectively (pp. 188, 183, 198).

Variable	Comparison of means from "pre-" to "post-" assessment (F(1,51)	
	CONTROL GRP.1 ("Heavy")	CONTROL GRP.2 ("Light")
EPQ		
Psychoticism	1.00 (N.S.)	0.63 (N.S.)
Extraversion	0.12 (N.S.)	0.06 (N.S.)
Neuroticism	1.98 (N.S.)	0.00 (N.S.)
Lie Score	0.02 (N.S.)	0.79 (N.S.)
SCL-90		
Somatic anxiety	0.67 (N.S.)	0.09 (N.S.)
Obs.compuls.	3.29 (N.S.)	1.27 (N.S.)
Interpers. Sensitiv.	2.80 (N.S.)	1.68 (N.S.)
Depression	2.77 (N.S.)	1.36 (N.S.)
Anxiety	3.80 (N.S.)	0.01 (N.S.)
Hostility	2.76 (N.S.)	1.41 (N.S.)
Phobic anxiety	0.00 (N.S.)	2.95 (N.S.)
Paranoid ideation	3.84 (N.S.)	4.00 (N.S.)
Psychoticism	1.07 (N.S.)	3.11 (N.S.)
Gen.Sympt. Index	11.25 ($p<0.025$)	0.21 (N.S.)
Pos. Sympt. Dis. Level	0.83 (N.S.)	0.02 (N.S.)
Pos. Sympt. total	15.97 ($p<0.025$)	0.04 (N.S.)
16PF		
Factor A	1.00 (N.S.)	0.63 (N.S.)
Factor B	0.29 (N.S.)	12.27 ($p<0.025$)
Factor C	0.65 (N.S.)	0.25 (N.S.)
Factor E	0.29 (N.S.)	0.00 (N.S.)
Factor F	0;63 (N.S.)	1.00 (N.S.)
Factor G	0.07 (N.S.)	0.17 (N.S.)
Factor H	2.50 (N.S.)	1.82 (N.S.)
Factor I	1.36 (N.S.)	0.65 (N.S.)
Factor L	2.50 (N.S.)	0.17 (N.S.)
Factor M	0.19 (N.S.)	1.92 (N.S.)
Factor N	12.27 ($p<0.025$)	4.00 (N.S.)
Factor O	5.00 (N.S.)	1.43 (N.S.)
Factor Q_1	2.14 (N.S.)	0.65 (N.S.)
Factor Q_2	1.36 (N.S.)	2.50 (N.S.)
Factor Q_3	10.00 ($p<0.05$)	2.14 (N.S.)
Factor Q_4	1.43 (N.S.)	0.09 (N.S.)

Table 5.25 (iii)

Control group changes over time - personality data

It can be seen from Table 5.25 (iii) that no significant changes occurred, within either control group, over time, on any of the EPQ personality factors.

However, certain changes did occur on the SCL-90. Control group 1, (the "heavy" smoking group) showed significant decreases on the measures of "General Symptomatic Index" and "Positive Symptom Total", between the two assessments ($p < 0.025$ in both cases). These changes are illustrated in Figures 5.55 and 5.59, respectively (p. 200 - 201).

On the 16PF, control group 1 showed a significant decrease between the first and second assessments on Factor N ("Forthrightness") ($p < 0.025$) and a significant increase on Factor Q_3 ("Self-control") ($p < 0.05$). Control group 2 showed a significant ($p < 0.025$) decrease on Factor B ("Intelligence"), between the two assessments. These changes are illustrated in Figures 5.81, 5.89 and 5.63, respectively (pp. 209, 211, 204).

f) "Use" and "Benefit" ratings for individual treatment techniques

(i) "Use"

Using the mean total subject ratings (0-5 scale) for the degree of use of each treatment technique, it was possible to rank-order these techniques according to their "usefulness". Table 5.26 shows the "use" weightings, thus derived, for each technique.

Rank Order	Technique	Degree of use index
1	Hierarchical reduction	19.15
2	Deprived response performance	16.38
3	Focussed relaxation	16.00
4	Coverant Control	14.85
5	Monetary Deprivation	14.50
6	Covert Sensitization	9.95
-	Contingency Contracting	Not applicable

Table 5.26

Degree of use of individual treatment techniques

A one-way analysis of variance yielded significant differences between the degrees of use of techniques ($F(5,42) = 4.22$ $p<0.01$). The Tukey (a) Test showed that both Hierarchical Reduction and Deprived Response Performance were used significantly more than was Covert Sensitization ($p<0.01$ and <0.05, respectively). (ANOVA Table 95; Appx.VI).

A series of analyses of variance with one repeated measure (ANOVA RI) was then performed to assess whether changes took place, over time, in the degree of use of each technique. These results are presented in Table 5.27, below.

Technique	Changes in degree of use, over time
Hierarchical reduction	$F(4,28) = 0.74$ (N.S.)
Deprived response performance	$F(4,28) = 1.05$ (N.S.)
Focussed relaxation	$F(4,12) = 2.05$ (N.S.)
Coverant control	$F(4,28) = 3.26$ ($p<0.05$)
Monetary deprivation	$F(4,28) = 2.03$ (N.S.)
Covert sensitization	$F(4,12) = 1.12$ (N.S.)

Table 5.27

Changes in the degree of use of each treatment technique, over time

It can be seen from Table 5.27 that only one technique was used, differentially, over time: the Tukey (a) Test showed that coverant control was used significantly more ($p<0.05$) over the fourth week of treatment than over the first week. (ANOVA Tables 96-101; Appx. VI).

(ii) "Benefit"

As with the "Use" ratings, the mean total subject ratings (0-5 scale) for the degree of benefit obtained from each treatment technique

were rank-ordered. The respective degrees of benefit obtained are thus evident from consulting Table 5.28, below.

Rank Order	Technique	Degree of benefit index
1	Hierarchical reduction	17.08
2	Focussed relaxation	15.95
3	Deprived response performance	15.20
4	Coverant Control	13.85
5	Monetary Deprivation	9.85
6	Covert Sensitization	9.70
-	Contingency contracting	Not applicable

Table 5.28

Degree of benefit obtained from individual treatment techniques

A one-way analysis of variance demonstrated significant differences between the degrees of benefit obtained from the use of each technique ($F(5,42) = 5.40$, $p<0.001$). The Tukey (a) Test showed that significantly more benefit was (seen as being) obtained from Hierarchical Reduction than from Covert Sensitization or Monetary Deprivation ($p<0.01$ in both cases) and further showed that more benefit was (seen as being) obtained from Focussed Relaxation than from Covert Sensitization or from Monetary Deprivation ($p<0.05$ in both cases). (ANOVA Table 102; Appx. VI).

A series of analyses of variance with one repeated measure (ANOVA RI) was then performed to assess whether changes occurred, in the degree of rated benefit obtained from each technique, over time. These results are presented in Table 5.29, below.

Technique	Changes in degree of benefit, over time
Hierarchical reduction	F (4,28) = 0.86 (N.S.)
Focussed relaxation	F (4,12) = 6.63 ($p<0.01$)
Deprived response performance	F (4,28) = 0.11 (N.S.)
Coverant control	F (4,28) = 4.63 ($p<0.01$)
Monetary deprivation	F (4,28) = 1.69 (N.S.)
Covert sensitization	F (4,12) = 0.35 (N.S.)

Table 5.29

Changes in the degree of benefit obtained from each treatment technique, over time.

It can be seen from Table 5.29 that, in the case of two treatment techniques, rated benefit changed over time. The Tukey (a) Test showed that, in the case of Focussed Relaxation, significantly more benefit was being obtained by the end of the fifth week of treatment than at the end of the first week ($p<0.01$) (the degrees of benefit at the first and fourth weeks differed at the $p<0.05$ level). In the case of Coverant Control, significantly more benefit was being obtained by the end of the fifth week than at the end of the first ($p<0.01$) (ANOVA Tables 103-108; Appx.VI).

(iii) Rated weekly benefit from treatment

Figure 5.93, below, illustrates the changes throughout the treatment programme in rated benefit obtained from treatment (over all groups).

An analysis of variance with one repeated measure (ANOVA RI) demonstrated a significant change in rated benefit over time ($F(4,28) = 4.54$ ($p< 0.01$). The Tukey (a) Test showed that the degree of benefit obtained from the treatment programme was significantly higher at the end of the fourth week than at the end of the first week ($p<0.05$) and significantly higher at the end of the fifth week than at the end of the first week ($p<0.01$). (ANOVA Table 109; Appx. VI).

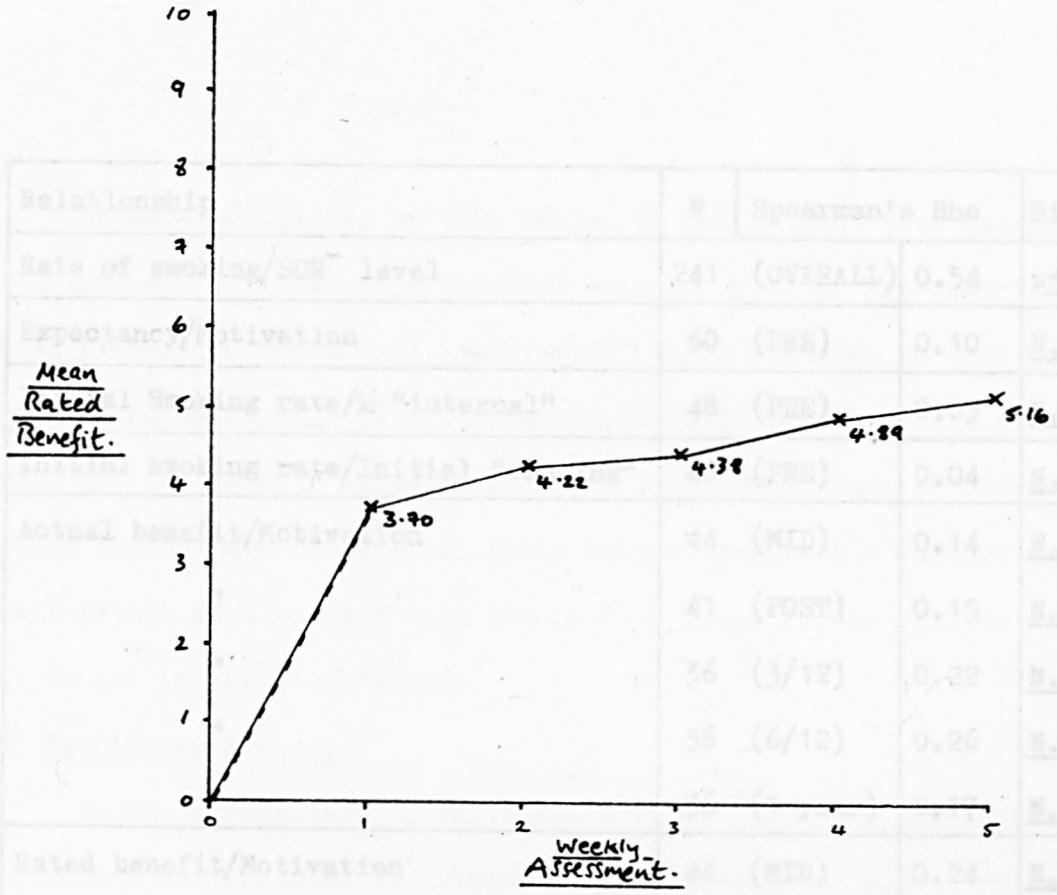


Fig. 5.93

Rated benefit from treatment, over the treatment period

g) Correlational Data

It was expected that certain of the variables examined in this study would correlate positively with one-another. Spearman's Rank Order Correlation Coefficients (Rho) were, therefore, computed, for a number of these variables. In all cases, as positive correlations were predicted, one-tailed tests were appropriate; where the value of Spearman's Rho was found to reach a level of significance of $p<0.05$, further analysis was performed to correct for ties (Siegel, 1956, p.206) and to correct for N's larger than 10 (op.cit. p.212). (In the case of non-significant values of Rho being obtained, further analysis was unnecessary, as the above corrections would have served only to further decrease the level of statistical significance).

The correlational data obtained is presented in Table 5.30, below. ("Actual benefit" is defined as the degree of movement towards target of the subject).

Relationship	N	Spearman's Rho	Significance
Rate of smoking/SCN^- level	241 (OVERALL)	0.54	p<0.001
Expectancy/Motivation	60 (PRE)	0.10	N.S.
Initial Smoking rate/% "internal"	48 (PRE)	0.05	N.S.
Initial smoking rate/Initial "craving"	48 (PRE)	0.04	N.S.
Actual benefit/Motivation	44 (MID)	0.14	N.S.
"	41 (POST)	0.15	N.S.
"	36 (3/12)	0.22	N.S.
"	36 (6/12)	0.26	N.S.
"	36 (1 year)	0.17	N.S.
Rated benefit/Motivation	44 (MID)	0.24	N.S.
"	41 (POST)	0.22	N.S.
"	32 (3/12)	0.14	N.S.
"	31 (6/12)	0.15	N.S.
"	33 (1 year)	0.13	N.S.
Actual benefit/Expectancy	44 (MID)	0.33	N.S.
"	41 (POST)	0.39	p<0.05
"	36 (3/12)	0.39	p<0.05
"	36 (6/12)	0.33	N.S.
"	36 (1 year)	0.28	N.S.
Rated benefit/Expectancy	44 (MID)	0.36	p<0.05
"	41 (POST)	0.26	N.S.
"	32 (3/12)	0.46	p<0.01
"	31 (6/12)	0.50	p<0.01
"	33 (1 year)	0.45	p<0.05
Rated benefit/Actual benefit	177 (OVERALL)	0.72	p<0.001
Actual benefit/% weight increase	176 (")	0.26	p<0.001
Actual benefit/Age	48 (")	0.10	N.S.

Table 5.30

Correlational Data

Commenting briefly on this table, it can be seen that significant positive correlations were obtained between level of expectancy and both self-rated and actual improvement (these measures in turn being significantly correlated), between improvement (decrease) in smoking rate and increase in gross body weight and between rate of smoking and serum thiocyanate blood level (this latter correlation validating the self-report smoking-rate data obtained). These issues will be addressed in detail in the Discussion.

h) Predictive Factors

By way of analysis of variance, all the variables examined at pre-treatment were related to outcome at each assessment point, in order to identify any pre-treatment characteristics which predicted a positive response to treatment. The method was as follows: a one-way analysis of variance was performed on the pre-treatment "scores" of the most "successful" subjects and on those of the least "successful" subjects, to see whether the scores of these two groups differed significantly; as complete data was available at one-year follow-up on 36 subjects, these two groups were each composed of 18 subjects. The results of this analysis are presented in Table 5.31, below.

It is evident from this table that certain pre-treatment characteristics predicted a positive treatment outcome. Noteworthy predictors were low SCL-90 scores on the measures of Somatic Anxiety, Obsessive Compulsiveness, Depression, Anxiety and Paranoid Ideation and on the Global Symptomatic Index and Positive Symptom Distress Level measures; a high score on 16PF factor Q_1 ("more experimenting/liberal") also predicted a positive outcome.

This issue will be addressed further in the Discussion.

VARIABLE	MID F(1,34)	Sig.	POST F(1,34)	Sig.	3/12 F(1,34)	Sig.	6/12 F(1,34)	Sig.	1 year F(1,34)	Sig.	COMMENTS
Smoking rate	0.04	NS	2.44	NS	1.73	NS	0.06	NS	0.65	NS	No sig. diffs.
Tar	2.53	NS	2.24	NS	0.72	NS	0.08	NS	0.10	NS	No sig. diffs.
Nicotine	0.01	NS	1.89	NS	0.75	NS	0.19	NS	0.35	NS	No sig. diffs.
Anxiety	0.00	NS	0.02	NS	0.73	NS	0.22	NS	1.10	NS	No sig. diffs.
Craving	0.11	NS	0.32	NS	0.78	NS	0.09	NS	0.78	NS	No sig. diffs.
"Internal/ External"	3.10	NS	6.91	$p<0.025$	2.56	NS	2.74	NS	1.12	NS	High S. do better at post only.
Expectancy	0.96	NS	0.94	NS	2.25	NS	0.24	NS	2.73	NS	No sig. diffs.
Motivation	0.00	NS	0.25	NS	0.01	NS	0.56	NS	0.06	NS	No sig. diffs.

Table 5.31 (i)

Predictive power of pre-treatment measures - self-report data

VARIABLE	MID F(1,34)	MID Sig.	POST F(1,34)	POST Sig.	3/12 F(1,34)	3/12 Sig.	6/12 F(1,34)	6/12 Sig.	1 year F(1,34)	1 year Sig.	COMMENTS
SCN^-	5.46	$p<0.05$	2.82	NS	1.43	NS	0.96	NS	2.19	NS	High S. do better at mid-treatment only
Gross body weight	0.07	NS	2.33	NS	0.89	NS	0.04	NS	0.01	NS	No sig. diffs.
Lung function:											
(i) FEV_1	0.35	NS	0.63	NS	0.32	NS	0.03	NS	1.35	NS	No sig. diffs.
(ii) FVC	0.26	NS	0.17	NS	0.01	NS	0.52	NS	1.46	NS	No sig. diffs
(iii) FEV/FVC	0.02	NS	0.37	NS	0.00	NS	0.75	NS	0.01	NS	No sig. diffs
(iv) TF	0.57	NS	0.02	NS	0.15	NS	0.16	NS	0.04	NS	No sig. diffs.
Age	0.01	NS	0.06	NS	0.00	NS	0.60	NS	1.63	NS	No sig. diffs.
Sex	0.90	NS	0.50	NS	0.00	NS	0.02	NS	0.00	NS	No sig. diffs.

Table 5.31 (ii)

Predictive power of pre-treatment measures - physiological data

VARIABLE	MID F(1,34)	MID Sig.	POST F(1,34)	POST Sig.	3/12 F(1,34)	3/12 Sig.	6/12 F(1,34)	6/12 Sig.	1 year F(1,34)	1 year Sig.	COMMENTS
EPQ											
Psychoticism	3.76	NS	1.05	NS	0.49	NS	0.20	NS	0.46	NS	No sig. diffs.
Extraversion	0.13	NS	0.01	NS	0.69	NS	1.73	NS	0.96	NS	No sig. diffs.
Neuroticism	0.48	NS	0.02	NS	0.22	NS	0.42	NS	0.08	NS	No sig. diffs.
Lie score	0.05	NS	0.03	NS	0.39	NS	0.75	NS	0.53	NS	No sig. diffs.
SCL-90											
Somatic anxiety	15.63	$p<0.001$	15.37	$p<0.001$	15.28	$p<0.001$	5.54	$p<0.025$	5.65	$p<0.025$	Low S. do better at all assessment points
Obsessive Compulsiveness	6.48	$p<0.025$	16.89	$p<0.001$	12.02	$p<0.01$	10.25	$p<0.01$	7.42	$p<0.025$	Low S. do better at all assessment points
Interpersonal Sensitivity	2.19	NS	4.09	NS	1.39	NS	0.75	NS	0.43	NS	No sig. diffs.
Depression	3.76	NS	4.56	$p<0.05$	5.16	$p<0.05$	3.26	NS	2.36	NS	Low S. do better at post & 3/12. No diffs.elsewhere
Anxiety	11.10	$p<0.01$	7.27	$p<0.025$	4.22	$p<0.05$	5.15	$p<0.05$	3.55	NS	Low S. do better at all points, except 1 yr.(no diffs)
Hostility	1.58	NS	3.89	NS	2.89	NS	6.01	$p<0.01$	6.35	$p<0.01$	Low S. do better only at 6/12 and 1 yr.
Phobic anxiety	0.89	NS	0.43	NS	0.10	NS	0.09	NS	0.29	NS	No sig. diffs.
Paranoid ideation	11.35	$p<0.01$	15.23	$p<0.001$	12.02	$p<0.01$	16.29	$p<0.001$	7.05	$p<0.025$	Low S. do better at all assessment points
Psychoticism	1.69	NS	2.70	NS	0.75	NS	0.04	NS	0.12	NS	No sig. diffs.
Global symptomatic index	7.46	$p<0.01$	15.37	$p<0.001$	14.21	$p<0.001$	6.50	$p<0.025$	2.12	NS	Low S. do better at all assessment points apart from 1 yr.
Positive symptom distress level	3.37	NS	7.36	$p<0.025$	6.81	$p<0.025$	1.27	NS	2.49	NS	Low S. do better at post & 3/12. No diffs. elsewhere
Positive symptom total	3.26	NS	1.65	NS	0.55	NS	0.41	NS	0.25	NS	No sig. diffs.

Table 5.31 (iii)

Predictive power of pre-treatment measures - personality data

VARIABLE	MID F(1,34)	Sig.	POST F(1,34)	Sig.	3/12 F(1,34)	Sig.	6/12 F(1,34)	Sig.	1 year F(1,34)	Sig.	COMMENTS
16PF											
Factor A	3.70	NS	0.44	NS	0.48	NS	0.25	NS	0.32	NS	No sig. diffs.
Factor B	0.30	NS	0.90	NS	1.69	NS	2.34	NS	5.14	$p<0.05$	High S. do better at 1 yr. only. (Intelligence)
Factor C	2.31	NS	7.67	$p<0.01$	2.71	NS	1.56	NS	2.95	NS	High S. do better at post only. (Emotional stability)
Factor E	1.03	NS	1.29	NS	4.11	NS	0.60	NS	0.07	NS	No sig. diffs.
Factor F	0.06	NS	0.46	NS	0.87	NS	0.44	NS	0.54	NS	No sig. diffs.
Factor G	2.10	NS	1.08	NS	1.05	NS	2.44	NS	4.21	$p<0.05$	Low S. do better at 1 yr. only. (Conscientiousness)
Factor H	0.36	NS	0.24	NS	0.10	NS	0.45	NS	0.79	NS	No sig. diffs.
Factor I	0.44	NS	0.34	NS	0.21	NS	0.00	NS	0.20	NS	No sig. diffs.
Factor L	0.04	NS	1.19	NS	0.43	NS	0.00	NS	0.04	NS	No sig. diffs.
Factor M	4.49	$p<0.05$	3.69	NS	1.47	NS	0.03	NS	0.12	NS	High S. do better at mid-treatment only. (More imaginative)
Factor N	1.12	NS	3.94	NS	1.83	NS	2.09	NS	2.51	NS	No sig. diffs.
Factor O	0.78	NS	0.90	NS	0.21	NS	0.11	NS	0.00	NS	No sig. diffs.
Factor Q_1	1.29	NS	3.19	NS	4.18	$p<0.05$	6.14	$p<0.025$	4.54	$p<0.05$	High S. do better at 3,6 & 1 yr. (More experimenting/liberal)
Factor Q_2	0.09	NS	0.11	NS	0.03	NS	0.35	NS	0.64	NS	No sig. diffs.
Factor Q_3	2.81	NS	2.09	NS	2.42	NS	2.49	NS	0.87	NS	No sig. diffs.
Factor Q_4	1.15	NS	2.86	NS	3.11	NS	5.29	$p<0.05$	3.63	NS	Low S. do better at 6 month only (More relaxed).

Table 5.31 (iii)(continued).

Predictive power of pre-treatment measures - personality data

i) Subject Attrition

Table 5.32, below, provides details of the numbers of subjects available for data collection, at each assessment point, for each of the variables measured. As stated earlier, the method of analysis used for outcome data allowed missing data to be taken into account (with the exception of certain physiological measures for Group 2).

Measure	Pre.	Mid.	Post.	3-month	6-month	1 year
	N (%)	N (%)	N (%)	N (%)	N (%)	N (%)
Self-report						
Smoking rate Tar intake Nicotine intake	48 (100)	44 (91.67)	41 (85.42)	37 (77.08)	37 (77.08)	37 (77.08)
*Anxiety rating *Craving intensity *% "Internal"	48 (100)	41 (85.42)	24 (50.00)	20 (41.67)	24 (50.00)	27 (56.25)
Overall Benefit Rating	48 (100)	44 (91.67)	43 (89.58)	35 (72.92)	35 (72.92)	35 (72.92)
Physiological						
SCN^-	48 (100)	43 (89.58)	42 (87.50)	32 (66.67)	32 (66.67)	31 (64.58)
Gross Body Weight	48 (100)	44 (91.67)	43 (89.58)	38 (79.17)	38 (79.17)	38 (79.17)
Lung Function	48 (100)	40 (83.33)	40 (83.33)	31 (64.58)	29 (60.42)	29 (60.42)
Personality						
EPQ SCL-90 16PF	48 (100)	44 (91.67)	43 (89.58)	33 (68.75)	33 (68.75)	33 (68.75)

Table 5.32

Subject Attrition Rates

(* Figures for these measures are artificially depressed, as abstinent subjects are included here, the measures not being applicable to non-smokers).

The mean percentage of the initial number of subjects available for assessment, at each assessment point, across all measures, are illustrated in Figure 5.94, below. Again, these figures are slightly depressed due to the non-applicability of certain measures to abstinent subjects, who are therefore included in this data.

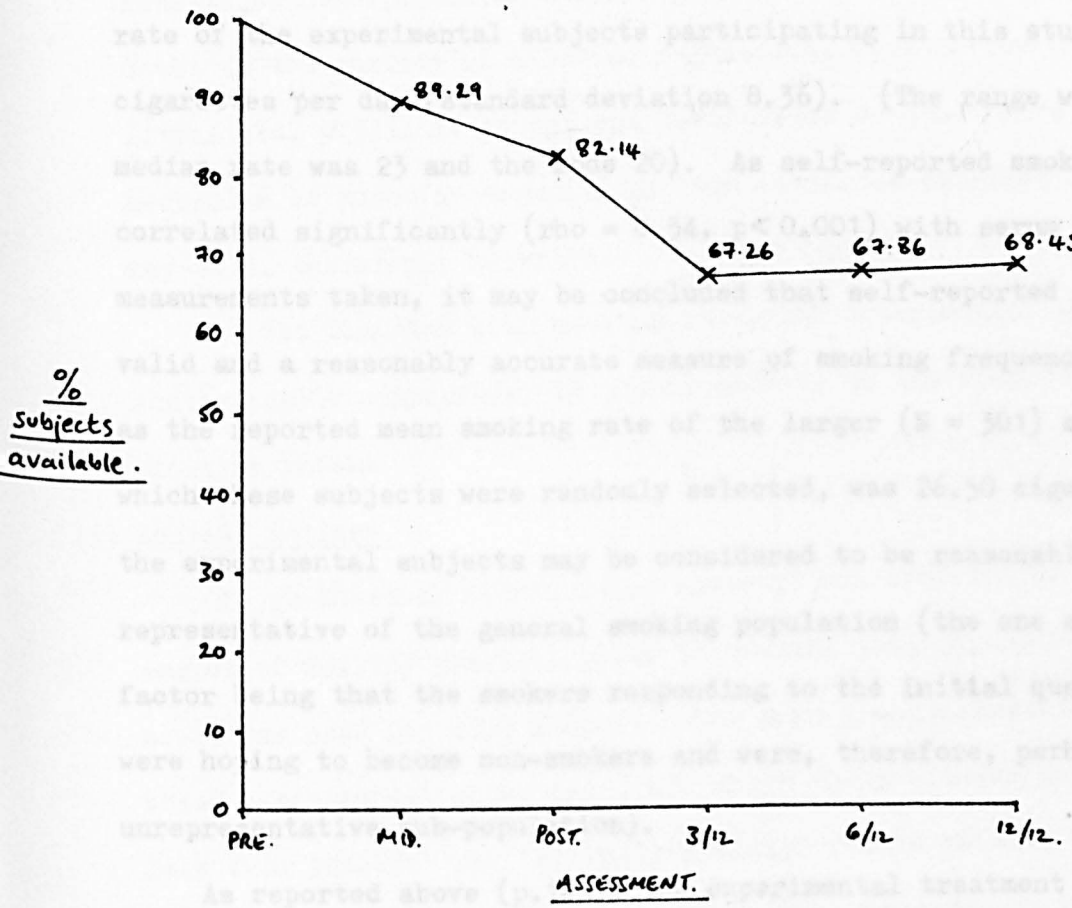


Fig. 5.94

Subject Attrition Rates

6. DISCUSSION

a) Interpretation and Discussion of the Results

(i) Pretreatment analyses

a) Self-report measures

(i) Smoking rate and tar and nicotine intake estimates

As reported in the Results section (p.140), the mean initial smoking rate of the experimental subjects participating in this study was 23.60 cigarettes per day (standard deviation 8.36). (The range was 8-45, the median rate was 23 and the mode 20). As self-reported smoking rate correlated significantly (rho = 0.54, $p < 0.001$) with serum thiocyonate measurements taken, it may be concluded that self-reported rates were valid and a reasonably accurate measure of smoking frequency. Moreover, as the reported mean smoking rate of the larger (N = 301) sample, from which these subjects were randomly selected, was 26.50 cigarettes a day, the experimental subjects may be considered to be reasonably representative of the general smoking population (the one extenuating factor being that the smokers responding to the initial questionnaire were hoping to become non-smokers and were, therefore, perhaps, an unrepresentative sub-population).

As reported above (p.145), the experimental treatment groups did not significantly differ from one-another on the measure of initial smoking-rate, at pre-treatment.

It has already been mentioned (p.141) that the estimated daily tar and nicotine intake levels of subjects were calculated directly from self-reported smoking rate, taking into account the brand of cigarettes smoked, and that H.D.U.K. tables were used for this purpose. Nicotine regulation or titration effects (and, by implication, "tar

regulation" effects) were not taken into account here, so it is possible that real nicotine and tar intake measures were underestimated (for those smokers who reduced their rate of smoking). However, bearing in mind the findings of Schachter (1977), that a 77% reduction in nicotine level led to only a 17-25% increase in cigarette consumption, and the results of other research studies previously mentioned which cast some doubt on the reality of nicotine regulation, (see Review of the Literature, p.33), it is not yet possible to be conclusive about the mechanism of nicotine-regulation; the estimates obtained in this study may well, therefore, be valid.

As was the case with smoking rate, the experimental groups were statistically comparable, at pre-treatment, on the measures of tar and nicotine intake.

(ii) Anxiety ratings

To re-iterate, "situational anxiety" was reported as being the degree of anxiety present, prior to smoking each cigarette. A 5 point scale was used for this purpose. The mean pre-treatment rating for the total experimental group was 1.63, which falls between the "totally free from anxiety" and "slightly anxious" points on this scale. The total experimental group can therefore be considered to be a relatively "anxiety-free" and "non-pathological" group, and comparable with the general (non-smoking) population.

As detailed in the Results section (p.147), however, experimental Group 6 obtained a significantly higher mean anxiety score, at pre-treatment, than did Groups 3 and 5. These latter Groups did not have especially low ratings, but the mean rating for Group 6 was relatively

high (2.48). This high score was attributable to the inordinately high anxiety levels of two of the six subjects in this group: one subject had a mean pre-treatment score of 3.01 and the other a score of 4.19. (The first subject was experiencing marital difficulties - for which behavioural counselling was offered, independently of her involvement in this project - and these difficulties were leading to a generally high level of anxiety; the second subject was clearly emotionally disturbed, was suffering from neurotic anxiety and dropped out of the treatment programme after initial assessment. This subject later received psychiatric treatment for his problems.)

It may be pointed out here that situational anxiety (ie. the level of anxiety experienced at the time of smoking)has been measured in very few previous studies in this field, despite it s therapeutic importance.

(iii) Craving Intensity Ratings

As with the Anxiety Ratings, degree of craving was recorded, using a 5-point scale, immediately before each cigarette was smoked. The mean rating (N = 48) of 2.39 reflected a "slight" to "moderate" degree of craving, according to the scale-point definitions. Craving may be considered, in turn, to reflect the degree of dependency (both physiologically and psychologically) on cigarettes and thus this experimental group were, taken as a whole, "slightly" to "moderately" dependent smokers, at the commencement of the treatment programme. There was, naturally, some individual variation in craving intensity ratings; (the range was 1.06-4.57, the median was 2.32 and the mode was 3.03).

The treatment groups, as mentioned previously (p.147), did not differ significantly at pre-treatment on the measure of craving intensity.

(iv) The "internal-external" smoking dimension

A relatively high proportion (72.85% - see Table 5.1(i), p.140) of the cigarettes smoked were classed, at pre-treatment, as "internal", by the total subject group. (The standard deviation of 22.86 does show, however, that considerable variability existed among individual smokers).

As previously discussed (pp.102-103), "internal" and "external" smokers supposedly smoke for different reasons: "internal" smokers in response to physiological cues and "external" smokers in response to environmental cues (Russell et al, 1974; Schachter, 1977). Moreover, this dimension has been related to rate of smoking (eg. Herman, 1974), "heavy" smokers being more "internal" than "light" smokers. Were this the case, the subjects participating in this study would have been expected to be relatively heavy smokers; although "heavy" is an arbitrary term, the mean rate of 23.6 cigarettes a day is not considered as being particularly heavy and so the relationship between "internal-external" smoking and smoking rate is perhaps not so clear as has been supposed. Moreover, the Spearman's Rho correlation between initial (pre-treatment) smoking rate and % "internal" cigarettes smoked was found to be 0.05, which is non-significant (see p.227) and this casts further doubt on the traditionally accepted nature of the "internal-external" dimension.

It was found that the experimental groups in this study did not differ significantly from one-another, at pre-treatment, on the measure of "internal vs. external" smoking (p.148).

(v) Expectancy

Expectancy, as stated previously, was measured on a 10 point scale and the mean expectancy rating of the subjects participating in this experiment was 8.69 (p.140). This can be interpreted as a relatively high mean score (and the standard deviation of 1.75 as signifying a narrow spread of expectancy ratings). The subjects, therefore, would have been expected, according to the results of at least one previous study in this field, (Blittner, Goldberg & Merbaum, 1978) to respond well to the treatment programme (perhaps regardless of the form of this programme) and to succeed in achieving their goals. This was, in fact, the case: this issue is addressed in more detail under the heading "Correlational Analyses", below (p.271).

The subject groups were equivalent on the measure of expectancy (see p.148).

(vi) Motivation

It has been mentioned earlier that the "motivation" questionnaire used in this study was intended to provide only a rough measure, not having been subjected to reliability of validity tests of any kind (see p.119). However, it was felt that, in view of the clear role of motivation as a "non-specific" factor in smoking control (McFall and Hammen, 1971; Raw, 1976), some measure was desirable, to assess whether motivation played some role in determining subjects' success in modifying their smoking behaviour, in this study.

As in the case of the measure of "expectancy", the issue of motivation will receive further attention in the context of the discussion of the correlational analyses which were performed (see p.271).

Suffice it to say, at this point, however, that no relationship was found between motivation, as measured, and response to treatment, but that the mean motivation score of 117.92 (see Table 5.1(i), p.140) of subjects before treatment commenced was equivalent to an approximate mean score of 6.3 on a 10 point scale and was therefore not especially high. Interestingly, "motivation" (again, as measured in this study) and "expectancy" did not correlate significantly (Rho = 0.10).

The "motivation" scores of the experimental groups did not differ significantly at pre-treatment and no differential group effects were therefore expected (see p.149).

b) Physiological measures

(i) Serum thiocyanate (SCN^-)

The mean SCN^- level obtained for subjects at pre-treatment was 152.94μ mol/litre (S.D. 37.98). This finding was commented upon briefly in the Results Section (p.142) and it is germane to reiterate, at this point, that this figure is comparable with the mean levels recorded in previous studies for equivalent mean rates of smoking. Butts et al (1974), for example, reported a mean of approximately 152 μ mol/litre for 20-a-day smokers and Vogt et al (1977) a mean of 175 μ mol/litre for 20+-a-day smokers (N not reported). The mean non-smoker SCN^- levels in these two studies were 44 and 65μmol/litre respectively: in the present study, the mean non-smoker SCN^- level was 49.33μ mol/litre.

Furthermore, it has been stated elsewhere (p.227) that the Spearman's Rho correlation between rate of smoking and SCN^- blood level was found to be a highly significant ($p<0.001$) 0.54 and this figure, too, compares well with the findings of previous studies -

a Spearman's Rho's of 0.46 (Butts et al, 1974) and 0.48 (Vogt et al, 1977).

The findings of the present study confirm the validity of using SCN^- as a molecular physiological measure in smoking research (it discriminates well between smokers and non-smokers) and also suggest that self-reported smoking rates are, perhaps, more accurate and reliable than has been supposed by some authors (eg. McFall, 1970); this is in line with the assertions of Frederiksen, Epstein and Kosevsky (1975) and Epstein and Collins (1977) who favoured self-report measures, reporting reliabilities of 0.85+.

The treatment groups did not differ significantly from one another, on the measure of SCN^-, at pre-treatment. (see p.149).

(ii) Gross Body Weight

The mean gross body weight of the subject group, at pre-treatment, was 67.51kg. (S.D. 10.23kg.). Sexes being equally represented, the expected weight for this age-group (mean age 42.35 years) was 64.4kg (Palmer, 1980). This finding was rather unexpected as, according to some studies (eg. Karvonen et al 1959; Bjelke, 1971; Khosia and Lowe, 1971; Goldbourt and Medalie, 1977) smokers, in general, weigh less than do non-smokers; the experimental subjects in this study were actually, on the average, over three kilograms above the expected weight.

A number of factors may account for this discrepency, (the dietary characteristics of the local population being one such factor) but here is not the place to investigate these: what was of more interest and importance in this study was the change over time in body weight, as a function of changes in smoking rate, and this will be discussed at a later point.

There were no differences between groups, at pre-treatment, on the measure of gross body-weight (see p.150).

(iii) Lung Function Measures

As stated earlier (p.142) the obtained mean FEV_1 (forced expiratory volume) of 89.25 for the total subject group at pre-treatment compares poorly with the expected (healthy) mean of 100. (The standard deviation of 15.08, moreover, does not reflect an especially wide degree of variation in individual FEV_1 readings); the FVC (forced vital capacity) figure mean of 100.02 (S.D. 15.70) was, however, "normal". This is an interesting and clinically significant finding and suggests that, although the lung capacity of the subjects was not diminished as a result of their smoking, their ability to expel air from the lungs was detrimentally influenced by smoking.

Low FEV_1 readings are characteristic of individuals with obstructive airways disease (eg. bronchitis, bronchial asthma or emphysema) (Bass, 1974) and the findings of this study suggests that the subject population, as a whole, were experiencing obstructive problems as a result of bronchial congestion due to smoking.

The more meaningful measure of FEV/FVC ratio was also found to be lower than the healthy value (which, for non-smokers, should be in the vicinity of 82.0 (Cotes, 1975)). For the subjects in this study, this value, at pre-treatment was 71.69 (S.D.9.88); a ratio of 70.00 or below is considered to be pathological (Bass, 1974), so the obtained mean ratio was very close to being indicative of pathological respiratory functioning.

Finally, the mean carbon monoxide (CO) transfer factor of 87.58 (S.D. 14.55) for the subject group also supports dysfunctional respiration - the healthy, expected value being 100.

The above findings serve to confirm the already well-established view that smoking is harmful to health (Doll and Peto, 1976; R.C.P. 1962, 1971, 1977; U.S.D.H.E.W. 1979).

On none of the lung-function measures did the treatment groups differ significantly, at pre-treatment assessment. (see pp.150-152).

c) Personality Measures

(i) Eysenck Personality Questionnaire (EPQ)

At pre-treatment, the mean scores of the total subject group on the three EPQ dimensions of Psychoticism, Extraversion and Neuroticism (and also on the fourth dimension of "Lie Score") all fell within the normal range for the age group studied, (ie. less than one standard deviation from the overall population mean - Eysenck and Eysenck (1975)). No significant inter-group differences existed at pre-treatment, (see Table 5.16(i), p.153). With respect to the parameters measured by the EPQ, therefore, the subject group was a "normal" population.

(ii) Symptom Check List (SCL) 90

On none of the factors examined by the SCL-90 was the mean T score for the total subject group, at pre-treatment, over 50, this being the "normal/pathological" cut-off point (Derogatis, Lipman and Covi, 1973). However, the mean score on the Somatic Anxiety factor did approach this cut-off point, being 46.35 (S.D. 14.58) and this suggests that the subjects participating in this study experienced a relatively high incidence of somatic symptoms of anxiety. It was

thought to be likely that the frequency and intensity of such symptoms would increase, at least initially, as smoking rate decreased, but it is difficult to explain the high mean score at baseline; it may be that the subjects, being, by virtue of their request for help with their smoking problem, a rather self-selected population, were more than normally anxious and concerned about their smoking behaviour and were therefore predisposed to experiencing somatic symptoms of anxiety (although their mean score on the factor of general anxiety was less "pathological" - 38.71 (S.D. 12.67)). Further investigation is perhaps warranted here.

A further point of interest was the (low) mean score of 22.81 on the "Phobic Anxiety" factor, with the remarkably high standard deviation of 25.08. The explanation for this exceptionally wide variation is that, whereas the majority (54.2%) of subjects participating in the study obtained a "Phobic Anxiety" score of zero, seven (14.6%) of the subjects (one male and six females, evenly dispersed among the eight treatment groups) obtained scores of over 50 (ie. scores which were "pathological"). All of these subjects experienced varying degrees of agoraphobia. As there is no reason to suppose that agoraphobic individuals are more likely to want to modify their smoking behaviour, nor that smokers wanting to stop are more likely to be agoraphobic, the data obtained perhaps serve to show how prevalent a disorder is agoraphobia. This is not the place to discuss this issue, which has been covered in depth elsewhere (Thorpe and Burns, 1983).

At pre-treatment, no significant differences were found between the eight treatment groups' mean scores on any of the SCL-90 factors (see Table 5.16(ii), p.154).

(iii) 16PF

As stated earlier (p.144) all the 16PF mean sten scores, for the total subject group, were within normal limits (the "normal" range being approximately 4.7 to 6.3), with the exception of the mean score of 7.52 on Factor B (Intelligence) (see Table 5.1 (iii) pp.143-144). This finding is not surprising, as it is reasonable to suppose that more intelligent individuals who smoke are more likely to make efforts to stop (or to obtain help with the problem) than are less intelligent smokers, who may be less aware of the harmful effects of smoking.

The relatively low standard deviation (1.64) obtained on Factor B, moreover, shows that the subject group was uniformly of above average intelligence (in fact, only seven (14.6%) of the subjects scored below the average (5.5) point on the scale, at pre-treatment).

It was mentioned in the Results Section (p.156) that, on one of the 16PF factors, a significant difference emerged between groups, an analysis, at pre-treatment. Group 3 had significantly higher mean Factor C (Emotional Stability) scores than all the other treatment groups, This was primarily due to three (50%) of the six subjects comprising Group 3, obtaining a C score of 9, at pre-treatment.

d) Age and Sex

It has already been noted that the treatment groups were statistically equivalent on the measure of age (the overall mean being 42.35 years (S.D. 10.35 years) (p.141 and p.156) and that the sexes were equally represented (p.144).

(ii) Outcome Analyses

a) Self-report measures

(i) Smoking Rate

As indicated earlier (pp.163 and 164), all experimental groups responded equally well to treatment: there were no significant inter-group differences in the degree of movement towards target (whether this be abstinence or reduction) and there were no groups x time interaction effects.

For all groups, treatment resulted in significant changes in smoking rate over time. Rate reduced significantly from baseline to mid-treatment and, again significantly, from mid-treatment to post-treatment. A significant increase in smoking rate then took place, for all groups, between post-treatment assessment and 3-month follow up and slower (non-significant) increases between 3 and 6 month and 6 and 12 month follow-up. However, the increase from 3 to 12 month follow-up was significant. It is important to state that, at one-year follow-up, the mean smoking rate for all groups was still significantly lower than at pre-treatment assessment.

Thus, regardless of whether subjects were "heavy" or "light" smokers (ie. baseline smoking rate), regardless of whether they aimed to abstain from smoking or to reduce their smoking rate to 25% of baseline and notwithstanding the differing designs of the treatment packages ("Self-control" versus "multicomponent"), subjects, overall, succeeded in obtaining long-term benefit from treatment.

(There was, naturally, some individual variation in response to treatment - some subjects were more "successful" than others; however, analysis of variance does, by definition, take into account this individual variation: wide variation yields lower (and therefore less significant) F ratios. The highly significant results obtained statistically on this outcome measure are, therefore, of equal clinical significance.).

It would appear, then, that several conclusions can be drawn from this study, with regard to factors influencing response to treatment. First, baseline smoking rate does not seem to be an especially important independent variable. Although it may still be the case that "heavy" and "light" smokers smoke for different reasons (Russell et al, 1974; Schachter, 1977), it seems that the type of treatment approach used in the present study would seem to be of equal effectiveness in modifying the behaviour of both "types" of smoker. The point made by Leventhal and Cleary (1980) that many light smokers have great difficulty in stopping smoking (as well as do heavy smokers) is therefore supported in an indirect way by this study: the finding here was that heavy smokers are as successful in modifying their smoking behaviour as are light smokers and differentiation with regard to baseline rate is, therefore, not necessary when considering likely response to intervention.

Secondly, the treatments used in this study enabled smokers to gain both short and long term benefit, whether the goal was total abstinence from smoking or whether the aim was to smoke at 25% of baseline-rate. (It must be remembered here that "benefit" is

defined as a reduction in smoking rate, a "movement towards target", and does not necessarily imply an achievement of that target. Abstinence and pre-planned reduction rates will be discussed below, where it will be seen that differences were found between abstaining and reducing subjects, with regard to their "success").

To re-iterate, all groups, regardless of the type of target, reduced their smoking rate significantly through treatment and were still smoking at a rate significantly lower than baseline, at one-year follow-up. It has been twice stated previously that "there is not yet sufficient evidence available to preclude treatment efforts which aim to establish a lower level of smoking in subjects, as an alternative to total abstinence" (pp 35 and 93); this assertion appears to be vindicated. (Vindication is also given to the conclusions drawn by researchers such as Martin et al (1981) that compensatory smoking behaviours - for example nicotine regulation - do not necessarily occur as a result of reduced rate; this is evidenced by the significantly reduced serum-thiocyanate levels found, at long-term follow-up, in the "reducing" subjects in this study. This issue is discussed below).

In the above respect, therefore, the results of this study are congruent with the findings of Schinke , Blythe and Doueck (1978), Elliott and Denney (1978), Foxx and Brown (1979) and Foxx and Axelroth (1983) in demonstrating that a reduced rate of smoking can be achieved and maintained in the long-term. Russell (1974) stated that "With (the) ..... more feasible goal (of controlled smoking),

success is not only possible but probable" (p.256) and this study supports this belief.

A third conclusion which may be drawn from this study, (and perhaps the most important one), is that the "self-control" package was of equal effectiveness as a method of treatment as was the more elaborate "multicomponent" package, this latter approach combining self-control with "therapist administered" techniques. An alternative conclusion which may be drawn here is that the additional techniques used in the multicomponent package - focussed relaxation training and covert sensitization - were of no inherent value in modifying smoking behaviour. This conclusion is rather untenable, however, as, in addition to there existing some empirical evidence that these techniques are infact effective (Ravensborg, 1976; Cautela, 1970; Sipich, Russell and Tobias, 1974), the subjects participating in this study rated at least one of the methods - focussed relaxation training - as being relatively highly beneficial as a treatment technique (see p.224).

It does not seem, then, that the direct treatment of subjects (at least with the techniques used in this study) is an essential requisite of therapy, but that a self control package of the type utilized is sufficient to elicit a favourable response to intervention. (This, of course, has implications for the cost-effectiveness of intervention with the problem of smoking; this matter will be discussed later.). It was suspected, from a review of the pertinent literature (see pp.87-91, above, and especially the studies by Chapman et al (1971), Morrow et al (1973) and Tongas,

Patterson and Goodkind (1976)) that an additive effect would be obtained by combining self-control and therapist administered procedures, subjects receiving both types of treatment being more successful in modifying their smoking behaviour. This was not the case and the statement by Franks and Wilson (1975) that "more is not always better" certainly gains support.

(ii) Abstinence rates and reduction "success" rates"

As indicated above (p.246), although all groups, regardless of their target, significantly reduced their smoking rates (and maintained this reduction) and although there were no inter-group differences in this respect, an analysis of whether "abstainers" were more successful in reaching their target than "reducers" was performed (See Results Section, pp.163-167) and this showed that, in absolute terms, the former were more successful than the latter.

To re-iterate, the abstinence rates for those subjects (N = 24) aiming to stop smoking completely, at each of the six assessment points, were: at pre-treatment, 0%; at mid-treatment, 4.17%; at post-treatment, 45.83%; at 3-month follow-up, 50.00%; and at both 6 and 12 month follow-up, 25.00%. The "success" rates for those subjects (N =24) aiming to smoke at 25% (or less) of their baseline rate were: at pre-treatment, 0%; at mid-treatment 16.67%; at post-treatment, 29.17%; at 3-month follow-up, 8.33%; at 6-month follow-up 16.67%; and at 12 month follow-up, 8.33%.

As stated on pp.165-166, the abstinence rates at all assessment points, with the exception of mid-treatment, were significantly higher than at baseline; the "success" rates for

reducers were, at no point, significantly higher than at pre-treatment. (Within these two major groups, no inter-sub-group differences were apparent for "abstainers" or for "reducers"). These data are not in any way remarkable but are nevertheless of considerable interest. First, with regard to the "abstaining" subjects, the abstinence rate at mid-treatment was disappointing (although, in fact, the nature of the treatment programme was such that total abstinence was not expected, or required, until the end of the treatment phase). Similarly, it was hoped that, at the end of treatment, the majority of subjects would be abstinent; in fact, just under half (N = 11) were. However, abstinence rate actually increased between the end of treatment and the first (3-month) follow-up assessment; this result was not expected (although it was hoped that abstinence would be maintained to a considerable degree, after treatment,) but shows, perhaps, that the treatment techniques comprising the packages used were of lasting benefit (or, alternatively, that the motivation of abstinent subjects to remain abstinent was high). This figure of 50% abstinent at 3-month follow-up compares well with the majority of previous studies in this field (see Lichtenstein and Danaher, 1976; Raw, 1978, p.175) but less well when measured against earlier studies employing self-control or multi-element packages (see pp.83-91, above). The same is true for the longer-term follow-up rates: an abstinence rate of 25% at one-year follow-up is superior to many previously obtained rates (Raw, op.cit), but inferior to the rates achieved, through the use of

self-control packages, by, for example, Brengelmann (1973), Flaxman (1974) and Delahunt and Curran (1976).

There is a clear need for treatment "booster" sessions after the three-month follow-up point, in order to maintain abstinence. This issue will be discussed in detail below.

With regard to the "reducing" subjects, a different, and less positive, picture emerges from the data obtained. Again, at no assessment point was there a significant statistical difference between reported smoking rate and smoking rate at pre-treatment, when 25% of baseline rate was used as the criterion of "success". The closest approach to significance came at post-treatment assessment, when 29.17% of subjects were smoking at the prescribed rate, or less (the value of p here was approximately 0.10) - $F(5,15) = 2.53$).

It is evident, then, that, where adherence to a pre-determined target rate is the goal, a target of 0% of baseline smoking rate is more easily attained (and maintained) than a "controlled smoking" target of 25%. This failure of the present experiment to effect controlled smoking will be discussed, in more detail, at a later point.

In conclusion, it must be remembered, however, that, when smoking rate, per se, is taken as the primary dependent variable, all groups in this study, regardless of target-rate, significantly reduced their rate and maintained this reduction at long-term follow-up.

To facilitate the comparison with previous studies of the results obtained here, below is reproduced a diagram (Fig. 6.1) originally appearing in Hunt and Bespalec (1974) and used later by Lichtenstein and Danaher (1976) to illustrate the typical rate-reductions and abstinence rates obtained from smoking treatment programmes. The data were collected from 89 such studies.

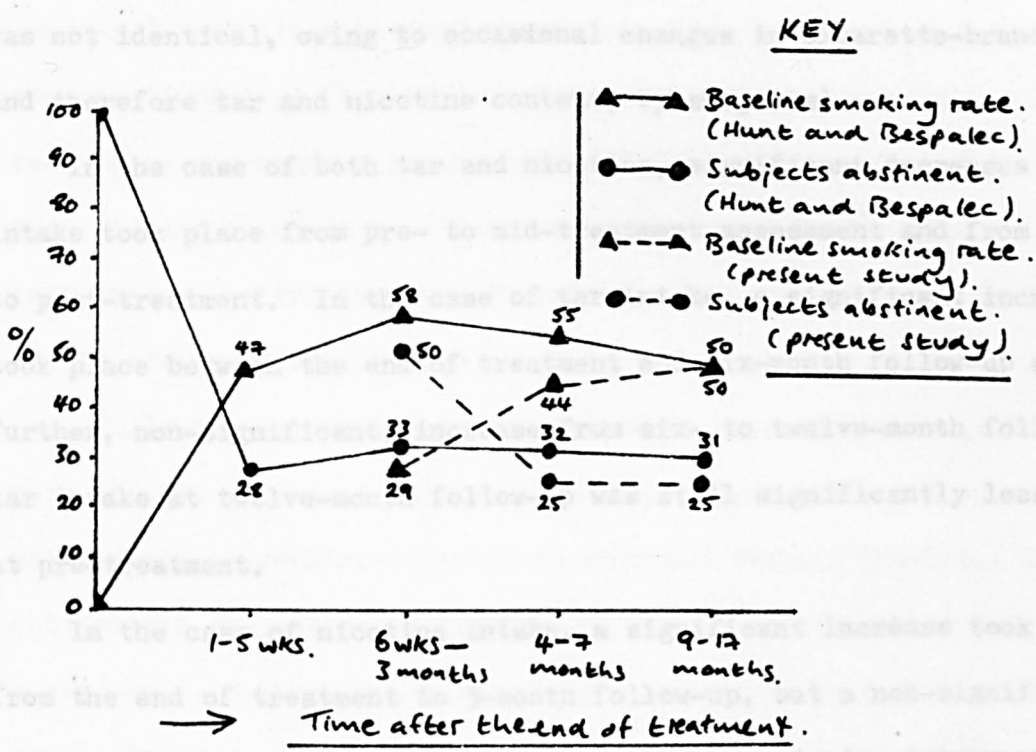


Fig.6.1
Comparison of outcome results - the present and previous studies.

It is important to point out that Lichtenstein and Danaher criticize Hunt and Bespalec's summary in that "the curves are based only on subjects who achieved abstinence at termination, thus yielding an overly optimistic picture of total program effectiveness" and referred to McFall and Hammen's (1971) conclusion that, typically, at 4-6 month follow-up, "percentage of baseline smoking averaged about 75% of baseline, and the percentage of abstinent subjects

ranged from 9% to 17% with a mean of 13%" (p.90)

(iii) Tar and Nicotine Intake

The question of the validity of the tar and nicotine measures obtained in this study has already been addressed (see p.236).

Measures taken at the post-baseline assessment points, in the case of both estimated tar and nicotine intake, followed a similar pattern to smoking rate (ie. movement towards target). (This pattern was not identical, owing to occasional changes in cigarette-brand, and therefore tar and nicotine content, by subjects).

In the case of both tar and nicotine, significant decreases in intake took place from pre- to mid-treatment assessment and from mid- to post-treatment. In the case of tar intake, a significant increase took place between the end of treatment and six-month follow-up and a further, non-significant, increase from six- to twelve-month follow-up; tar intake at twelve-month follow-up was still significantly less than at pre-treatment.

In the case of nicotine intake, a significant increase took place from the end of treatment to 3-month follow-up, but a non-significant increase from three- to twelve-month follow-up. Again, intake at one-year follow-up was significantly less than at pre-treatment.

Neither with tar nor nicotine outcome measures were any inter-group differences found.

It can be argued from this data, taking into account the harmfulness of tar and nicotine as substances contained in cigarette smoke (R.C.P. 1962, 1971, 1977; USDHEW, 1979), that long-term benefit, with regard to physical health, was obtained by all groups participating in this study.

(iv) Overall Benefit Ratings

The mean Overall Benefit Ratings reported by each treatment group reflected the objective measures of benefit taken. A significant degree of benefit was apparent by mid-treatment assessment; in fact, the amount of benefit obtained during the first half of the treatment phase was represented by the greatest change in degree of benefit obtained, between two adjacent assessment points (see Fig. 5.10, p.171). Subjectively rated benefit subsequently increased from mid-treatment to post-treatment assessment (although non-significantly), implying that the treatment programmes used were seen as having maximum impact over the first three weeks of treatment. This finding is again a reflection of the changes over time in smoking rate, where maximum decrease in rate was evident over the first half of the treatment phase.

Following asymptote at post-treatment assessment, the total group mean benefit rating at this point being approximately 7.7 on a 10-point scale, a decline occurred in reported Overall Benefit. This decline was statistically significant between post-treatment assessment and three-month follow-up (as was the case with smoking-rate), but non-significant from three-month to one-year follow-up.

No inter-group differences in overall rated benefit were apparent, at any assessment point, and no groups x time interaction effects occurred.

The data obtained here seem to demonstrate that a very close relationship exists between actual benefit obtained from treatment (ie. reduction is smoking rate) and subjectively perceived benefit. (The Spearman's Rho correlation between these two measures, over all subjects and assessments, was found to be 0.72, this being significant

at the $p \leq 0.001$ level). It may, therefore, be concluded that, from a clinical viewpoint, subjects who respond well to treatment do actually feel to have done well and may thus be described as having benefited psychologically, as well as physiologically, from treatment.

(v) Anxiety Ratings

Some interesting, although expected, findings took place when the anxiety ratings over time for all treatment groups were examined. (Again, no inter-group differences were evident at any assessment point, other than pre-treatment - see pp.172 and 236).

Mean anxiety ratings increased slightly (non-significantly) during the first half of treatment, but significantly during the second half. Then, in line with the pervasive "relapse", or movement towards baseline of smoking-rate, anxiety ratings again decreased significantly; a further (non-significant) decrease took place between the three- and six-month follow-up assessments.

As predicted, therefore, (see p.118), an "inverted-U" shaped change took place, over time, in subjectively experienced anxiety, measured prior to smoking each cigarette, and this "inverted-U" peaked at post-treatment assessment, when smoking-rate was at its lowest.

The clinical implications of this phenomenon are clear, as is the bearing of these findings on future efforts to modify smoking behaviour, and this issue will be discussed under a later heading.

(vi) Craving Intensity

As was the case with "Anxiety Ratings" (see above), it was expected than an "inverted-U" shape would be apparent, when mean "craving intensity" ratings were examined over time (either because smoking rate would follow the same pattern or because abstinence or

reduced rate would eventually become more tolerable). This expectation was borne out: craving intensity increased (non-significantly) from pre- to mid-treatment assessment, significantly from mid- to post-treatment assessment and then decreased significantly until three-month follow-up. No further significant changes then took place. The explanation for this pattern is most likely related to a combination of the factors mentioned above - a degree of "relapse", together with adjustment to lower levels of bodily nicotine.

No significant differences existed between mean craving intensity ratings of the eight treatment groups, at any assessment point.

The implications of these findings will be discussed, along with those relating to reported anxiety, at a later stage.

(vii) "Internal/External" Smoking

Contrary to expectations (see p. 118), no changes took place, over time, in the proportion of cigarettes which were rated as being smoked in response to "internal" or "external" stimuli. Further, no inter-group differences were apparent, at any assessment point, on this measure.

It was thought that a reduction in smoking-rate would lead to an increase in the number of cigarettes judged as "internal", partly because several treatment techniques were specifically designed to weaken the power of external, environmental stimuli in eliciting smoking behaviour, but also because, given the importance of the capacity of smoking to maintain the required level of nicotine in the body (Russell, 1971; Raw, 1978), it was

expected that nicotine level depletion would intensify internal "signals" to smoke.

Clearly, this was not the case. This rather negative finding, along with the equally negative finding that "internal" smoking and smoking rate were uncorrelated (see p.227) seems to cast doubt on the utility and validity of "internal/external" smoking as a measure in smoking research. Herman's (1974) finding that "light" smokers were equally responsive to change in nicotine levels as were "heavy" smokers does seem to gain some indirect support from this study.

b) Physiological Measures

(i) Serum Thiocyanate (SCN^-)

As mentioned earlier (p.175), some difficulties arose with the analysis of repeated SCN^- measures for the treatment groups in this study, owing to the failure of several subjects in one group (Group 2) to provide blood-samples when required. This finding is, in itself, however, of interest. Despite the fact that, before treatment, all subjects undertook to provide blood-samples (see Contract, Appendix III), this intrusive procedure was evidently sufficiently aversive to interfere, to some degree, with data collection. The clear implication then, is that assessment procedures in smoking research (an area of research where subject attrition rates are, in any case, notoriously high)(Merbaum and Rosenbaum, 1980) should be as non-aversive and painless as possible: although SCN^- levels measured by blood-sampling is a highly reliable physiological measure (Butts et al, 1974), perhaps saliva SCN^- sampling is a more suitable

method for this type of research (Prue, Martin and Hume, 1980).

As previously described, two separate analyses were, then, performed on SCN^- outcome data: the data obtained from all groups were subjected to analysis of variance at six-month follow-up and the data from seven of the eight groups were analysed at twelve-month follow-up. In neither case was there any significant between groups difference, at any assessment point; this was, it will be remembered, the case with smoking-rate reduction. Furthermore, as in the case of smoking-rate, all post-baseline SCN^- assessments indicated significantly lower blood SCN^- levels than were evident at pre-treatment; this finding applied to analysis at both six- and twelve-month follow-up. The increase in mean SCN^- levels from six- to twelve-month follow-up was non-significant.

This is an important finding. Serum-thiocyanate measurement was considered to be the primary physiological measure used in this study, and positive changes on this measure are seen as most accurately representing a true improvement from a health viewpoint (Prue, Krapfl and Martin, 1981). The fact that, even at one-year follow-up, mean SCN^- levels were significantly lower than at pre-treatment, suggests that the subjects treated gained considerable long-term health benefit from their involvement in this experiment.

(ii) Gross Body-Weight

The analysis of body-weight changes over time produced some interesting findings, these being of especial relevance clinically and having considerable implications for further work in this field.

As stated earlier, (p.120), reduced smoking has been identified with increased weight (eg. Blitzer, Rimm & Giefer, 1977) and so this "molar" measure was not only considered to be useful as a secondary physiological check on self-report (SCN^- being the primary such measure) but was also seen as worthy of further investigation in its own right.

It appears from the findings that weight change can, indeed, be used as a reliable check on self-report (the Spearman's Rho correlation between change in smoking-rate and change in weight was 0.26, which was, with an "N" of 176, significant at the $p < 0.001$ level). As described earlier (pp.179-181), although there were no reliable differences in mean weight between adjacent assessment points, a significant increase in weight did occur between pre-treatment assessment and three-month follow-up and the difference between weight at post-treatment assessment and six-month follow-up was also significant. A (non-significant) decrease in weight took place between six- and twelve-month follow-up. There were no significant inter-group differences, and no groups x time interaction effects. In this study, then, reduction in smoking rate was clearly associated with an increase in body weight. The slowness of the increase in weight (significant differences appearing only over relatively extended periods of measurement) is, perhaps, to be expected with a "molar" measure, as opposed to "molecular" measures such as serum-thiocyanate level.

As weight increase is frequently undesirable, from a subject's point of view (and from a more objective, health point of view) it would seem to be wise to offer dietary advice during and following treatment programmes which are designed to reduce or eliminate

smoking behaviour. The subjective aversiveness of weight increase is a factor which should be taken into consideration in smoking behaviour modification programmes, if the "drop-out" rate is to be minimized and if abstinence or reduction in smoking-rate are to be permanently achieved.

This issue will be addressed further in a later section of this discussion.

(iii) Lung Function

The same difficulties as were experienced in the case of SCN^- measurement applied to obtaining lung-function data from the subjects in this experiment: analysis was possible of all groups' data up to three-month follow-up, but the failure of several subjects in one group (again, Group 2) to attend for lung-function assessment at six- and twelve-month follow-up resulted in the data from only seven of the eight treatment groups being analysed at the last two assessment points.

The reason for this failure to attend, by some subjects, is likely to be the same as for SCN^- assessment, namely, the aversiveness of the procedure; it was reported by some individuals that measurement of the CO transfer factor was a rather unpleasant procedure (involving the use of a nose-clip). This result serves to substantiate the above statement that assessment procedures in smoking research need to be as "painless" as possible, if subject attendance rates for assessment are to be maximized.

The lung-function results obtained are rather anomalous; this is possibly a function of the relatively small number of subjects available for long-term follow-up assessment. No inter-group

differences were found, nor were any significant changes over time, on any of the measures taken (FEV, FVC, FEV/FVC ratio and CO transfer factor).

Taking all groups together, non-significant improvements in lung-function were apparent on the measures of FEV, (from pre-treatment to mid-treatment, mid-treatment to post-treatment and post-treatment to three-month follow-up, FVC (from pre-treatment to mid-treatment and, again, from mid-treatment to post-treatment) and CO transfer factor (as for FEV). The improvement in FEV, from pre-treatment to three-month follow-up assessment did, in fact, approach significance ($F(3,69) = 1.30$; $p = 0.28$ at three-month follow-up and $F(5,110) = 1.80$; $p = \underline{0.12}$ at twelve-month follow-up, looking at the pre- to three-month changes in the context of the subsequent data obtained). If more subjects had been available for six- and twelve-month follow-up assessment, perhaps this latter result would have reached statistical significance; certainly, the trend was in that direction.

There was, therefore, some tentative evidence that benefit was obtained by subjects, as a result of modifying their smoking behaviour, with respect to respiratory functioning, and, in particular, with regard to the measure of FEV_1. To reiterate, this is a measure of the individuals' ability to expire air quickly from the lungs, and a low FEV_1 reading is indicative of airways obstruction.

It is clear that, if, as seems likely, respiratory changes were, in fact, occurring over time, in the total treatment group, these

were slow. It would seem probable that follow-up at longer duration after the end of treatment (assuming, of course, that improvement maintained with respect to smoking-rate) may reveal statistically significant differences in lung-function, when measures at this time are compared with pre-treatment measures. This conclusion does seem to be rather inconsistent with previous findings, however. For example, Paxton and Scott (1981) noted a significant improvement in FEV_1 in subjects who had abstained from smoking for only two months. It may be, therefore, that the low "N" sampled at 12-month follow-up was responsible for the lack of significance found in the present study.

c) Personality Measures

The rationale for administering personality questionnaires, as part of the assessment procedure in this study, was described earlier. To reiterate, "the relationship between smoking and personality factors is unclear and confusing. This is .... (an) area where further investigation is needed" (pp.101-102) and, further, "(it was hoped that) .... certain personality characteristics would be found to predict a positive response to treatment (or otherwise) and .... that certain changes over time may be apparent in certain "personality" characteristics .... as a result of modified smoking behaviour". (pp.121-122).

The present section concerns the latter question - do certain personality characteristics (or characteristic patterns of behaviour) change, over time, as smoking behaviour changes? (The issue of predictive personality factors will be addressed below).

With regard to the Eysenck Personality Questionnaire (EPQ), on none of the four factors examined were any changes over time apparent, as smoking-rate changed; (nor were any inter-group differences or interaction effects present). (see p.194).

On the Symptom Check List (SCL)-90, although, again, there were no significant inter-group differences, some significant changes did occur, over time, along with a reduction in smoking rate. Most notably, group mean scores on the factors of both "Anxiety" and "Hostility" were found to be significantly lower at one-year follow-up than at pre-, mid- or post-treatment assessment, ($p = \underline{0.008}$ and $p = \underline{0.013}$, respectively).

Less remarkably, group mean scores on the factor of "psychoticism" were significantly lower at mid-treatment than at pre-treatment assessment ($p = \underline{0.043}$) and also significantly lower at one-year follow-up than at pre-treatment and post-treatment assessment ($p = 0.023$).

In the cases of Anxiety and Hostility, slight (non-significant) increases in scores took place between the beginning and the end of the six-week treatment programme and these increases served to accentuate the lower twelve-month follow-up scores. It appears, therefore, that reductions in smoking (or abstinence from smoking) and consequently reduced nicotine intake had the effect of raising subjects' overall anxiety levels (this being in contrast to "situational" anxiety levels, which also increased throughout the treatment period (see p.172)) and increasing feelings of hostility or aggressiveness. This finding is concordant with the recognition of a "withdrawal syndrome" which is characterized by certain

psychological phenomena, including restlessness and irritability (Larson and Silvette, 1971; Brecher, 1972) and clearly has implications for improving the nature and effectiveness of treatment programmes. This will be discussed later.

Where the changes in "Psychoticism" scores are concerned, interpretation is more difficult.

Detailed examination of individual subjects' scores on this factor shows that, in most treatment groups, at least one subject, who obtained a positive score on the factor of "Psychoticism" either at pre-treatment assessment or at post-treatment assessment, obtained a score of zero at either (or both) mid-treatment assessment or twelve-month follow-up. Unlike the majority of SCL-90 factors, "Psychoticism" is a very "sensitive" scale, in that a "moderate" score (of 2 on a 5-point scale) on, for example, three of the factor items, produced a T score of approximately 47 (which is close to the "pathological" cut-off point of 50). The reason for this is that psychotic symptomatology is very characteristic and of considerable clinical significance and, as the SCL-90 is a clinical assessment tool, it is seen as important to identify psychosis to facilitate intervention. It may be, then, that a high proportion of "false-positive" identifications is considered preferable to overlooking patients with psychotic disorders; indeed, none of the subjects in the present experiment who obtained a high "Psychoticism" score was, in fact, psychotic.

Why the high "Psychoticism" scores obtained by some subjects should have occurred at pre- and post-treatment assessment (and,

to a lesser degree, at three and six-month follow-up) is difficult to explain and this must perhaps be attributed to an artifact of the assessment method.

The third personality measure employed in this study was the 16 Personality Factor Questionnaire (16PF). Taking all groups together, no significant changes over time, on any of the 16PF factors, took place. There were, however, several significant inter-group differences, which are detailed in the Results Section (p.202). As the groups whose scores differed were not in any way distinguished, with respect to the design of the experiment (see Fig.3.2, p.112), these differences cannot be attributed to any of the independent (treatment, type of smoker or target) variables and are therefore of little significance. The differences obtained thus simply reflect the variability of within-group (individual) scores.

Of possible significance, however, is the fact that, on one of the factors measured - "Toughmindedness" - a significant groups x time interaction did emerge (scores decreasing, in all cases, over time, for three treatment groups) (see pp.202-203). Again, from the point of view of the experimental design, the "groups" element here is of no significance; but the changes over time for some groups perhaps suggest that a decrease in smoking-rate, and the concomitant increases in anxiety and hostility (see above), leads to certain individuals' becoming more "tender-minded, clinging and sensitive" (IPAT, 1970).

The conclusions drawn here, especially in relation to the significant changes which were found to occur on certain SCL-90 and 16PF factors, are tentative. Unfortunately the issue of personality and smoking is still "unclear and confusing" (p.121)

and it is evident that further, more specific, research is needed to clarify the findings obtained in this study.

(iii) "Use" and "Benefit" Ratings

a) "Degree of Use"

The utilization of "use" and "benefit" ratings by the subjects participating in this study allowed several analyses to be performed, on the data obtained.

First, rank ordering the total subject ratings for each treatment technique used and analysing the "degree of use indices" thus obtained showed that techniques were used differentially (see pp.222-223). More specifically, a significant difference was found to exist between the "use indices" for Hierarchical Reduction and Deprived Response Performance and the index for Covert Sensitization. This suggests that the former two techniques were applied more than was the method of covert-sensitization (which was one of the "therapist-administered" techniques, but which subjects were required to practice alone between treatment sessions).

It is not clear why covert-sensitization was used at a relatively low level. Perhaps the very aversiveness of the technique was responsible for its "unpopularity". The technique's characteristic of being rather time-consuming, if used properly, may be a factor which possibly weighed against its degree of use, although this argument is attenuated by the fact that Focussed Relaxation Training, which was equally time-consuming, was ranked relatively high on the list.

Rather more clear are the reasons why Hierarchical Reduction and Deprived Response Performance were used to a significantly higher degree. Both of these techniques were "well received" by subjects, being firmly based on proven and easily understood psychological principles (classical conditioning). Moreover, using the method of Hierarchical Reduction did not require any positive action on the part of the subject; rather, it was the non-performance of smoking behaviour that characterized the technique.

The methods of Focussed Relaxation, Coverant Control and Monetary Deprivation were all used to a moderate degree, although the latter method, again being subjectively aversive (as is covert sensitization), was found to be next to the lowest on the list.

Secondly, changes in the degree of use of the various techniques, over time, were examined (see p.223). It was found that, with only one technique - coverant control - was a significant change found; the method was used significantly more over the fourth week of treatment than over the first week. This finding is explainable in that subjects, initially and on the whole, had some difficulty in understanding the psychological principles underlying the use of this technique and commented that it seemed over-complex. However, continuing encouragement and clarification resulted in its being more willingly accepted and this is reflected in the above result.

To conclude this discussion of "degree of use" ratings, it is interesting to note that the maximum "index" obtained was 19.15 (for Hierarchical Reduction) (see Table 5.26, p.222) and the mean rating for all techniques was 15.14; the maximum possible index was 25.00,

showing that no individual technique was (subjectively rated as being) used fully. This finding leads one to enquire whether the treatment programme(s), as a whole, would have been more effective, were maximum use made of the individual methods. Finally, it must be emphasised that "degree of use" does not reflect "degree of usefulness"; this measure is associated more with "degree of benefit" obtained from techniques, which is discussed below.

b) "Degree of Benefit"

As with "use" ratings, "degree of benefit (obtained from individual techniques)" ratings were rank ordered according to the indices computed for all subjects (see pp.223-224). Significantly greater benefit was (seen as being) obtained from Hierarchical Reduction (which was the most "beneficial" method) than from Covert Sensitization or from Monetary Deprivation. This differential effect is concordant with "degree of use" ratings for the various techniques, where these three methods were identically positioned when rank-ordered.*

It is interesting that the two "aversive techniques used (Monetary Deprivation and Covert Sensitization) were seen as providing the least benefit. This may have been because of their inherent lack of effectiveness as treatment techniques or, perhaps more likely,

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*An almost perfect match was found between rank-orderings for the treatment techniques relative "degrees of use" and their "degrees of benefit". (Spearman's Rho correlation = 0.94 $p = 0.01$). The only exception here was the reversal of the positions of Deprived Response Performance and Focussed Relaxation. It may be considered axiomatic to say that, the more a technique is used, the more benefit will be obtained from that technique, but an alternative explanation for the above findings may be that some techniques were found to be of so little benefit, that they were subsequently made little use of. It is difficult to ascertain which of these explanations applies to these findings.

because they were used to a lesser degree than was desirable. This remains an empirical question.

In the case of two treatment techniques, rated benefit obtained changed over time, (over the duration of the treatment programme). More benefit was being obtained towards the end of treatment than at the beginning, from the techniques of Focussed Relaxation and Coverant Control (see p.225). With regard to the former method, this change was expected; efficient relaxation is a skill which must be learned and it is logical that this skill is refined, through practice, over time. In the case of Coverant Control, however, a different explanation is likely; this was provided above, in the context of "degree of use" ratings, when it was suggested that the technique became more comprehensible over time and was therefore used more, thus leading to increased benefit.

The fact that the remaining techniques employed did not yield a differential degree of benefit, over time, does not imply that they are less useful, but that they perhaps become less appropriate as smoking behaviour is brought under increasing control. (Take, for example, the technique of Hierarchical Reduction: once the most important high-probability smoking situations have been dissociated from smoking behaviour, the technique will become less powerful and less relevant to the subject's further attempts to reduce smoking).

c) <u>Rated weekly benefit from treatment</u>

In addition to analysing the rated benefit obtained from different treatment techniques (b), above), and examining the "overall benefit" obtained from treatment, up to the twelve-month

follow-up assessment point, analysis was also conducted on subjects' "weekly benefit ratings", throughout the duration of treatment. These data were presented earlier (pp.225-226) and clearly show that benefit increased as treatment progressed (see Figure 5.93, p.226). The greatest increase in benefit was apparent between the commencement of treatment and the end of the first treatment week; a statistically significant difference also emerged at the end of the fourth week of treatment (when obtained benefit was greater than at the end of the first week).

This shows that, although immediate benefit can be obtained from treatment, yet further benefit accrues from continuing treatment, over a period of weeks. This, of course, was expected as a function of the nature of the treatment programme, but still underlines the fact that treatments of a moderately long duration, using self-control packages, are likely to be more effective than short-term interventions.

(iv) Correlational Analyses

Several of the correlational findings of this study have already received attention, under the appropriate headings. The findings already discussed can be summarized as follows : self-reported smoking rate correlated significantly (Rho = 0.54, $p < 0.001$) with serum thiocyanate measurements, showing that self-reported rates were valid; baseline smoking rate did not correlate significantly (Rho = 0.05, N.S.) with % "internal" cigarettes smoked, thus casting doubt on the validity of the traditionally accepted nature of the "internal/external" dimension; "motivation" and "expectancy" scores did not correlate significantly (Rho = 0.10, N.S.) (this possibly being a

function of the lack of validity of the "motivation" scores computed for subjects); change (decrease) in smoking-rate correlated significantly (Rho = 0.26, $p < 0.001$) with change (increase) in gross body-weight; and the degree to which individual treatment techniques were used by subjects correlated significantly (Rho = 0.94, p = 0.01) with the degree of benefit reported as being attained from these techniques.

Some additional, interesting correlational findings emerged from this study. First, it was found that initial smoking rate did not correlate significantly (Rho = 0.04, N.S.) with initial "craving intensity" (contrary to expectations). This is of significance from a clinical perspective, in that it seems that it would be mistaken to assume that "heavy" smokers are more likely to experience more intense craving when reducing or abstaining from smoking than are "light" smokers; the corollary of this conclusion is that "light" smokers may be just as likely to experience intense craving, as "heavy" smokers. It is considered important to bear these probabilities in mind when devising treatment programmes aimed at modifying smoking behaviour.

Secondly, no significant correlation was found between level of motivation (at pre-treatment) and a "successful" response to treatment, as measured at any assessment point (from mid-treatment to one-year follow-up). This was surprising, in that previous studies have clearly identified motivation as an important, "non-specific" factor, influencing the outcome of smoking treatment programmes (McFall and Hammen, 1971; Raw, 1976, 1978) (see p.98 above). The most rational conclusion which can be drawn from

the negative finding of the present study, regarding motivation, is that this psychological characteristic was not measured properly; it has been suggested elsewhere that the questionnaire used was likely to be invalid.

Thirdly, significant, positive correlations were found between level of expectancy at pre-treatment and a successful response to treatment, as measured by examining smoking-rate, at both post-treatment assessment and three-month follow-up (Rho = 0.39, $p < 0.05$, in both cases) (Values of Spearman's Rho at other assessment points failed to reach statistical significance). This suggests that the "non-specific" factor of expectancy is, indeed, one of importance in determining response to treatment. This conclusion is consolidated by considering the correlations between subjectively rated (overall) benefit from treatment and initial expectancy, at each assessment point; the value of Spearman's Rho failed to reach statistical significance at only one point (post-treatment assessment) and was as high as 0.50 at six-month follow-up assessment. (See p.227). (Note that the correlation between "actual" and "self-rated" benefit was highly significant - Rho = 0.72, $p \leq 0.001$ - but not perfect, hence the inconsistencies described above).

This finding lends support to that of Blittner, Goldberg and Merbaum (1978) and serves to underline the importance of expectancy as an independent variable in the modification of smoking behaviour. (The deliberate manipulation (enhancement) of expectancy would, taking the above finding into account, seem to have a role to play in the treatment of smoking; this will be discussed in a later section, in the context of future research).

Finally, response to treatment and the variable of age were not found to correlate significantly (Rho = 0.10, N.S.). It was suggested that a significant, negative correlation would be discovered here (greater improvement being achieved by younger subjects), as it seemed rational to suppose that, the longer an individual's smoking history (assuming, of course, that age and smoking history are positively correlated), the more difficulty that individual would have in modifying his/her smoking behaviour (the behaviour being over-learned and the addiction to nicotine well-established). The non-significant correlation found is encouraging, the conclusion being that individuals who have smoked for a long period are equally likely to benefit from treatment as are younger smokers, who have a shorter smoking history.

(v) Predictive Factors

(a) Self-report measures

On only one of the self-report measures used in this study was a significant relationship discovered between pre-treatment characteristics of subjects and response to treatment: a high proportion of "internal" smoking was associated with a more successful outcome at post-treatment assessment, (see Table 5.31(i), p.229). This result is difficult to explain. If the "internal/external" dimension has any validity (at least, as measured in this study) - and there is some reason to believe that this validity is doubtful (see p. 238 , above) - then it would have been more understandable if the opposite relationship had been found (ie. less physio-

logically dependent, more "external" smokers responding better to psychological treatment). It must therefore be concluded that the significant predictive relationship found here is probably an artifact, perhaps being due to error of measurement or to unidentified chance factors.

Neither initial smoking rate nor initial situational anxiety or craving levels predicted the outcome of treatment. This finding can be interpreted positively, in that, regardless of baseline smoking rate and degree of dependency on cigarettes (as measured, indirectly, by the latter two parameters), subjects can successfully control their smoking behaviour. It has already been stated that, whether individuals are "heavy" or "light" smokers does not appear to be a significant factor in determining response to treatment (see p.246). Level of motivation did not predict outcome, but the lack of validity of the motivation questionnaire is almost undoubtedly the reason for this finding; this matter had already been discussed (see p.239).

A rather more surprising, and, at first sight, paradoxical finding was that "expectancy" was not found to be a predictive factor. The apparent paradox here is that expectancy and actual benefit obtained from treatment correlated positively and significantly (see previous section - Correlational Analyses). However, closer examination of these results resolves this paradox. To reiterate, the predictive power of the pre-treatment variables was examined by performing an analysis of variance on the "scores" on each variable of the most successful and least successful subjects,

at each assessment point; in the case of the variable of "expectancy", many subjects (50%) obtained a score of 10 (on a 0-10 scale), but considerable variation existed in the degrees of "success" of these subjects, the mean "success" or benefit of this group being, in fact, only moderate. Although the difference between the mean expectancy scores of the "successful" and "unsuccessful" (arbitrary) groups was in the right direction, this difference was not sufficient to yield a high enough "F" ratio to reveal a statistically significant difference, at any assessment point. Correlational analysis (Spearman's Rho), on the other hand, examines the trend relating to two variables and takes all scores into account. The problem of clustering of one set of scores at the same level along one axis, as found with analysis of variance, is therefore eliminated. It can be therefore concluded that the correlational analysis performed on the data obtained produced the "truer" picture of the inter-relationship between the variables of expectancy and benefit, and that expectancy is, in that case, an important factor.

The statistical problem described above is perhaps best understood by referring to Figure 6.2. This diagram illustrates the clustering of subjects' expectancy scores along the horizontal ("benefit") axis; assessment at mid-treatment is chosen for illustration purposes.

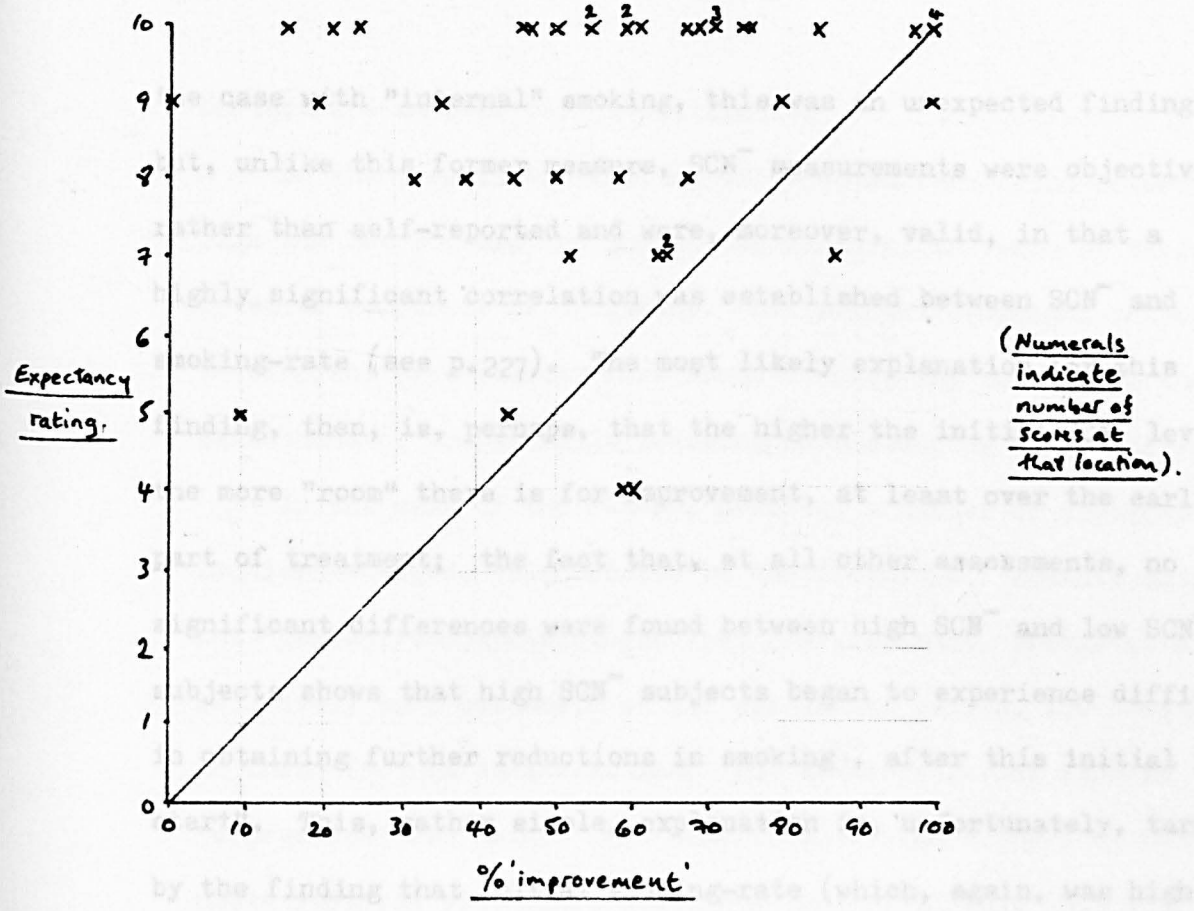

Fig.6.2
The relationship between "expectancy" and mid-treatment benefit: an illustration of paradoxical results of analysis

(b) Physiological measures

Neither body-weight, lung-function, age nor sex were found to predict response to the treatment packages employed in this study (see Table 5.31(ii), p.230). Regardless, therefore, of standing on these variables, smoking behaviour can be brought under control by many individuals.

One physiological measure, serum thiocyanate level at pre-treatment, however, did predict outcome: subjects with higher initial SCN^- levels tended to benefit more over the first three weeks of treatment, than did subjects with low levels. As was

the case with "internal" smoking, this was an unexpected finding, but, unlike this former measure, SCN^- measurements were objective rather than self-reported and were, moreover, valid, in that a highly significant correlation was established between SCN^- and smoking-rate (see p.227). The most likely explanation for this finding, then, is, perhaps, that the higher the initial SCN^- level, the more "room" there is for improvement, at least over the early part of treatment; the fact that, at all other assessments, no significant differences were found between high SCN^- and low SCN^- subjects shows that high SCN^- subjects began to experience difficulty in obtaining further reductions in smoking , after this initial "fast start". This, rather simple, explanation is, unfortunately, tarnished by the finding that initial smoking-rate (which, again, was highly correlated with SCN^- levels) did not predict outcome in any way. It may well be, therefore, that the finding in question is simply the result of unidentified extraneous factors, such as a relatively small sample size (N = 18 in each group).

(c) Personality Measures

So far the discussion of predictive factors and the explanation of causal relationships apparently found to exist have been characterized by uncertainty and inconclusiveness; some paradoxical findings have also been reported. In the case of personality factors, however, far more conclusive statements can be made, as higher levels of significance for certain factors were found, high conformity of results, over assessments, was evident (see Table 5.31(iii), p.231) and logical relationships between personality

factors and outcome were identifiable.

In the case of the EPQ, no differences were found between high and low scorers, on any of the factors, in terms of their relative responses to treatment. Neither extraversion, introversion not psychoticism were found to have any bearing on the likelihood of successfully reducing or abstaining from smoking.

In the case of the SCL-90, however, a number of important findings emerged. High scores on the factors of "somatic anxiety" and "obsessive-compulsiveness", at pre-treatment, predicted a relatively poor outcome; at all assessment points, low scorers on these factors benefited more from treatment. Low scorers on "depression" had gained more benefit than high scorers at both post-treatment assessment and three-month follow-up (but were equivalent at all other assessment points). High ("general", as opposed to "situational" - see above) anxiety scorers showed less improvement with respect to smoking rate reduction, at all assessment points apart from one-year follow-up, than did low "anxiety" scorers. Low "hostility" scorers did better at six- and twelve-month follow-up. At all points, a high "paranoid ideation" score was found to be a predictor of poor improvement with regard to smoking control. Finally, the individual's "Global Symptomatic Index" (GSI) was a good predictor (low scorers responding more successfully to treatment, as was the "Positive Symptom Distress Level" (PSDL) (at least when success at the post-treatment and three-month follow-up points is considered).

Some specific and some more general statements may be made,

by way of interpreting these results. First, the relationship between the factor of "somatic anxiety" and outcome is clinically explainable: reducing smoking is known to lead to withdrawal symptoms, many of which are somatic in nature (Weybrew and Stark, 1967; Brecher, 1972); if an individual already suffers from a high level of somatic anxiety, then the symptoms experienced will be intensified and will become more frequent as smoking-rate reduces. This aversive consequence of reducing smoking will therefore operate instrumentally, punishing reduced smoking, and so decreasing the probability of further reduction.

Secondly, it is likely that individuals having "obsessive-compulsive" personality characteristics, being relatively rigid and inflexible, will be less likely to be able to modify their smoking behaviour, which, in most cases, is an extremely well-learned (and perhaps overlearned) behaviour (Hunt and Matarazzo, 1970). Again, therefore, this "predictive" finding lends itself to a logical, clinical explanation.

Thirdly, with regard to the SCL-90 factors of "(general) anxiety" and "depression", explanations pertain for their predictive power, which are the same as for "somatic anxiety", in that both feelings of anxiety and depressed mood-state have been clearly associated with withdrawal from smoking (Weybrew and Stark, 1967). Anxiety and depression therefore intensify as smoking-rate reduces, to the point where the cost outweighs the gain, and further reduction becomes difficult or impossible.

A final, specific conclusion can be drawn in relation to the

SCL-90 factor of "hostility", and this is, again, consistent with the conclusions regarding "somatic anxiety", "anxiety" and "depression". Irritability has been found to increase as smoking decreases (Larson and Silvette, 1971) and it is therefore understandable that smokers who are already relatively "hostile" individuals (hostility and irritability being related) will find their efforts at reducing smoking (or maintaining a reduction) inhibited by still more extreme feelings of hostility; as well as being subjectively distressing such behaviour is likely to be punished by others, thus affording additional punishment to a low level of smoking behaviour. This operant process was evidenced by the fact that low "hostility" scorers were more "successful" at long-term follow-up (six and twelve months) than were high scorers.

Commenting more generally on the SCL-90 findings, it is clear that, the more psychologically "disturbed" or "distressed" an individual (viz. The ability of "paranoid ideation" and "global symptomatic index" scores to predict the outcome of treatment), the less likely is that individual to benefit from a primarily psychological treatment programme for smoking.

Several 16PF personality factors were found to relate to treatment outcome. The more "successful" subjects were those who scored <u>higher</u> on Factor B ("intelligence") (these subjects had benefited more by twelve-month follow-up), on Factor C ("emotional stability") (more benefit at three-month follow-up), on Factor M ("more imaginative") (more benefit at mid-treatment) and on Factor Q_1 ("more experimenting/liberal") (more benefit at three, six and

twelve-month follow-up) and those subjects who obtained lower scores on Factor G ("conscientiousness") (these subjects benefiting more by twelve-month follow-up) and on Factor Q_4 ("anxious/tense") (more benefit at six-month follow-up).

These findings were, largely, not unexpected. It is logical that more intelligent and emotionally stable individuals should prove to be more successful candidates for a smoking control programme and equally understandable that less rigid (more "liberal") and tense individuals should respond better to treatment. Less clear is the discovered relationship between "success" and the characteristics of "conscientiousness" and "imaginativeness"; further investigation will, perhaps, clarify these findings.

To conclude this section, it is rather difficult, with reference to the results obtained in this study, to identify either self-report or physiological variables, measured at pre-treatment, which predict whether the response to treatment will be successful or otherwise. Where personality attributes are concerned, however, there seems little doubt that certain characteristics are related to outcome. This issue will receive further attention when the implications of this study are discussed, at a later point.

(vi) The Control Groups

(a) Self-report measures

The purpose of the Control groups in this study was to provide a comparison between treated and untreated groups. As the control groups were "waiting-list" controls, rather than groups receiving treatment of a minimal nature, it is not possible to draw any conclusions as to which aspects of the treatment programme were effective; for example, it may be that "non-specific" treatment factors were responsible for some of the results obtained. This matter will receive further discussion in a later section.

At pre-treatment assessment, Control Group 1, the "heavy-smoking" control group, along with treatment groups 1 and 5, obtained a significantly lower "anxiety" score than did the other "heavy-smoking" groups (Groups 2 and 6); Control Group 2 (the "light-smoking" control group), along with treatment groups 2, 4 and 8, obtained a significantly lower "motivation" score than Group 7. (See Table 5.18(i), p.157). These inter-group differences, evident at pre-treatment, were not, in any way, reflected in groups' responses to treatment (and neither "anxiety" nor "motivation" scores were found to predict outcome) and so the comparison between the control groups and the treatment groups on self-report measures, at later assessment points, was not invalidated.

Table 5.25(i) (p.219) shows that neither control group showed a significant change over time, from "pre-treatment" to "post-treatment" equivalents, on any self-report measure. It can therefore be concluded that the changes on these measures which were

evident in the treatment groups were due to the implementation of the treatment programme.

(b) Physiological measures

No significant differences were apparent between the control groups and the treatment groups, on any of the physiological measures taken at pre-treatment assessment. (See Table 5.18(ii), p.158).

However, it has been reported (p.220) that, although control group SCN^- levels and body-weight measures showed no change from "pre-treatment" to "post-treatment", significant changes were evident on some lung-function measures over this eight-week period. To re-iterate, Control Group 1 (the "heavy-smoking" control group) demonstrated a highly significant increase (improvement) in FEV/FVC ratio, between the two assessment points and Control Group 2 (the "light-smoking" control group) showed a significant decrease (deterioration) in FEV, and in FEV/FVC ratio. These changes in lung-function measures were not related to a significant change, in either direction, of smoking-rate, over this same period.

In the case of the first finding , every subject in Control Group 1, without exception, showed an increase in FEV/FVC ratio from "pre-" to "post-treatment" assessment. It is unlikely that such a significant change within this group should occur over a relatively short period, which was due to a real improvement in lung-functioning, especially as no significant changes were evident in the treatment groups, as a result of decreased smoking-rate, over

a much longer period. The most plausible explanation for this finding, then, is that some error in measurement occurred, perhaps for all subjects in this group (who were assessed on the same day and under similar environmental conditions), at either the first or second assessment.

This same explanation may well apply to the other anomalous finding reported earlier and above, with regard to the control groups' lung function measures. Whether similar measurement error was a factor influencing the obtained lung-function data for the treatment groups is not known; if this was the case, then it is possible that, where no change over time for any treatment groups was reported, a significant change in lung-function may, in fact, have occurred. However, taking into account the uniformity of these results, it is felt that this is unlikely and that the error in measurement was probably limited to the control groups.

(c) Personality measures

At pre-treatment assessment, it was found that, on one personality measure, 16PF Factor Q_2 ("self-sufficiency"), Control Group 1 (the "heavy-smoking" control group) obtained a significantly higher score than did treatment Group 2 (see p.160). As no differential responses to treatment subsequently took place between Group 2 and the remaining treatment groups, and as pre-treatment 16PF Factor Q_2 scores were not found to predict outcome, it can justifiably be concluded that this finding is immaterial.

Certain changes took place, between assessments, on certain of the control groups' personality measures. (See Table 5.25(iii),p.221).

Control Group 1 (the "heavy-smoking" control group) showed a significant decrease on the SCL-90 measures of "Global Symptomatic Index" and "Positive Symptom Total". This same group also showed a significant decrease on 16PF Factor N ("Forthrightness") and a significant increase on Factor Q_3 ("Self-Control"); Control Group 2 showed a significant decrease on 16PF Factor B ("Intelligence"), over time.

These changes on the measures of GSI and PST are difficult to explain, as are those on the 16PF factors. On none of these measures did any treatment group demonstrate a change over time. Only two explanations seem possible: either a decrease in smoking-rate serves to maintain stability on these personality measures or some error took place in the measurement of these factors. The former explanation is implausible, so it is likely that measurement-error is responsible for these findings in the control groups. This conclusion is reinforced by the fact that one of the changes occurring was on the measure of "intelligence" (Control Group 2 decreasing over time); this finding is untenable.

The implication of these findings is to cast some doubt on the conclusions drawn from the analysis of the personality outcome data of the treatment groups; if measurement-error occurred in the case of the control groups it is not unlikely that this was also the case with the treatment groups. This point will be re-emphasized in the later section on the implications of this study.

(b) Validation of the Hypotheses

This section is designed to complement the earlier section

"Hypotheses" (Section 4, pp.137-139). For clarity, therefore, the original hypotheses are reproduced as they first appeared; the experimental findings are then stated.

(i) Type of treatment

"It was hypothesized that those groups receiving treatment consisting of the self-control package plus therapist-administered methods would obtain greater benefit from treatment than those groups receiving the self-control package alone, but that all treatment groups would significantly reduce their rate of smoking, regardless of treatment condition".

The first part of this hypothesis was not upheld; no significant differences emerged between those groups receiving the "self-control" package and those receiving this package plus "therapist-administered" techniques, with regard to reductions in smoking rate.

The second part of this hypothesis was upheld; all treatment groups, regardless of treatment condition, significantly reduced their rate of smoking.

(ii) Goals of Treatment

"It was hypothesized that those groups having a goal of reduced or controlled smoking would be as successful in achieving their goal as would the groups whose desire was to abstain totally from cigarette smoking".

This hypothesis was not supported. A greater proportion of subjects who aimed at abstaining from smoking achieved their goal, compared with the proportion of subjects aiming at smoking at 25% of their baseline rate. (However, rate reductions across groups, regardless of treatment goal, did not significantly differ).

(iii) "Heavy" versus "Light" smokers

"It was hypothesized that heavy smokers would be less successful in achieving their goal than would light smokers".

This hypothesis was not supported. No significant differences

were apparent between the relative "success" rates of "heavy" and "light" smokers.

(iv) Physiological correlates

"It was hypothesized that: a) serum thiocyanate (SCN^-) levels would be significantly reduced, as a result of treatment, in all groups, but that this reduction would be more pronounced in the groups receiving the 'additional' treatment techniques and less pronounced in groups aiming at 'controlled' smoking; b) respiratory functioning would significantly improve in all groups, but that this improvement would be more pronounced in those groups receiving the 'additional' treatment techniques and less pronounced in those groups aiming at 'controlled' smoking; and, c) all groups would significantly increase in weight as a result of treatment, but that this increase would be more pronounced in those groups receiving the 'additional' treatment techniques and less pronounced in those groups aiming at 'controlled' smoking".

Hypothesis a) was partially upheld: SCN^- levels were significantly reduced, as a result of treatment, in all groups; however, no inter-group differences were found, as a result of treatment condition or of treatment goal.

Hypothesis b) was not upheld: in no treatment group was any significant improvement in respiratory functioning evident, regardless of treatment condition or treatment goal.

Hypothesis c) was partially upheld: all groups increased significantly in weight, as a result of treatment; however, no inter-group differences were found, the independent variables of treatment condition and treatment goal thus being of no relevance.

(v) Personality measures

"It was hypothesized that no significant changes would take place over time, as a result of treatment, on any of the personality factor measures".

This hypothesis was not supported. Although no significant changes occurred, over time, on any of the Eysenck Personality

Questionnaire factors, significant changes were apparent (over all groups) on certain SCL-90 and 16PF factors. Specifically, long-term decreases on the SCL-90 factors of "Anxiety", "Hostility" and "Psychoticism" were found; and a Groups x Time interaction effect was discovered on 16PF factor "I" ("Toughmindedness"), Groups 2, 4 and 7 decreasing more, over time, than the remaining treatment groups.

(vi) Predictive Factors

"It was hypothesized that, of the measures taken, pre-treatment level of motivation and level of expectancy would significantly and positively correlate with degree of reduction is smoking rate. It was hypothesized that none of the remaining pre-treatment measures would predict outcome".

The first hypothesis, here, was partially upheld: although level of motivation did not significantly correlate with degree of reduction in smoking rate, level of expectancy did correlate positively and significantly.

The second hypothesis was not upheld. Some predictive power was (tentatively) attributed to the pre-treatment measure serum-thiocyanate level, and more conclusively to the SCL-90 measures of "Somatic Anxiety", "Obsessive-Compulsiveness", "Depression", "Anxiety", "Paranoid Ideation", "Global Symptomatic Index" and "Positive Symptom Distress Level". The 16PF factors of "Intelligence" (Factor B), "Emotional Stability" (Factor C), "Anxiety/Tension" (Factor Q_4) and "more experimenting/liberal" (Factor Q_1) were found to have some predictive power. (More tentative conclusions were drawn regarding the ability of the 16PF factors of "Conscientiousness" (Factor G) and "Imaginativeness" (Factor M) to predict outcome).

(vii) Additional Correlates

"It was hypothesized that: a) self-reported rate of smoking and serum thiocyanate level measurements would correlate significantly and positively, and, b) baseline (pre-treatment) smoking rate and extent of 'internal' smoking (cigarettes smoked as a response to internal rather than external cues) would co-relate significantly and positively".

The first part of this hypothesis was validated: a significant, positive correlation was found between self-reported rate of smoking and serum thiocyanate level measurements.

The second part was not validated: smoking-rate and extent of "internal" smoking were not found to correlate significantly.

(viii) "Use" and "Benefit" ratings

"It was hypothesized that all treatment techniques would be used by subjects to the same degree and that subjects would obtain equal benefit from all treatment techniques.

It was further hypothesized that degree of use of and degree of benefit obtained from treatment techniques would be significantly and positively correlated and that rated degree of overall benefit obtained from treatment would correlate significantly and positively with self-reported reductions in smoking-rate".

The first hypothesis stated here was not supported: subjects were found to use treatment techniques to differing degrees and differential benefit was found to be obtained from the various treatment techniques.

Both aspects of the second hypothesis were supported: the degree of use of and the degree of benefit obtained from treatment techniques correlated positively and significantly; and rated degree of overall benefit obtained from treatment correlated significantly and positively with self-reported reductions in smoking rate.

(ix) Maintenance of change

"It was hypothesized that the benefits obtained from treatment would still be evident at 3 month, 6 month and 1 year follow-up assessment".

This hypothesis was upheld. Significant reductions in smoking rate were evident, for all treatment groups, at all follow-up assessments.

(x) Control Groups

"It was hypothesized that no significant changes would occur within the control groups, on any of the measures taken, between their first and second assessments".

This hypothesis was not fully upheld. Although no significant group changes, over time, were found on any of the self-report measures taken, significant changes were found on two of the lung-function measures and on certain factors of the SCL-90 and 16PF questionnaires. (These changes were, however, attributed to measurement-error).

(c) Methodological Considerations and Shortcomings

(i) Subject and group-composition factors

As the subjects participating in this study were individuals who requested help to stop/reduce smoking and were sufficiently motivated to complete the initial questionnaire, which was quite lengthy, it may be concluded that these subjects did not represent the general smoking population. Moreover, those subjects who remained in treatment, having undergone full pre-treatment assessment (this being in some respects aversive), were, by the same token, even less likely to be typical smokers. Despite the inconclusive results obtained from the analyses of "motivation" questionnaire scores, it is safe to conclude that the subjects

treated, on the whole, were well-motivated.

Although this is a methodological problem which can be clearly defined, it is also one which, with an outcome study of this type, is impossible to eliminate. Any treatment programme entailing the participation or co-operation of volunteers must recognize the fact that its subjects are distinguished by virtue of their very co-operation and by the fact that they are volunteers.

It can only be concluded that, with a group of unmotivated smokers, the results of this and similar studies may not have been as positive.

Perhaps the most serious shortcoming of this study was its use of relatively small groups of subjects. It has already been explained (p.113) that, initially, eight subjects were allocated to each treatment group, but that, following pre-treatment assessment, in the case of five of the eight groups, only six subjects attended for the first treatment session; it was therefore decided to randomly eliminate two subjects from the remaining three groups, to balance the number of subjects in each group (to facilitate analysis).

This problem was magnified by the fact that further subject attrition occurred, both during the treatment phase and during the follow-up period, in the case of some treatment groups.

Although outcome analysis was precluded, because of insufficient data at long-term follow-up, for only serum thiocyanate and lung-function measures, (Treatment Group 2 providing very

sparse data), the results obtained for certain other groups, for some variables, should be regarded, perhaps, as being tentative. The main method of analysis used in this study was analysis of variance; although this is a relatively powerful parametric technique, the validity of the results thus obtained is attenuated by low "n's" in the groups compared. More specifically, analysis of variance compares the mean of groups and, by definition, takes variance into account; if wide variation occurs in the "scores" of individuals in a group, on a certain measure, analysis of variance is less likely to yield a significant F ratio. The smaller the group, the more easily contaminated are the results of analysis of variance by wide individual variation in scores (See, for example, the results obtained on the measure of anxiety, at pre-treatment, pp. 236-237).

In the case of the results obtained in this study which were based on small group "n's", therefore, it must be stated that these need to be interpreted with caution, as do the conclusions which have been drawn from a consideration of these results.

With hindsight, a higher initial number of subjects per group should have been used in this study; the supposition that "Eight subjects ..... was felt to be the optimum number" (p.113) was not warranted.

With regard to the control groups, certain methodological problems are apparent. Firstly, as with the treatment groups, a higher number of subjects per group would, perhaps, have been more appropriate (although no attrition occurred between the two

assessments). Secondly, and more importantly, the nature of the control groups used in this study needs to be taken into account in examining the effectiveness of the treatment programmes used with the experimental groups. The control groups were "waiting-list" controls; they received no treatment whatever during the eight-week period which elapsed between their two assessments. The implication of this is that no conclusions can be drawn, with any absolute conviction, regarding which elements or aspects of treatment were effective with the treatment groups: the results obtained, in terms of the modification of smoking behaviour, may have been as much due to the influence of "non-specific" factors (such as motivation, expectancy, personality, etc. - see pp.96-104) as to specific treatment factors (ie. the treatment packages, per se). (The one "non-specific" factor which did apply to the control groups was that of self-monitoring - recording the number of cigarettes smoked over a 10-day period prior to assessment. This factor was, therefore, effectively controlled for).

As the issue of what type of control group is best used in this type of study has been addressed in depth, elsewhere (Bernstein, 1969; Raw, 1978), no further discussion will take place here. It must be said, however, that the conclusions drawn from this study may have been less open to alternative interpretation had "minimal treatment" control groups been used, in an effort to control for "non-specific" factors, rather than "waiting-list" controls. The avoidance of over-complexity was the primary reason why the latter type of group was employed,

but perhaps this disadvantage is outweighed by the advantages of more appropriate controls.

(ii) Treatment factors

It is believed that certain modifications to the treatment methods used in this study may have enhanced the overall effectiveness of the treatment programme used. Firstly, although Hills (1983) has confirmed the present author's earlier belief that, in using the method of hierarchical reduction, it is best to eliminate "hard" hierarchy items before "easy" ones, on consideration, the reverse may be preferable. If a smoker is required to stop smoking in a situation where this behaviour is of very high probability (the associative bond being strong), the anxiety/stress which may result is perhaps likely to discourage further attempts at smoking reduction. Conversely, stopping smoking in a less difficult, but nevertheless high-probability, situation is likely to be less stressful and more easily accomplished; further reduction efforts may, therefore, be facilitated as a result of this early success. Further research is necessary to clarify this issue.

Secondly, one of the methods used in this experiment was contingency contracting of a social nature. Specifically, subjects were asked to inform others that they were embarking on a programme designed to reduce or eliminate their smoking behaviour. No check was made, to ascertain whether this request was followed. It would, perhaps, have been wise to confirm that this was the case, by interviewing subjects' spouses, friends, etc. In the event, this possibility was overlooked. (This same validation

technique could, perhaps, have been used, with respect to other methods of treatment supposedly being utilized; this would have provided a more objective measure than the "use" ratings employed. However, the reliability of spouse's reports would also have to be questioned, assuming the co-operation between spouse and experimenter in the first place).

Thirdly and finally, it is necessary to re-iterate a point made earlier (p.127) with regard to the use of the technique of "monetary deprivation". It is acknowledged that the donation of "fines" (as a result of smoking) to a "charity of one's choice" may not be a subjectively aversive act. Thus this method, as used in the present study, was perhaps not, in reality, the "self-punishment" technique which it was intended to be. The giving of money forfeited to a strongly disliked charity or organization would certainly have been perceived as more aversive, but the ethical implications of this weighed against the use of this contingency. Perhaps an act such as simply destroying the money accumulated as a result of smoking would have been more effective than the actual procedure followed. In addition to this methodological problem, it also needs to be emphasized that no objective check was made on whether individuals were actually "fining" themselves, as instructed, for smoking; whether this method was, in fact, used as directed, is not known.

(iii) Assessment factors

The assessment methodology of this study was characterized by certain problems, these being, most notably, the reliability

and/or validity of some of the measures used.

The one measure which was almost certainly invalid was the "motivation" score calculated for each subject. Motivation failed to predict outcome and, as there exists ample evidence that motivation is an important "non-specific" factor influencing response to treatment of smoking (McFall and Hammen, 1971; Raw 1976, 1978), it seems safe to conclude that the lack of predictive power was due to the measure's invalidity.

A similar argument may be applied to the lung-function findings; no significant changes were found to take place, over time, in the treatment groups' respiratory functioning, despite their having decreased their smoking rate and despite lung-function measures being taken at one-year follow-up; conversely, significant changes did occur in the case of the control groups, who were assessed twice over a short duration and who did not alter their smoking rate. The reliability of the lung-function measures taken is, therefore, questionable, and more accurate assessment would have been desirable.

With regard to the various personality factors measured, too, high reliability (and perhaps validity) also seem to have been absent in some instances. It has already been stated (pp.263-267) that, on certain SCL-90 and 16PF factors, the control groups showed significant changes over time and, further, that these changes were either due to the fact that a decrease in smoking-rate (as evidenced by the treatment groups) serves to maintain stability regarding scores on these factors or due to error in measurement. This latter explanation is more plausible and so it must be concluded that the

personality characteristic measures in question were lacking in reliability. As previously stated, this finding casts some doubt on the conclusions drawn, with respect to the treatment groups, where personality measurement is concerned.

A final area where the methodological problems of validity and reliability are salient is the utilization of "degree of use" ratings in this experiment. These ratings were entirely subjective and self-report was the method of assessment. As no objective check (eg. experimenter or spouse observation) was conducted on whether subjects were, indeed, using the methods employed in the study, the honesty of the subjects in completing these ratings was of paramount importance. As a high positive correlation was found between "use" and "benefit" ratings for individual techniques, it can probably be justifiably concluded that the reliability of the "degree of use" ratings was relatively high; only an examination of the correlation between self-reported "use" and more objective measures of "use", however, could have firmly established the reliability of the former.

The last point which needs to be made, concerning assessment, is that, although follow-up was conducted up to one-year after the termination of treatment, no further data was collected from subjects thereafter. Benefit from treatment was still clearly apparent after a year, for all treatment groups, and it was assumed that this benefit was permanent. However, this assumption may be unfounded. Long-term studies, such as those conducted by Colletti and Stern (1980) and Buchkremer (1982) are admirable, and the results of this study would have been more conclusive, had extended follow-up assessment been performed.

(d) Conclusions and Implications for Future Research

It is possible to draw several major conclusions from the results of this study. The study's primary aim was to compare the efficacy of a comprehensive, but nevertheless simple, self-control treatment package with that of a more complex package incorporating "therapist-administered" treatment techniques. The unmistakeable conclusion is that these treatment packages were equally effective in bringing about positive, lasting change in individuals' smoking behaviour.

This finding can be interpreted in two ways. Firstly, it may be that any treatment "package", using more than one technique, will result in a significant change in smoking behaviour. Raw (1977) has said that "Almost any 'treatment' technique ..... can serve as a stimulus to stop smoking"(p.200). In the absence of control groups who undergo a form of treatment consisting of all but the methods being examined experimentally (ie. "non-specific" factors are made common to all groups), it is impossible to reach a firm conclusion as to whether the treatment methods are inherently effective. This "non-specific" element of treatment may possibly have accounted for the improvement evident in this experiment.

The second interpretation is that the additional, "therapist-administered" techniques may have added nothing to what was already a truly effective treatment package.

The fact that subjects were actually able to specify which treatment techniques they found beneficial, (and the fact that

differential benefit was described) strongly suggests that it was the very nature of the treatment package(s) that led to significant reductions in smoking-rate. Raw's statement does not, therefore, seem to be supported; (even if this were the case, however, what is important, in the final analysis, is whether subjects do in fact benefit from treatment, rather than what are the specific aspects of treatment which lead to this benefit. As long as the procedure and the methods used are replicable, as they are in the case of this study, the methods of treatment used must be considered to be of value).

It seems, then, that the "behavioural contingency" model of smoking, as proposed by Frederiksen and Simon (1979) (see pp.18-21, above) is an accurate and valid model; the rationale underlying the nature of the treatment packages utilized in the present study stemmed from a consideration of the virtues of this model, and the results obtained do provide validation for this view of smoking behaviour.

It is certainly worth stating that the finding regarding the equality of the two treatment packages used, in terms of efficacy, has implications with regard to the cost-effectiveness of programmes designed to help smokers; direct therapist involvement in treatment does not seem to be necessary (apart from the therapist's initiating and guiding self-control efforts and perhaps reinforcing progress) and this means that far more clients may be dealt with, simultaneously, in a clinical setting, than has previously been supposed.

A second issue which this study investigated was that of the feasibility of reduced or "controlled" smoking, in the long term. Once again, it was found that all groups aiming to reduce their rate of smoking did so, significantly (and maintained this reduction in the long-term); however, difficulty was found in reducing to the prescribed rate of smoking (a quarter of the initial rate). It has already been stated (pp. 250-254) that, if the criterion of "successful" participation in the intervention programme is considered, by a subject, to be the attainment and maintenance of a specified target rate of smoking, then a target of zero seems to be more easily attainable and maintainable than does a target of 25% of baseline-rate.

Whether this study lends support to the concept of controlled smoking, therefore, depends on the "success" criteria used. Subjects were, on the whole, unable to control their smoking at a relatively low, pre-specified rate, relative to their initial rate; however, all groups were able to reduce (and keep at a lower level) their smoking frequency, to a significant degree (approximately 50% of baseline rate at 1-year follow-up; see p. 253).

Russell (1974) has opined that the goal of abstinence is far too difficult for most smokers to achieve and that the goal of intervention should, perhaps, be to make smoking less harmful, for every smoker. Russell's opinion and suggested goal are supported by the findings of the present study - abstinence was hard to achieve, but all groups in the study gained long-term benefit, from a health viewpoint, from their participation in the

experiment. This health benefit is clearly apparent (at least indirectly) by considering the significantly decreased serum thiocyanate levels for all groups in the study, as a result of treatment. (It may be added, parenthetically, at this point, that, as a high correlation was found between self-reported smoking-rate and objectively measured SCN levels, the former type of measure can be considered more reliable, in this type of experiment, than has formerly been suspected, and physiological corroboration is therefore perhaps unnecessary; it was felt that requiring subjects to provide blood-samples contributed to the high drop-out rate in this study. Furthermore, the fact that SCN levels dropped along with a decrease in smoking rate casts doubt on the validity of the nicotine-regulation hypothesis; were nicotine regulation to have occurred, SCN levels would not have decreased significantly - See p.26-35, above).

A final point, regarding the comparison of groups' responses to treatment in this study, is that baseline smoking rate was found to be of no consequence in predicting the likelihood of successfully reducing smoking. Both "light" and "heavy" smokers obtained equal benefit from the treatment packages used.

(Attention must be drawn again, however, when considering the above conclusions, to the methodological issue raised earlier (p. 292), namely, that the non-significant differences in the treatment groups' responses to intervention may have been, at least in part, due to the low numbers - and therefore relatively high within group variances - comprising the individual treatment groups).

Although it is clear from an appraisal of the results of this study that significant benefit was obtained from treatment by all groups and that this benefit was lasting, it is equally clear that significant relapse took place over time, after treatment was formally terminated. More specifically, a statistically significant relapse occurred between the end of treatment and three-month follow-up assessment and (less significantly) between three-month and one-year follow-up assessment. The conclusion to be drawn from this is that it is necessary for subjects who have succeeded in reducing their rate of smoking (or abstaining from smoking) by the end of treatment to continue to employ the methods used, thereafter. It would be certainly desirable, taking the above results into account, to offer regular (but perhaps fairly infrequent) "booster" sessions for subjects, to reinforce continuing progress and to remind subjects of the techniques being employed. Such booster sessions would seem to be of especial value during the first three-months after treatment ends, but there is an argument for booster sessions over a longer period.

In addition to the major conclusions drawn above, a number of further important findings emerged from this study. Firstly, the level of subjects' expectancy played a clear role in determining response to treatment. This finding is concordant with previous studies (eg. Blittner, Goldberg and Merbaum, 1978). It follows, from this, that the deliberate enhancement of expectancy may well facilitate the treatment of smoking behaviour and this issue

certainly deserves empirical study.

Secondly, the weight-increase factor appears to be of importance and interest both from a clinical and a theoretical perspective. Clinically, the importance lies in the fact that weight increase is often seen by clients/patients as aversive and this may undermine efforts to abstain from or reduce smoking, which would otherwise have proved successful. It will be remembered that, in this study, all groups showed a significant weight increase, over time, as smoking rate reduced, and that it was suggested that this contributed to the high rate of subject-attrition. It seems necessary, therefore, to incorporate in any smoking control programme advice aimed at preventing (or minimizing) weight-increase (at least for those subjects who view this as a problem). The weight increase phenomenon remains of interest theoretically, as it is unclear to what extent physiological, metabolic factors are responsible, as compared to factors relating to diet and eating behaviour. There is no doubt that reducing smoking leads to metabolic changes, which in turn may lead to weight increase (see Russell, 1980, pp.254-255) but there is equally little doubt that changes in smoking behaviour lead to changes in eating behaviour (see pp.49-52, above). Further, empirical research is needed to elucidate the relative contributions of these mechanisms to weight-increase.

Thirdly, reference must be made to the changes which took place, over time, in this study, on the measures of "situational anxiety" and "craving intensity", as smoking rate decreased and

then again increased. In both cases, "inverted U" shaped patterns emerged, showing that anxiety and craving are very much dependent on rate of smoking (and deprivation from nicotine). It is clear that any intervention programme which takes these parameters into account, and offers assistance to the individual in dealing with the subjectively negative affect resulting from reduced smoking, will be more effective than programmes ignoring these phenomena. Perhaps a more intensive form of anxiety management training would be beneficial to many subjects; this warrants further investigation.

Fourthly, it seems from the results of this study that certain personality factors can predict response to treatment. There are two implications here: one is that, as those subjects who score highly on scales measuring degree of somatic anxiety, depression, obsessive-compulsiveness, etc. (see p. 231) seem to be poor candidates for smoking-treatment programmes, it may be that the limited resources available should be directed towards individuals who do not exhibit signs and symptoms of this kind; the second, perhaps more constructive (and ethical), implication is that, before offering help with smoking to individuals with personality characteristics (or behavioural tendencies) which predict a poor response to treatment, help should be given to change these characteristic ways of behaving. Perhaps smoking intervention programmes should be more broadly based than has been typical, in that other aspects of individual behaviour should be dealt with concurrently.

Fifthly and finally, some pertinent conclusions can be drawn from the examination of the relationship between "degree of use of" and "degree of benefit obtained from" the various treatment techniques used in this study. Apart from the obvious conclusion that certain techniques were rated as being more effective than others, it is evident that, the more a technique is used, the more benefit accrues from that technique. Although it perhaps seems to be stating the obvious, it is worth saying that stringent efforts should be made to ensure that subjects participating in smoking control programmes are indeed utilizing the techniques they are supposed to be utilizing. Enlisting the co-operation of spouses or other relevant individuals may be one way of ensuring adherence to advice and instructions given.

Recommendations for the direction of future clinical work and research in the field of smoking behaviour modification can be summarised as follows.

A valid, working model of smoking behaviour is essential. Only by reference to such a model, which must incorporate the various facets of smoking behaviour (both physiological and psychological), can effective means of treatment be devised. There seems little doubt, from this conclusion, that "package" treatments are the method of choice; (there is also a need for constant interplay between models and treatment, the former being revised appropriately in the light of treatment results).

The evidence suggests that self-control packages, which are used independently of direct therapist treatment, are effective. However, it is clear that "treatment" must continue over an extended period of time, if relapse is to be minimized; booster treatment sessions would seem to be necessary.

The concept of controlled smoking is worth pursuing. Total abstinence does seem to be a difficult goal for most smokers to achieve, but this study has shown that significant reductions in smoking rate, and subsequent improvement in physical health, can be attained and maintained over an extended period. The ability of individuals to learn to smoke at a relatively low, pre-specified rate seems to be an even more elusive goal than total abstinence and efforts need to be made to determine at what level, relative to baseline smoking-rate, difficulty in maintaining improvement begins to be experienced: a reduction of 50% in rate seems relatively easy to establish; a reduction of 75%, far less so.

Specific issues which need to be addressed and subjected to further, experimental investigations are those of the role of expectancy in determining an individual's response to treatment (the deliberate manipulation of expectancy being a promising line of research to follow) and the phenomenon of weight increase, as a result of decreased smoking rate. The mechanisms underlying weight-increase need further elucidation and the phenomenon requires control, in smoking-treatment programmes, through the application of appropriate techniques, if subject co-operation is to be maximized and subject-attrition minimized.

The subjective experiencing of craving and of an increase in anxiety level, as smoking-rate decreases, are likely to be factors which undermine what would otherwise be effective treatment programmes: direct attention needs to be paid to these aversive psychophysiological states in formulating efficient modes of intervention.

Further work needs to be carried out to elucidate the power of specific "personality" factors to predict the outcome of treatment of smoking behaviour. This is still an unclear area. However, what is clear is that certain individual characteristics can inhibit a positive response to treatment (for example a tendency towards depression, obsessive-compulsiveness or the experiencing of symptoms of somatic anxiety) and these characteristics need to be taken into account when devising and executing treatment programmes.

Finally, it is likely that some enhancement of treatment effectiveness would result from the involvement, in treatment, of subjects' spouses, friends, associates, or others who may be explicitly designated as observers or co-therapists, with the express purpose of ensuring that the advice and instructions offered by the therapist are followed by the smoker. There is evidence that the conscientious application of the treatment methods suggested contributes significantly towards a successful response to treatment; increasing this degree of conscientiousness, perhaps through external pressure, should prove to be of value.

In conclusion, it is hoped that the information gained from this study and the above suggestions for future research will stimulate the design and implementation of increasingly effective treatment programmes for what, unfortunately, still remains the "single largest preventable cause of premature death in the United Kingdom" (R.C.P., 1983).

To quote from Frederiksen and Simon (1979):

> "Smoking is a complex behaviour likely to be maintained by a variety of factors..... Our models of smoking are increasingly comprehensive and realistic. Investigators are starting to break with old habits with regard to treatment goals and measurement procedures. We are also finally starting to pay more than lip service to the concepts of comprehensive treatment and long-term maintenance. With the vigorous and systematic pursuit of these trends, continued progress in the control of smoking behaviour is achievable"(p.541).

It is believed that this study has represented such "continued progress" and that a further step has been taken towards the prevention of avoidable severe illness and premature death.

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APPENDIX I

a) The smoking questionnaire

RM A:

me:.....

ldress:.....

.....

ate of Birth:..... Age:..... Sex: M/F.

ccupation.....

lease answer all the following questions by putting a tick in the appropriate box:-

arital Status:

- Single.
- Married and living with spouse.
- Separated or divorced.
- Widowed.

1. Are you:-

- In paid employment?
- A housewife?
- Retired?
- Permanently sick or disabled?
- Unemployed due to temporary illness?
- Unemployed due to other circumstances?

2. Which of the sections below best describes your job (or describes your last job if you are not working now):-

- Semi-skilled or unskilled work.
- Clerical, secretarial.
- Foreman, supervisor.
- Professional, managerial or administrative.
- Skilled work, craftsman.
- Never had paid employment.

If in paid employment

3 Are you allowed to smoke during working hours?

- Yes whenever I want to
- Yes, but only during breaks.
- No, not at all.

4. Do you suffer from any of the following, (you may tick more than one box if appropriate)?

- Chronic bronchitis.
- Heart disease.
- Stomach ulcer.
- High blood pressure.
- Coughing - with sputum.
- Coughing - without sputum.
- Pain in limbs during/after exercise.
- Breathlessness.
- Chest pain during/after exercise.
- Chest pain at rest.
- Palpitations.
- Tremulousness, shaking of hands.
- Indigestion.

WOMEN:

(i) Do you have any children?

Yes. ☐
No. ☐

(ii) If YES, were any of your children born prematurely?

Yes. ☐
No. ☐

(iii) If YES,

1 - 2 weeks premature. ☐
3 - 4 weeks premature. ☐
5 - 6 weeks premature ☐
More than 6 weeks premature. ☐

(iv) Have you had a low birth weight baby?

Yes. ☐
No. ☐

(v) If YES, weight(s) at birth: 1.....
2.....
3.....

ALL PATIENTS:

Have there been any of the following major changes in your life in the past year? (You may tick more than one box if appropriate).

Getting married. ☐
Separation/divorce. ☐
Significant difficulties within marriage. ☐
Moving house. ☐
New family member, (birth, adoption, etc.) ☐
Son/daughter marrying and/or leaving home. ☐
Changing own job. ☐
Unemployment/redundancy. ☐
Retirement. ☐
Personal illness or injury requiring hospital treatment (including out-patient visits). ☐
Surgical operation. ☐
Death or serious illness in close member of family. ☐
Menopause. ☐
Significant change in financial situation, (e.g. increase or decrease in income). ☐
Taking out a major loan (e.g. mortgage) ☐

At what age did you start smoking regularly (once a day or more)?

.....

How much were you smoking each day approximately one year after you began smoking?

Pipe: .....ounces.
Cigars ..... (number)
Cigarettes ..... (number)

What is the greatest amount you have ever smoked daily?

Pipe .....ounces.
Cigars .....(number)
Cigarettes.....(number)

How much do you usually smoke in a day?

Pipe .....ounces.
Cigars .....(number)
Cigarettes.....(number)

How much money do you spend on cigarettes/tobacco in one week?

Less than £1. ☐
Between £1 and £3. ☐
Between £3 and £5 ☐
Between £5 and £7. ☐
More than £7. ☐

CIGARETTE SMOKERS ONLY:

(i) What type of cigarettes do you usually smoke?

Filter tipped. ☐
Plain. ☐
Hand-rolled. ☐

(ii) What is the FULL BRAND NAME, (e.g. Embassy, Extra Mild), of your usual cigarette?

.....

(iii) Is it:-

King size? ☐
Regular Size? ☐
Small Size? ☐

(iv) Is it:

Low tar? ☐
Low to middle tar? ☐
Middle tar? ☐
Middle to high tar? ☐
High tar? ☐
Don't know. ☐

ALL PATIENTS:

When you smoke do you inhale:-

Very much? ☐
A fair amount? ☐
Just a little? ☐
Not at all? ☐

How much of a cigarette or cigar do you usually leave?

Very little. ☐
A quarter. ☐
A half. ☐
Three quarters. ☐

continued.....

Is the person who is most important to you a smoker?

Yes. ☐
No. ☐

if YES, how much per day does s/he smoke?

Pipe ..... ounces.
Cigars ..... (number)
Cigarettes ..... (number).

Did any of the following members of your family smoke when you lived in your parents' home? (You may tick more than one box if appropriate).

Father. ☐
Mother. ☐
Brother(s). ☐
Sister(s). ☐
Not applicable. ☐

About how many times have you tried seriously to stop smoking in the past?

Never. ☐
Once. ☐
2 - 4 times. ☐
5 times or more. ☐

Not counting times when you were ill or in hospital, what is the longest time you have ever gone without smoking in the past five years?

Less than 1 day. ☐
1 - 6 days. ☐
1 - 4 weeks. ☐
1 - 3 months. ☐
4 - 11 months. ☐
1 - 3 years. ☐
More than 3 years. ☐

The last time you stopped smoking for more than one week, how long did you stay off cigarettes?

2 - 4 weeks. ☐
1 - 3 months. ☐
4 - 11 months. ☐
1 - 3 years. ☐
More than 3 years. ☐
Not applicable. ☐

How did you stop? Describe. .....
.....

What major difficulties did you find in stopping? .....
.....

continued.....

Have you ever tried to CUT DOWN the number of cigarettes you smoke?

Yes. ☐
No. ☐

If YES, and you succeeded in maintaining your reduction for longer than three months, how much did you reduce by?

By one quarter. ☐
By one half. ☐
By three quarters. ☐

In the past, have you put on weight as a result of stopping or cutting down smoking?

None. ☐
A small amount. ☐
Quite a lot. ☐
Very much. ☐

In the past year:-

(i) has the amount of tobacco/cigarettes you smoke changed significantly, (i.e. by at least one quarter)?

Increased. ☐
Decreased. ☐
No change. ☐

(ii) have you increased or decreased the amount of alcohol you drink, (by at least one quarter)?

Increased. ☐
Decreased. ☐
No change. ☐

(iii) have you attempted to change your eating habits?

To lose weight. ☐
To gain weight. ☐
No change. ☐

you for completing this questionnaire. Could you now please check that you answered all the questions appropriate to you and that you have not missed any.

APPENDIX I (contin.)

b) Demographic information

Below is presented the information obtained from an analysis of respondents' questionnaire answers. Three hundred and ten smokers completed questionnaires, but, occasionally, some items were omitted. The number of individuals completing each item is stated in parentheses. The presentation of this information follows the format of the questionnaire.

Age	Mean 39.99 years. (SD 10.24 years) (N = 310)
Sex	60% Female (N = 186) 40% Male (N = 124)
Mean Female Age	38.81 years (SD 10.13 years) (N = 186)
Mean Male Age	41.77 years (SD 10.20 years) (N = 124)

Marital Status

Single	: 9.7% (N = 30)	(N = 310)
Married and living with spouse	: 79.9% (N = 247)	
Separated or divorced	: 7.8% (N = 24)	
Widowed	: 2.6% (N = 8)	

Q1 Employment

In paid employment	: 85.8% (N = 266)	(N = 310)
A housewife	: 8.1% (N = 25)	
Retired	: 1.0% (N = 3)	
Permanently sick/ disabled	: 1.6% (N = 5)	
Unemployed due to temporary illness	: 0.6% (N = 2)	
Unemployed due to other circumstances	: 2.9% (N = 9)	

Q2. Type of employment

Semiskilled/unskilled	: 24.1%	(N = 74)	(N = 307)
Clerical/secretarial	: 18.6%	(N = 57)	
Foreman/supervisor	: 9.1%	(N = 28)	
Professional/Managerial/ Administrative	: 36.8%	(N = 113)	
Skilled work/craftsman	: 11.1%	(N = 34)	
Never had paid employment	: 0.3%	(N = 1)	

Q3. Allowed to smoke at work

Yes, whenever I want to	: 69.3%	(N = 190)	(N = 274)
Only during breaks	: 30.7%	(N = 84)	
Not at all	: 0	0	

Q4. Physical symptoms

Chronic bronchitis	: 9.0%	(N = 28)
Heart disease	: 2.3%	(N = 7)
Stomach ulcer	: 3.2%	(N = 10)
High blood pressure	: 4.2%	(N = 13)
Coughing - with sputum	: 33.9%	(N = 105)
without sputum	: 21.6%	(N = 67)
Pain in limbs during/after exercise	: 23.5%	(N = 73)
Breathlessness	: 49.4%	(N = 153)
Chest pain during/after exercise	: 13.5%	(N= 42)
Chest pain at rest	: 10.3%	(N = 32)
Palpitations	: 19.4%	(N = 60)

Tremulousness/shaking of hands : 11.3% (N = 35)

Indigestion : 25.5% (N = 79)

(All respondents answered this section (N = 310). 88.4% of respondents (N = 274) reported the occurrence of one or more of the symptoms listed).

Q5. Motherhood

86% (N = 160) of the females responding were mothers.

20% (N = 32) of these women had had premature babies.

Of these 32,
- 1-2 weeks premature 31% (N = 10)
- 3-4 weeks premature 44% (N = 14)
- 5-6 weeks premature 12.5% (N = 4)
- 6+ weeks premature 12.5% (N = 4)

Q7. Onset of smoking

Mean age 16.96 years (N = 308)

Q8. Smoking rate after one year

Mean rate 9.4 cigarettes per day (N = 298)

Q9. Maximum rate

Mean maximum rate 37.78 cigarettes per day (N = 307)

Q10. Mean self-reported rate

26.5 cigarettes per day (N = 301).

Q12. Type of cigarette

Filter tipped	:	93.06% (N = 282)	(N = 303)
Plain	:	6.27% (N = 19)	
Hand-rolled	:	0.66% (N = 2)	
King size	:	61.05% (N = 185)	(N = 303)
Regular size	:	25.41% (N = 77)	
Small size	:	13.53% (N = 41)	
Low tar	:	34.32% (N = 104)	(N = 303)
Low to Middle tar	:	7.92% (N = 24)	
Middle tar	:	51.81% (N = 157)	
Middle to high tar	:	3.30% (N = 10)	
High tar	:	0.99% (N = 3)	
Don't know	:	1.65% (N = 5)	

Q13. Inhaling

Very much	:	53.46% (N = 166)	(N = 310)
A fair amount	:	41.91% (N = 130)	
Just a little	:	4.3% (N = 13)	
Not at all	:	0.33% (N = 1)	

Q14. Amount of cigarette left

Very little	:	82.77% (N = 255)	(N = 308)
A quarter	:	16.87% (N = 52)	
A half	:	0.33% (N = 1)	
Three quarters	:	0 0	

Q15. Is the person most important to you a smoker?

Yes	:	53.08% (N = 163)	(N = 307)
No	:	46.90% (N = 144)	

Q16. Other smokers in family (when lived at home)

a) Father only	:	29.05% (N = 86)	
b) Mother only	:	5.06% (N = 15)	
c) Brother(s) only	:	3.04% (N = 9)	
d) Sister(s) only	:	1.35% (N = 4)	
e) N/A	:	5.06% (N = 15)	

a) & b)	:	15.20%	(N = 45)
a) & c)	:	9.79%	(N = 29)
a) & d)	:	6.75%	(N = 20)
a) & b) & c)	:	5.74%	(N = 17)
a) & b) & c) & d)	:	5.06%	(N = 15)
a) & c) & d)	:	5.40%	(N = 16)
b) & c)	:	0.67%	(N = 2)
c) & d)	:	1.01%	(N = 3)
a) & b) & d)	:	4.72%	(N = 14)
b) & d)	:	1.68%	(N = 5)
b) & c) & d)	:	0.33%	(N = 1)

(Total number responding to this item : 296)

Q17. Number of attempts to stop smoking

Never	:	12.57%	(N = 39)	(N = 310)
Once	:	19.99%	(N = 62)	
2-4 times	:	52.89%	(N = 164)	
5+ times	:	14.51%	(N = 45)	

Q18. Longest period without smoking (last five years)

1 day	:	26.38%	(N = 81)	(N = 307)
1-6 days	:	26.70%	(N = 82)	
1-4 weeks	:	15.30%	(N = 47)	
1-3 months	:	15.63%	(N = 48)	
4-11 months	:	11.72%	(N = 36)	
1-3 years	:	3.58%	(N = 11)	
3 years +	:	0.65%	(N = 2)	

Q19. How long off cigarettes (last time stopped)?

2-4 weeks	:	33.63%	(N = 75)	(N = 223)
1-3 months	:	23.31%	(N = 52)	
4-11 months	:	10.76%	(N = 24)	
1-3 years	:	4.93%	(N = 11)	
3 years +	:	2.69%	(N = 6)	
N/A	:	24.66%	(N = 55)	

Q22. Ever tried to reduce

Yes	:	86.10%	(N = 267)	(N = 310)
No	:	13.90%	(N = 43)	

Q23. Weight increase as a result of reducing or stopping smoking

None	:	48.09% (N = 127)	(N = 264)
A small amount	:	29.15% (N = 77)	
Quite a lot	:	15.90% (N = 42)	
Very much	:	6.81% (N = 18)	

APPENDIX II

Assessment and Treatment Schedule

SESSION NO.	ASSESSMENT	TREATMENT
AI (Pre-treatment assessment)	Explanation of rationale and commitment to treatment. Signing of therapeutic contract. EPQ/SCL-90/16PF Questionnaires. SCN level. Respiratory functioning (FEV/FVC/TF) Weight Expectancy rating Drawing up of hierarchy and 'covert statements'. Begin 10 day baseline record. (see card for variables measured)	-
TI a) Individual	Benefit rating (weekly) Individual method rating of benefit and degree of use.	Inform others of intent to stop/control smoking. Relaxation/Covert sensitization and focussed relaxation training.
TI b) Group	-	First hierarchy item eliminated. First phase of deprived response performance. First phase of self-punishment. Coverant control introduced.
TII a) Individual	Benefit rating (weekly). Individual method rating of benefit and degree of use.	Relaxation/Covert Sensitisation and focussed relaxation training.
TII b) Group	-	Second hierarchy item eliminated. Second phase of DRP Second phase of self-punishment.

SESSION NO.	ASSESSMENT	TREATMENT
TIII a) Individual	Benefit rating (weekly). Individual method rating of benefit and degree of use.	Relaxation/Covert Sensitisation and focussed relaxation training
b) Group	Give 10 days' record cards. (see card for variables to be measured)	Third item of hierarchy eliminated. Third phase of DRP Third phase of self-punishment.
AII (Mid-treatment assessment)	EPQ/SCL-90/16PF. SCN^{-} level. Respiratory functioning Weight Overall Benefit Rating Expectancy rating.	-
TIV a) Individual	Benefit rating (weekly) Individual method of rating benefit and degree of use.	Relaxation/Covert Sensitisation and focussed relaxation training.
b) Group	-	Fourth item of hierarchy eliminated. Fourth phase of DRP Fourth phase of self-punishment.
TV a) Individual	Benefit rating (weekly) Individual method rating of benefit and degree of use.	Relaxation/Covert Sensitisation and focussed relaxation training.
b) Group	-	Fifth item of hierarchy eliminated. Fifth phase of DRP Fifth phase of self-punishment.

SESSION NO.	ASSESSMENT	TREATMENT
TVI a) Individual	Benefit rating (weekly) Individual method rating of benefit and degree of use.	Relaxation/Covert Sensitisation and focussed relaxation training.
b) Group	Give 10 days' record cards. (see card for variables to be measured).	Instructions to utilize all methods in order to now adhere to target rate of smoking.
AIII (Post-treatment assessment)	EPQ/SCL-90/16PF SCN level. Weight. Overall benefit rating. Respiratory functioning	-
AIV (3 months F.U.)	As for post-treatment assessment	-
AV (6 months F.U.)	As for post-treatment assessment	-
AVI (12 months F.U.)	As for post-treatment assessment	-

APPENDIX III

Materials used in the Study

(i) Smoking questionnaire
(ii) Motivation questionnaire
(iii) Daily record card
(iv) "About your treatment"
(v) "The Treatment Programme"
(vi) The Contract
(vii) "Self-Control Techniques"
(viii) "Direct Training Techniques"
(ix) Focussed Relaxation and Covert Sensitization guide
(x) Response Probability Hierarchy
(xi) Coverant Control Statements
(xii) Expectancy Rating
(xiii) Weekly Benefit Rating
(xiv) Overall Benefit Rating
(xv) Degree of use ratings
(xvi) Degree of Benefit Ratings
(xvii) Weekly instructions

ORM A:

ame:.....

ddress:.....

.....

ate of Birth:..... Age:..... Sex: M/F.

ccupation.....

lease answer all the following questions by putting a tick in the appropriate box:-

arital Status:
- Single. ☐
- Married and living with spouse. ☐
- Separated or divorced. ☐
- Widowed. ☐

Are you:-
- In paid employment? ☐
- A housewife? ☐
- Retired? ☐
- Permanently sick or disabled? ☐
- Unemployed due to temporary illness? ☐
- Unemployed due to other circumstances? ☐

Which of the sections below best describes your job (or describes your last job if you are not working now):-
- Semi-skilled or unskilled work. ☐
- Clerical, secretarial. ☐
- Foreman, supervisor. ☐
- Professional, managerial or administrative. ☐
- Skilled work, craftsman. ☐
- Never had paid employment. ☐

If in paid employment

Are you allowed to smoke during working hours?
- Yes whenever I want to ☐
- Yes, but only during breaks. ☐
- No, not at all. ☐

Do you suffer from any of the following, (you may tick more than one box if appropriate)?
- Chronic bronchitis. ☐
- Heart disease. ☐
- Stomach ulcer. ☐
- High blood pressure. ☐
- Coughing - with sputum. ☐
- Coughing - without sputum. ☐
- Pain in limbs during/after exercise. ☐
- Breathlessness. ☐
- Chest pain during/after exercise. ☐
- Chest pain at rest. ☐
- Palpitations. ☐
- Tremulousness, shaking of hands. ☐
- Indigestion. ☐

WOMEN:

(i) Do you have any children?

Yes. ☐
No. ☐

(ii) If YES, were any of your children born prematurely?

Yes. ☐
No. ☐

(iii) If YES,

1 - 2 weeks premature. ☐
3 - 4 weeks premature. ☐
5 - 6 weeks premature ☐
More than 6 weeks premature. ☐

(iv) Have you had a low birth weight baby?

Yes. ☐
No. ☐

(v) If YES, weight(s) at birth: 1.....
2.....
3.....

ALL PATIENTS:

Have there been any of the following major changes in your life in the past year? (You may tick more than one box if appropriate).

Getting married. ☐
Separation/divorce. ☐
Significant difficulties within marriage. ☐
Moving house. ☐
New family member, (birth, adoption, etc.) ☐
Son/daughter marrying and/or leaving home. ☐
Changing own job. ☐
Unemployment/redundancy. ☐
Retirement. ☐
Personal illness or injury requiring hospital treatment (including out-patient visits). ☐
Surgical operation. ☐
Death or serious illness in close member of family. ☐
Menopause. ☐
Significant change in financial situation, (e.g. increase or decrease in income). ☐
Taking out a major loan (e.g. mortgage) ☐

At what age did you start smoking regularly (once a day or more)?

.....

How much were you smoking each day approximately one year after you began smoking?

Pipe: .....ounces.
Cigars ..... (number)
Cigarettes ..... (number)

continued.....

What is the greatest amount you have ever smoked daily?

Pipe .....ounces.
Cigars .....(number)
Cigarettes.....(number)

How much do you usually smoke in a day?

Pipe .....ounces.
Cigars .....(number)
Cigarettes.....(number)

How much money do you spend on cigarettes/tobacco in one week?

Less than £1. ☐
Between £1 and £3. ☐
Between £3 and £5 ☐
Between £5 and £7. ☐
More than £7. ☐

CIGARETTE SMOKERS ONLY:

(i) What type of cigarettes do you usually smoke?

Filter tipped. ☐
Plain. ☐
Hand-rolled. ☐

(ii) What is the FULL BRAND NAME, (e.g. Embassy, Extra Mild), of your usual cigarette?

.....

(iii) Is it:-

King size? ☐
Regular Size? ☐
Small Size? ☐

(iv) Is it:

Low tar? ☐
Low to middle tar? ☐
Middle tar? ☐
Middle to high tar? ☐
High tar? ☐
Don't know. ☐

ALL PATIENTS:

When you smoke do you inhale:-

Very much? ☐
A fair amount? ☐
Just a little? ☐
Not at all? ☐

How much of a cigarette or cigar do you usually leave?

Very little. ☐
A quarter. ☐
A half. ☐
Three quarters. ☐

continued.....

15. Is the person who is most important to you a smoker?

Yes. ☐
No. ☐

if YES, how much per day does s/he smoke?

Pipe ..... ounces.
Cigars ..... (number)
Cigarettes ..... (number).

16. Did any of the following members of your family smoke when you lived in your parents' home? (You may tick more than one box if appropriate).

Father. ☐
Mother. ☐
Brother(s). ☐
Sister(s). ☐
Not applicable. ☐

17. About how many times have you tried seriously to stop smoking in the past?

Never. ☐
Once. ☐
2 - 4 times. ☐
5 times or more. ☐

18. Not counting times when you were ill or in hospital, what is the longest time you have ever gone without smoking in the past five years?

Less than 1 day. ☐
1 - 6 days. ☐
1 - 4 weeks. ☐
1 - 3 months. ☐
4 - 11 months. ☐
1 - 3 years. ☐
More than 3 years. ☐

19. The last time you stopped smoking for more than one week, how long did you stay off cigarettes?

2 - 4 weeks. ☐
1 - 3 months. ☐
4 - 11 months. ☐
1 - 3 years. ☐
More than 3 years. ☐
Not applicable. ☐

20. How did you stop? Describe. .....
.....

21. What major difficulties did you find in stopping? .....
.....

continued.....

22. Have you ever tried to CUT DOWN the number of cigarettes you smoke?

Yes. ☐
No. ☐

If YES, and you succeeded in maintaining your reduction for longer than three months, how much did you reduce by?

By one quarter. ☐
By one half. ☐
By three quarters. ☐

23. In the past, have you put on weight as a result of stopping or cutting down smoking?

None. ☐
A small amount. ☐
Quite a lot. ☐
Very much. ☐

24. In the past year:-

(i) has the amount of tobacco/cigarettes you smoke changed significantly, (i.e. by at least one quarter)?

Increased. ☐
Decreased. ☐
No change. ☐

(ii) have you increased or decreased the amount of alcohol you drink, (by at least one quarter)?

Increased. ☐
Decreased. ☐
No change. ☐

(iii) have you attempted to change your eating habits?

To lose weight. ☐
To gain weight. ☐
No change. ☐

Thank you for completing this questionnaire. Could you now please check that you have answered all the questions appropriate to you and that you have not missed any.

FORM B:

Name:.....

Date:.....

Listed below are various examples of the sort of thing smokers sometimes say about their smoking. We would like to know how much you agree or disagree with the statements; there are no right or wrong answers to please answer honestly.

Please put a tick in one of the four columns after each statement.

	Not at all how I feel	Only slightly how I feel	Quite like how I feel	Very much how I feel
1. I am frightened about what smoking may be doing to me.				
2. Even if I stopped smoking for a while, I am sure that other people would persuade me to start again.				
3. I resent other people telling me that I should not smoke.				
4. I do not think I am really prepared to give up smoking if it proves too difficult or distressing.				
5. I have never made a serious effort to give up smoking completely.				
6. If life was easier, I would have less need to smoke.				
7. I feel I am constantly being 'got at' nowadays because I am a smoker.				
8. I know that some people die because they smoke but I think most smokers stay just healthy as non-smokers.				
9. I would like to give up smoking if I could do so easily.				
10. If I really wanted to I could give up smoking.				
11. I am not going to be able to give up smoking unless someone helps me.				
12. I think you have to smoke a lot more than I do to put your health at serious risk.				

continued .....

FORM B: —continuation— 1

	Not at all how I feel	Only slightly how I feel	Quite like how I feel	Very much how I feel
13. I would feel very ashamed of myself if I tried to give up smoking and failed.				
14. If I give up smoking I would expect to feel a lot healthier than I do now.				
15. I find smoking helps me cope when I have problems.				
16. I think of my smoking as a sickness which needs to be cured.				
17. I think that the government should do more to persuade people not to smoke				
18. What I feel I really need is a pill or some sort of medicine that will stop me wanting to smoke.				
19. I feel that other people are partly to blame for the fact that I became a smoker.				
20. I really want to stop smoking but I need somebody to tell me how to do it.				
21. I am aware that smoke has unpleasant effects on non-smoking friends.				
22. It is a good idea to divide public places, such as cinemas, into smoking and non-smoking areas.				
23. I am aware that my non-smoking friends disapprove of me smoking.				
24. Smoking makes my clothes and hair smell unpleasantly.				
25. I do not want to give up smoking if it means I will put on more than one stone in weight.				

Please answer the following questions by putting a tick in the appropriate box:-

26. How enjoyable is smoking for you?

Very enjoyable ____
Mildly enjoyable ____
No strong feelings ____
Mildly distasteful ____
Very distasteful ____

FORM B: —continuation—
2

27. How unpleasant do you find it if you cannot smoke for an hour or two?

Very unpleasant ☐
Mildly unpleasant ☐
No strong feelings ☐
Mildly pleasant ☐
Very pleasant ☐

28. Do you think you are addicted to smoking?

Extremely ☐
Fairly ☐
Slightly ☐
Not at all ☐
Do not know ☐

29. How important is it to you to stop smoking?

Extremely important ☐
Very important ☐
Fairly important ☐
Slightly important ☐
Not at all important ☐
Do not know ☐

30. List in order of importance any of the following reasons for giving up smoking which apply to you (including any other reasons you may have specified in the blank spaces provided). Place the number 1 in the box next to the most important reason, number 2 in the box next to the second most important reason et cetera.

Health ☐
Women: pregnancy ☐
It is expensive ☐
It is a dirty habit ☐
It is not fair on non-smokers ☐
I do not like being addicted ☐
Some other reason(s) (please specify):
.....
.....
.....

31. Do you have any concerns or worries about your health while you continue to smoke?

YES/NO

If YES, please list, in order of importance, your greatest concern(s):-

Greatest concern .....
2nd greatest concern .....
3rd greatest concern .....
4th greatest concern .....

continuod .....

FORM B: -continuation-
3

32. Listed below are some possible consequences of smoking and stopping smoking. Please say how important each of the consequences are to you by ticking one box for each consequence.

How important to you is it:-	Not important to me	Quite important to me	Extremely important to me
- to have enough spending money			
- not to get lung cancer			
- not to get short of breath			
- Not to annoy people who do not smoke			
- to live to a ripe old age			
- not to get bronchitis			
- to feel you have control over yourself			
- to set a good example to children			
- not to get heart disease			
- to be able to calm down when you are tense or angry			
- not to cough a lot in the mornings			
- not to have your hair and clothes smell of smoke			
- not to have stained teeth or fingers			

* **Triggering Stimuli** fall into two categories - (a) Internal Stimuli or (b) External Stimuli.

(a) Internal Stimuli. These stimuli will include thoughts such as "Ah! It would be nice to have a cigarette" or a physical craving.
(b) External Stimuli. These stimuli will include items such as seeing some-one else smoking, being offered a cigarette, reading an advertisement, a particular place, (e.g. a pub, armchair in the lounge etc.), having a drink, etc. Try to determine what triggered off your smoking behaviour. Was it an internal or external stimulus? Put a tick in the appropriate column (INT or EXT) and specify the stimulus, e.g. a strong craving, a particular thought, being offered a cigarette, etc.

** **Intensity of Craving.** Code:-

1 = No craving at all.
2 = Slight craving.
3 = Moderate craving.
4 = Strong craving.
5 = Extreme craving.

*** **Mood.** Code:-

ANN = Annoyed.
ANX = Anxious.
BOR = Bored.
IRR = Irritable..
MIS = Miserable..
PLE = Pleasant mood.
TEN = Tense.
TIR = Tired.

**** **Anxiety Level.** Code:-

1 = Totally free from anxiety.
2 = Slightly anxious.
3 = Moderately anxious.
4 = Strongly anxious.
5 = Extremely anxious.

Research No.

RESEARCH INTO THE CONTROL OF SMOKING

DAILY RECORD CARD

Name .....

Day .....

Date .....

INSTRUCTIONS TO CLIENTS

1. Please keep details on the record card of each cigarette smoked.
2. Complete the record card BEFORE the cigarette is smoked.
3. Use a new record card for each day.
4. Go on to a second record card if more than 25 cigarettes are smoked each day.
5. Bring your record cards with you to each treatment session.

DAILY RECORD

No.	Time	Brand	? Triggering Stimuli			Place	Activity	? Alone Yes/No	Intensity of craving **	Mood ***	Anxiety Level ****
			INT.	EXT.	Specify *						
1											
2											
3											
4											
5											
6											
7											
8											
9											
10											
11											
12											
13											
14											
15											
16											
17											
18											
19											
20											
21											
22											
23											
24											
25											

SMOKING RESEARCH PROJECT: About your treatment.

The treatment which you will be receiving over the coming weeks is designed to help you to give up smoking, or to reduce your smoking to a minimal level, depending on which goal you have set for yourself.

Although the methods which you will be asked to use have been found to be effective in helping smokers, it must be emphasized that they will do just that, and only that, - i.e. help you to control your smoking. The main aspect of treatment is your determination to alter your smoking behaviour and, if you are determined to be successful, there is a high likelihood that you will be.

It is of vital importance that the instructions which you are given during the course of treatment are adhered to as closely as possible. This adherence, combined with good motivation on your part, will, once again, ensure a good chance of your achieving your desired goal without undue discomfort.

It is appreciated that you may feel the assessment sessions to be rather lengthy and vigorous but you are assured that this vigour is absolutely necessary if you are to obtain maximum benefit from treatment. Your patience in this respect will be appreciated.

Finally, it must be strongly emphasized that the end of the six weeks treatment programme does not signify the end of the need to continue applying the methods you will be using. Your therapist will wish to see you, periodically, for up to a year following treatment and these methods may have to be continually applied during part, or all, of this period.

Work hard at your treatment and you will reach your goal!

MOKING RESEARCH PROJECT: The Treatment Programme.

After your initial assessment sessions, which will include measurements of our lung-functioning, your weight and levels of harmful substances in your blood, n addition to the completion of several questionnaires, you will be asked to keep n exact record of the number of cigarettes smoked over a minimum of ten days. It s important at this point not to try to alter the number of cigarettes you smoke ut simply to continue as usual.

Following this "baseline" period your twice-weekly treatment sessions will egin. From the very start of treatment you will be requested to reduce your evel of smoking as much as you possibly can; although the methods you will be sing as part of treatment will help you to reach your desired goal, the main ffect will come from your determination to control your impulses to smoke.

You will have been given the choice of aiming for a 75% reduction in your moking level, (i.e. learning to control your smoking efficiently), or giving up moking completely, (i.e. a 100% reduction). It is hoped that, by the end of the ifth week of treatment you will have reached your goal. However, if you have ot done so you will be instructed, at this point, to begin to stick to your chosen evel of smoking and to continue to use the techniques which you will have learned, o maintain this level.

You will be seen on several occasions, for assessment, after the termination of treatment by your therapist, and you will be encouraged, when seen, to continue orking at overcoming your problem, if you have not already done so.

SMOKING RESEARCH PROJECT: Contract.

In view of the cost of this research project in terms of the finances of Rochdale Area Health Authority and of the therapist's time, only those individuals who are willing to abide by the following conditions will be included in the treatment programme.

(a) Unless unavoidable, due to illness or other extreme circumstances, subjects will attend all assessment and treatment sessions. If any appointments do have to be missed, additional ones will be arranged. There will be a total of twelve treatment sessions, (six during the daytime and six during the evening), and a total of twelve assessment sessions. Assessment will take place prior to treatment, half-way through treatment, at the end of treatment, three months after treatment, six months after treatment and one year after treatment.

(b) Subjects must carry out, to the best of their ability, the instructions given during treatment. Subjects are assured that, if the therapist's instructions are followed as closely as possible, the probability of their participation in treatment being successful will be maximized.

(c) Separate instruction sheets will be given to you each week. These, too, must be followed to the best of your ability.

I wish to participate in the SMOKING RESEARCH treatment programme and agree to abide by the above conditions.

Signed.....

Date.....

Witnessed.....

SMOKING RESEARCH PROJECT. "Self-Control" Techniques.

The majority of the techniques you will be using during, and following, your treatment programme are known as "Self-Control" techniques. They are so-called because they enable you to control and modify your own behaviour, (in this case, smoking behaviour), by manipulating your environment in certain ways and by making certain consequences contingent upon, (i.e. depend upon), smoking and "non-smoking" behaviour.

Below is a list of the methods you will be asked to use - these will be discussed and explained in far greater detail, of course, during your treatment sessions.

(1) Hierarchical Reduction:

During your initial assessment sessions you will have been asked to draw up a list of five situations in which there exists a strong likelihood of your smoking a cigarette. As treatment progresses, from week to week, you will be asked to refrain from smoking in these particular situations, in a systematic way. More specifically, during the first week of treatment you will not smoke in the situation you have rated first on your list, during the second week you will not smoke, additionally, in your second most likely situation, and so on until, by the final session, you will have ceased smoking in all of your "high likelihood" situations. Thus, by this means, the association which you have built up over a long period between smoking and certain situations will be weakened. This will enable you to gain increasing control of your smoking behaviour.

(2) Deprived Response Performance.

The use of this method involves making smoking systematically less enjoyable by having your surroundings become increasingly less pleasant.

continued.....

SMOKING RESEARCH PROJECT: "Self-Control" Techniques.

(2) Deprived Response Performance. (continued)

During the first week of treatment you will be prohibited from smoking whilst performing pleasurable activities such as watching T.V., reading, listening to the radio or to records, et cetera.

During the second week you will be, additionally, prohibited from smoking in the company of other people.

During the third week, smoking will be further restricted to one room of your house (or work).

During the fourth week of treatment smoking may occur only in the lavatory, (both at work and at home), and, during the fifth week, only whilst standing up in the lavatory.

It should be obvious, here, that, eventually, smoking will afford you such little pleasure that you will really wonder whether its worthwhile!

(3) Self-Punishment/Monetary Deprivation

This method is based on the principle that, the higher the "cost" of smoking a cigarette, the less likely you will be to smoke.

Thus, during the first week of treatment, each cigarette you smoke will cost you 1p. During the second week, each will cost you 3p; during the third week, 5p; during the fourth, 7p and during the final week, 10p.

The money forfeited in this way will be sent to a charity of your choice at the end of each week. (It is suggested that you consider the Cancer Research Fund as the charity to which your money is sent).

continued.....

SMOKING RESEARCH PROJECT "Self-Control" Techniques

(4) Social Contracting

At the commencement of treatment you will be required to inform your relatives, friends and work-colleagues of your intention to give up/reduce smoking (whichever applied in your particular case).

(5) Coverant Control

During the initial assessment you will have been asked to draw up two separate lists of five statements, one list being "anti-smoking" statements and the other being "pro-no-smoking" statements.

Each time you feel the desire for a cigarette, during and after treatment, you will be required to rehearse (to yourself) and think carefully about, a pair of these statements. If you then successfully avoid having a cigarette you are to make a "rewarding" statement to yourself. This method will be explained more fully, and in more detail, during your treatment sessions.

KING RESEARCH PROJECT. Direct Training Techniques.

- Daytime Sessions.

In addition to the self-control techniques listed on your handout, you will o be trained, by your therapist, in two methods which have been found to be cessful in helping people to give up/limit their smoking. These methods are cribed briefly below and will be explained more fully during your actual atment.

(a) Focussed Relaxation Training.

You will be asked, at the beginning of treatment, to describe the physical feelings you experience when "craving" for a cigarette. You will then be trained how to totally relax yourself, physically, with special emphasis being placed on those areas of your body where the craving sensations are felt. You will be asked to practise this method at home, daily, until you are able to alleviate the craving sensations.

(b) Covert Sensitization.

This method will be used in combination with your "Focussed Relaxation" technique and you will also be asked to practise this daily, at home.

The aim of the method is to encourage you to associate the thought of smoking, (and smoking itself), with unpleasant physical sensations. This will be done, in practical terms, by your being trained by your therapist to clearly visualize (imagine) "smoking situations" and to imagine, at the same time, certain unpleasant events occurring.

Conversely, you will be trained to associate the avoidance of smoking with the relief from unpleasant thoughts and with more relaxing physical sensations.

Once again, this method will, of course, be explained to you in far greater detail during treatment.

SMOKING RESEARCH PROJECT.

DAYTIME INDIVIDUAL TREATMENT SESSIONS - FOCUSSED RELAXATION TRAINING AND COVERT SENSITIZATION.

A. RELAXATION PHASE:

(i) Active relaxation.

(a) Breathe in deeply, tensing the LEFT HAND: breathe out, relax (x 5).
(b) " " " " " RIGHT HAND: " " " "
(c) " " " " " LEFT ARM: " " " "
(d) " " " " " RIGHT ARM: " " " "
(e) " " " " " LEFT LEG: " " " "
(f) " " " " " RIGHT LEG: " " " "
(g) " " " " " LOWER BACK & SEAT: " " " "
(h) " " " " " STOMACH: " " " "
(i) " " " " " SHOULDERS & UPPER BACK: " " " "
(j) " " " " " NECK & THROAT: " " " "
(k) " " " " " FACE & SCALP: " " " "
(l) " " " " " WHOLE BODY: " " " "

(ii) Passive relaxation.

(2 minutes) (a) Concentration on general feelings of warmth, heaviness and relaxation.

(b) Concentration on specific, (chest, throat and stomach), feelings of warmth and relaxation. Imagine feelings of cigarette - craving, then relax these areas, noticing that craving disappears. (x 3).

B. COVERT SENSITIZATION PHASE:

(1) Approach (x 2).

Visualize comfortable chair, at home.

Feel desire for cigarette (craving sensations).

Cigarette packet and matches/lighter are on the floor next to chair.

(i) Reach for pack .....mild nausea.

(ii) Take out cigarette .....nausea increases, mouth becomes dry.

(iii) Put cigarette in mouth...nausea increases further, dry mouth, bad taste.

(iv) Light cigarette and inhale .....intense nausea, dry mouth, very unpleasant taste.

WEEK ONE:

(v) Inhale ..... feel like heaving (vizualise in detail)

WEEK TWO:

(vi) Inhale ..... retch (vizualize in detail).

WEEK THREE:

(vii) Inhale ..... vomit on self and on cigarettes (vizualise in detail).

WEEKS FOUR, FIVE AND SIX.

(2) Avoidance (x 2)

(i) Put cigarettes out in ashtray (feelings begin to subside).

(ii) Put cigarettes and matches/lighter away (subside further).

(iii) Concentrate on feelings of relaxation and comfort.

(iv) Walk out of room to bathroom (vizualise and feel relaxed).

(v) Drink of water / undress, wash if necessary.

(vi) Concentrate on pleasant, relaxed sensations.

SMOKING RESEARCH PROJECT: Response Probability Hierarchy.

Name.....

Please list below five situations in which you are likely to have a cigarette.

When you have done this please number these situations according to their likelihood to be associated with smoking. (No. 1 to be the most likely situation, No. 2 to be the next most likely, et cetera).

	SITUATION	NO.
a		
b		
c		
d		
e		

MOKING RESEARCH PROJECT. Coverant Control Statements.

Name.....

lease enter in the table below five reasons why you wish to stop/reduce smoking, i.e. why you feel it is bad to continue smoking as you do).

1	
2	
3	
4	
5	

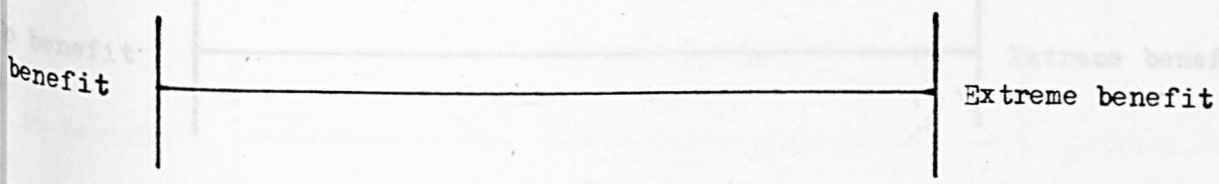
Please enter in the table below five advantages of not smoking.

1	
2	
3	
4	
5	

SMOKING RESEARCH PROJECT. Expectancy Rating.

Name.....

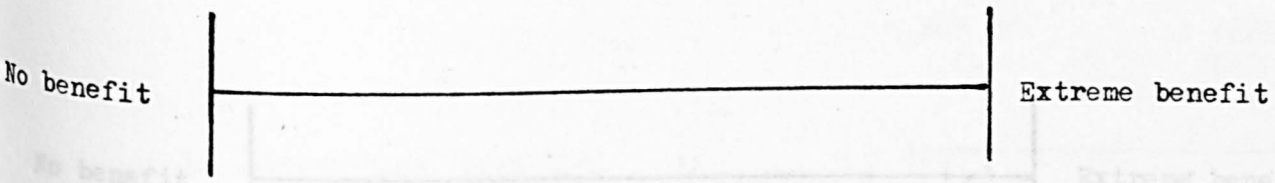
Please mark, with a vertical line, on the scale below, the amount of benefit you expect to derive from treatment.



SMOKING RESEARCH PROJECT. Weekly Benefit Rating.

Name.....

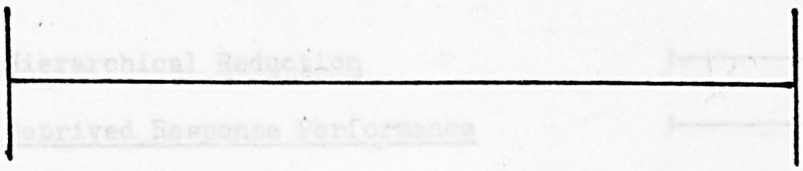
Please mark, with a vertical line, on the scale below, the amount of benefit you have derived over the past week, from treatment.



SMOKING RESEARCH PROJECT. Overall Benefit Rating.

Name.....

Please mark, with a vertical line, on the scale below, the amount of benefit you have derived from the treatment programme to date.

No benefit  Extreme benefit

SMOKING RESEARCH PROJECT

Name .....

Date .....

Please mark on the scales below the degree to which you have used each particular method over the last week.

	NOT AT ALL	A GREAT DEAL
1. Hierarchical Reduction	⊢——————⊣	
2. Deprived Response Performance	⊢——————⊣	
3. Monetary Deprivation	⊢——————⊣	
4. Coverant Control	⊢——————⊣	
5. Covert Sensitization	⊢——————⊣	
6. Focussed Relaxation Training	⊢——————⊣	

SMOKING RESEARCH PROJECT

Name .....

Date .....

Please mark on the scales below the amount of benefit which you have derived from each particular method over the last week.

1. Hierarchical Reduction ——————————
2. Deprived Response Performance ——————————
3. Monetary Deprivation ——————————
4. Coverant Control ——————————
5. Covert Sensitization ——————————
6. Focussed Relaxation Training ——————————

Smoking Research

Treatment week one

1) Deprived response performance

No smoking whilst watching T.V., reading, listening to the radio or records.

2) Monetary Deprivation

1p per cigarette to be forfeited to a charity of your choice at the end of the week.

3) Eliminate the first item on your hierarchy

4) Coverant Control

Use pairs of your statements when you desire a cigarette. If the temptation to smoke is resisted congratulate yourself on your success.

5) Inform others of your intention to stop smoking

SMOKING RESEARCH

Treatment week two

1) Deprived response performance

No smoking whilst watching T.V., reading, listening to the radio or records. No smoking in the company of other people.

2) Monetary deprivation

3p per cigarette to be forfeited to a charity of your choice at the end of the week.

3) Eliminate the second item on your hierarchy

4) Continue to use your pairs of 'coverant control' statements

Smoking Research Treatment Programme

Treatment Week Three

1) Deprived response performance

No smoking whilst watching T.V., reading, listening to the radio or records.

No smoking in the company of other people.

Smoking only allowed in one room at home or at work.

2) Monetary deprivation

5p per cigarette to be forfeited to a charity of your choice at the end of the week.

3) Eliminate the third item on your hierarchy

4) Continue to use your pairs of 'coverant control' statements

Smoking Research Treatment Programme

Treatment week four

1) Deprived response performance

No smoking whilst watching T.V., reading, listening to the radio or records.

No smoking in the company of other people.

Smoking only allowed in the lavatory (at work or at home)

2) Monetary Deprivation

7p per cigarette to be forfeited to a charity of your choice at the end of the week.

3) Eliminate the fourth item on your hierarchy

4) Continue to use your pairs of coverant control statements

Smoking Research Treatment Programme

Treatment week five

1) Deprived response performance

No smoking whilst watching T.V., reading, listening to the radio or records.

No smoking in the company of other people.

Smoking only allowed whilst STANDING in the lavatory at work or at home.

2) Monetary deprivation

10p per cigarette to be forfeited to a charity of your choice at the end of the week.

3) Eliminate the fifth item on your hierarchy

4) Continue to use your pairs of "coverant control" statements

APPENDIX IV

Transcript of Focussed Relaxation and Covert Sensitization tape

APPENDIX IV

Transcript of Focussed Relaxation and Covert Sensitisation Tape.

(i) Active relaxation
(ii) Passive relaxation
(iii) Covert sensitisation

(i) Just lie back, close your eyes and relax as well as you can do. Now I want you to spend a few seconds getting into a nice steady breathing rhythm. Breathe nice and deeply and when you breathe in really fill your lungs up and when you breathe out really empty them. Next time you breathe in I want you to clench your left fist as tightly as you can. Really clench it, and as you breathe out let your fist relax and breathe in and clench it and breathe out and relax. Breathe in, tense it up and relax. Again breathe in and out and once more with your fist breathe in and now relax it. And now I want you to move on to doing exactly the same thing with your right fist. So in your own time breathe in and tense up and breathe out and relax. Now once more with your fist and now relax yourself completely. And now I want you to carry on doing the same thing and this time tense up your left arm as you breathe in and as you breathe out let it relax completely. Breathe in and tense it up and relax. Breathe in again, really tense it up - the more you tense it up the better and breathe out and relax. And again and relax. Once more with that arm and relax. Now do exactly the same with your right arm. Now let your arms relax completely. Carry on breathing nice and deeply, carry on lying there with your eyes closed and notice that your whole body is starting to relax. And now I want you to do exactly the same thing with your left leg, as you breathe in really tense your left leg up and as you breathe out let it relax completely. Breathe in and make your whole leg go rigid and tense and breathe out and relax. In ... and relax. Tense ... and relax. Once more with that leg, breathe in and really tense it up and now relax. And now move on to doing exactly the same with your right leg. Breathe in, tense it up and breathe out and relax. Breathe in and tense and relax. And again ... relax. Tense ... and relax. And one more time with that leg breathe in and now let it relax completely. Lie there for a few seconds and notice now that your arms and legs are feeling really heavy and relaxed and perhaps a little bit warm and tingly. And now I want

you to concentrate on the muscles in the lower part of your back and around your bottom and as you breathe in I want you to tense those muscles up as tightly as you can and as you breathe out I want you to relax them. Breathe in, and tense ...and relax. Tense ... and relax. In ... tense ... out ... and relax. Once more with those muscles, breathe in and now let them relax completely. And now we will move on to your stomach muscles. As you breathe in I want you to make your stomach muscles as hard as you can and as you breathe out really let them go soft and relax. Breathe in, tense them up and breathe out and relax. Breathe in ... and out so that the muscles in your stomach are really soft and relaxed. Breathe in again ... and breathe out and let them go soft. Notice how soft they are becoming each time you breathe out. One more time with those muscles - breathe in and then breathe out and let them relax completely. Now lie there and take two or three nice easy deep breaths and notice how relaxed the whole of your body is becoming. And now I would like you to concentrate on the muscles in your shoulders and your chest and the top part of your back: the whole of the top of your torso. And again as you breathe in I would like you to tense all those muscles by shrugging your shoulders and making your back go tight and as you breathe out let them relax. Breathe in again, tense them up - really screw the muscles up and breathe out, let your shoulders fall back. Breathe in ... tense, and out, letting the muscles go soft and heavy. Breathe in again and now really let them relax this time. Let you whole body relax and sink back into the bed. Now breathe nice and evenly and deeply for a few seconds. In a few seconds we will move on to the muscles in your neck and your throat and once again, in a few seconds, as you breathe out I want you to tense those muscles up. Really let the muscles in your neck and throat go hard and tense and as you breathe out completely relax. So breathe in ... tense those muscles up and breathe out and relax them. Breathe in ... tense and breathe out and relax. In ... and out. One more time breathe in and now relax them and let your head loll back on its own. Notice how heavy your head feels and just let yourself relax as much as you possibly can do. And now I want you to move on to the muscles in your face

and your scalp. And in a few seconds I will ask you to tense those muscles up as you breathe in and relax them as you breathe out and when you do so I want you to screw your eyes up, screw the muscles in your mouth up and press your tongue against the roof of your mouth, wrinkle your forehead. Try and tense all the muscles in your facial area up as you breathe in and as you breathe out let them all relax. Breathe in ... tense them up and breathe out and relax them. And breathe in ... breathe out and relax. In ... and tense and out and relax. Again breathe in and out and relax. Once more with those muscles breathe in and this time really let your face muscles go completely relaxed as you breathe out. Now just lie there for a few seconds and notice how relaxed the whole of your body is becoming. And now I would like you to think about all the muscles in the whole of your body - arms and legs, shoulders, stomach, face - all the muscles you have already concentrated on and this time as you breathe in I want you to make all those muscles tense up. Make the whole of your body go tense and rigid and as you breathe out let your whole body relax completely and let it sink back into the bed. So breathe in ... tense all those muscles up and breathe out and relax completely. Breathe in ... tense up, screw all the muscles up and as you breathe out let yourself sink back into the bed. Again breathe in ... and out. Breathe in, and tense up as hard as you can and breathe out, and relax. Now one last time, breathe in deeply, screw all the muscles up and this time as you breathe out let your whole body relax totally. Breathe out, let all the muscles relax completely. Now just lie back and think about the sensations in your body and notice how relaxed the whole of your body feels. Your arms and legs should feel nice and warm and heavy and your breathing should be nice and even and deep and regular. Notice how relaxed and calm and comfortable you feel. You are so relaxed that you could quite easily drift off to sleep. You are completely relaxed and all your muscles feel soft and heavy.

(ii) And now I want you to concentrate on the feelings that you have in the muscles in your stomach and chest and throat. Notice that at the moment you feel nice and soft and relaxed. I want you to lie back now with

your eyes closed and try and imagine that you really really want a cigarette. Imagine that you really desire a cigarette - it's a long time since you had a smoke. Try and imagine that you haven't had a cigarette all day long and that you really really crave for a cigarette. And notice that as you start to get the feelings of wanting a cigarette coming on, the muscles in your chest and stomach and throat start to tense up a little bit. Notice that the feelings of craving for a cigarette are associated with tension in the muscles in your stomach and chest and throat and that the stronger the feelings of craving you get, the more tension arises in those muscles. And now when you reach the stage that you can imagine really desiring a cigarette I want you to deliberately concentrate on relaxing those muscles. Breathe nice and deeply - take two or three really deep breaths and relax the muscles in your stomach and your chest and in your throat. Make the muscles go soft and warm and heavy. And notice that if you relax those muscles, the feelings of craving gradually start to diminish and die away. Relax the muscles even further. Breathe nice and deeply and let your whole body relax. Notice how soft your stomach muscles are becoming and the muscles in your chest and shoulders and the muscles in your throat. And notice that as those muscles do become soft that the feelings of craving for a cigarette die away more and more until you have controlled those feelings completely. And now that those muscles are relaxed I want you to bring about the same feelings again. Imagine again that those feelings of craving for a cigarette are building up. Imagine that its hours and hours since you last had a smoke and you really would like a cigarette. Try and imagine that you have just had a great big meal and try and recall the feelings you get of wanting a cigarette after a big meal. Notice that as those feelings start to come on again the muscles in your stomach and chest and shoulders and throat start to become a little bit more tense. Perhaps a feeling of tightness starts to come into your stomach as the feelings of craving for a cigarette get stronger and stronger. And now relax those muscles ... breathe nice and deeply. Take two or three really deep breaths and notice that as you breathe out each time those muscles relax and become

heavy and warm and soft. And as they do so the feelings of craving and desiring a cigarette again gradually become less and less until eventually you are going to be able to relax those muscles so much, they are going to become so warm and soft that the feelings of wanting a cigarette are totally controlled. Just lie there for a few seconds and notice how relaxed and comfortable the whole of your body feels.(iii) Now I want you to imagine as clearly and vividly as you can sitting in a nice comfortable chair at home. Try and imagine that you are sitting in your most comfortable, favourite chair at home. Lie there with your eyes closed and really try and visualise that very clearly. Now I want you to imagine that you really fancy a cigarette and craving sensations are in your stomach, chest and throat and you really would like a cigarette and try and picture on the floor next to the chair your packet of cigarettes and a box of matches and an ashtray. Try and visualise that scene as clearly and as vividly as you can. Now imagine reaching down for the packet of cigarettes and as you reach down and pick up the packet of cigarettes a feeling of queasiness, nausea and sickness suddenly comes across your stomach. As you reach down and get hold of the cigarettes your stomach seems to turn over and a wave of sickness comes across you. Now I want you to imagine taking a cigarette out of the packet and as you pull the cigarette out of the packet this feeling of queasiness and sickness in your stomach becomes stronger, just as though you have had far too much to drink or something really bad to eat and your stomach starts to feel really unpleasant and unsettled. And at the same time notice that your mouth has started to become dry and it is becoming difficult to swallow. Now imagine as vividly and clearly as you can putting a cigarette into your mouth. Put the cigarette to your lips (it is not lit yet) and as you do so the feeling of nausea and sickness suddenly starts to become very strong, just as though there is a heavy, cold weight in your stomach. Your mouth becomes drier and it starts to become really difficult to swallow and your tongue is sticking to the roof of your mouth and a very unpleasant taste is starting to develop in your mouth. A very unpleasant, sour, bitter taste. Next I want you to imagine that you

are lighting the cigarette. You light the cigarette and inhale and as the smoke goes down into your chest and some of it gets into your stomach a feeling of very intense nausea, a very strong feeling of sickness comes across you. Your stomach feels really unpleasant and disturbed, your mouth is extremely dry, it is almost impossible to swallow and you have a very, very bad bitter unpleasant taste in your mouth. You really do feel terrible but nevertheless you carry on. Now you take another drag on the cigarette, inhaling again and this time as the smoke goes down you find yourself on the verge of actually being sick. You feel your stomach muscles contract as though your stomach wants to expel all its contents. Your stomach feels really bad and uncomfortable. You feel very, very sick, the room is spinning around a little bit, you come out in a cold sweat and this unpleasant taste in your mouth is very very strong now and it is being made far worse by the smoke that you are inhaling. And now once again you put the cigarette in your mouth and you inhale. Try and imagine the smoke going down into your lungs, some of it getting into your stomach and as it does so you can actually feel your stomach muscles contract again and you actually wretch. You can feel bile coming into the back of your throat. You are right on the verge of being sick. Your whole body is incredibly tense, your forehead is covered in sweat and you feel really, really ill. Just try to imagine the bitter taste of the bile in the back of your throat mixing with the tobacco smoke. Your mouth is really dry, its almost impossible to swallow and you really do feel very ill. Now imagine inhaling once more on the cigarette, this time as you inhale and the smoke goes into your chest, lungs and stomach you actually vomit. Try and imagine as vividly and clearly as you can sitting in your chair, tasting the smoke in your mouth and actually vomiting. Imagine yourself looking down and seeing your clothes covered in vomit and seeing your hands covered in vomit. Imagine the smell of it, the terrible taste of it in the back of your throat and the back of your nose. Your eyes are watering and your nose is running and you feel really really ill. All you can taste in your mouth is the bitter taste of cigarettes and smoke and the taste of vomit. And now imagine

leaning forward and stubbing the cigarette out, the cigarette that's wet and damp, pushing the ashtray away from you and immediately you do that you start to feel better. Imagine lying back in the chair and closing your eyes. You have got rid of the cigarette and you close your eyes and you start to relax. Your stomach starts to settle down and you stand up and walk out of the smoky room and walk towards the bathroom. Imagine yourself walking into the bathroom and taking off your vomit-drenched clothes and having a really nice warm wash, washing all the sickness away, washing the smell away. Try and imagine yourself actually going through those motions and imagine yourself standing there in the bathroom feeling completely refreshed, feeling very well, having a drink, washing the bad taste away completely. And imagine thinking to yourself "How could I possible have sat there and smoked half a cigarette when that was the effect it had on me". Now we are going to run through the very same procedure again and this time really try and visualise it as clearly as you possibly can. The more clearly you can picture yourself going through these various stages, the more this is going to help you to stop smoking. You will start to find the idea of smoking quite repulsive. So imagine yourself sitting in your chair at home. Imagine yourself reaching down, picking your cigarette packet up and as you lift the cigarette packet up that now familiar feeling of queasiness and dizziness comes over you and your stomach starts to feel a little bit tender and unsettled. Imagine taking a cigarette out of the packet. Picture yourself taking the cigarette out of the packet and as you do so that feeling of queasiness in your stomach becomes a little bit stronger and your mouth starts to get dry. As you are lying there now try and imagine your mouth becoming dry. Imagine your tongue starting to stick to the roof of your mouth. Imagine your throat becoming dry. Picture yourself sitting in your chair at home, putting the cigarette in your mouth and as you do so your mouth becomes drier still and a feeling of tension starts to grip your whole body and your stomach starts to feel definitely unpleasant and unsettled. Now imagine striking a match and holding the match to the cigarette and inhaling. As the smoke goes down, a wave of nausea, a wave of sickness, quite suddenly comes over you. The smoke tastes

really vile and you really do feel like putting the cigarette out, but you don't do. You inhale on the cigarette again and this time as the smoke goes down your stomach starts to contract and you feel like heaving up and you start to feel quite dizzy and light-headed. You feel very unpleasantly light-headed but still you carry on and you inhale again and this time it makes you wretch. You can actually feel the contents of your stomach start to move up towards your mouth and you have a real job holding it down. The smoke is trickling out of your nose and you breathe it out as quickly as you can because its having such a terrible effect on you. It tastes really really vile, very bitter, very acrid and your mouth is very dry and your stomach really does feel very bad. But still you make yourself carry on and you put the cigarette to your mouth and you make yourself inhale again and breathe the smoke down into your lungs and immediately you have finished breathing the smoke in you suddenly vomit - all the contents of your stomach fly out of your mouth. Imagine yourself sitting there in the chair, vomit spewing out and spilling out all over your clothes, all over your hands, all over the cigarette that you are holding in your hand. Try and imagine the horrible sticky wet feeling of it and the smell of it as you are sitting there, combining with the cigarette smoke; and you just go on being sick. Each time you breathe in and smell the cigarette smoke in the air and on your hands, all around you, it makes your stomach feel worse and worse and you really do feel terrible. Now imagine yourself stubbing the cigarette out and throwing it down and closing your eyes and immediately you have got rid of the cigarette you start to feel better again. Imagine standing up and walking out of the room into the fresh air where there is no tobacco smoke. Imagine walking to the bathroom. Imagine going into the bathroom and stripping your vomit-laden clothes off and having a nice warm wash, cleaning your teeth and getting rid of the taste of tobacco smoke and imagine having a nice long cool drink and standing there in the bathroom feeling clean and fresh and healthy. Your stomach now feels completely settled because you have escaped from the room that is full of tobacco smoke and you are away from your cigarettes and you stand there and wonder why you had a cigarette

when you knew it was going to make you feel so bad; and the last thing you want to do is to go back into that room and pick a cigarette up and start to smoke it because it would have exactly the same effect on you again. You are quite happy going completely without a cigarette. And now just lie there for a few seconds and concentrate on feelings of total relaxation. Really let yourself relax. Let your whole body go heavy and let all your muscles go soft and just lie there and think that you don't really need a cigarette. You could quite easily go for as long as you want to without one.

APPENDIX V

The Motivation Questionnaire

A copy of the Motivation Questionnaire is included in Appendix III, (materials used in the study). On the following copy, the weights attached to each alternative response have been inserted, showing how the total "Motivation Score" was computed. The score range is 42-158.

FORM B:

Name:.....

Date:.....

Listed below are various examples of the sort of thing smokers sometimes say about their smoking. We'd like to know how much you agree or disagree with the statements: there are no right or wrong answers so please answer honestly.

Please put a tick in one of the four columns after each statement.

	Not at all how I feel.	Only slightly how I feel.	Quite like how I feel.	Very much how I feel.
1. I'm frightened about what smoking may be doing to me.	1	2	3	4
2. Even if I stopped smoking for a while, I'm sure that other people would persuade me to start again.	4	3	2	1
3. I resent other people telling me that I shouldn't smoke.	1	2	3	4
4. I don't think I'm really prepared to give up smoking if it proves too difficult or distressing.	4	3	2	1
5. I've never made a serious effort to give up smoking completely.	4	3	2	1
6. If life was easier, I'd have less need to smoke.	4	3	2	1
7. I feel I'm constantly being 'got at' nowadays because I'm a smoker.	1	2	3	4
8. I know that some people die because they smoke, but I think most smokers stay just as healthy as non-smokers.	4	3	2	1
9. I'd like to give up smoking if I could do so easily.	1	2	3	4
10. If I really wanted to, I could give up smoking.	1	2	3	4
11. I'm not going to be able to give up smoking unless someone helps me.	4	3	2	1
12. I think you have to smoke a lot more than I do to put your health at serious risk.	4	3	2	1
13. I'd feel very ashamed of myself if I tried to give up smoking and failed.	1	2	3	4

continued.....

	Not at all how I feel.	Only slightly how I feel.	Quite like how I feel.	Very much how I feel
14. If I give up smoking, I'd expect to feel a lot healthier than I do now.	1	2	3	4
15. I find smoking helps me cope when I've got problems.	4	3	2	1
16. I think of my smoking as a sickness which needs to be cured.	1	2	3	4
17. I think that the government should do more to persuade people not to smoke.	1	2	3	4
18. What I feel I really need is a pill or some sort of medicine that'll stop me wanting to smoke.	1	2	3	4
19. I feel that other people are partly to blame for the fact that I became a smoker.	4	3	2	1
20. I really want to stop smoking, but I need somebody to tell me how to do it.	1	2	3	4
21. I am aware that smoke has unpleasant effects on non-smoking friends.	1	2	3	4
22. It is a good idea to divide public places, such as cinemas, into smoking and non-smoking areas.	1	2	3	4
23. I am aware that my non-smoking friends disapprove of me smoking.	1	2	3	4
24. Smoking makes my clothes and hair smell unpleasantly.	1	2	3	4
25. I do not want to give up smoking if it means I will put on more than 1 stone in weight.	4	3	2	1

Please answer the following questions by putting a tick in the appropriate box:-

26. How enjoyable is smoking for you?

Very enjoyable.	1
Mildly enjoyable.	2
No strong feelings.	3
Mildly distasteful.	4
Very distasteful.	5

continued.....

27. How unpleasant do you find it if you can't smoke for an hour or two?

Very unpleasant.	1
Mildly unpleasant.	2
No strong feelings.	3
Mildly pleasant.	4
Very pleasant.	5

28. Do you think you are addicted to smoking?

Extremely.	1
Fairly.	2
Slightly.	3
Not at all.	4
Don't know.	/

29. How important is it to you to stop smoking?

Extremely important.	5
Very important.	4
Fairly important.	3
Slightly important.	2
Not at all important.	1
Don't know.	/

30. List in order of importance any of the following reasons for giving up smoking which apply to you, (including any other reasons you may have specified in the blank spaces provided). Place the number 1 in the box next to the most important reason; No. 2 in the box next to the second most important reason, et cetera.

Health.	
Women: pregnancy.	
Its expensive.	
Its a dirty habit.	
It isn't fair on non-smokers.	
I don't like being addicted.	

Some other reason(s) (please specify):
.....
.....
.....

31. Do you have any concerns or worries about your health while you continue to smoke. YES/NO.

If "YES" please list, in order of importance, your greatest concern(s):-

Greatest concern.....

2nd greatest concern.....

3rd greatest concern.....

4th greatest concern.....

continued.....

32. Listed below are some possible consequences of smoking and stopping smoking. Please say how important each of the consequences are to you by ticking one box for each consequence.

How important to you is it:-	Not important to me.	Quite important to me.	Extremely important to me.
- to have enough spending money?	1	2	3
- not to get lung cancer?	1	2	3
- not to become short of breath?	1	2	3
- not to annoy people who don't smoke?	1	2	3
- to live to a ripe old age?	1	2	3
- not to get bronchitis?	1	2	3
- to feel you have control over yourself?	1	2	3
- to set a good example to children?	1	2	3
- not to get heart disease?	1	2	3
- to be able to calm down when you're tense or angry?	1	2	3
- not to cough a lot in the mornings?	1	2	3
- not to have your hair and clothes smell of smoke?	1	2	3
- Not to have stained teeth or fingers?	1	2	3

APPENDIX VI

ANOVA Summary Tables

(In cases where Analysis of Covariance was employed, dummy covariates being used to represent missing values (see p.161), the degrees of freedom (d.f.) were adjusted accordingly.

As "Sum of Squares" is the product of mean square and degrees of freedom SoS are omitted from the Tables, for the sake of conciseness.

Figures are rounded to two decimal places).

Source	d.f.	M.S.	F.	Sig.
Between groups	3	51.68	0.73	N.S.
Error	44	71.10		

ANOVA Table 1

Pretreatment smoking rate

Source	d.f.	M.S.	F.	Sig.
Between Groups	3	2052.87	0.09	N.S.
Error	44	21623.05		

ANOVA Table 2

Pretreatment tar intake

Source	d.f.	M.S.	F.	Sig.
Between groups	3	74.30	0.57	N.S.
Error	44	130.64		

ANOVA Table 3

Pretreatment nicotine intake

Source	d.f.	M.S.	F.	Sig.
Between groups	7	10333.62	2.45	$p < 0.05$
Error	40	4211.92		

ANOVA Table 4

Pretreatment anxiety ratings

Source	d.f.	M.S.	F.	Sig.
Between groups	7	4749.08	1.02	N.S.
Error	40	4662.76		

ANOVA Table 5

Pretreatment craving intensity

Source	d.f.	M.S.	F.	Sig.
Between Groups	7	279.32	0.50	N.S.
Error	40	561.22		

ANOVA Table 6

Pretreatment internal/external smoking

Source	d.f.	M.S.	F.	Sig.
Between groups	7	3.43	1.13	N.S.
Error	40	3.04		

ANOVA Table 7

Expectancy ratings

Source	d.f.	M.S.	F.	Sig.
Between groups	7	179.34	1.46	N.S.
Error	40	123.13		

ANOVA Table 8

Motivation scores

Source	d.f.	M.S.	F.	Sig.
Between groups	7	992.04	0.65	N.S.
Error	40	1521.36		

ANOVA Table 9

Pretreatment SCN^-

Source	d.f.	M.S.	F.	Sig.
Between Groups	7	17397.23	1.88	N.S.
Error	40	9244.07		

ANOVA Table 10

Pretreatment gross body-weight

Source	d.f.	M.S.	F.	Sig.
Between groups	7	236.95	1.05	N.S.
Error	40	225.76		

ANOVA Table 11

Pretreatment FEV_1

Source	d.f.	M.S.	F.	Sig.
Between groups	7	201.07	0.79	N.S.
Error	40	254.34		

ANOVA Table 12

Pretreatment FVC

Source	d.f.	M.S.	F.	Sig.
Between groups	7	98.19	0.91	N.S.
Error	40	107.58		

ANOVA Table 13

Pretreatment FEV/FVC

Source	d.f.	M.S.	F.	Sig.
Between Groups	7	186.95	0.87	N.S.
Error	40	215.98		

ANOVA Table 14

Pretreatment Co Transfer Factor

Source	d.f.	M.S.	F.	Sig.
Between groups	7	2.14	0.51	N.S.
Error	40	4.20		

ANOVA Table 15

Pretreatment EPQ(P)

Source	d.f.	M.S.	F.	Sig.
Between groups	7	20.71	0.81	N.S.
Error	40	25.62		

ANOVA Table 16

Pretreatment EPQ(E)

Source	d.f.	M.S.	F.	Sig.
Between groups	7	25.31	0.99	N.S.
Error	40	25.60		

ANOVA Table 17

Pretreatment EPQ(N)

Source	d.f.	M.S.	F.	Sig.
Between Groups	7	17.66	1.25	N.S.
Error	40	14.13		

ANOVA Table 18

Pretreatment EPQ(L)

Source	d.f.	M.S.	F.	Sig.
Between groups	7	193.16	0.90	N.S.
Error	40	215.82		

ANOVA Table 19

Pretreatment SCL-90 (Somatic Anxiety)

Source	d.f.	M.S.	F.	Sig.
Between groups	7	97.69	0.68	N.S.
Error	40	143.43		

ANOVA Table 20

Pretreatment SCL-90 (Obs.-Compulsiveness)

Source	d.f.	M.S.	F.	Sig.
Between groups	7	60.05	0.48	N.S.
Error	40	125.69		

ANOVA Table 21

Pretreatment SCL-90 (Interpers. sensitivity)

Source	d.f.	M.S.	F.	Sig.
Between Groups	7	170.26	1.69	N.S.
Error	40	100.53		

ANOVA Table 22

Pretreatment SCL-90 (Depression)

Source	d.f.	M.S.	F.	Sig.
Between groups	7	98.80	0.58	N.S.
Error	40	171.46		

ANOVA Table 23

Pretreatment SCL-90 (Anxiety)

Source	d.f.	M.S.	F.	Sig.
Between groups	7	275.61	1.58	N.S.
Error	40	174.77		

ANOVA Table 24

Pretreatment SCL-90 (Hostility)

Source	d.f.	M.S.	F.	Sig.
Between groups	7	154.74	0.22	N.S.
Error	40	711.90		

ANOVA Table 25

Pretreatment SCL-90 (Phobic Anxiety)

Source	d.f.	M.S.	F.	Sig.
Between Groups	7	439.57	1.02	N.S.
Error	40	429.17		

ANOVA Table 26

Pretreatment SCL-90 (Paranoid Ideation)

Source	d.f.	M.S.	F.	Sig.
Between groups	7	280.19	0.81	N.S.
Error	40	344.52		

ANOVA Table 27

Pretreatment SCL-90 (Psychoticism)

Source	d.f.	M.S.	F.	Sig.
Between groups	7	101.47	1.42	N.S.
Error	40	71.28		

ANOVA Table 28

Pretreatment SCL-90 (GSI)

Source	d.f.	M.S.	F.	Sig.
Between groups	7	128.67	1.85	N.S.
Error	40	69.61		

ANOVA Table 29

Pretreatment SCL-90 (PSDL)

Source	d.f.	M.S.	F.	Sig.
Between Groups	7	82.37	0.85	N.S.
Error	40	96.43		

ANOVA Table 30

Pretreatment SCL-90 (PST)

Source	d.f.	M.S.	F.	Sig.
Between groups	7	6.14	1.61	N.S.
Error	40	3.83		

ANOVA Table 31

Pretreatment 16PF (A)

Source	d.f.	M.S.	F.	Sig.
Between groups	7	2.26	0.82	N.S.
Error	40	2.75		

ANOVA Table 32

Pretreatment 16PF (B)

Source	d.f.	M.S.	F.	Sig.
Between groups	7	9.99	2.79	$p < 0.025$
Error	40	3.58		

ANOVA Table 33

Pretreatment 16PF (C)

Source	d.f.	M.S.	F.	Sig.
Between Groups	7	3.90	1.03	N.S.
Error	40	3.78		

ANOVA Table 34

Pretreatment 16PF (E)

Source	d.f.	M.S.	F.	Sig.
Between groups	7	3.04	0.84	N.S.
Error	40	3.60		

ANOVA Table 35

Pretreatment 16PF (F)

Source	d.f.	M.S.	F.	Sig.
Between groups	7	3.19	0.86	N.S.
Error	40	3.70		

ANOVA Table 36

Pretreatment 16PF (G)

Source	d.f.	M.S.	F.	Sig.
Between groups	7	3.47	0.79	N.S.
Error	40	4.40		

ANOVA Table37

Pretreatment 16PF (H)

Source	d.f.	M.S.	F.	Sig.
Between Groups	7	3.95	1.11	N.S.
Error	40	3.55		

ANOVA Table 38

Pretreatment 16PF (I)

Source	d.f.	M.S.	F.	Sig.
Between groups	7	6.23	1.36	N.S.
Error	40	4.59		

ANOVA Table 39

Pretreatment 16PF (L)

Source	d.f.	M.S.	F.	Sig.
Between groups	7	2.43	0.77	N.S.
Error	40	3.14		

ANOVA Table 40

Pretreatment 16PF (M)

Source	d.f.	M.S.	F.	Sig.
Between groups	7	0.89	0.48	N.S.
Error	40	1.84		

ANOVA Table 41

Pretreatment 16PF (N)

Source	d.f.	M.S.	F.	Sig.
Between Groups	7	1.09	0.22	N.S.
Error	40	4.95		

ANOVA Table 42

Pretreatment 16PF (O)

Source	d.f.	M.S.	F.	Sig.
Between groups	7	5.07	1.50	N.S.
Error	40	3.37		

ANOVA Table 43

Pretreatment 16PF (Q_1)

Source	d.f.	M.S.	F.	Sig.
Between groups	7	7.23	2.23	N.S.
Error	40	3.23		

ANOVA Table 44

Pretreatment 16PF (Q_2)

Source	d.f.	M.S.	F.	Sig.
Between groups	7	4.38	1.08	N.S.
Error	40	4.05		

ANOVA Table 45

Pretreatment 16PF (Q_3)

Source	d.f.	M.S.	F.	Sig.
Between Groups	7	8.33	1.72	N.S.
Error	40	4.84		

ANOVA Table 46

Pretreatment 16PF (Q_4)

Source	d.f.	M.S.	F.	Sig.
Between groups	7	64.12	0.58	N.S.
Error	40	110.23		

ANOVA Table 47

Age

Source	d.f.	M.S.	F.	Sig.
groups	7	4969.66	1.08	N.S.
Error	28	4614.16		
Assessments	5	22455.35	39.85	$p < 0.001$
Assessments x groups	35	675.33	1.20	N.S.
Error	144	563.46		

ANOVA Table 48

Smoking rate - Analysis at one-year follow-up

Source	d.f.	M.S.	F.	Sig.
Groups	3	1.11	2.05	N.S.
Assessments	5	6.10	11.30	$p < 0.001$
Error	15	0.54		

ANOVA Table 49

Abstinence rates - Analysis at one-year follow-up

Source	d.f.	M.S.	F.	Sig.
Groups	3	1.38	2.42	N.S.
Assessments	5	1.44	2.53	N.S.
Error	15	0.57		

ANOVA Table 50

Successful reduction rates - Analysis at one-year follow-up

Source	d.f.	M.S.	F.	Sig.
Groups	7	141893.94	1.65	N.S.
Error	28	85752.30		
Assessments	5	203985.38	30.96	$p < 0.001$
Assessments x groups	35	7982.94	1.21	N.S.
Error	144	6587.93		

ANOVA Table 51

Tar intake - Analysis at one-year follow-up

Source	d.f.	M.S.	F.	Sig.
Groups	7	1104.01	1.74	N.S.
Error	28	634.98		
Assessments	5	1462.32	29.70	$p < 0.001$
Assessments x groups	35	52.08	1.06	N.S.
Error	144	49.24		

ANOVA Table 52

Nicotine intake - Analysis at one-year follow-up

Source	d.f.	M.S.	F.	Sig.
Groups	7	41.59	1.33	N.S.
Error	22	31.16		
Assessments	5	215.40	56.41	$p < 0.001$
Assessments x groups	35	3.59	0.94	N.S.
Error	127	3.82		

ANOVA Table 53

Overall Benefit Ratings - Analysis at one-year follow-up

Source	d.f.	M.S.	F.	Sig.
Groups	7	23012.85	0.49	N.S.
Error	5	47116.84		
Assessments	5	11366.10	5.71	$p < 0.001$
Assessments x groups	35	3109.89	1.56	N.S.
Error	73	1991.82		

ANOVA Table 54

Anxiety ratings - Analysis at one-year follow-up

Source	d.f.	M.S.	F.	Sig.
Groups	7	42786.32	1.40	N.S.
Error	5	30651;18		
Assessments	5	4324.02	2.71	$p < 0.05$
Assessments x groups	35	1830.81	1.15	N.S.
Error	73	1529.91		

ANOVA Table55

Craving intensity - Analysis at one-year follow-up

Source	d.f.	M.S.	F.	Sig.
Groups	7	4864.37	1.00	N.S.
Error	6	4866.44		
Assessments	5	304.27	1.96	N.S.
Assessments x groups	35	153.62	0.99	N.S.
Error	78	154.91		

ANOVA Table 56

Internal/External smoking - Analysis at one-year follow-up

Source	d.f.	M.S.	F.	Sig.
Groups	7	6845.93	2.13	N.S.
Error	20	3208.70		
Assessments	4	8269.63	15.03	$p < 0.001$
Assessments x groups	28	632.91	1.15	N.S.
Error	89	550.16		

ANOVA Table 57(a)

SCN^- levels - Analysis at six-month follow-up

Source	d.f.	M.S.	F.	Sig.
Groups	6	9021.93	2.34	N.S.
Error	20	3858.55		
Assessments	5	6044.53	9.10	$p < 0.001$
Assessments x groups	30	636.36	0.96	N.S.
Error	112	664.21		

ANOVA Table 57(b)

SCN^- levels - Analysis at six-month follow-up

Source	d.f.	M.S.	F.	Sig.
Groups	7	92369.62	1.29	N.S.
Error	24	71470.60		
Assessments	5	1610.20	6.77	$p < 0.001$
Assessments x groups	35	156.25	0.66	N.S.
Error	135	237.99		

ANOVA Table 58

Gross Body-Weight - Analysis at one-year follow-up

Source	d.f.	M.S.	F.	Sig.
Groups	7	518.74	0.49	N.S.
Error	22	1066.41		
Assessments	3	33.99	1.30	N.S.
Assessments x groups	21	18.71	0.72	N.S.
Error	68	26.07		

ANOVA Table 59(a)

FEV_1 - Analysis at three-month follow-up

Source	d.f.	M.S.	F.	Sig.
Groups	6	715.38	0.46	N.S.
Error	21	1546.76		
Assessments	5	62.04	1.80	N.S.
Assessments x groups	30	29.92	0.87	N.S.
Error	109	34.50		

ANOVA Table 59 (b)

FEV_1 - Analysis at one-year follow-up

Source	d.f.	M.S.	F.	Sig.
Groups	7	670.09	0.66	N.S.
Error	22	1016.40		
Assessments	3	16.81	0.92	N.S.
Assessments x groups	21	16.39	0.90	N.S.
Error	68	18.31		

ANOVA Table 60 (a)

FVC - Analysis at three-month follow-up

Source	d.f.	M.S.	F.	Sig.
Groups	6	1000.64	0.63	N.S.
Error	21	1598.58		
Assessments	5	6.30	0.40	N.S.
Assessments x groups	30	15.97	1.00	N.S.
Error	109	15.94		

ANOVA Table 60 (b)

FVC - Analysis at one-year follow-up

Source	d.f.	M.S.	F.	Sig.
Groups	7	159.96	0.51	N.S.
Error	22	316.62		
Assessments	3	6.68	0.36	N.S.
Assessments x groups	21	14.80	0.80	N.S.
Error	68	18.44		

ANOVA Table 61 (a)

FEV/FVC - Analysis at three-month follow-up

Source	d.f.	M.S.	F.	Sig.
Groups	6	272.90	0.58	N.S.
Error	21	474.55		
Assessments	5	21.28	1.08	N.S.
Assessments x groups	30	17.67	0.89	N.S.
Error	109	19.77		

ANOVA Table 61 (b)

FEV/FVC - Analysis at one-year follow-up

Source	d.f.	M.S.	F.	Sig.
Groups	7	606.83	0.90	N.S.
Error	22	676.11		
Assessments	3	2.60	0.22	N.S.
Assessments x groups	21	14.85	1.28	N.S.
Error	68	11.62		

ANOVA Table 62 (a)

CO Transfer Factor - Analysis at three-month follow-up

Source	d.f.	M.S.	F.	Sig.
Groups	6	1077.19	1.05	N.S.
Error	21	1023.20		
Assessments	5	14.03	0.78	N.S.
Assessments x groups	30	18.85	1.05	N.S.
Error	109	18.00		

ANOVA Table 62 (b)

CO Transfer Factor - Analysis at one-year follow-up

Source	d.f.	M.S.	F.	Sig.
Groups	7	5.42	0.30	N.S.
Error	20	18.11		
Assessments	5	0.07	0.05	N.S.
Assessments x groups	35	1.17	0.86	N.S.
Error	117	1.36		

ANOVA Table 63

EPQ (P) - Analysis at one-year follow-up

Source	d.f.	M.S.	F.	Sig.
Groups	7	201.11	1.44	N.S.
Error	20	139.60		
Assessments	5	3.93	0.93	N.S.
Assessments x groups	35	3.68	0.87	N.S.
Error	117	4.24		

ANOVA Table 64

EPQ (E) - Analysis at one-year follow-up

Source	d.f.	M.S.	F.	Sig.
Groups	7	117.01	0.77	N.S.
Error	20	151.89		
Assessments	5	3.56	0.50	N.S.
Assessments x groups	35	6.32	0.89	N.S.
Error	117	7.08		

ANOVA Table 65

EPQ (N) - Analysis at one-year follow-up

Source	d.f.	M.S.	F.	Sig.
Groups	7	105.47	1.66	N.S.
Error	20	63.49		
Assessments	5	3.94	1.47	N.S.
Assessments x groups	35	2.57	0.96	N.S.
Error	117	2.68		

ANOVA Table 66

EPQ (L) - Analysis at one-year follow-up

Source	d.f.	M.S.	F.	Sig.
Groups	7	446.83	0.45	N.S.
Error	19	983.89		
Assessments	5	121.43	1.01	N.S.
Assessments x groups	35	92.78	0.77	N.S.
Error	116	120.14		

ANOVA Table 67

SCL-90 (Somatic Anxiety) - Analysis at one-year follow-up

Source	d.f.	M.S.	F.	Sig.
Groups	7	509.30	1.59	N.S.
Error	19	320.85		
Assessments	5	64.50	0.86	N.S.
Assessments x groups	35	75.97	1.01	N.S.
Error	116	75.07		

ANOVA Table 68

SCL-90 (Obsess.-Compulsiveness) - Analysis at one-year follow-up

Source	d.f.	M.S.	F.	Sig.
Groups	7	362.36	0.81	N.S.
Error	19	448.10		
Assessments	5	77.35	1.22	N.S.
Assessments x groups	35	75.22	1.19	N.S.
Error	116	63.40		

ANOVA Table 69

SCL-90 (Interpers.-Sensitivity) - Analysis at one-year follow-up

Source	d.f.	M.S.	F.	Sig.
Groups	7	914.70	1.68	N.S.
Error	19	543.12		
Assessments	5	89;19	1.19	N.S.
Assessments x groups	35	75.81	1.01	N.S.
Error	116	74.76		

ANOVA Table 70

SCL-90 (Depression) - Analysis at one-year follow-up

Source	d.f.	M.S.	F.	Sig.
Groups	7	508.62	1.18	N.S.
Error	19	430.02		
Assessments	5	235.29	3.29	$p < 0.01$
Assessments x groups	35	83.17	1.16	N.S.
Error	116	71.46		

ANOVA Table 71

SCL-90 (Anxiety) - Analysis at one-year follow-up

Source	d.f.	M.S.	F.	Sig.
Groups	7	418.33	0.58	N.S.
Error	19	724.96		
Assessments	5	467.75	3.02	$p < 0.025$
Assessments x groups	35	176.67	1.14	N.S.
Error	116	154.78		

ANOVA Table 72

SCL-90 (Hostility) - Analysis at one-year follow-up

Source	d.f.	M.S.	F.	Sig.
Groups	7	1091.26	0.55	N.S.
Error	19	1974.14		
Assessments	5	319.17	1.64	N.S.
Assessments x groups	35	183.81	0.94	N.S.
Error	116	195.06		

ANOVA Table 73

SCL-90 (Phobic Anxiety) - Analysis at one-year follow-up

Source	d.f.	M.S.	F.	Sig.
Groups	7	2099.77	1.44	N.S.
Error	19	1459.36		
Assessments	5	102.13	0.43	N.S.
Assessments x groups	35	182.76	0.76	N.S.
Error	116	240.14		

ANOVA Table 74

SCL-90 (Paranoid ideation) - Analysis at one-year follow-up

Source	d.f.	M.S.	F.	Sig.
Groups	7	1193.51	0.89	N.S.
Error	19	1337.78		
Assessments	5	559.15	2.71	$p < 0.025$
Assessments x groups	35	153.24	0.74	N.S.
Error	116	206.03		

ANOVA Table 75

SCL-90 (Psychoticism) - Analysis at one-year follow-up

Source	d.f.	M.S.	F.	Sig.
Groups	7	204.36	1.62	N.S.
Error	19	163.20		
Assessments	5	49.17	1.29	N.S.
Assessments x groups	35	31.44	0.82	N.S.
Error	116	38.26		

ANOVA Table 76

SCL-90 (GSI) - Analysis at one-year follow-up

Source	d.f.	M.S.	F.	Sig.
Groups	7	267.96	2.41	N.S.
Error	19	110.98		
Assessments	5	73.97	1.63	N.S.
Assessments x groups	35	37.10	0.82	N.S.
Error	116	45.32		

ANOVA Table 77

SCL-90 (PSDL) - Analysis at one-year follow-up

Source	d.f.	M.S.	F.	Sig.
Groups	7	268.59	0.92	N.S.
Error	19	240.49		
Assessments	5	29.00	0.72	N.S.
Assessments x groups	35	32.45	0.81	N.S.
Error	116	40.27		

ANOVA Table 78

SCL-90 (PST) - Analysis at one-year follow-up

Source	d.f.	M.S.	F.	Sig.
Groups	7	18.77	0.87	N.S.
Error	20	21.66		
Assessments	5	1.04	0.75	N.S.
Assessments x groups	35	1.27	0.91	N.S.
Error	117	1.39		

ANOVA Table 79

16PF (A) - Analysis at one-year follow-up

Source	d.f.	M.S.	F.	Sig.
Groups	7	9.23	0.79	N.S.
Error	20	11.64		
Assessments	5	0.63	0.68	N.S.
Assessments x groups	35	0.80	0.86	N.S.
Error	117	0.93		

ANOVA Table 80

16PF (B) - Analysis at one-year follow-up

Source	d.f.	M.S.	F.	Sig.
Groups	7	16.75	0.61	N.S.
Error	20	27.34		
Assessments	5	1.93	1.84	N.S.
Assessments x groups	35	1.11	1.05	N.S.
Error	117	1.05		

ANOVA Table 81

16PF (C) - Analysis at one-year follow-up

Source	d.f.	M.S.	F.	Sig.
Groups	7	15.59	0.83	N.S.
Error	20	18.84		
Assessments	5	1.00	0.89	N.S.
Assessments x groups	35	1.21	1.08	N.S.
Error	117	1.12		

ANOVA Table 82

16PF (E) - Analysis at one-year follow-up

Source	d.f.	M.S.	F.	Sig.
Groups	7	6.88	0.28	N.S.
Error	20	25.22		
Assessments	5	0.40	0.39	N.S.
Assessments x groups	35	0.86	0.85	N.S.
Error	117	1.02		

ANOVA Table 83

16PF (F) - Analysis at one-year follow-up

Source	d.f.	M.S.	F.	Sig.
Groups	7	10.16	0.73	N.S.
Error	20	13.95		
Assessments	5	0.64	0.87	N.S.
Assessments x groups	35	0.93	1.27	N.S.
Error	117	0.74		

ANOVA Table 84

16PF (G) - Analysis at one-year follow-up

Source	d.f.	M.S.	F.	Sig.
Groups	7	25.39	1.19	N.S.
Error	20	21.38		
Assessments	5	1.29	1.79	N.S.
Assessments x groups	35	1.06	1.48	N.S.
Error	117	0.72		

ANOVA Table 85

16PF (H) - Analysis at one-year follow-up

Source	d.f.	M.S.	F.	Sig.
Groups	7	23.82	1.65	N.S.
Error	20	14.45		
Assessments	5	1.75	1.61	N.S.
Assessments x groups	35	1.78	1.64	$p < 0.05$
Error	117	1.09		

ANOVA Table 86

16PF (I) - Analysis at one-year follow-up

Source	d.f.	M.S.	F.	Sig.
Groups	7	44.69	3.31	$p < 0.025$
Error	20	13.47		
Assessments	5	1.26	1.00	N.S.
Assessments x groups	35	0.74	0.59	N.S.
Error	117	1.26		

ANOVA Table 87

16PF (L) - Analysis at one-year follow-up

Source	d.f.	M.S.	F.	Sig.
Groups	7	5.57	0.48	N.S.
Error	20	11.69		
Assessments	5	1.77	1.33	N.S.
Assessments x groups	35	1.31	0.98	N.S.
Error	117	1.33		

ANOVA Table 88

16PF (M) - Analysis at one-year follow-up

Source	d.f.	M.S.	F.	Sig.
Groups	7	3.93	0.39	N.S.
Error	20	9.96		
Assessments	5	0.63	0.53	N.S.
Assessments x groups	35	1.20	1.01	N.S.
Error	117	1.20		

ANOVA Table 89

16PF (N) - Analysis at one-year follow-up

Source	d.f.	M.S.	F.	Sig.
Groups	7	31.04	1.27	N.S.
Error	20	24.41		
Assessments	5	0.11	0.10	N.S.
Assessments x groups	35	0.82	0.73	N.S.
Error	117	1.13		

ANOVA Table 90

16PF (O) - Analysis at one-year follow-up

Source	d.f.	M.S.	F.	Sig.
Groups	7	47.34	2.76	$p < 0.05$
Error	20	17.16		
Assessments	5	1.36	0.80	N.S.
Assessments x groups	35	2.34	1.38	N.S.
Error	117	1.70		

ANOVA Table 91

16PF (Q_1) - Analysis at one-year follow-up

Source	d.f.	M.S.	F.	Sig.
Groups	7	16.50	0.67	N.S.
Error	20	24.60		
Assessments	5	1.65	1.54	N.S.
Assessments x groups	35	1.10	1.03	N.S.
Error	117	1.07		

ANOVA Table 92

16PF (Q_2) - Analysis at one-year follow-up

Source	d.f.	M.S.	F.	Sig.
Groups	7	13.04	0.89	N.S.
Error	20	14.65		
Assessments	5	1.37	1.23	N.S.
Assessments x groups	35	1.04	0.93	N.S.
Error	117	1.12		

ANOVA Table 93

16PF (Q_3) - Analysis at one-year follow-up

Source	d.f.	M.S.	F.	Sig.
Groups	7	21.90	0.81	N.S.
Error	20	26.89		
Assessments	5	1.67	1.51	N.S.
Assessments x groups	35	1.38	1.25	N.S.
Error	117	1.11		

ANOVA Table 94

16PF (Q_4) - Analysis at one-year follow-up

Source	d.f.	M.S.	F.	Sig.
Between techniques	5	1820.64	4.22	$p < 0.01$
Error	42	431.45		

ANOVA Table 95

Differential degree of use of treatment techniques

Source	d.f.	M.S.	F.	Sig.
Assessments	4	14.90	0.74	N.S.
Groups	7	87.81		
Error	28	20.03		

ANOVA Table 96

Changes in degree of use of Hierarchical Reduction over time

Source	d.f.	M.S.	F.	Sig.
Assessments	4	12.12	1.05	N.S.
Groups	7	110.48		
Error	28	11.48		

ANOVA Table 97

Changes in degree of use of Deprived Response Performance over time

Source	d.f.	M.S.	F.	Sig.
Assessments	4	29.13	2.05	N.S.
Groups	3	12.94		
Error	12	14.23		

ANOVA Table 98

Changes in degree of use of Focussed Relaxation over time

Source	d.f.	M.S.	F.	Sig.
Assessments	4	39.72	3.26	$p < 0.05$
Groups	7	59.48		
Error	28	12.22		

ANOVA Table 99

Changes in degree of use of Coverant Control over time

Source	d.f.	M.S.	F.	Sig.
Assessments	4	54.07	2.03	N.S.
Groups	7	179.72		
Error	28	26.64		

ANOVA Table 100

Changes in degree of use of Monetary Deprivation over time

Source	d.f.	M.S.	F.	Sig.
Assessments	4	13.30	1.12	N.S.
Groups	3	174.32		
Error	12	11.90		

ANOVA Table 101

Changes in degree of use of Covert Sensitization over time

Source	d.f.	M.S.	F.	Sig.
Between Techniques	5	1968.89	5.40	$p < 0.001$
Error	42	364.84		

ANOVA Table 102

Differential degree of benefit from treatment techniques

Source	d.f.	M.S.	F.	Sig.
Assessments	4	10.29	0.86	N.S.
Groups	7	95.09		
Error	28	12.01		

ANOVA Table 103

Changes in degree of benefit from Hierarchical Reduction over time

Source	d.f.	M.S.	F.	Sig.
Assessments	4	38.43	6.63	$p < 0.01$
Groups	3	15.92		
Error	12	5.80		

ANOVA Table 104

Changes in degree of benefit from Focussed Relaxation over time

Source	d.f.	M.S.	F.	Sig.
Assessments	4	1.85	0.11	N.S.
Groups	7	124.92		
Error	28	18.31		

ANOVA Table 105

Changes in degree of benefit from Deprived Response Performance over time

Source	d.f.	M.S.	F.	Sig.
Assessments	4	60.97	4.63	$p < 0.01$
Groups	7	31.25		
Error	28	13.17		

ANOVA Table 106

Changes in degree of benefit from Coverant Control over time

Source	d.f.	M.S.	F.	Sig.
Assessments	4	20.28	1.69	N.S.
Groups	7	105.99		
Error	28	12.01		

ANOVA Table 107

Changes in degree of benefit from Monetary Deprivation over time

Source	d.f.	M.S.	F.	Sig.
Assessments	4	3.55	0.35	N.S.
Groups	3	172.07		
Error	12	10.32		

ANOVA Table 108

Changes in degree of benefit from Covert Sensitization over time

ADDENDUM

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1) Introduction

A consideration of the design and the statistical analysis employed in this study led to the conclusion that further discussion of certain methodological and conceptual features was warranted and that a reanalysis of the main outcome data using a different analytical model may produce results which either contrasted with or confirmed those originally obtained.

Dealing with this latter point first, it was felt that, whereas in the main analysis, no inter-group differences were found on the primary outcome measures (smoking-rate, overall benefit rating and physiological measures), a reanalysis of combined group data for the three main independent variables (treatment condition, baseline smoking rate and treatment-target) may reveal such differences. It will be seen that this was, in fact, the case.

With respect to the further methodological issues, it was believed that additional discussion was merited regarding certain contaminating factors inherent in the experimental design, the constraints placed upon truly random allocation of subjects to treatment groups and the effects of these constraints, the characteristics of some of the correlational data presented and the nature of the control groups used in the study. Furthermore, additional discussion concerning the conceptual nature of controlled smoking and the conceptual analysis of relapse in smoking beaaviour, especially in relation to ideas developed in the modification of alcohol usage and to the "Abstinence Violation Effect" (Marlatt, 1978; Marlatt and Gordon, 1985), was felt to be pertinent.

2) Further statistical analysis

a) Results

A one-way analysis of variance with one repeated measure was used to compare the outcome of the two sets of four treatment groups, for each of the three independent variables constituting the design of the experiment (see p.110), on each of the main dependent measures taken. The results of these analyses are presented below, under the appropriate dependent variable heading, and are summarized in Table A.1. (p.A.8)

(i) Smoking rate

(a) Self control package (SC) vs. self control plus therapist administered (SC+) package

The groups receiving the SC+ package reduced their smoking rate to a significantly greater extent than those receiving the SC package alone ($F\ (1,5) = 10.66$, $p<0.025$).

The improvement over time for both groups combined was highly significant ($F\ (5,5) = 88.42$, $p<0.001$). The Tukey (a) test showed that all assessment points differed reliably ($p<0.01$) from pre-treatment assessment.

(See ANOVA table A.1. and Fig. A.1., pp. A.40 and A.28)

(b) "Light" vs. "Heavy" Smokers

No significant group differences emerged from the analysis ($F\ (1,5) = 0.00$, NS).

A significant change over time, for both groups combined, occurred ($F\ (5,5) = 35.12$, $p<0.001$). The Tukey (a) test showed that all assessments differed reliably from pre-treatment assessment ($p<0.01$)

(See ANOVA table A.2. and Fig. A.2., pp A40 and A28)

(c) 100% vs. 75% reducers

No significant group differences emerged from the analysis ($F(1,5) = 5.20$, NS).

For both groups combined, a significant change over time was evident ($F(5,5) = 14.11$, $p<0.01$). The Tukey (a) test showed that all assessments, with the exception of 12 month follow-up, differed reliably ($p<0.05$) from pre-treatment assessment.

(See ANOVA table A.3. and Fig. A.3., p.p.A40 and A29).

(ii) Overall benefit ratings

(a) SC package vs. SC+ package

No significant group differences emerged from the analysis ($F(1,5) = 3.00$, NS).

A significant change over time, for both groups combined, was apparent ($F(5,5) = 19.43$, $p<0.01$). The Tukey (a) test showed that all assessment points differed reliably ($p<0.05$) from pre-treatment assessment.

(See ANOVA table A.4. and Fig. A.4., p.p.A.41 and A.29).

(b) "Light" vs. "Heavy" smokers

No significant group differences were found ($F(1,5) = 1.64$, NS).

A significant change over time was found, for both groups combined ($F(5,5) = 40.81$, $p<0.001$). The Tukey (a) test showed that all assessment points differed reliably ($p<0.01$) from pre-treatment assessment.

See ANOVA table A.5. and Fig. A.5., p.p.A.41 and A.30).

(c) 100% vs. 75% reducers

A significant difference between groups was evident ($F(1,5) = 19.06$, $p<0.01$), the 100% reducing group reporting having obtained

more benefit from treatment than the 75% reducing group.

A significant change over time, for both groups combined, also emerged ($F(5,5) = 30.48$, $p<0.001$). The Tukey (a) test showed that all assessment points differed reliably ($p<0.01$) from pre-treatment assessment.

(See ANOVA table A.6. and Fig. A.6., p.p.A.41 and A.30).

(iii) Serum thiocyanate (SCN^-)

(a) SC package vs. SC+ package

No significant difference between groups was evident ($F(1,5) = 0.47$, NS).

No significant changes over time, for both groups combined, emerged from the analysis ($F(5,5) = 4.80$, NS).

(See ANOVA table A.7. and Fig. A.7., p.p.A.42 and A.31).

(b) "Light" vs. "Heavy" smokers

The two groups differed significantly on this measure ($F(1,5) = 30.11$, $p<0.01$); the "heavy" smoking group had higher SCN^- levels than did the "light" smoking group.

A significant change over time, for both groups combined, also occurred ($F(5,5) = 21.07$, $p<0.01$). The Tukey (a) test showed that all post-baseline SCN^- levels were reliably lower than at pre-treatment ($p<0.05$).

(See ANOVA table A.8. and Fig. A.8., p.p. A.42 and A.31).

(c) 100% vs. 75% reducers

No significant difference between groups was found ($F(1,5) = 3.58$, NS).

A significant change over time, for both groups combined, occurred ($F(5,5) = 5.82$, $p<0.05$). The Tukey (a) test showed a reliable

difference between pre-treatment assessment and post-treatment and three-month follow-up assessments ($p<0.05$).

(See ANOVA table A.9. and Fig. A.9., p.p.A.42 and A.32).

(iv) Lung Function Measures

(a) SC package vs. SC+ package

On the measures of FEV_1 and FVC, significant inter-group differences were found ($F(1,5) = 39.19$, $p<0.01$ and $F(1,5) = 7.66$, $p<0.05$, respectively), the SC+ group having higher scores in both cases. No significant differences were found between groups on the measures of FEV/FVC or Carbon Monoxide Transfer Factor ($F(1,5) = 0.63$, NS and $F(1,5) = 5.71$, NS, respectively).

No significant changes over time, for both groups combined, were found on the measures of FEV_1, FVC or TF ($F(5,5) = 2.81$, NS; $F(5,5) = 0.62$, NS and $F(5,5) = 2.14$, NS, respectively). On the measure of FEV/FVC, a significant negative change over time was found to have occurred, ($F(5,5) = 6.00$, $p<0.05$). The Tukey (a) test, however, showed that no reliable between assessment differences existed ($p>0.05$).

(See ANOVA tables A.10, A.11, A.12 and A.13 and Figs. A.10, A.11, A.12 and A.13, pp.A.43, A.44 and pp. A.32, A.33 and A.34).

(b) "Light" vs. "Heavy" smokers

On the measures of FEV_1, FVC and CO Transfer Factor, significant inter-group differences were found ($F(1,5) = 23.03$, $p<0.01$; $F(1,5) = 19.03$, $p<0.01$; and $F(1,5) = 7.08$, $p<0.05$), respectively). In all cases, the "light" smoking group obtained higher scores than the "heavy" smoking group. No significant group difference was found on the measure of FEV/FVC. ($F(1,5) = 0.19$, NS).

Only on the measure of FEV_1 was a significant change over time, for both groups combined, found. ($F(5,5) = 6.39$, $p<0.05$); the Tukey (a) test showed that the twelve-month follow-up FEV_1 score was reliably lower than the three-month follow-up score ($p<0.05$). No significant differences over time emerged on the measures of FVC, FEV/FVC or TF ($F(5,5) = 0.35$, NS; $F(5,5) = 1.15$, NS and $F(5,5) = 0.83$, NS, respectively).

(See ANOVA tables A.14, A.15, A.16 and A.17 and Figs. A.14, A.15, A.16 and A.17, p.p. A.44, A.45 and p.p. A.34, A.35 and A.36).

(c) 100% vs. 75% reducers

Significant inter-group differences were apparent on the measures of FEV_1 and FVC ($F(1,5) = 9.14$, $p<0.025$ and $F(1,5) = 11.72$, $p<0.025$, respectively), the 75% reducing group obtaining higher scores in both cases. No significant differences were found on the measures of FEV/FVC or TF ($F(1,5) = 0.03$, NS and $F(1,5) = 0.24$, NS, respectively).

On none of the lung function measures was a significant change over time, for both groups combined, observed (FEV_1 - $F(5,5) = 1.83$, NS; FVC - $F(5,5) = 0.37$, NS; FEV/FVC - $F(5,5) = 1.14$, NS; TF - $F(5,5) = 0.30$, NS).

(See ANOVA tables A.18, A.19, A.20 and A.21 and Figs A.18, A.19, A.20 and A.21, p.p. A.45, A.46 and p.p. A.36, A.37 and A.38).

(v) Gross Body Weight

(a) SC package vs. SC+ package

A highly significant difference ($F(1,5) = 387.89$, $p<0.001$) was found between the two groups, the SC package group weighing more than the SC+ package group. The amounts and rates of weight

increase for the two groups, however, were comparable (the SC only group weighing more at the outset).

A significant increase in weight over time, for the two groups combined, was observed ($F(5,5) = 10.22$, $p<0.025$). The Tukey (a) test showed that weight at three-, six- and twelve-month follow-up assessments was reliably higher than at pre-treatment assessment ($p<0.05$).

(See ANOVA table A.22 and Fig. A.22, p.p. A.47 and A.38).

(b) "Light" vs. "Heavy" smokers

A significant inter-group difference was observed ($F(1,5) = 20.19$, $p<0.025$), the "heavy" smoking group being higher on the measure of weight; this difference was, however, apparent at pre-treatment.

No significant change in weight over time occurred, for both groups combined ($F(5,5) = 1.09$, NS).

(See ANOVA table A.23 and Fig. A.23, p.p. A.47 and A.39).

(c) 100% vs. 75% reducers

A highly significant between groups difference was found on the measure of body weight ($F(1,5) = 143.96$, $p<0.001$); the 75% reducers weighed more than did the 100% reducers. The former group weighed more at the outset of treatment.

A significant change over time occurred ($F(5,5) = 6.81$, $p<0.05$), for both groups combined. The Tukey (a) test showed that weight at three- and six-month follow-up was reliably higher than at pre-treatment (p 0.05).

(See ANOVA table A.24 and Fig.A.24, p.p. A.47 and A.39).

Table A.1 - Summary of results of analysis of variance for combined groups on the three main independent variables

		Between groups difference		Over assessments	
ANALYSIS	Outcome Measure	F (1,5)	sig.	F (5,5)	sig.
Self-control groups (1,2,3,4)	Overall benefit rating	3.00	NS	19.43	p<0.01
	SCN–	0.47	NS	4.80	NS
-vs-	FEV_1	39.19	p<0.01	2.81	NS
	FVC	7.66	p<0.05	0.62	NS
	FEV/FVC	0.63	NS	6.00	p<0.05
	TF	5.71	NS	2.14	NS
Multi-Element groups (5,6,7,8)	Gross Body weight	387.89	p<0.001	10.22	p<0.025
	Smoking rate	10.66	p<0.025	88.42	p<0.001
"Light" Smoking groups (1,2,5,6)	Overall benefit rating	1.64	NS	40.81	p<0.001
	SCN–	30.11	p<0.01	21.07	p<0.01
-vs-	FEV_1	23.03	p<0.01	6.39	p<0.05
	FVC	19.03	p<0.01	0.35	NS
	FEV/FVC	0.19	NS	1.15	NS
	TF	7.08	p<0.05	0.83	NS
"Heavy" Smoking groups (3,4,7,8)	Gross Body weight	20.19	p<0.025	1.09	NS
	Smoking rate	0.00	NS	35.12	p<0.001
100% reducing groups (1,3,5,7)	Overall benefit rating	19.06	p<0.01	30.48	p<0.001
	SCN–	3.58	NS	5.82	p<0.05
-vs-	FEV1	9.14	p<0.05	1.83	NS
	FVC	11.72	p<0.025	0.37	NS
	FEV/FVC	0.03	NS	1.14	NS
	TF	0.24	NS	0.30	NS
75% reducing groups (2,4,6,8)	Gross Body weight	1143.96	p<0.001	6.81	p<0.05
	Smoking rate	5.20	NS	14.11	p<0.01

b) Discussion of Results

(i) Smoking-rate

The analysis conducted confirmed the previous result (p.163) that, regardless of initial smoking rate and regardless of goal of treatment (abstinence or reduction), groups were equally successful in reducing their smoking rate. However, in contrast to this comparability and to the original results, the re-analysis showed that those groups receiving the self control package plus therapist-administered techniques reduced their smoking rate to a significantly greater extent than did those groups receiving the self-control package alone. This finding lends some credence to the supposition that "multicomponent packages (may) be superior in their effectiveness to less comprehensive packages" (p.106); some support is therefore given to previous findings that an additive affect may accrue from combining self-control and therapist administered procedures (Chapman et al, 1971; Morrow et al, 1973; Tongas et al, 1976).

The differential effect discovered is clearly illustrated in Fig. A.1., (p. A.28). (An alternative explanation for this finding is presented below: see section 3 (a), p.A.13).

(ii) Overall benefit ratings

The original findings (p.169), that groups receiving different treatment packages and groups with differing baserates of smoking reported equivalent degrees of overall benefit from treatment, were confirmed. It was found, however, that the groups aiming at total abstinence from smoking reported having obtained greater benefit at all assessment points, than those aiming at reduction to 25% of baseline. This finding is not consistent with the fact that these two groups were equally successful in reducing their smoking rate (see above) and suggests

that subjective feelings of achievement did not reflect objective improvement, for the reduced smoking groups; it is in keeping, however, with the earlier finding (p.166 and pp.250-252) that, in absolute terms, total abstinence is a more viable goal than 75% reduction from baseline.

Notwithstanding the above finding, it must be emphasized that the reanalysis of the data demonstrated that for all three main factors, all groups reported significantly and reliably higher benefit ratings at all assessment points, compared with pre-treatment.

(iii) Serum thiocyanate (SCN^-)

Re-analysis confirmed that for the factors "heavy" versus "light" smoking and 100% versus 75% reduction, post-baseline measures differed significantly (in a positive direction) from pre-treatment. (In the case of "abstinence" versus "reduction", however, only the post-treatment and three-month follow-up measures were reliably lower than pre-treatment). When the self-control package only and the self-control plus therapist administered package groups were examined, however, the F-ratio obtained just failed to reach significance at the 0.05 level, for both groups combined, over time. With regard to inter-group differences, none were apparent, except for "heavy" versus "light" smokers (the heavier smokers having higher SCN^- levels); this result serves to confirm the validity of SCN^- as a physiological correlate of smoking rate and was not unexpected. This latter finding did not emerge from the finer analysis conducted initially (see pp. 175 and 179), perhaps because of the low number of subjects remaining in some individual groups at long-term follow-up.

(iv) Lung Function Measures

Some anomalous findings emerged here and some were, at first

sight, inconsistent with the original findings. When the self-control package and self-control plus therapist administered package groups were compared, it was apparent that the latter groups had higher scores on the measures of FEV_1 and FVC; this was not the case for the FEV/FVC ratio nor for CO Transfer Factor. However, close examination of the data showed that for both FEV_1 and FVC, the multicomponent group had higher initial readings than did the self-control package only group, so the finding cannot be attributed to the effects of treatment. Although analysis of variance suggested that these groups, looked at together, showed a deterioration over time on the measure of FEV/FVC, the Tukey (a) test did not confirm the reliability of these changes.

Comparing the "heavy" and "light" smoking groups, between-group differences existed on the measures of FEV_1, FVC and CO Transfer Factor; once more, however, these differences were apparent at the onset of treatment and were probably not attributable to treatment. Only in the case of FVC was it likely that the difference was treatment-related. (See Fig. A.15, p.A.35). It is interesting and also clinically significant that the "light" smoking group scored higher on all of these indices than the "heavy" smoking group, corroborating evidence that smoking is associated with impaired lung-function (McCarthy et al, 1976; U.S.D.H.E.W., 1979). On the measure of FEV_1, it was found that, for both of these groups combined, significantly lower scores were obtained at one-year follow-up than at three-month follow-up. Although this could perhaps be associated with the parallel increase (ie. relapse) in smoking-rate, the fact that one-year follow-up rate was still lower than at pre-treatment makes this finding difficult to explain.

Comparing the 100% reducing and 75% reducing groups on lung-function measures, the group differences found on FEV_1 and FVC were again

attributable to different group characteristics at pre-treatment, rather than to any effects of treatment. On none of the measures was a significant change over assessments found.

(v) Gross Body Weight

For all three main treatment factors, significant group differences were found on the measure of weight; in all cases, however, these differences were evident at the outset of treatment and were not a result of changes in smoking rate or any other factors associated with treatment.

It was initially found (pp.179-181) that a significant increase in body weight, for all groups, over time, occurred; this finding was partially substantiated by re-analysis of the data. Looking at the self-control and multi-element groups, weight at three-, six- and twelve-month follow-up was found to be reliably higher than at pre-treatment; similarly, examining the "abstaining" and "reducing" groups, weight at three- and six-month follow-up was higher than at the beginning of treatment. However, in the case of "heavy" and "light" smokers, no such significant change over time was apparent. It may be concluded from these results, however, that the association between smoking rate reduction and body-weight increase is largely confirmed (Gordon et al, 1975; Blitzer et al, 1977).

c) Conclusions

The results obtained from re-analysis of the data for the main outcome measures of this study are, on the whole, consistent with the original findings. Regardless of treatment condition, goal of treatment or baseline smoking rate, all groups improved over time on the measures of smoking rate and overall benefit ratings; however, there is some

evidence that the multicomponent package may have been more effective than the self-control package alone and that "abstaining" subjects perceived themselves as having obtained more benefit from treatment than did "reducing" subjects.

Serum thiocyanate was confirmed as being a reliable correlate of smoking rate. When the primary independent variable of treatment condition was examined, however, less marked changes over time, in SCN^- levels, were apparent, despite significant decreases in rate.

Analysis of lung-function measures corroborated the relationship between smoking and impaired function. However, there was little evidence that changes over time occurred as a result of decreased smoking nor any evidence that the three main treatment factors led to any differential changes in lung function; with the possible exception of the measure of FVC the differences which were apparent were due to incomparability of groups at the onset of treatment (this finding did not emerge from the initial data analysis - pp.150-152).

Finally, no interactive relationship was found between weight increase on any of the three main independent variables (group differences again being attributable to pre-treatment inequality), but further considerable support was afforded to the original finding that, looking at combined groups, weight increased over time as a result of a reduction in smoking-rate.

3 Further methodological issues

(a) Contaminating factors inherent in the experimental design

The time constraints placed upon the experimenter in conducting this research meant that the practical, data collection phase of the experiment had a duration of over three-and-a-half years (May 1979 to

January 1983), when one-year follow-up assessment on the last-run groups is included. Two groups were run concurrently ("light" and "heavy" smoking groups for each treatment condition and each treatment target), but the four "self-control package only" groups were run before the "multicomponent package" groups.

It may thus have been the case that contamination occurred, in that certain environmental influences, operating differentially over time, were not controlled for. Such influences may have had an impact upon non-specific factors relating to treatment outcome, such as degree of motivation, degree of expectancy, knowledge of the harmful effects of smoking, etc. Financial factors may also have operated. Although such influence may have been in either direction (augmenting or detracting from the efficacy of intervention), it is more likely that a positive effect would be exerted (groups run later in treatment being more likely to succeed than earlier groups). The increased level of public knowledge about the harmful effects of smoking (mediated by more attention on television and in the written media and by official publications such as the report of the Royal College of Physicians (e.g. R.C.P. 1983)), the increased price of cigarettes and the higher visibility of "stop-smoking" clinics and organizations such as Action on Smoking and Health (ASH), are all factors which may be expected to facilitate treatment programmes designed to modify smoking behaviour.

In the event, no clear knowledge was attributed to the multicomponent package used, as compared to the self-control package; however, it will be remembered from the re-analysis of the results (pp. A.12-A.13), that the former treatment package was found to be significantly more effective in bringing about reductions in smoking rate

and it is possible that this effect was due to such contaminating factors as are mentioned above, rather than to active ingredients of the treatment package itself.

(b) Non-random allocation of subjects to treatment groups

As described in the "Procedure" section of this thesis (p.130-131), subjects were randomly selected from the pool, to be allocated to treatment groups. Allocation to groups was not random, however, as all subjects were initially asked whether they wished to attempt to abstain from smoking or to aim at controlling their rate of smoking at 25% of baseline, this distinction being an essential part of the experimental design. Thus it may have been the case that the two subject sub-populations (abstainers and reducers) differed on certain parameters, as a function of their aim of involvement in the treatment programme. The most obvious such parameter is that of motivation. Although treatment groups were not found to differ, at pre-treatment, on the measure of motivation (see p.149), it was concluded (pp.272-273 and p.275) that the "motivation questionnaire" employed in the study was perhaps neither a reliable nor valid assessment tool; differences in degree of motivation may, in fact, have been present.

It is difficult to envisage how this methodological problem could be overcome in a study comparing different treatment goals; ethical implications would be associated with giving subjects no choice as to their goal, so only through the use of an accurate measure of motivation (thereby ensuring the equality of groups on this "non-specific" measure at pre-treatment) could the difficulty be overcome. The reality of this methodological problem is supported by the findings that "controlled smoking" subjects were, in fact, less successful in

achieving and maintaining their goal than were "abstaining" groups (pp. 163-167), in absolute terms, and that the former group reported having obtained less subjective benefit from treatment than the latter (p.A.13).

A similar argument could be made where "abstainers" and "reducers" are concerned, regarding the measure of "expectancy". However, this measure, unlike that of motivation, was considered to be both valid and reliable, correlating significantly with treatment outcome, and the two types of groups did not differ significantly on expectancy at pre-treatment.

c) Nature of the correlational data presented

Table 5.30 (p.227) presented the correlational data obtained in this study. Two of the correlational analyses performed purported to examine the relationship between reduction in smoking rate and other dependent measures ("Rated benefit/Actual benefit" and "Actual benefit/% weight increase"). These analyses were based on pooled "between-subject" and "within-subject data" (ie. every measure taken for each subject) and, as a result, the "N's" used to compute the correlational values were relatively high.

It was felt that different results may be obtained by examining between-subject data only, at different assessment points, (the higher the "N", in computing Spearman Rho values, the lower the value of Rho needs to be, to reach a statistically significant level), and this was, in fact, the finding in some cases. The re-computed correlational data (corrected for ties and for "large" N's) are presented in Table A.2 (p.A.17). For the "Rated benefit/Actual benefit" relationship, the revised correlational data confirm the original. Whereas, in the case of pooled data, the value of Spearman's Rho was found to be 0.72, this

Relationship	N	Spearman's Rho	Significance level
Rated benefit/Actual benefit			
Mid-treatment	44	0.361	p<0.01
Post-treatment	41	0.828	p<0.001
3/12 follow-up	30	0.790	p<0.001
6/12 follow-up	29	0.602	p<0.001
12/12 follow-up	31	0.701	p<0.001
Actual benefit/%wt.increase			
Mid-treatment	42	0.184	NS
Post-treatment	38	0.122	NS
3/12 follow-up	32	0.465	p<0.01
6/12 follow-up	32	0.381	p<0.025
12/12 follow-up	35	0.183	NS

Table A.2

Between-subjects correlational data

being significant at the $p<0.001$ level, in the case of between-subject only data, the correlations at all assessment points, with the exception of mid-treatment assessment, were also significant at the $p<0.001$ level (mean Spearman's Rho value = 0.66, mean N = 35). The Rho value at mid-treatment assessment was relatively low (0.36) but this was, nevertheless, significant at the $p<0.01$ level. These data clearly confirm that reduction in smoking rate was reflected in self-reported benefit obtained from treatment.

For the "Actual benefit/% weight increase" relationship, the re-worked data provided less unequivocal support for the original finding that these two factors were positively correlated at a high level of significance. In the original analysis, although the value of Spearman's Rho was only 0.26, the high N of 176 resulted in a level of significance of $p<0.001$. For between subject data only, non-significant positive correlations were obtained for mid- and post-treatment and twelve-month follow-up assessments; however, significant correlations of Rho = 0.465 ($p<0.01$) were obtained for three-month follow-up data, Rho = 0.381 ($p<0.025$) for six-month follow-up data. These findings serve to confirm the suggested relationship between reduction in smoking rate and increase in gross-body weight, but also suggest that this relationship was, in this study, most marked at medium-long-term follow-up points.

d) <u>The nature of the control groups</u>

The problems described in section a), above, relating to contamination of groups as a result of uncontrolled external factors, may, it can be argued, have also applied to the control groups used in this study. As these two groups (one "heavy" and one "light"

smoking group) were run concurrently, no such differences would have existed between these groups. However, the control groups were assessed and then reassessed following the termination of treatment with the last two experimental groups; a long period passed, between the running of the earlier experimental groups and the assessment of the controls. As mentioned earlier, characteristics such as motivation, expectancy and knowledge of the effects and treatment of cigarette smoking may have been present to different degrees in these groups. Although this methodological problem needs to be identified, it must, however, be noted that the control groups and treatment groups were not found to differ from one-another at pre-"treatment", on the measures of expectancy or motivation. (See p.157).

A further methodological criticism regarding the control groups is that, after recruitment, the period of more than two-and-a-half years which passed before "treatment" was offered may, in itself, have led to a decrease in motivation. Again, however, the data obtained do not support this supposition.

4) Further theoretical issues

(a) The concept of controlled smoking: further discussion

The concept of "controlled smoking" was first advanced by Frederiksen, Peterson and Murphy (1976). This alternative goal in the field of smoking behaviour modification stemmed firstly from the practical issue that, where the treatment goal of total abstinence from smoking applied, exceptionally high relapse rates typically resulted (Murray and Hobbs, 1981), (the phenomenon of relapse is discussed in detail below), and, secondly, from the conceptual issue that, whereas a "disease" model of smoking would not accept any goal

other than abstinence, a "self-control" model does accommodate "controlled" smoking (Frederiksen et al, 1976).

Two common beliefs are attacked by the concepts, the first one being that, if an individual smokes at all, after gaining control over the behaviour, his or her smoking rate will ultimately return to baseline; this belief may, in part, have developed from post hoc rationalizations by relapsed (abstaining) smokers. The second belief is that, since the use of tobacco is unhealthy, it should not be used at all. Few would argue that abstinence, when achievable and maintainable, is not preferable to controlled smoking; however, where individuals are either unwilling or unable to abstain, controlled smoking is a dersirable alternative goal.

The term "controlled" smoking has often been equated with "safer" smoking (Russell, 1974), and, indeed, a reduced smoking rate does lead to attenuation of the health-risks incurred; there is no conclusive evidence that nicotine-compensatory behaviours negate the benefits obtained from reducing smoking rate (see section on Nicotine Regulation pp.26-35). However, rate-reduction is not the only way in which smoking can be made safer: topographical changes in smoking behaviour (eg. shorter puff duration, longer inter-puff intervals, less inhalation, etc.) and changes in the substance used (eg. low tar content cigarettes) can also lead to the individual's benefiting from a health viewpoint. (As the present study was concerned with rate-reduction only, the latter two methods of safer smoking were, however, not addressed).

In conclusion, traditional abstinence-oriented procedures, which are based on self-control or self-management skills, lend themselves to the treatment goal of controlled smoking. As in the field of alcohol

abuse (eg. Strickler et al, 1976; Rollnick and Heather, 1982) perhaps shifting the focus of attention and intervention to the risks incurred by the behaviour, rather than focussing on the behaviour, per se, may yield more successful results than have been typical in the field of smoking behaviour modification.

(b) The phenomenon of relapse and the Abstinence Violation Effect

Little has been said, in the main body of this thesis, about the phenomenon of relapse, other than remarking that it is, perhaps, the "bête noir" of smoking research. Furthermore, no mention has been made of the "Abstinence Violation Effect" (AVE) (Marlatt, 1978) a concept which has recently undergone an increase in importance in this field.

Before relating the above issues closely to smoking behaviour, a conceptual analysis will be presented and reference made to the application of findings to the problem of alcohol abuse.

Webster's New Collegiate Dictionary (1983) offers two definitions of "relapse": a) "a recurrence of symptoms of a disease after a period of improvement" (ie. an "all or more" conceptualization) and b) "the act or instance of backsliding, worsening or subsiding" (ie. a "lapse"). Modern thinking, with regard to smoking behaviour quite rightly shuns the disease model; the second definition is therefore the more germane where intervention with smoking is concerned.

The "relapse process" may be conceptualized as follows: whilst maintaining acquired abstinence, the individual experiences a sense of perceived control, or self-efficacy (Bandura, 1977); the longer the period of abstinence, the greater the sense of self-efficacy. If a "high-risk" situation is then encountered, the individual will either demonstrate an effective (cognitive and behavioural) coping

response, or otherwise. (Cummings et al (1980) identified three main high-risk categories - negative emotional states, such as anxiety, depression or boredom, interpersonal conflict, for example within the family or work-setting, and social pressure, which may be either direct or indirect). If effective coping occurs, self-efficacy increases and the risk of relapse is reduced; if, however, the behaviour in question occurs, the opposite may result.

The AVE is closely related to the subjective belief by the former abstainer that "once over the line, there's no going back". The process is believed to be as follows: prior to the first lapse, the individual is committed to abstinence; if a lapse occurs, the AVE will come into play and the intensity of this effect will be determined by a number of factors, such factors being the degree of external justification, the strength of prior committment , the duration of prior abstinence and the subjective importance of the prohibited behaviour. The intensity of the AVE is augmented by two key cognitive-affective elements - cognitive dissonance (Festinger, 1964), which is related to feelings of conflict and guilt, and the personal-attribution effect, related to self-blame and the perception of the self as being weak-willed or low in self-sufficiency.

The essence of the AVE is that, the greater its intensity, as determined by the above factors, the greater the probability of complete relapse, as opposed to a temporary lapse, if the prohibited behaviour occurs. To summarize the process: if a lapse is attributed to internal causes (eg. lack of willpower or weakness) feelings of guilt and conflict will engender a perceived loss of control; this will in turn lead to a redefinition of the self as "relapsed" and the behaviour will become "out of control".

Marlatt (1985a) has presented a "relapse-prevention" model based upon the premise that the cognitive/affective reactions to the first slip, or lapse, after a period of abstinence, determine whether this lapse is followed by a full return to the former behaviour. The goal of relapse prevention is teaching individuals to anticipate and deal with pertinent problems which may arise. Marlatt (1985b) has advocated the use of role-playing and "relapse-rehearsal" (this being closely related to the method of "programmed relapse" (Haley, 1977) which entails abstinent individuals indulging in the relevant behaviour and being helped to deal with the cognitive/affective behaviours resulting). Earlier, Chaney et al, (1978), suggested the use of skill-training procedures such as relaxation training, stress-management and efficacy-enhancing imagery, as ways of preventing relapse.

Relapse prevention needs to take into account the AVE. Cognitive-restructuring techniques are of importance in this respect: a lapse needs to be seen by the individual as a mistake, a slip, rather than a disaster, and attribution of the lapse to the situation or environment is preferable to attribution to internal factors. Marlatt (1985a) noted that the AVE is reduced in intensity if the individual attributes a lapse to external, unstable and specific factors, rather than to internal, stable, global factors. He further states that the "prognosis" is more favourable when the subject understands the nature of the AVE and when he or she is able to keep calm, renew the commitment to abstinence and to review the circumstances which led to the lapse, planning for recovery.

The earliest application of relapse-prevention techniques were in the field of alcohol abuse. As with tobacco-smoking, the reduction of the probability of relapse has been an issue of paramount importance. (Hunt et al, (1971) noted that, after treatment, 50-60% of alcoholics relapsed within the first three months). Efforts at preventing relapse in ex-drinkers have been based upon relapse prevention skill training as described above; cognitive-behavioural interventions to increase self-efficacy for abstainers (and cognitive restructuring regarding the ability to exert control over any future drinking), have been successful (Gorski and Miller, 1979). The influence of family support has been stressed in relapse-prevention programmes (McCrady et al, 1985). Finally, Rollnick and Heather (1982) made the important comment that controlled drinking, as opposed to total abstinence, is likely to reduce relapse probability, attenuating the effects of the AVE. Taking the foregoing into account, a number of points can now be made with regard to relapse prevention, the AVE and cigarette-smoking interventions. As stated by Shiffman et al (1985), the core of relapse prevention in smoking is the anticipation of and effective coping with "relapse-crises". Situational antecedents and emotional/cognitive consequences (eg. the AVE) must be taken into account in training programmes; Shiffman and his colleagues identified three areas of focus - education (orienting the smoker to the "quitting process" and encouraging realistic expectations), assessment (the identification of likely relapse situations and the individual's deficiencies in his or her "coping repertoire") and coping-skill development (for example, how to combat the AVE). They also emphasized the importance of detailed debriefing following relapse crises.

It is hoped that further concentration on the relapse process and further efforts to develop effective relapse-prevention techniques will lead to the increased potency of interventions aimed at smoking abstinence or smoking control.

References

Those references contained in the Addendum and not appearing below will be found in the main Reference list, pages 310-338.

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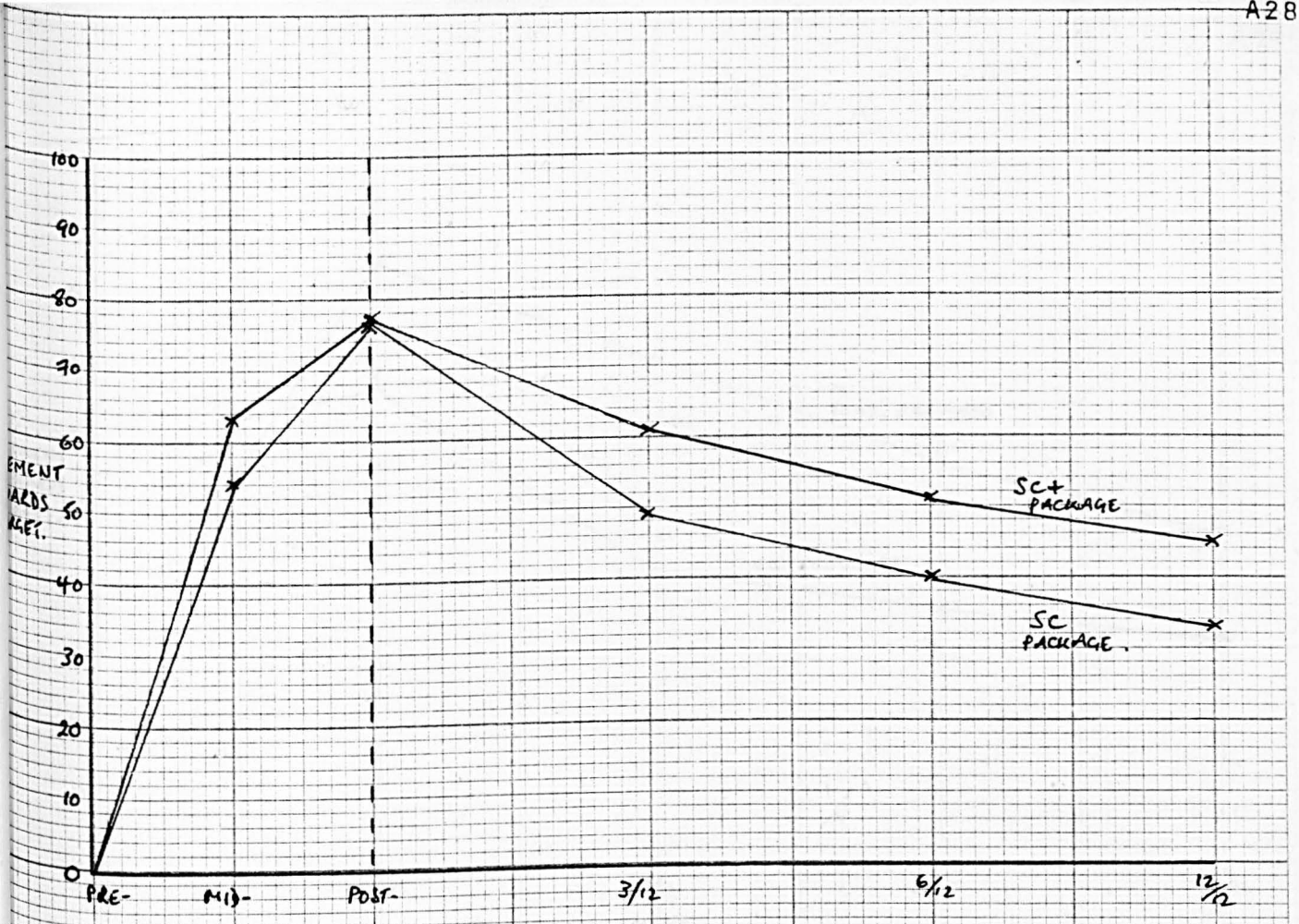

FIG. A.1.

SMOKING RATE : SELF-CONTROL PACKAGE VS. SELF-CONTROL PLUS THERAPIST ADMINISTERED PACKAGE.

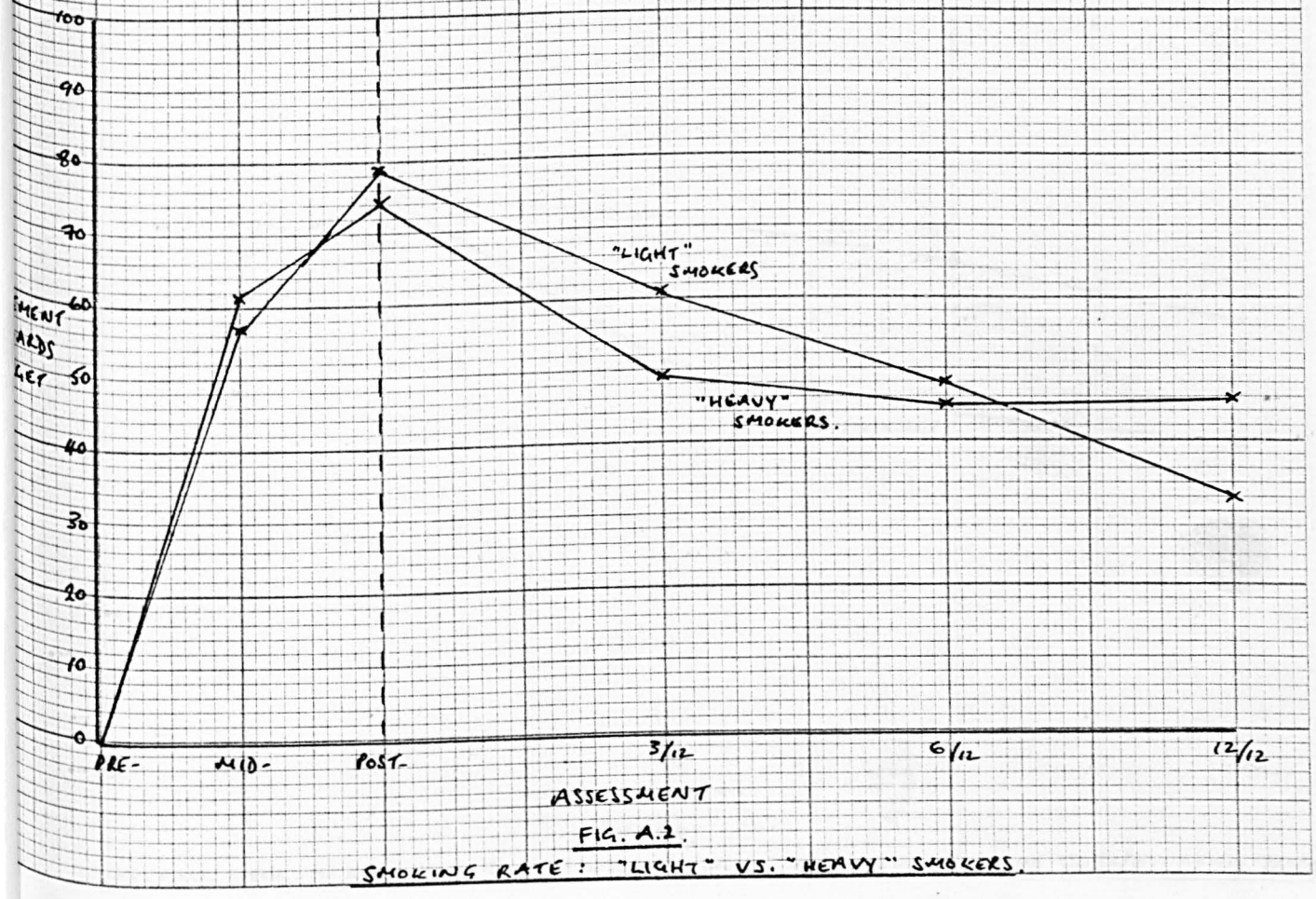

FIG. A.2.

SMOKING RATE : "LIGHT" VS. "HEAVY" SMOKERS.

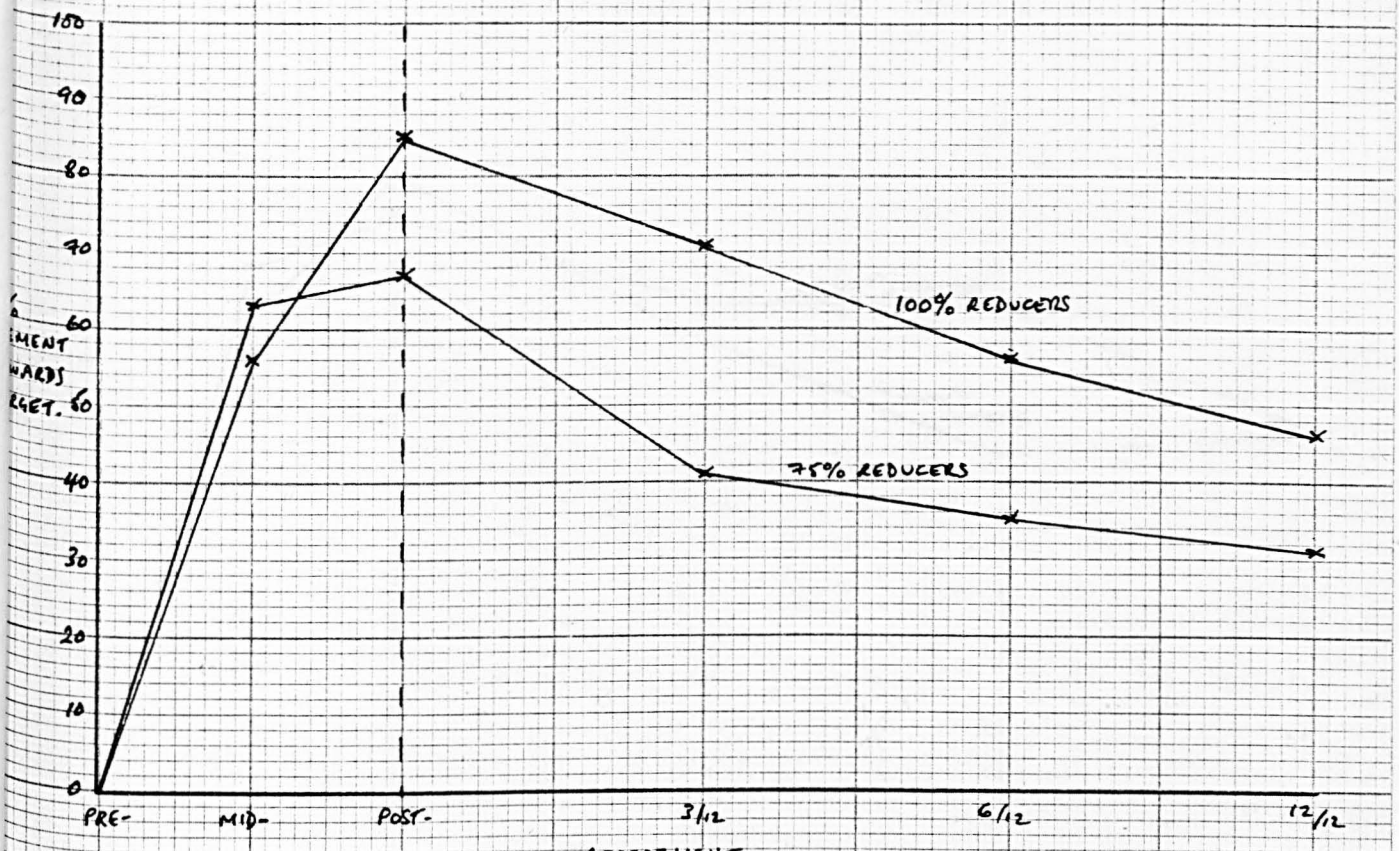

FIG. A3.

SMOKING RATE : 100% VS. 75% REDUCERS

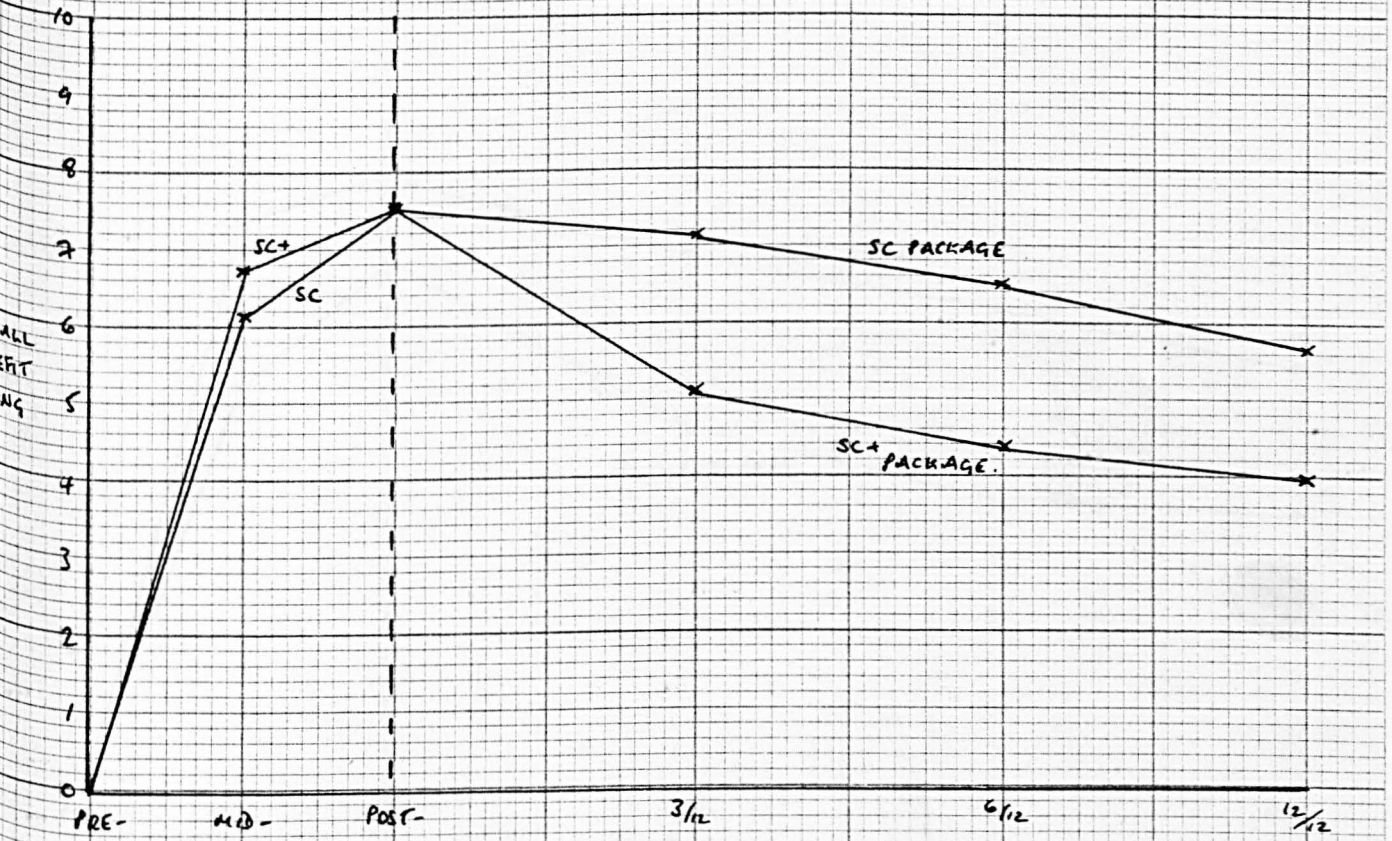

FIG. A4

OVERALL BENEFIT RATINGS : SELF-CONTROL PACKAGE VS. SELF-CONTROL PLUS THERAPIST ADMIN. PACKA

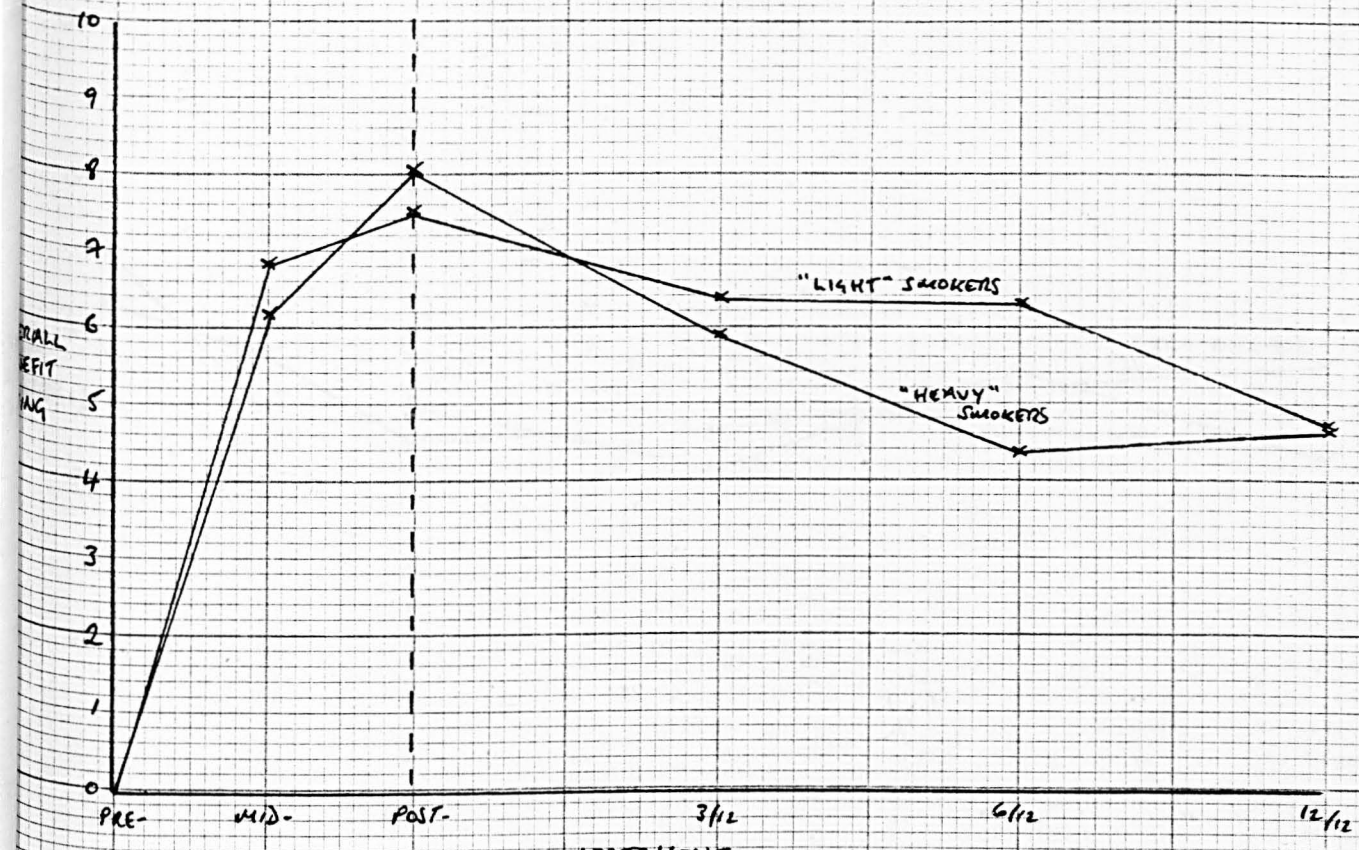

FIG. A5.

OVERALL BENEFIT RATINGS: "LIGHT" VS. "HEAVY" SMOKERS

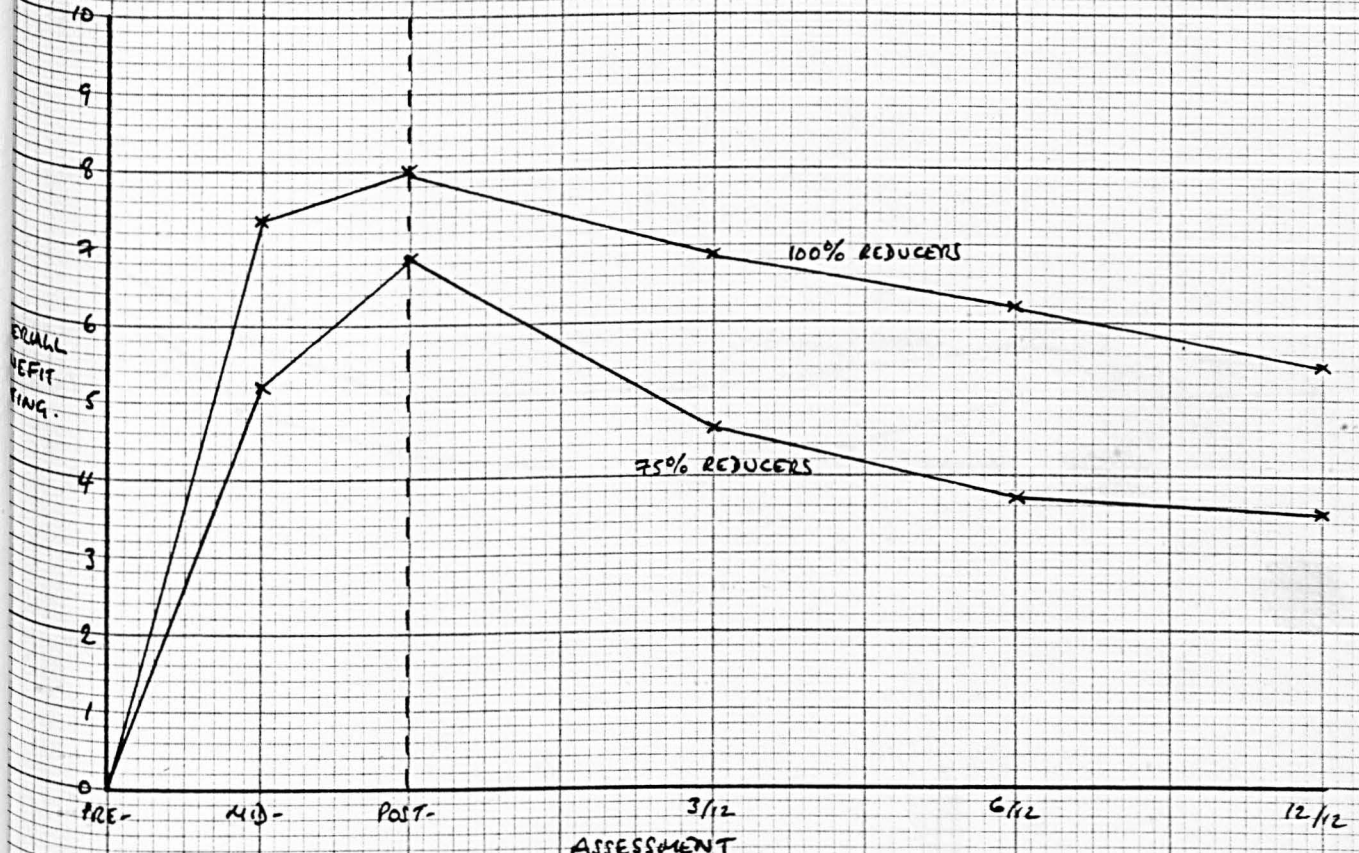

FIG. A6.

OVERALL BENEFIT RATINGS: 100% VS. 75% REDUCERS.

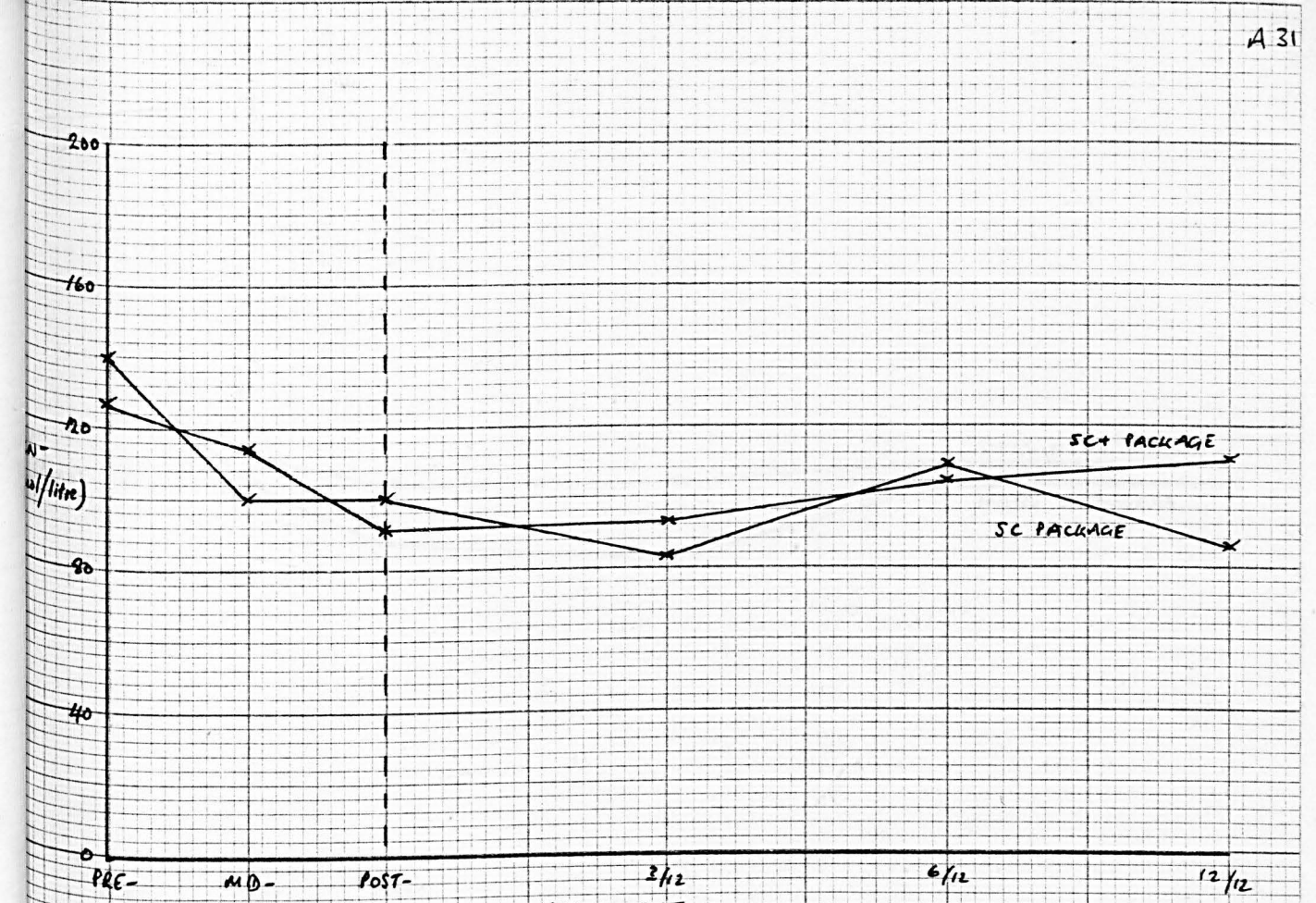

FIG. 17.

SCN⁻ : SELF-CONTROL PACKAGE VS. SELF-CONTROL PLUS THERAPIST ADMINISTERED PACKAGE.

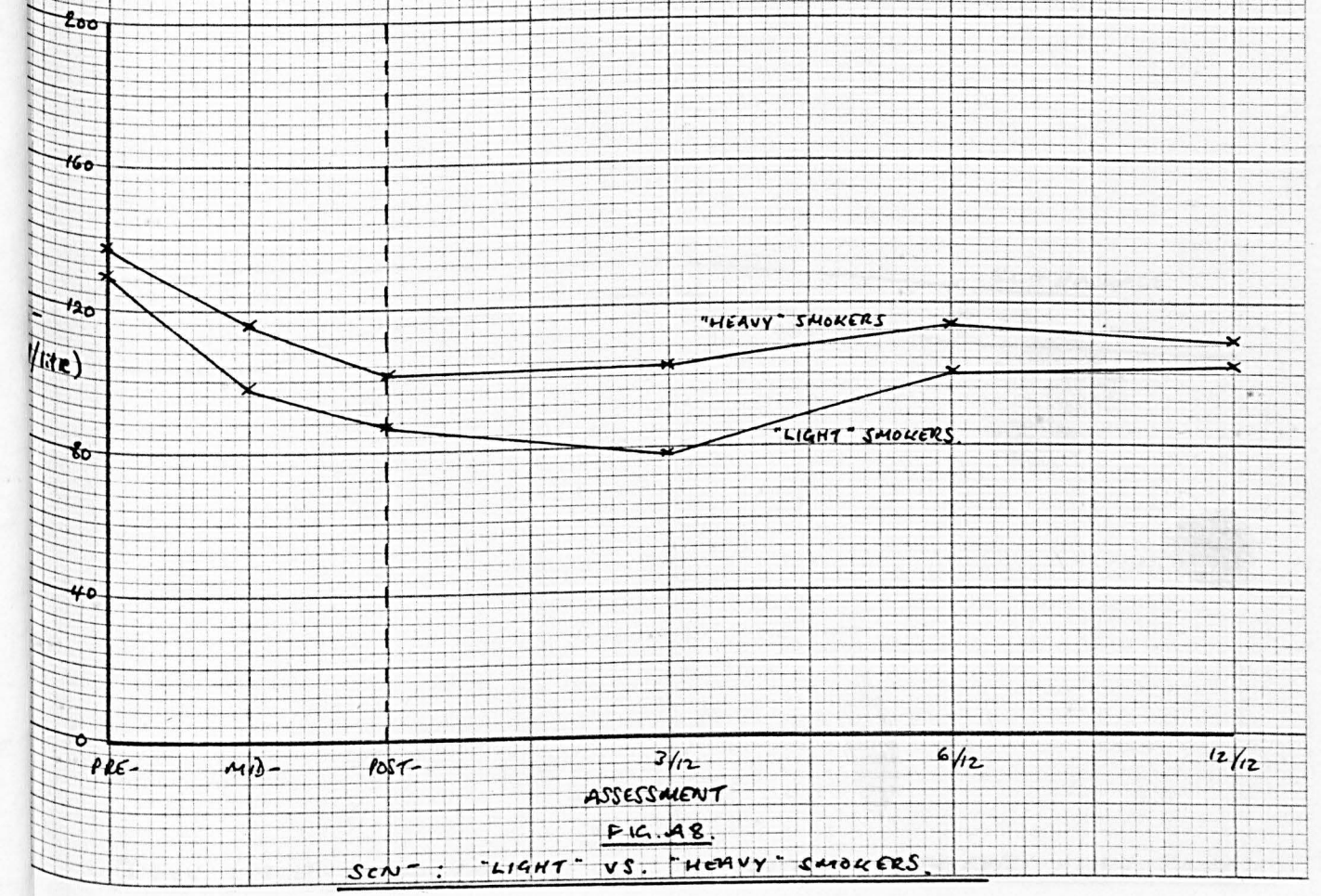

FIG. 18.

SCN⁻ : "LIGHT" VS. "HEAVY" SMOKERS.

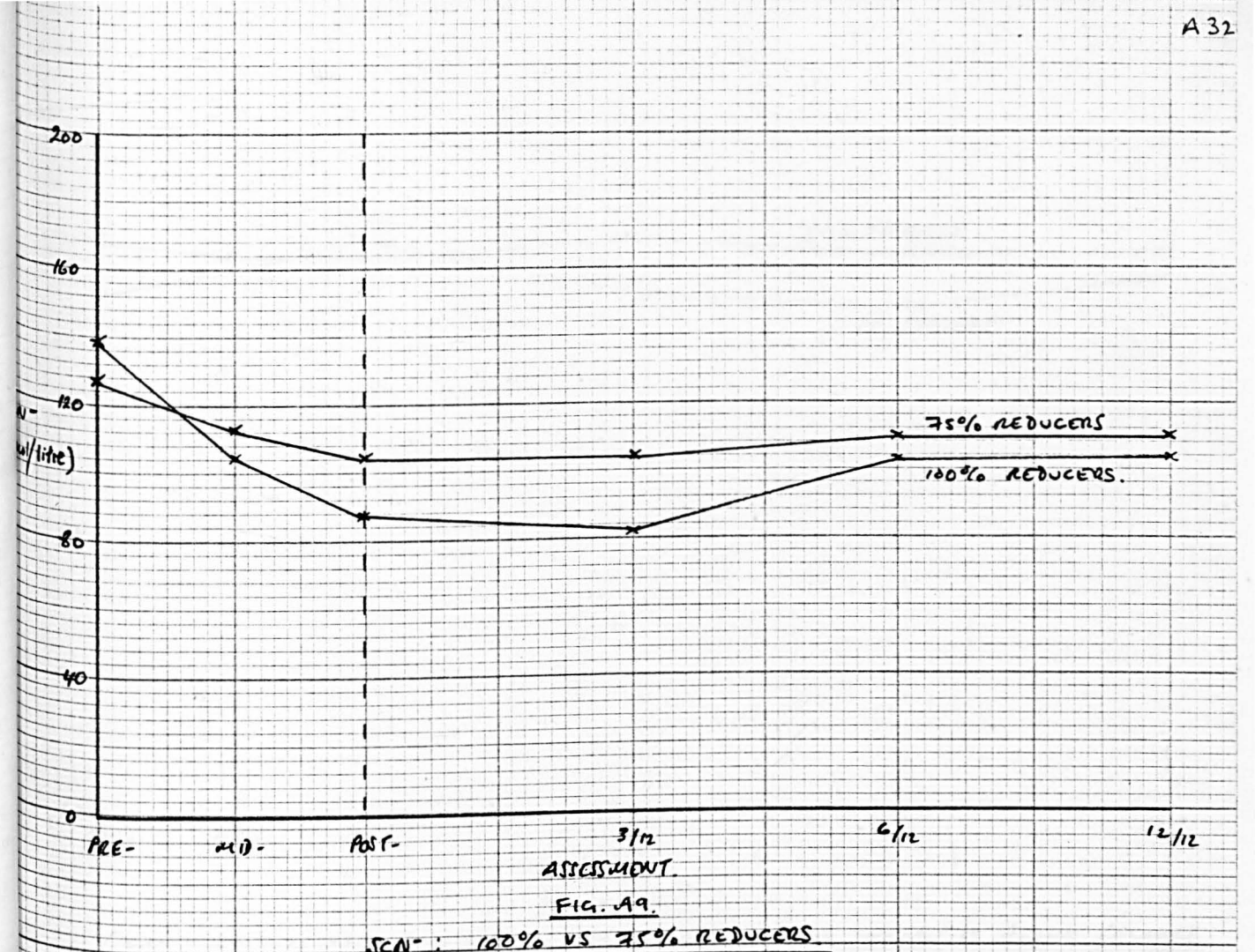

FIG. A9.

SCN⁻ : 100% VS 75% REDUCERS.

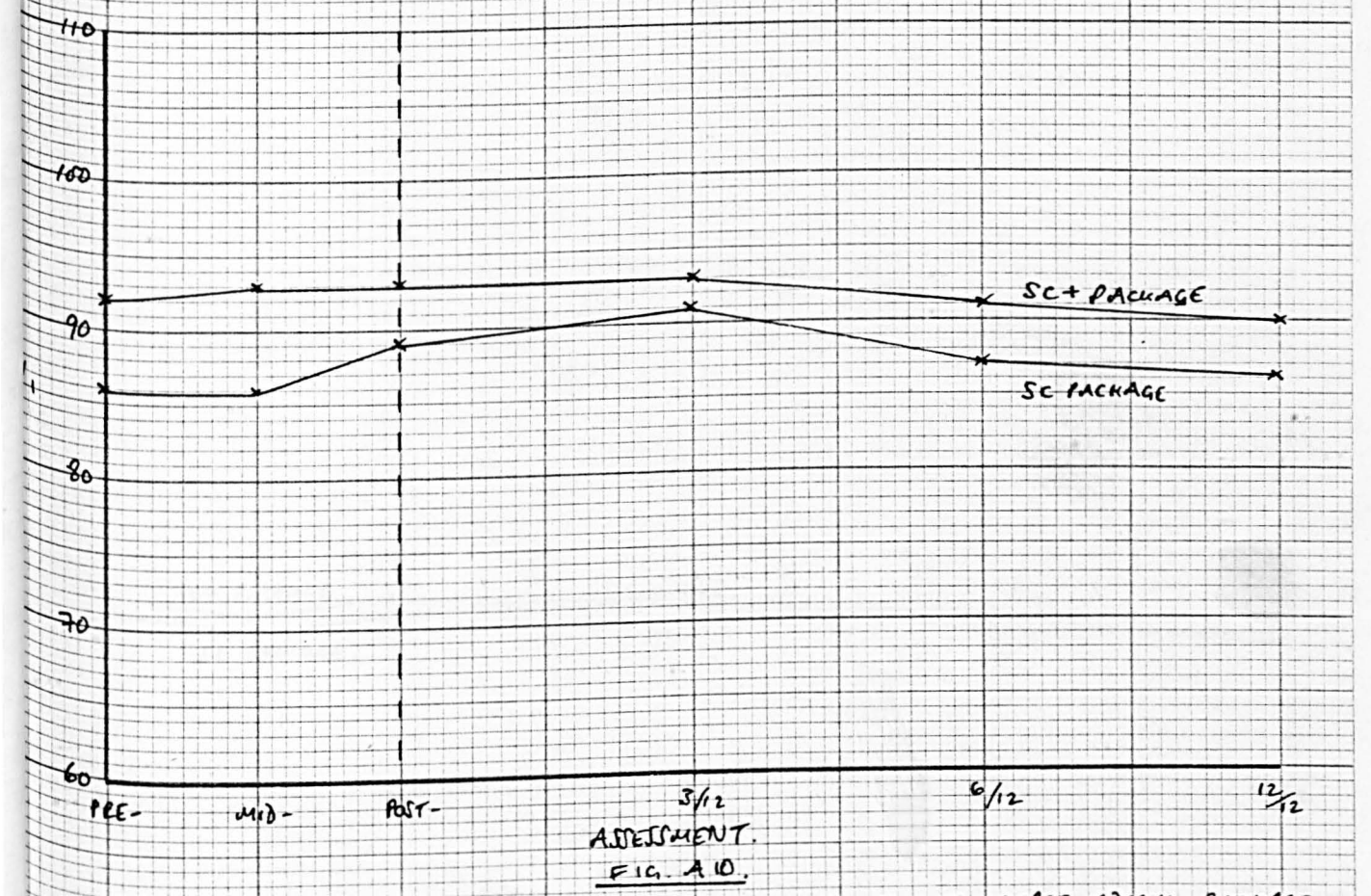

FIG. A10.

% FEV_1 : SELF-CONTROL PACKAGE VS. SELF-CONTROL PLUS THERAPIST ADMIN. PACKAGE.

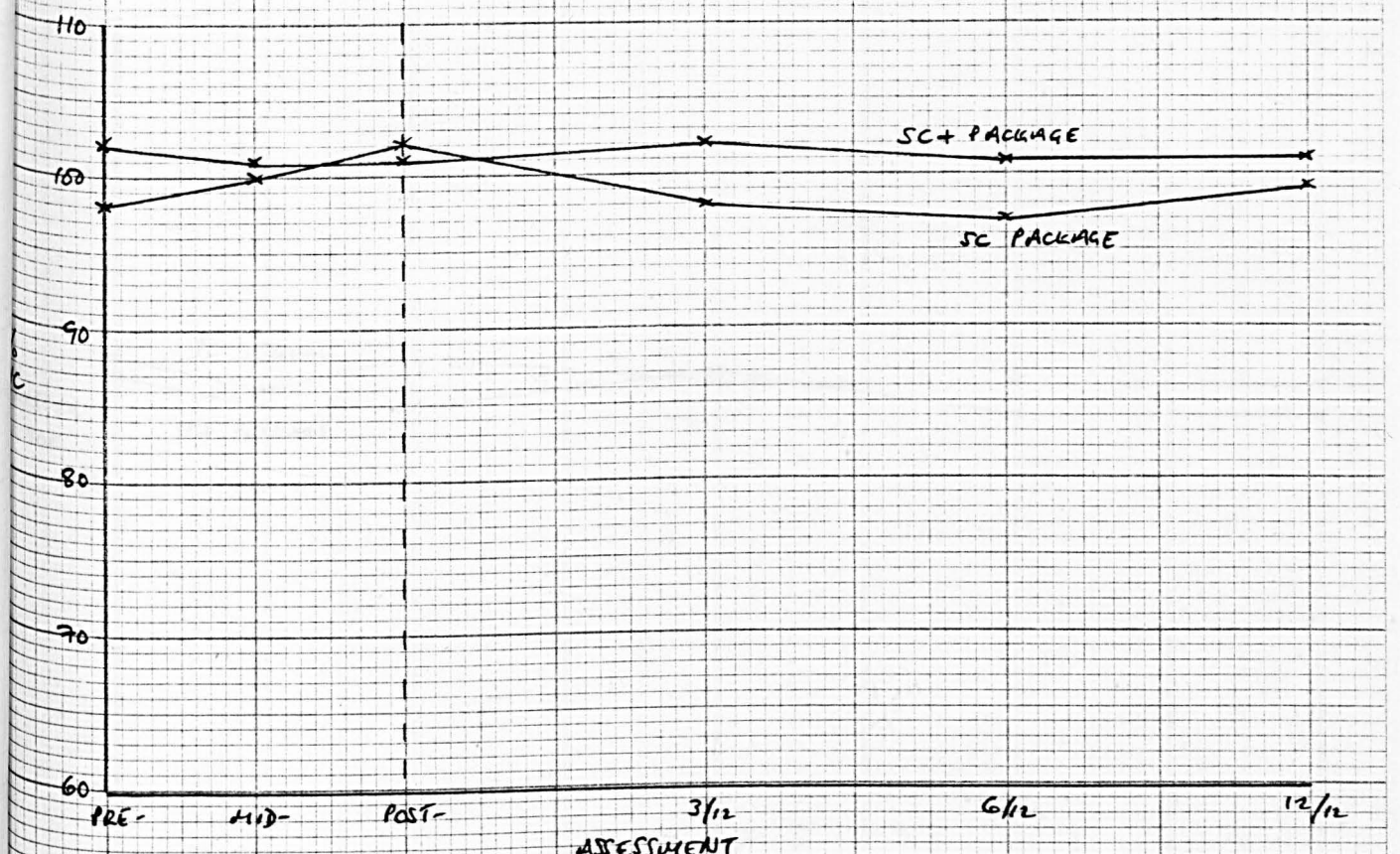

FIG. A11.

%FVC : SELF-CONTROL PACKAGE VS. SELF-CONTROL PLUS THERAPIST ADMIN. PACKAGE.

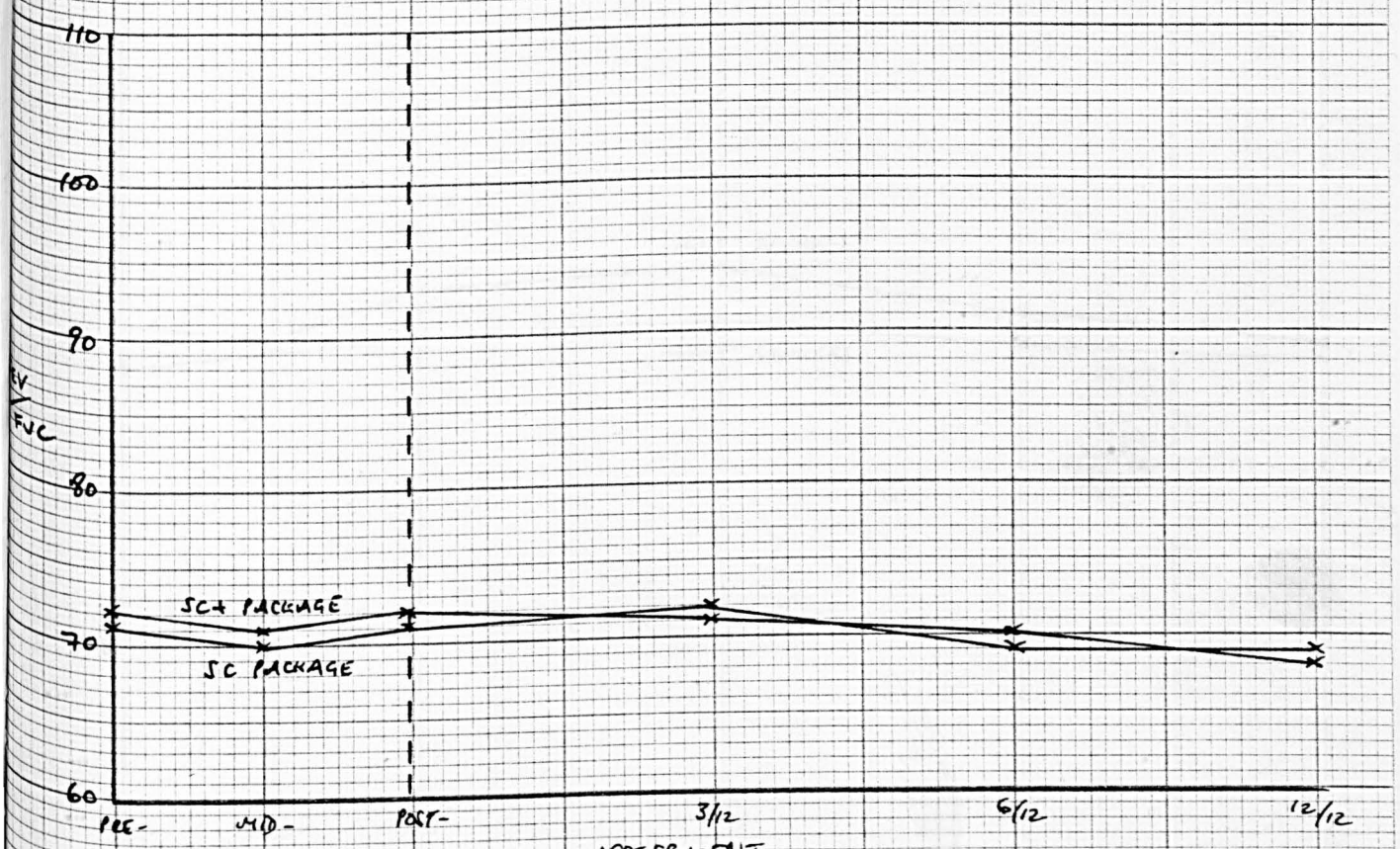

FIG. A12.

FEV/FVC : SELF-CONTROL PACKAGE VS. SELF-CONTROL PLUS THERAPIST ADMINISTERED PACKAGE.

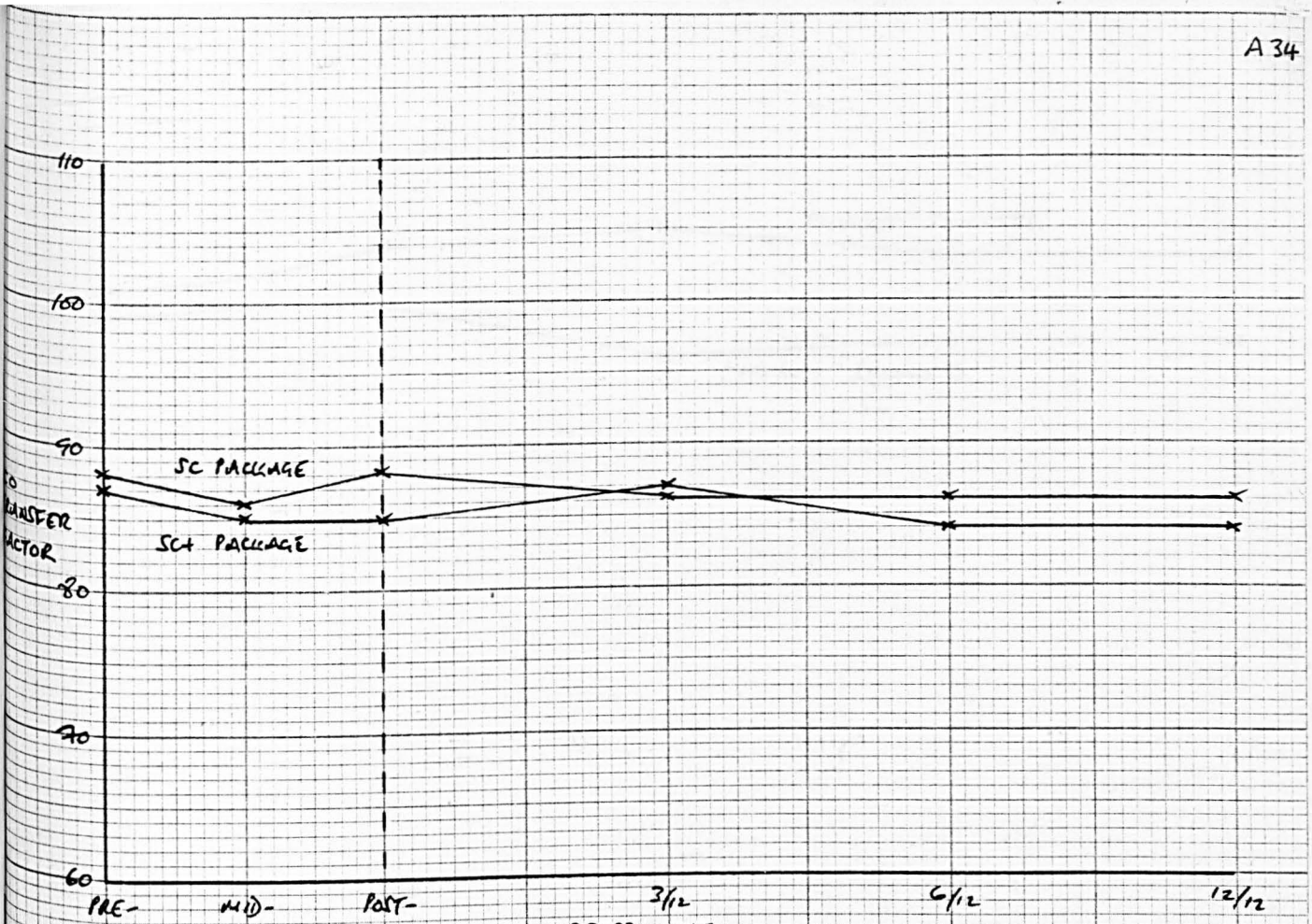

FIG. A 13.

CO TRANSFER FACTOR : SELF-CONTROL PACKAGE VS. SELF-CONTROL PLUS TIGERMIST ADMIN. PACKAGE.

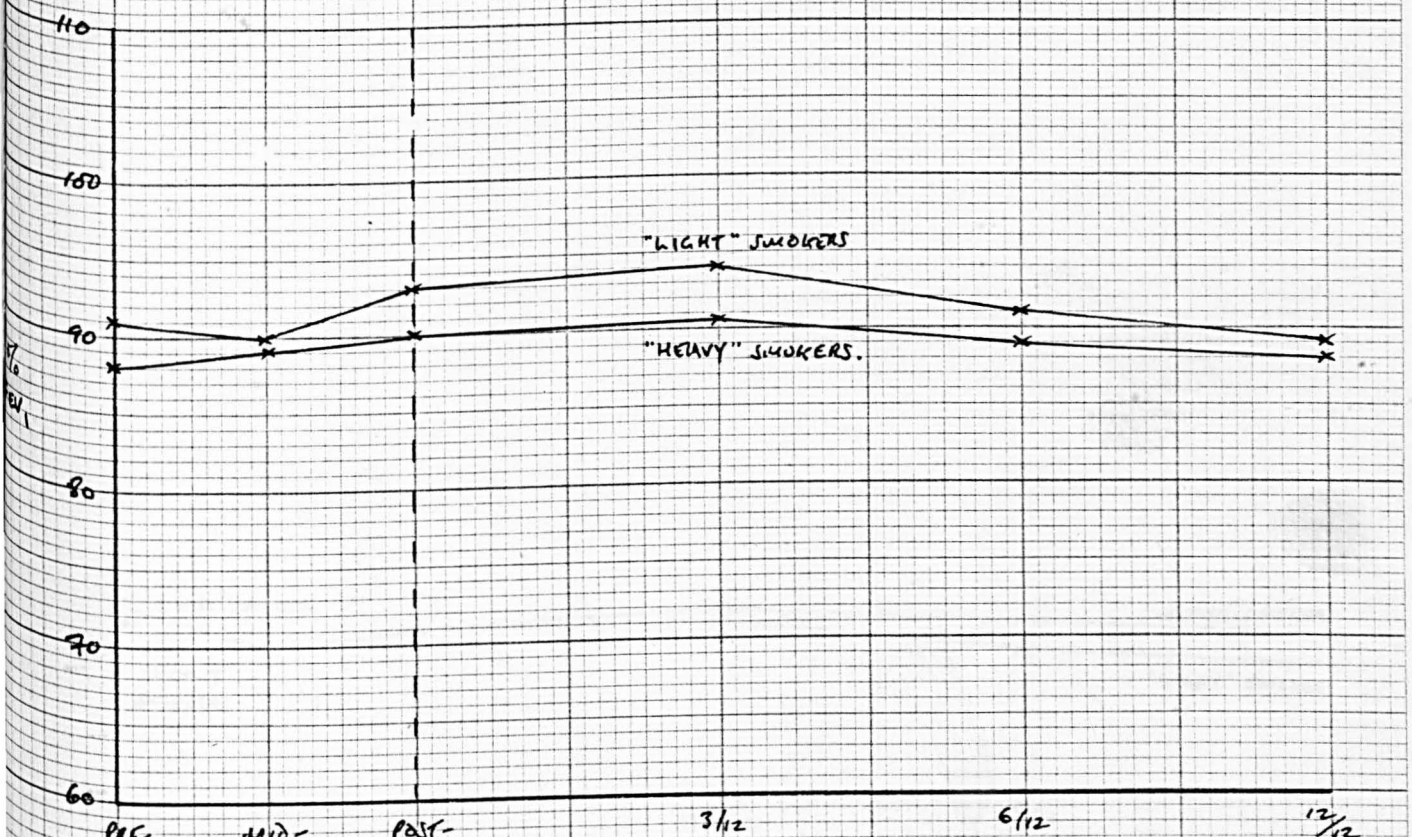

FIG. A 14.

% FEV$_1$: "LIGHT" VS. "HEAVY" SMOKERS.

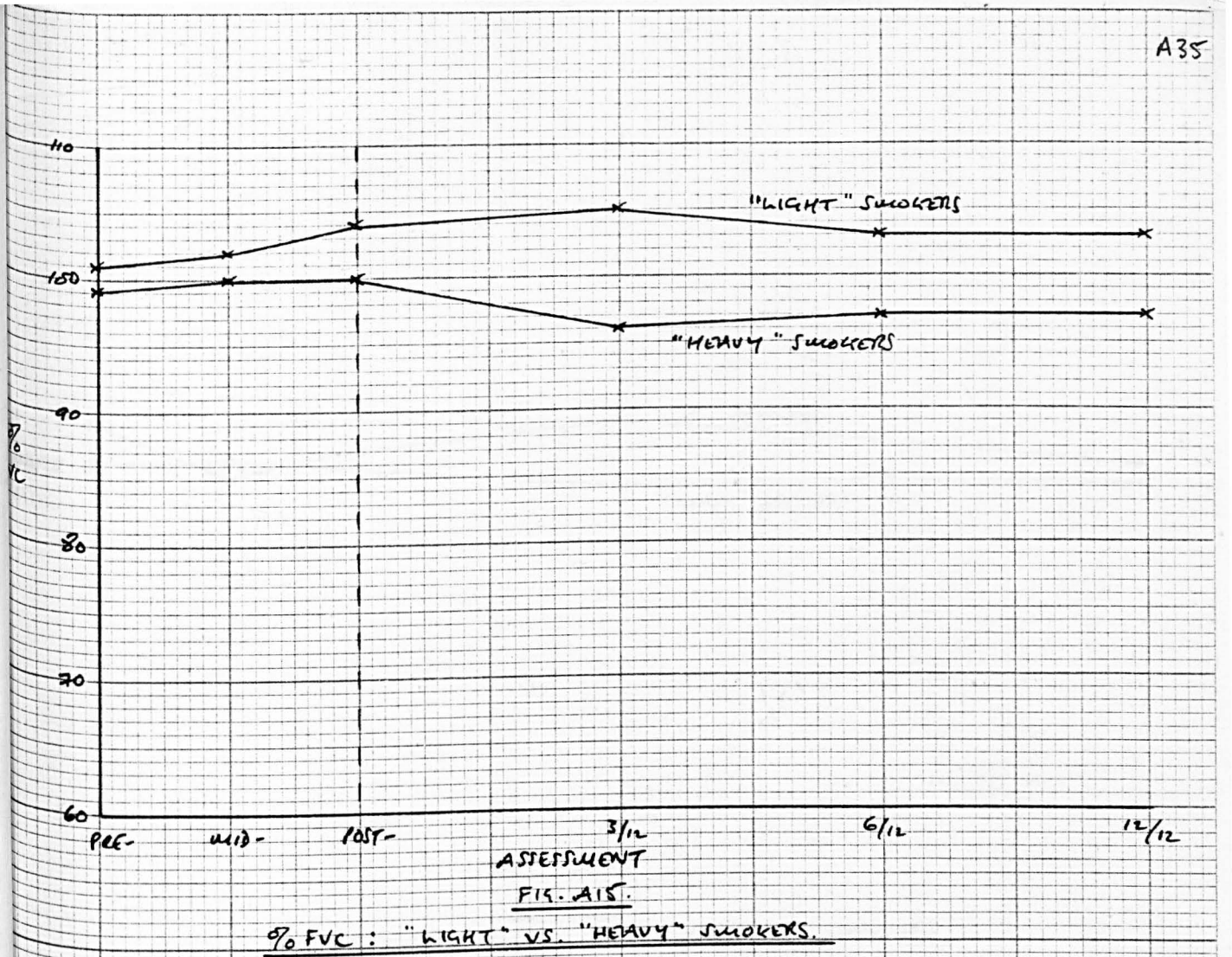

FIG. A15.

% FVC : "LIGHT" VS. "HEAVY" SMOKERS.

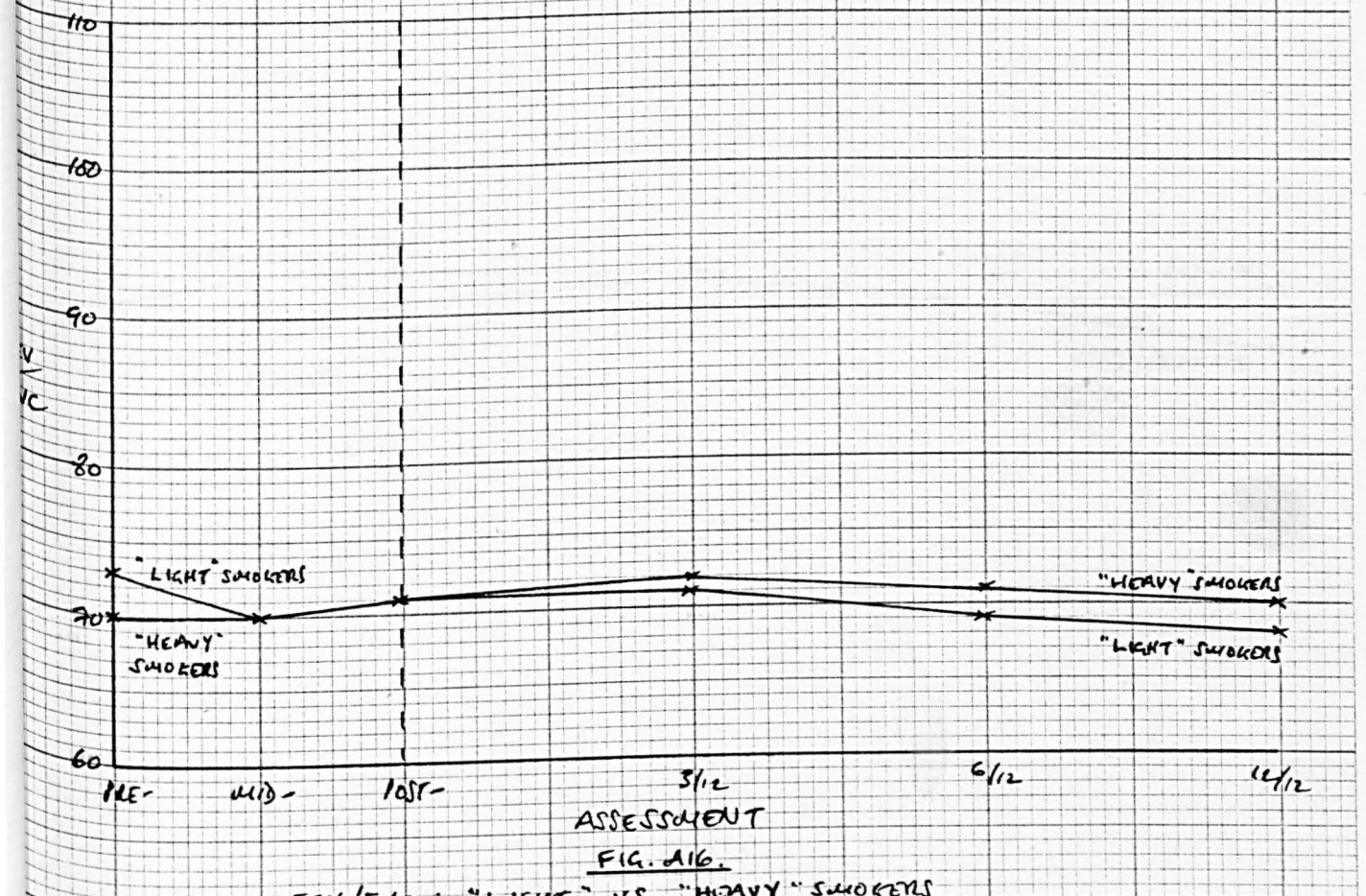

FIG. A16.

FEV/FVC : "LIGHT" VS. "HEAVY" SMOKERS

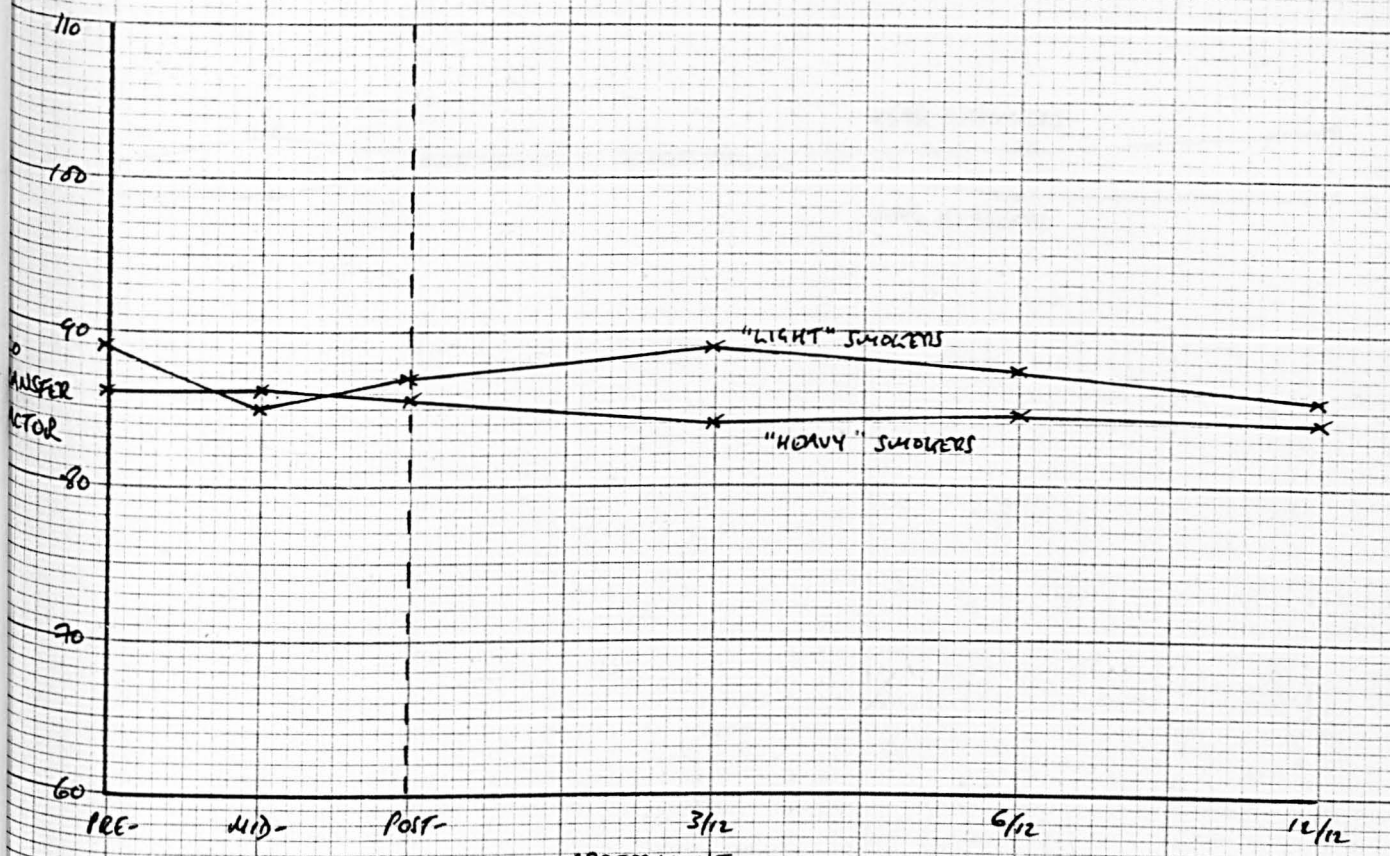

FIG. A17.

CO TRANSFER FACTOR: "LIGHT" VS. "HEAVY" SMOKERS.

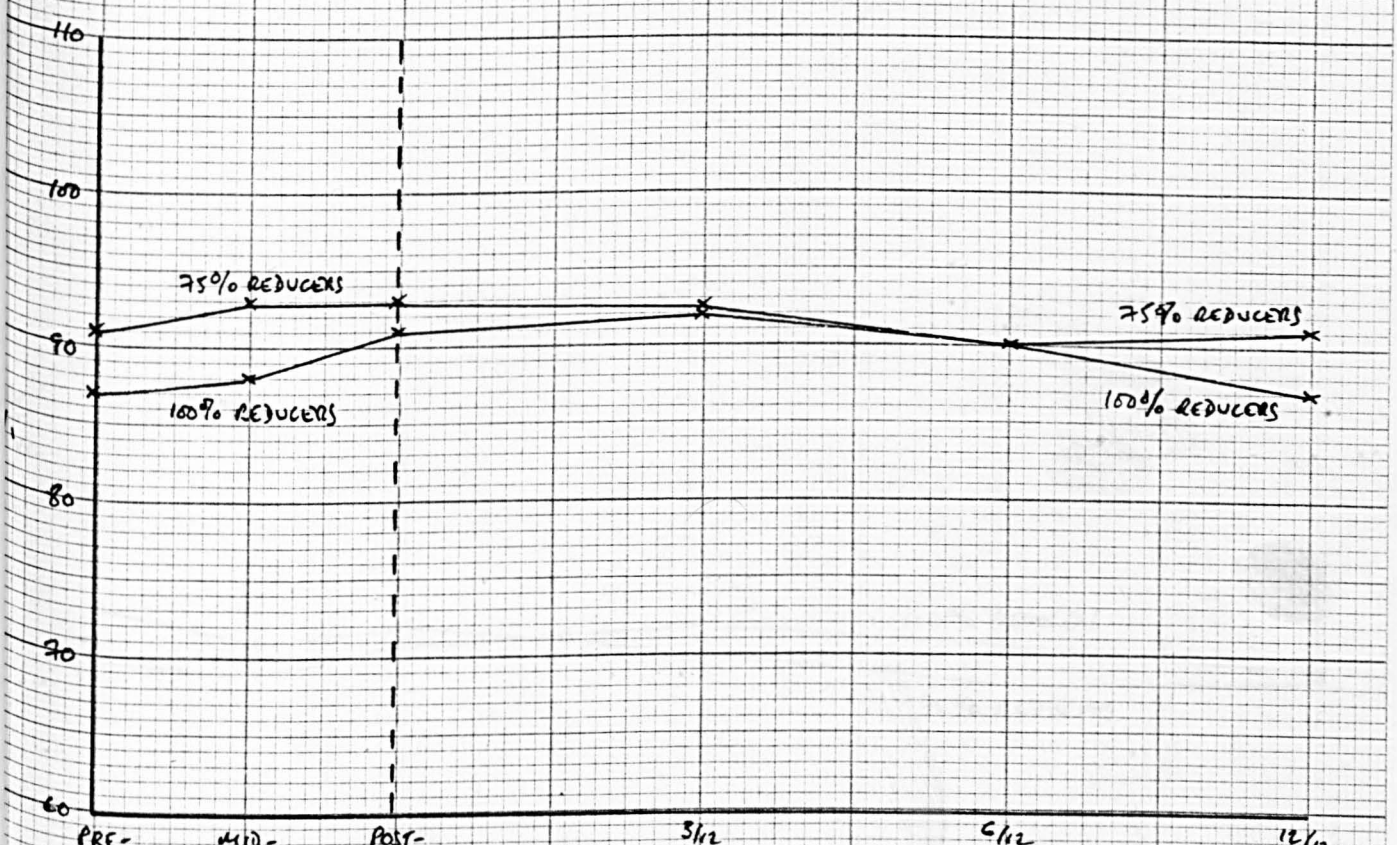

FIG. A18.

% FEV_1 : 100% VS. 75% REDUCERS

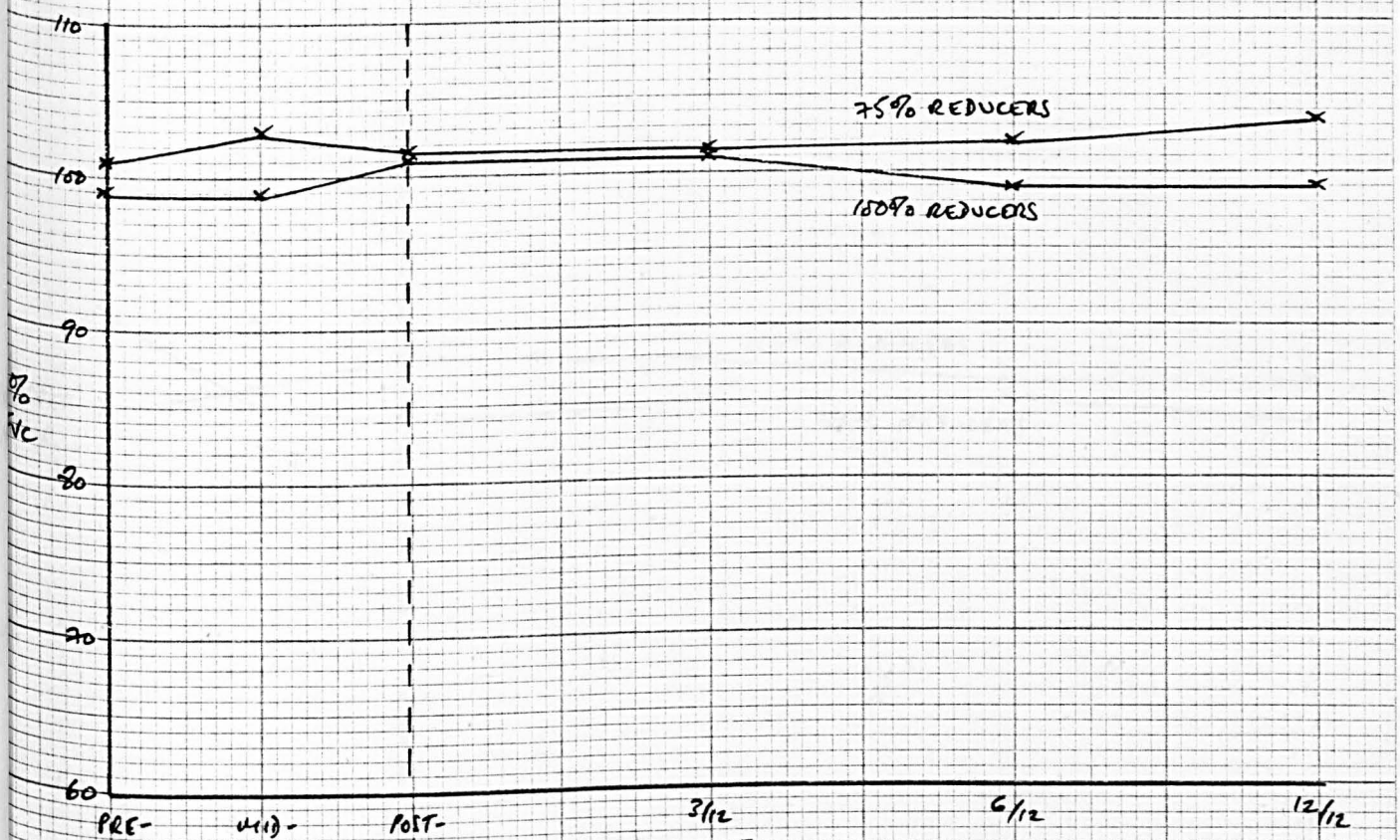

FIG. A19.

% FVC : 100% VS. 75% REDUCERS.

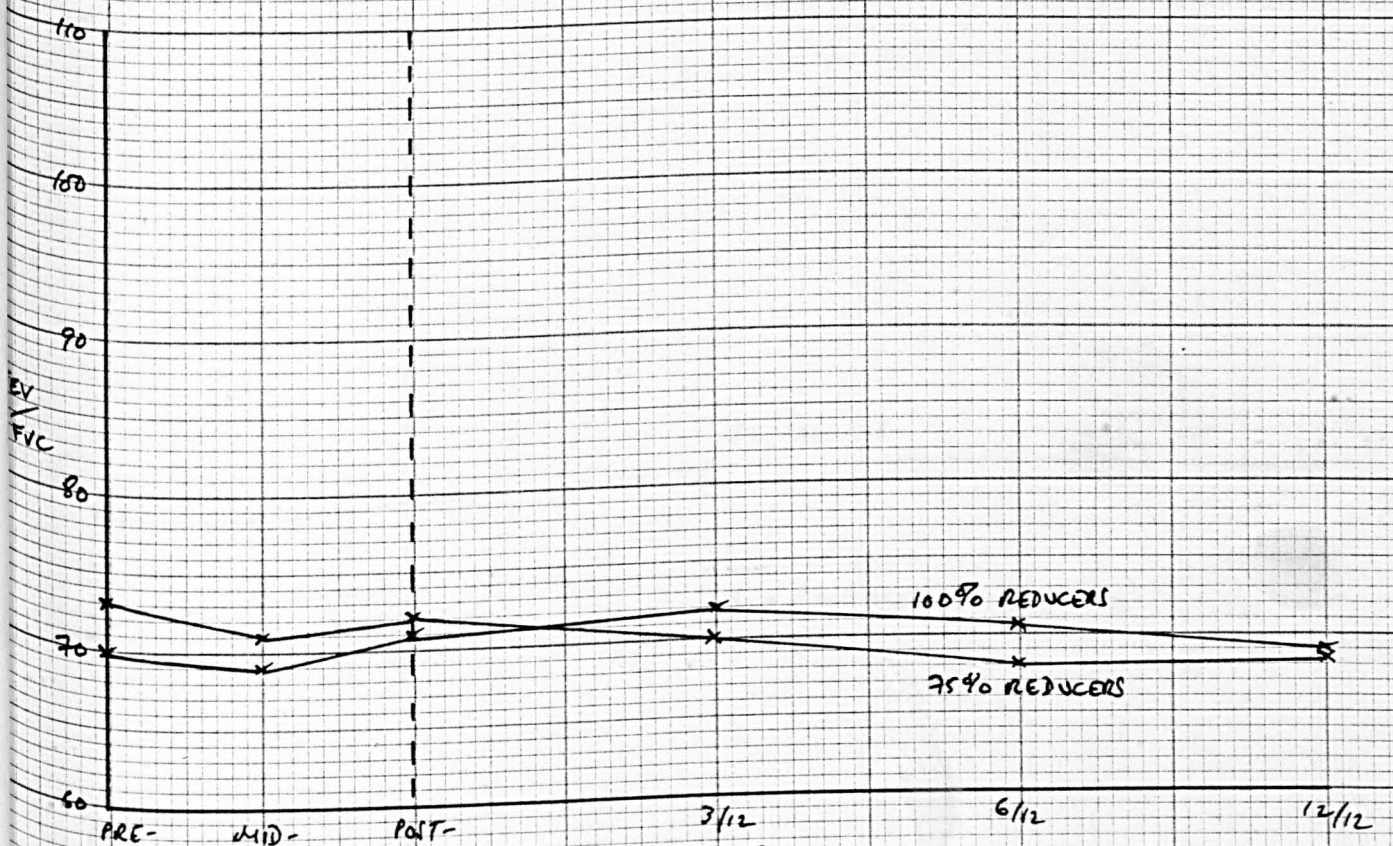

FIG. A20.

FEV / FVC : 100% VS. 75% REDUCERS.

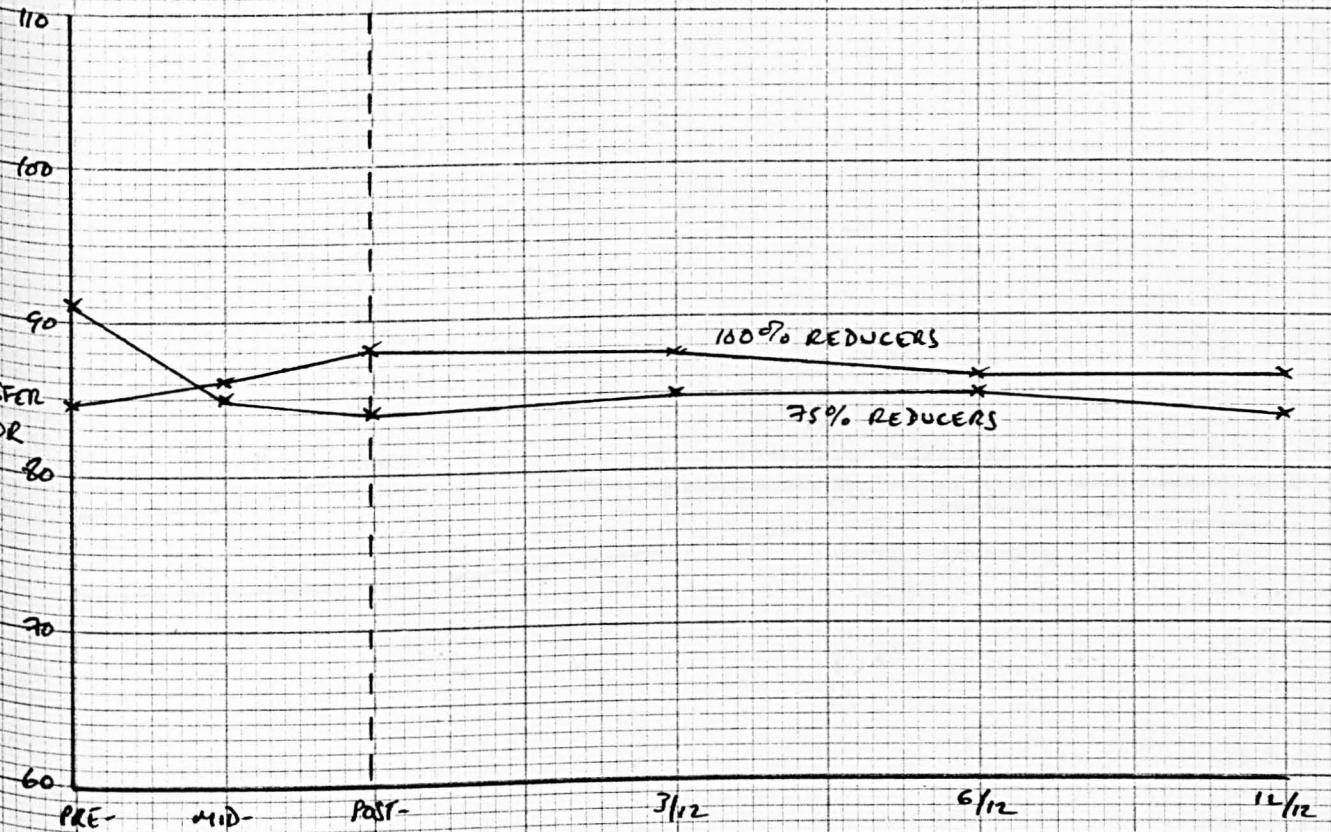

FIG. A 21.

CO TRANSFER FACTOR : 100% VS. 75% REDUCERS.

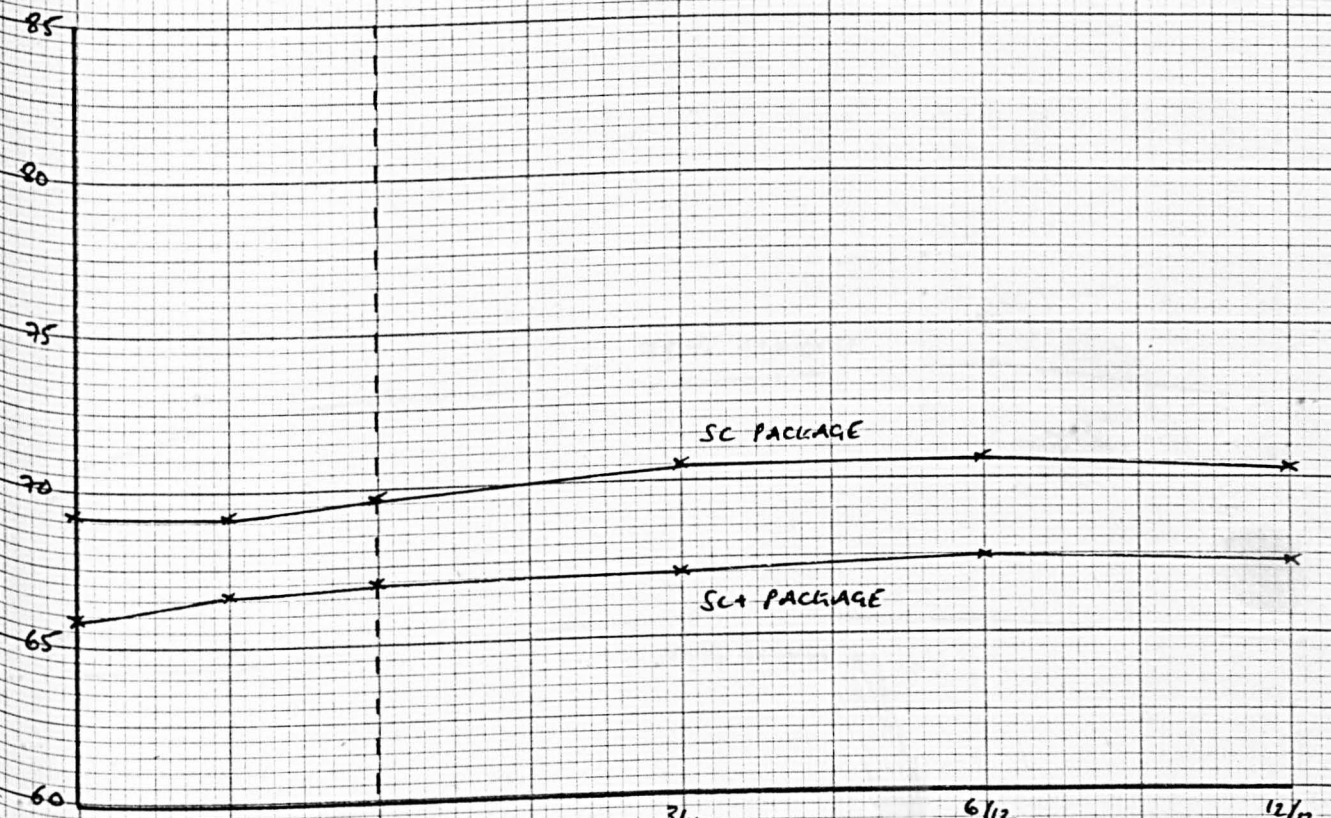

FIG. A 22.

GROSS BODY-WEIGHT : SELF-CONTROL PACKAGE VS. SELF-CONTROL PLUS THERAPIST ADMIN. PACKAGE

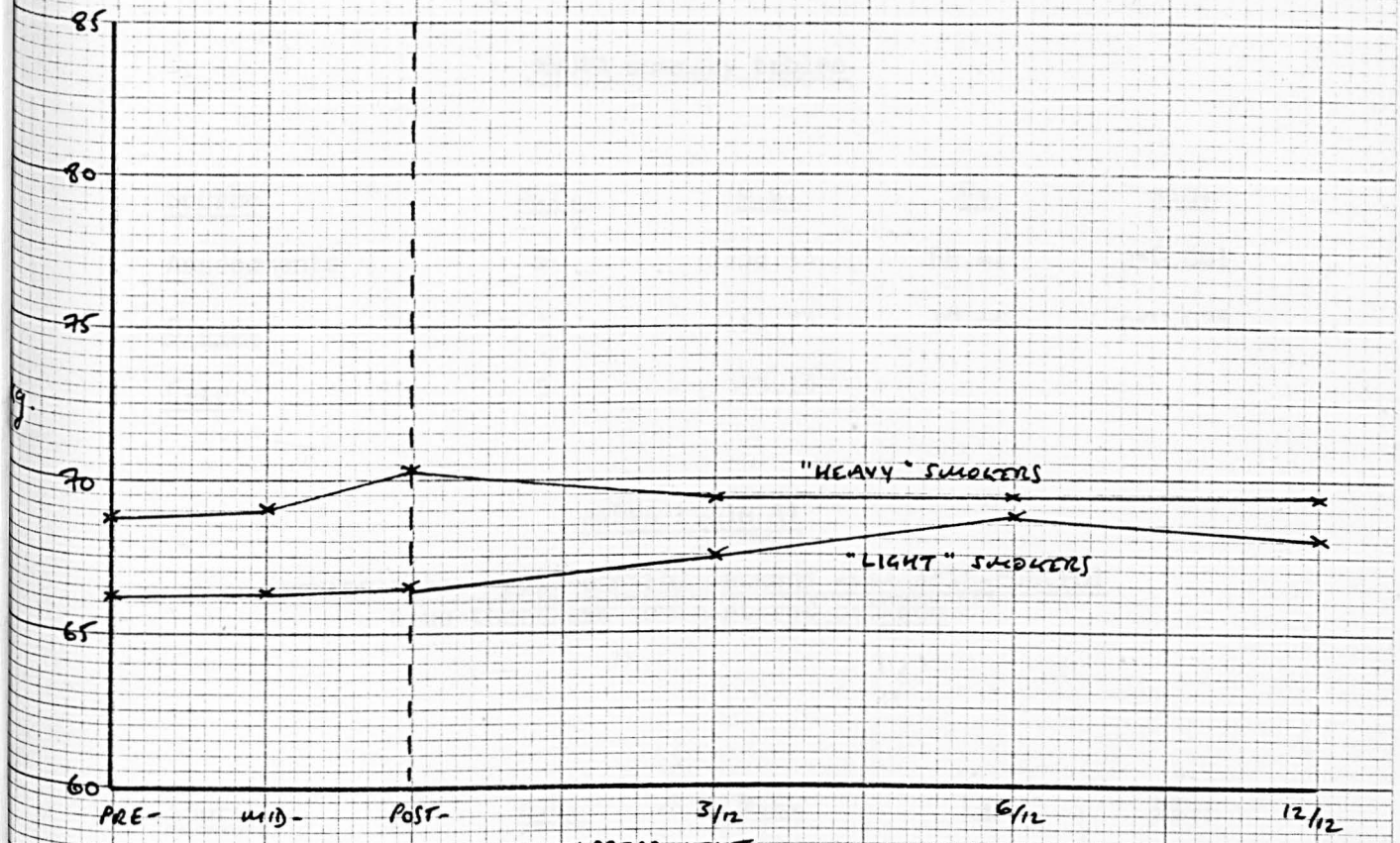

FIG. A 23.

GROSS BODY-WEIGHT: "HEAVY" VS. "LIGHT" SMOKERS.

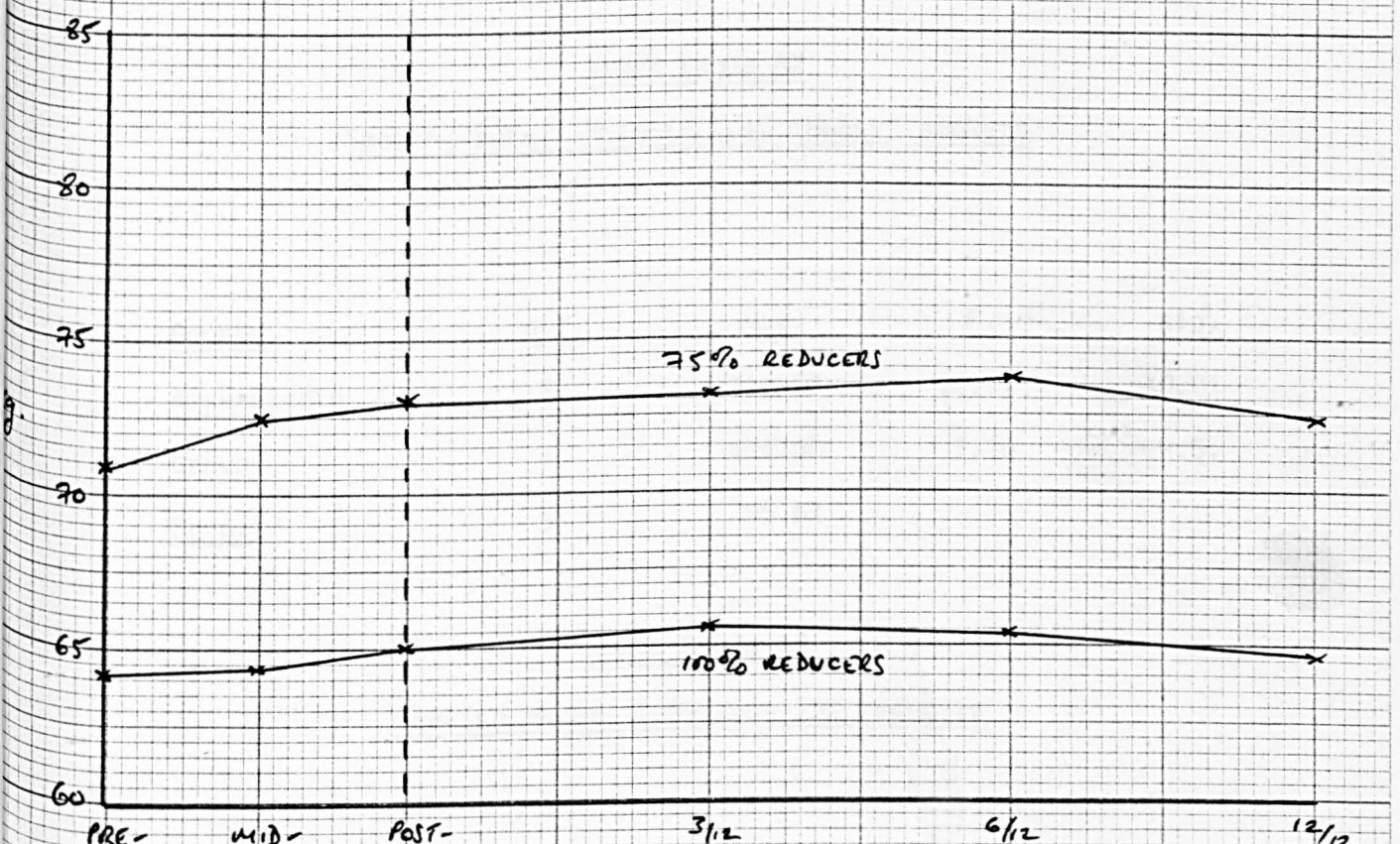

FIG. A 24

GROSS BODY-WEIGHT: 100% VS. 75% REDUCERS.

ANOVA summary tables

Source	d.f.	m.s.	F.	sig.
Assessments	5	1338.13	88.42	p<0.001
Groups	1	161.33	10.66	p<0.025
Error	5	15.13		

ANOVA table A1

Smoking rate: self-control package -vs- self-control plus therapist administered package

Source	d.f.	m.s.	F.	sig.
Assessments	5	1342.63	35.12	p<0.001
Groups	1	0.08	0	N.S.
Error	5	38.23		

ANOVA table A2

Smoking rate: "Light" -vs- "Heavy" smokers

Source	d.f.	m.s.	F.	sig.
Assessments	5	1341.88	14.11	p<0.01
Groups	1	494.08	5.20	N.S.
Error	5	95.08		

ANOVA table A3

Smoking rate: 100% -vs- 75% reducers

Source	d.f.	m.s.	F.	sig.
Assessments	5	138561.48	19.43	p<0.01
Groups	1	21420.75	3.00	N.S.
Error	5	7130.95		

ANOVA table A4

Overall benefit ratings: self-control package -vs- self-control plus therapist administered package

Source	d.f.	m.s.	F.	sig.
Assessments	5	145679.48	40.81	p<0.001
Groups	1	5852.09	1.64	N.S.
Error	5	3569.88		

ANOVA table A5

Overall benefit ratings: "Light" -vs- "Heavy" smokers

Source	d.f.	m.s.	F.	sig.
Assessments	5	132729.53	30.48	p<0.001
Groups	1	83000.33	19.06	p<0.01
Error	5	4354.53		

ANOVA table A6

Overall benefit ratings: 100% -vs- 75% reducers

Source	d.f.	m.s.	F.	sig.
Assessments	5	490.13	4.80	N.S.
Groups	1	48.00	0.47	N.S.
Error	5	102.20		

ANOVA table A7

SCN^-: self-control package -vs- self-control plus therapist administered package

Source	d.f.	m.s.	F.	sig.
Assessments	5	441.48	21.07	$p<0.01$
Groups	1	630.75	30.11	$p<0.01$
Error	5	20.95		

ANOVA table A8

SCN^-: "Light" -vs- "Heavy" smokers

Source	d.f.	m.s.	F.	sig.
Assessments	5	387.47	5.82	$p<0.05$
Groups	1	238.52	3.58	N.S.
Error	5	66.57		

ANOVA table A9

SCN^-: 100% -vs- 75% reducers

Source	d.f.	m.s.	F.	sig.
Assessments	5	4.35	2.81	N.S.
Groups	1	60.75	39.19	$p<0.01$
Error	5	1.55		

ANOVA table A10

$\%FEV_1$: self-control package -vs- self-control plus therapist administered package

Source	d.f.	m.s.	F.	sig.
Assessments	5	1.33	0.62	N.S.
Groups	1	16.33	7.66	$p<0.05$
Error	5	2.13		

ANOVA table 11

%FVC: self-control package -vs- self-control plus therapist administered package

Source	d.f.	m.s.	F.	Sig.
Assessments	5	3.20	6.00	$p<0.05$
Groups	1	0.33	0.63	N.S.
Error	5	0.53		

ANOVA table A12

FEV/FVC: self-control package -vs- self-control plus therapist-administered package

Source	d.f.	m.s.	F.	sig.
Assessments	5	2.00	2.14	N.S.
Groups	1	5.33	5.71	N.S.
Error	5	0.93		

ANOVA table A13

CO transfer factor: self-control package -vs- self-control plus therapist administered package

Source	d.f.	m.s.	F.	sig.
Assessments	5	3.97	6.39	$p<0.05$
Groups	1	14.30	23.03	$p<0.01$
Error	5	0.62		

ANOVA table 14

$\%FEV_1$: "Light" -vs- "Heavy" smokers

Source	d.f.	m.s.	F.	sig.
Assessments	5	1.28	0.35	N.S.
Groups	1	70.08	19.03	$p<0.01$
Error	5	3.68		

ANOVA table A15

%FVC: "Light" -vs- "Heavy" smokers

Source	d.f.	m.s.	F.	sig.
Assessments	5	2.00	1.15	N.S.
Groups	1	0.33	0.19	N.S.
Error	5	1.73		

ANOVA table 16

FEV/FVC: "Light" -vs- "Heavy" smokers

Source	d.f.	m.s.	F.	sig.
Assessments	5	1.68	0.83	N.S.
Groups	1	14.30	7.08	$p<0.05$
Error	5	2.02		

ANOVA table 17

CO transfer factor: "Light" -vs- "Heavy" smokers

Source	d.f.	m.s.	F.	sig.
Assessments	5	3.95	1.83	N.S.
Groups	1	19.76	9.14	$p<0.05$
Error	5	2.16		

ANOVA table A18

$\%FEV_1$: 100% -vs- 75% reducers

Source	d.f.	m.s.	F.	sig.
Assessments	5	0.56	0.37	N.S.
Groups	1	17.76	11.72	$p<0.025$
Error	5	1.52		

ANOVA table A19

%FVC: 100% -vs- 75% reducers

Source	d.f.	m.s.	F.	sig.
Assessments	5	2.78	1.14	N.S.
Groups	1	0.08	0.03	N.S.
Error	5	2.43		

ANOVA table A20

FEV/FVC: 100% -vs- 75% reducers

Source	d.f.	m.s.	F.	sig.
Assessments	5	2.07	0.30	N.S.
Groups	1	1.69	0.24	N.S.
Error	5	6.94		

ANOVA table A21

CO transfer factor: 100% -vs- 75% reducers

Source	d.f.	m.s.	F.	sig.
Assessments	5	70.35	10.22	p<0.025
Groups	1	2670.08	387.89	p<0.001
Error	5	6.88		

ANOVA table A22

Gross body-weight: self-control package -vs- self-control plus therapist administered package

Source	d.f.	m.s.	F.	sig.
Assessments	5	76.92	1.09	N.S.
Groups	1	1419.19	20.19	p<0.025
Error	5	70.29		

ANOVA table A23

Gross body-weight: "Light" -vs- "Heavy" smokers

Source	d.f.	m.s.	F.	sig.
Assessments	5	108.49	6.81	p<0.05
Groups	1	18213.02	1143.96	p<0.001
Error	5	15.92		

ANOVA table A24

Gross body-weight: 100% -vs- 75% reducers