# A CONTROLLED COMPARATIVE INVESTIGATION OF PSYCHOLOGICAL TREATMENTS FOR CHRONIC SLEEP-ONSET INSOMNIA

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## DECLARATION

I declare that this thesis is entirely my own work. The work has not been submitted for any other degree either at this institution or elsewhere.

Colin A Espie

## ABSTRACT

Seventy, GP-referred, sleep-onset insomniacs were randomly assigned to either progressive relaxation, stimulus control, paradoxical intention, placebo or no treatment control groups. Following baseline assessment of sleep pattern and quality subjects received 8 weeks of treatment, comprising 4 weeks under counterdemand and 4 weeks under positive demand instruction to control for demand characteristics and expectancy effects. A further 14 patients were allocated consecutively to a tailored therapy condition as a development of the main study. Measures of treatment process and outcome were obtained from self-report instruments validated against objective monitoring via the "Somtrak" Sleep Assessment Device. Follow-up data were collected at 6 weeks, and 3, 6 and 17 months post-treatment.

Stimulus control was rapidly effective in reducing sleep latency and increases in sleep duration also emerged after a number of weeks. Changes in sleep satisfaction did not, however, parallel sleep pattern change for this group. By contrast, relaxation was associated with marked improvement in qualitative measures and was only modestly effective in altering sleep pattern. Paradox exhibited an initially variable response but, by the fourth week, subjects demonstrated significant sleep latency reduction and some increment in subjective satisfaction. The tailored therapy group did not compare favourably with the randomly allocated groups, suggesting that selection according to individual presentation is less important than the "power" of the treatment applied. Placebo and no treatment subjects did not improve significantly although between group statistical analyses did not always achieve statistical significance. Time within treatment group sub-effects of the MANOVA programme proved particularly useful in data analysis. Treatment effects were maintained at lengthy follow-up,

although a proportion of initially drug-dependent patients resumed medication.

Results are discussed with reference to the literature and to measures of clinical as opposed to statistical significance. The selective impact of the treatments is related to a hypothesised model of differential treatment effect. Guidelines for clinical practice are presented and some suggestions are made for further research.

## LIST OF ABBREVIATIONS

| | |
|---|---|
| ANOVA | Analysis of Variance |
| ARS | Analogue Rating Scale |
| ARS 1 to ARS 10 | Analogue Rating Scale Items 1 to 10 |
| BMDP | Biomedical Data Package |
| C | Self-referred Client (Tables 1-4) |
| CEQ | Credibility Evaluation Questionnaire |
| df | Degrees of Freedom |
| DIMS | Disorders of Initiating and Maintaining Sleep |
| DSQ | Daily Sleep Questionnaire |
| EEG | Electroencephalogram |
| EMG | Electromyogram |
| ENJOY | Rating Scale Measure of Sleep Enjoyment |
| FA | Factor Analysis |
| GP | General Practitioner |
| IRRIT | Rating Scale Measure of Irritability |
| MANOVA | Multivariate Analysis of Variance |
| MMPI | Minnesota Multiphasic Personality Inventory |
| MSA | Measure of Sampling Adequacy |
| MSLT | Multiple Sleep Latency Test |
| NAPDAY | Rating Scale Measure of Daytime Napping |
| P | Patient Presenting at Clinic (Tables 1-4) |
| Prob | Probability |
| R | Recruited Client (Tables 1-4) |
| RAD | Relaxation Assessment Device |
| RAS | Reticular Activating System |
| REM | Rapid Eye Movement Sleep |
| REPTH | Rating Scale Measure of Repetitive Thoughts |
| RESTED | Rating Scale Measure of Sleep Restedness |
| S | Student Sample (Tables 1-4) |
| SAD | Sleep Assessment Device |
| SBRS | Sleep Behaviour Rating Scale |
| SD | Standard Deviation |
| SDT | Signal Detection Theory |
| SE diff | Standard Error of Difference Between Means |
| Sig Level | Significance Level |
| SOL | Sleep Onset Latency |
| SOL SD | Variability (SD) of SOL |
| SPF | Split Plot Factorial |
| SPSSX | Statistical Package for the Social Sciences |
| SWS | Slow Wave Sleep |
| TMAS | Taylor Manifest Anxiety Scale |
| TOT | Total Sleep Duration |
| TOT SD | Variability (SD) of TOT |
| WAKE | Wakening Frequency |
| WAKE SD | Variability (SD) of WAKE |
| WASO | Wake Time After Sleep Onset |
| ZAS | Zung Anxiety Scale |
| ZDS | Zung Depression Scale |

# INTRODUCTION

Insomnia is a common complaint at the GP's surgery. Traditional medical management involves the prescription of hypnotic drugs, but these are often ineffective in the long-term and may pose problems upon withdrawal. The literature on insomnia, however, contains a number of alternative models for the aetiology, maintenance, and management of chronic insomnia. Various behavioural approaches have been investigated and been reported as useful, particularly in the reduction of lengthy sleep-onset latency, and a recent review paper has concluded that "most psychological treatments are helpful, and no one treatment emerges as superior, although stimulus control has an edge" (Lichstein and Fischer, 1985; p. 346).

Most of the research upon psychological therapies has been conducted in the USA using subjects solicited by means of media advertisement. Problems of generalizability to spontaneously presenting clinical populations are, therefore, substantial and the practising clinician remains uncertain of the applicability of psychological procedures to his/her patients, most of whom have chronic and severe sleep problems. In addition, sleep complaints generally reflect both quantitative and qualitative concerns and there is need to investigate the impact of such treatments upon overall sleep pattern and its perceived quality.

The present study was conducted to provide systematic and controlled evaluation, using physician-referred subjects, of three promising psychological treatments for initial insomnia; namely, progressive relaxation, stimulus control and paradoxical intention therapies. The positive selection of the patient group permitted valid assessment of the clinical usefulness of the treatments. Methodological protocols for the management of chronic drug-using patients were also developed based upon the pharmacological and psychological literature.

# CHAPTER 1

## NORMAL SLEEP AND THE PROBLEM OF INSOMNIA

### NORMAL SLEEP PATTERNS

Extensive sleep laboratory studies, employing all-night polysomnographic monitoring, have revealed that there is a typical "architecture" of sleep comprising a number of different sleep stages which present at various times during the night and conform to a common pattern. These stages of sleep fall into two distinct categories; that of orthodox sleep (also known as non-REM sleep) and that of paradoxical sleep (usually termed REM sleep). Within orthodox sleep four separate stages of sleep pattern have been identified, and REM sleep, so called because of the presence of rapid eye movements which are peculiar to this sleep phase, alternates regularly with orthodox sleep approximately five times during the night. These sleep stages have been recognised for at least the past 20 years and the reader is referred to any major textbook on sleep research for detailed description (A recent book by Kales and Kales, 1984, is an excellent source). A brief summary of normal sleep is, however, provided below.

During waking life, the electroencephalogram (EEG) is characterised by high-frequency, low-amplitude brain activity called beta waves. When the eyes are closed, and as relaxation continues, the frequency of this activity reduces and a state of drowsiness is associated with the development of alpha rhythms. The onset of sleep is commonly defined as the point at which these alpha rhythms disappear, although this has been an issue of some dispute (see Chapter 4). Stages 1 and 2 of orthodox sleep then develop with further slowing of EEG frequency and increases in wave amplitude. Theta waves predominate during both of these early stages, although it is possible to discriminate stage 2

sleep by identifying the appearance of either sleep spindles (short bursts of rhythmic, high-frequency waves of uniform amplitude) or K complexes (sudden high-amplitude bipolar spikes of approximately two seconds duration). As sleep deepens further still, larger and even slower delta waves appear within sleep stages 3 and 4 which taken together are known as slow-wave sleep (SWS) (Rechtschaffen and Kales, 1968).

The stages described thus far constitute non-REM sleep. Approximately 1.5 hours after sleep-onset, however, the EEG trace changes and closely resembles that of the waking state except for the presence of regular, low-amplitude, saw-toothed waves, accompanied by electromyogram (EMG) records which indicate an absence of muscle activity. The individual is functionally paralysed. Rapid, darting eye movements are, however, commonly observed, and if subjects are wakened from REM sleep they frequently report vivid and detailed dreams (Dement and Kleitman, 1957). Physiological correlates of REM sleep include increases in blood pressure, heart and respiration rate, body temperature and oxygen consumption. Penile erections also occur in conjunction with REM sleep (summary from Kales and Kales, 1984). The loss of muscle tone and the increased levels of autonomic and neuronal activity have led to REM sleep being described as "an awake brain in a paralysed body" (Coates and Thoresen, 1980).

Clearly sleep is not simply an absence of wakefulness but is a period of continual mental, physiological and behavioural activity, full of complexity, yet following a relatively predictable pattern. It is also worth noting that sleep should not be considered in isolation from wakefulness, but rather as part of a 24-hour wake-sleep biological cycle which varies uniquely from individual to individual.

## SLEEP AND THE AGEING PROCESS

Sleep patterns and sleep needs should be considered within a developmental context. A strong daily cycle of sleeping and waking develops with age. Babies gradually sleep by night and are awake during the day, until as children a routine similar to that of adults is established. The average time spent asleep becomes progressively shorter with increasing age; with newborn infants requiring around 16 hours, compared with 8 hours in young adults (Kleitman and Engelman, 1953; Williams, Karacan and Hursch, 1974). The elderly obtain even less sleep, largely because of more frequent and more prolonged wakenings during the night (Webb and Campbell, 1980; Spiegel, 1981; Hayaschi and Endo, 1982; Webb, 1982). McGhie and Russell (1962) found, in their study of a large sample of subjects in central Scotland, that wakenings were more concentrated during the second half of the night, and were especially problematic for females of middle age or older. The major change apparent in EEG records with increased age is a dramatic reduction in the "deepest" of sleep stages, ie. stages 3 and 4, correlating highly with this propensity toward wakening in older subjects (Coates and Thoresen, 1979). It would seem reasonable, therefore, to regard these age-related changes in sleep as constituting a type of "developmental insomnia".

Descriptive studies have been generally consistent in their findings permitting some confidence that current descriptions of what sleep is are fairly adequate. Understanding of the why of sleep, however, proves more problematic. Since this issue is not of central importance to this thesis it will be dealt with only briefly.

## THE FUNCTIONS OF SLEEP

Several theories regarding the function of sleep have been proposed.

The first of these is that sleep is needed for physical and psychological restoration (Hartmann, 1973; Adam and Oswald, 1977). Evidence for this restorative function comes from studies demonstrating that sleep facilitates the synthesis of protein, and that the secretion of growth hormone is sleep dependent (eg. Adam, 1982). Childrens growth takes place largely during the night and they grow faster at times of good sleep. Since proteins are the building blocks of development it is also thought that tissue restoration progresses at a faster rate during sleep. Horne (1983) has, however, pointed out that, although there is strong evidence for this being so for brain tissue, there is some doubt about the generality of the process.

As far as psychological functioning is concerned studies of sleep deprivation have shown that disorders of thought and perception resembling schizophrenia are provoked by total deprivation of several days' sleep (eg. Johnson, 1969). Fatigue and decrements in performance tasks (eg. reaction time) are also observed during prolonged wakefulness, particularly during the early morning hours when subjects would normally be asleep. Selective deprivation of REM sleep has been said to produce a "hyper-response state" with increased irritability and emotional lability (Agnew, Webb and Williams, 1967). It may be that the recovery of REM sleep is most essential to adequate functioning since REM sleep increases considerably beyond its usual proportion during recovery nights (Dement, 1960). This may reflect the widely held view that REM sleep plays a critical role in memory and learning (eg. Dewan, 1970), and indeed a second theory concerning the function of sleep is that it is central to the human capacity to learn, unlearn and remember. Information may be programmed and memory consolidated during sleep, and, in particular, REM sleep may be responsible for this integration process (Greenberg and Leiderman, 1966).

A further hypothesis, which has been termed the phylogenetic theory, regards sleep as a mechanism for conserving energy. REM periods serve to bring about brief wakenings during this period of defenceless vulnerability, and prepare the organism for fight or flight without disturbing sleep continuity (Snyder, 1966). The finding that humans waken more readily from REM sleep than slow wave sleep is consistent with this theory (Rechtschaffen, Hauri and Zeitlin, 1966), however, other workers have reported contrary findings in that arousal from REM sleep is more difficult in animals, and some defenceless animals have considerably less REM sleep than do their predators (Drucker-Colin, 1979). In a similar vein, Webb (1979) has forwarded the ethological theory which is another attempt to establish the "survival value" of sleep. He suggests that ecological pressures exerted upon the organism make non-responding important, and that sleep effectively removes the individual from the environment at times when waking activities would be dangerous or otherwise maladaptive.

In reviewing the literature on the "why" of sleep it is evident that no single explanation enjoys general acceptance, although the theories are not necessarily in competition and are not mutually exclusive. Having considered thus far the pattern and role(s) of normal sleep, the background is in place to examine the sleep disorders themselves.

## DEFINITIONS OF INSOMNIA

For the purposes of this thesis, those sleep disorders which do not have a direct bearing upon insomnia itself will be referred to only briefly. The Association of Sleep Disorders Centres has published a "Diagnostic Classification of Sleep and Arousal Disorders" (Sleep, 1979, 2(1)) which is also summarised in the American Psychiatric Association's (1980) Diagnostic and Statistical Manual of Mental Disorders (DSM III). This system separates the insomnias, defined as

"disorders of initiating and maintaining sleep" (DIMS), from three other major categories of sleep disturbance. These are a) disorders of excessive somnolence (including narcolepsy and hypersomnolence), b) disorders of the sleep-wake schedule (exemplified by jet-lag and the effects of shift-work upon the circadian rhythm), and c) the parasomnias (a heterogeneous group including sleep-walking, sleep terrors and sleep-related enuresis). Under the DIMS classification nine sub-types are identified as follows:-

1. Psychophysiological DIMS
2. DIMS associated with psychiatric disturbance
3. DIMS associated with drug and alcohol use
4. DIMS associated with sleep-induced respiratory impairment
5. DIMS associated with myoclonus and "restless legs" syndrome
6. DIMS associated with medical, toxic, environmental conditions
7. Childhood-onset DIMS
8. DIMS associated with other conditions
9. No DIMS abnormality (according to EEG criteria)

The reader's attention is directed towards the primary insomnias, that is those which entail "a persistent inability to obtain adequate sleep" (Williams et al, 1974; Ribordy and Denney, 1977; Bootzin and Nicassio, 1978), and which are not secondary to medical or psychiatric illness. In terms of the classification, therefore, sub-groups 1, 2 and 9 provide the focus of interest, with the remainder being of concern solely for the purposes of exclusion. Psychophysiological insomnia can be differentiated from subjective insomnia (sub-type 9) in that the former is diagnosable upon examination of EEG records; that is polygraphic evidence confirms self-report. Subjective insomnia, however, refers to a sleep problem, not substantiated by EEG, which has led some workers to doubt the validity of the complaint. Recent evidence, however, has questioned the accuracy with which EEG can identify sleep-onset within the insomniac group. (These issues will be fully addressed in Chapter 4, when the relationship between subjective and objective assessment will be considered. Criteria for the exclusion of other DIMS categories will also be dealt with at that point). DIMS

associated with psychiatric disturbance (sub-type 2) are also of considerable interest since this category covers sleep disorders where neurotic profiles are elevated in simple association with poor sleep as well as those which are an expression of underlying psychopathology. The relationship between personality, anxiety, depression, and clinical insomnia is discussed in Chapter 3 .

A number of studies have compared the sleep patterns of normal subjects with insomniacs using patient samples rarging from 11 to 18 per group (Monroe, 1967; Karacan, Williams, Salis and Hursch, 1971; Karacan, Williams, Littell and Salis, 1973; Frankel, Coursey, Buchbinder and Snyder, 1976; Gillin, Duncan, Pettigrew, Frankel and Snyder, 1979; Gaillard, 1978). The most extensive study, however, has been that of Kales, Bixler, Vela-Bueno, Cadieux, Soldatos and Kales (1984) who evaluated the sleep of 150 insomniacs (age range 19-90 years) and compared sleep characteristics with 100 normal control subjects. Insomniacs were found to have significantly longer mean sleep-onset latencies (SOL), within each of the age-bands studied, thus confirming the results of the earlier, less extensive reports. This increased sleep latency accounted for the greater "total wake time" also experienced by the insomniac group. No significant differences were found, however, on the amount of wakefulness after sleep-onset, a result which confirms the findings of Karacan et al (1971), but contrasts with those of Monroe (1967) and Gaillard (1978). Kales et al also considered the duration of nightly wakenings, however, and found that more protracted wakenings were typical of the insomniac group. In other words, insomniacs had greater difficulty returning to sleep. In common with the research literature on age-related changes in sleep patterns, these authors also found that wakenings after sleep-onset increased with age in both experimental groups, suggesting that this type of developmental, sleep-maintenance insomnia is, in fact, non-

pathological, despite the distress which it causes to many. Kales et al, therefore, concluded that "the insomniac's primary difficulty is initiating sleep, whether at the beginning of the sleep period or following awakenings during the night".

## THE PREVALENCE OF INSOMNIA

It has been reported that more than 50% of adults complain of current or past sleep disorder and 38% experience current difficulties with sleep (Bixler, Kales, Soldatos, Kales and Healey, 1979). Insomnia was by far the most prevalent problem in Bixler et al's study, accounting for 32% of the present sleep disorders; a finding which is in broad agreement with other reports where 10-15% of subjects experienced mild insomnia, and a further 10-15% severe or frequent insomnia (Kales, Bixler, Leo, Healey and Slye, 1974; Montgomery, Perkin and Wise, 1975) In an American national prospective study of over 1 million men and women, Hammond (1964) reported that 13% of men and 26.4% of women complained of insomnia, and, in the United Kingdom, samples have yielded prevalence rates of 18-25% (Shepherd, Cooper, Brown and Katton, 1966; Dunnell and Cartwright, 1972). In addition, McGhie and Russell (1962) reported that more than 15% of their Scottish sample suffered from chronic insomnia. Difficulties in sleeping are not, however, confined to the adult population since Price, Coates, Thoresen and Grinstead (1978) found that 13% of high school students were sleep-disturbed on four or more nights per week, with a further 33% complaining of occasional sleep difficulty.

Apart from these prevalence figures, evidence for the widespread problems posed by poor sleep can be drawn from studies which have reported on the prescribing of sleep medication. In spite of the now total withdrawal of barbiturates, other non-barbiturate and

benzodiazepine hypnotics are commonly prescribed. These have remained the medical treatment of choice, although there is increasing evidence against the long-term effectiveness of chemotherapy (Kales, Bixler, Tan, Scharf and Kales, 1974; Oswald, 1979; Kales, Soldatos, Bixler and Kales, 1983). In the USA, studies have found that as many as 82% of patients presenting at sleep clinics make regular use of night-time medication (Roth, Kramer and Lutz, 1976), and Cooper (1977) reported on the alarming 27 million prescriptions which were written for hypnotic drugs during 1976 alone. In that same year, a UK survey indicated that 70% of all prescriptions for hypnotics had been written by the receptionist, compared with only 22% for other types of drugs (Freed, 1976).

Having recognised the prevalence of sleep disorders and the considerable management and financial issues involved in their treatment, it is now important to consider some useful theoretical models of the causation and maintenance of insomnia.

# CHAPTER 2

## AETIOLOGICAL AND THEORETICAL FACTORS

In this chapter a number of factors of proposed aetiological and theoretical significance, for primary insomnia, will be discussed in some detail. It will become clear that no definitive explanation can be given regarding the causation and maintenance of insomnia, but recent authors are agreed that models based upon the following theoretical positions are meritorious and worthy of further investigation (Coates and Thoresen, 1980; Turner and DiTomasso, 1980; Borkovec, 1982).

1. Physiological hyperarousal
2. Maladaptive operant learning
3. Heightened anxiety, especially performance anxiety
4. Pre-sleep cognitive intrusion
5. Sleep pattern variability and impoverished self efficacy
6. Dysfunction of the sleep-wake system

A review of the evidence supporting each of these theories will be presented.

### PHYSIOLOGICAL HYPERAROUSAL

In 1967 Monroe conducted an extremely influential study comparing good and poor sleepers on a number of physiological and psychological variables. His results gave rise to the view that poor sleepers exhibit heightened autonomic arousal (higher rectal temperature, vasoconstrictions per minute, perspiration rate and skin conductance, body movements per hour) both prior to and during sleep. The intuitive appeal of these findings along with the common use in clinical practice of progressive relaxation techniques (based upon Jacobson, 1938) may, however, have afforded this model a somewhat overvalued status since adequate replication studies have not been forthcoming. Johns, Gay, Masterton and Bruce (1971) did find higher levels of adrenocortical activity in their insomniac group, but other workers have failed to reproduce this effect (Frankel, Buchbinder, Coursey and Snyder, 1973).

The relationship between EMG-defined muscle tension and insomnia was investigated by Haynes, Follingstad and McGowan (1974) and Good (1975), however, neither study found a significant association. Similarly, Slama (see Borkovec, 1979) found no differences in skin conductance and heart rate levels between insomniacs and good sleepers, although the data from this study do refer to observations made during a day-time nap session. Borkovec also quotes other unpublished studies on student populations which yield inconsistent results, and he draws attention to research from two related areas which pose serious problems for the potential mediational role of physiological hyperactivity.

Firstly, length of latency to sleep-onset has not correlated with heart rate or frontalis EMG levels (Haynes et al, 1974; Good, 1975; Browman and Tepas, 1976) or even with physiological activity purposefully elevated by physical excercise (Hauri, 1968); and secondly, numerous outcome investigations of relaxation techniques have failed to yield significant correlations between sleep improvement and within-therapy assessments of change on various physiological measures, including heart and respiration rate, and forearm and frontalis EMG (Borkovec and Fowles, 1973; Haynes, Sides and Lockwood, 1977; Freedman and Papsdorf, 1976; Lick and Heffler, 1977; Borkovec, Grayson, O'Brien, and Weerts, 1979; Coursey, Frankel, Gaarder and Mott, 1980; Hauri, 1981). In addition, Borkovec (1982) has suggested a further potentially confounding variable. He observed that a number of his sleep disturbed cases exhibited phase-shift desynchronies, which implies that alterations in circadian rhythm may be a sufficient explanation for observed arousal differences.

There are also a number of methodological difficulties with the research reports in this area. Firstly, there are relatively few reports and most have not differentiated adequately between good and

problem sleepers in order to be convincing comparisons of two distinct groups. Secondly, studies have approached insomnia as a unitary phenomenon and have failed to consider specific sub-groups of insomniacs, some of whom may be more typically "physiological responders" than others. It may be that the Three Systems Model (Lang, Rice and Sternbach, 1972), as employed elsewhere in the psychological literature, could be fruitfully applied to the analysis of sleep disorders by careful consideration of each individual's characteristic behavioural, cognitive and physiological responses. Finally, the clinical utility of relaxation techniques has afforded a validity to the hyperarousal theory which has not been amply justified. The reported success of this treatment may not be presumed to operate via autonomic de-arousal. The role of a physiological mechanism in the aetiology and maintenance of insomnia does, therefore, await further innovative study.

MALADAPTIVE OPERANT LEARNING

Bootzin (1972) has proposed an operant learning concept of sleep disturbance where the conditioning of sleep-incompatible behaviours to bed-related stimuli may play a significant role in the creation and maintenance of insomnia. Thus a treatment which eliminates such associations and pairs rapid sleep-onset with bed cues should be therapeutic. Within this stimulus control model, primary insomnia is thought to result from bed-time behaviour which vitiates the bed's "cueing potential" for sleep ; that is its potential to act as a discriminatory stimulus for sleep. Stimulus control treatment, therefore, aims to disassociate activities such as reading, watching television, eating and smoking from the bedroom environment, since these are most appropriately conducted elsewhere, and, thereby, to establish a bedtime routine which is conducive to sleep. The emphasis

is, therefore, upon altering habitual maladaptive patterns to facilitate the development of a strong sleep rhythm. See Chapter 7 (Methodology) for details of the therapeutic instructions.

Zwart and Lisman (1979) conducted a component control analysis aimed at isolating the critical mechanism involved, and reported that the active ingredient of stimulus control may be that of doing "something else" other than trying to sleep, if sleep has not rapidly occurred. This seemed to be the case whether the chosen activity could be deemed sleep-compatible or sleep-incompatible. Furthermore, their temporal control strategy (do not nap during the day and rise at the same time each morning) proved to be as effective as the complete set of stimulus control instructions, which suggests that the stimulus control strategy may function through an improved harmonisation of the individual's circadian cycle rather than via the hypothesised reconditioning of responses to the environment. In further contrast to the predictions of the theory insomniacs have not been found to engage more frequently in overt sleep-incompatible behaviours in bed than non-insomniacs (Haynes et al, 1974).

Another, alternative explanation for the impact of stimulus control is that it may preclude arousing cognitive events from the bedroom and the immediate pre-sleep period, and achieve its therapeutic effect through the reduction of worry and arousing efforts to initiate sleep. Turner and Ascher (1979b), for example, have reported that all six subjects in their study found that the therapy instructions served to "break up lying in bed and thinking behaviour". Many adults appear to use the first minutes after retiring to ponder the day's activities and to plan strategies for the next day, and Turner and Ascher concluded that "this reflection and problem-solving activity became habitual (for their clients) and out of the individual's control ——— The stimulus control

instructions seemed to provide them with a means of asserting self control over their bedtime cognitions by simply getting out of bed".

It seems, therefore, that in spite of the encouraging results from treatment studies of stimulus control procedures, the theoretical rationale for its mode of action has yet to be satisfactorily explained. The applicability of the concept of stimulus control as a description of the therapeutic process is certainly in some doubt.

HEIGHTENED ANXIETY - ESPECIALLY PERFORMANCE ANXIETY

Research studies have consistently found that poor sleepers score more highly on measures of anxiety and neurotic depression than do good sleepers (see Chapter 3). In addition, it is a common clinical observation that insomniacs experience considerable anticipatory anxiety as bedtime approaches. Some researchers have focused, therefore, upon the role of anxiety as a potential aetiological factor in primary insomnia. Anxiety-based models can be readily sub-divided into two categories; namely, that of performance anxiety mediation, where the technique known as paradoxical intention has been employed as a therapeutic strategy; and that of pre-sleep cognitive intrusion where methods derived from cognitive-behavioural theory have recently gained some popularity. It is the purpose of this section to consider the former, and the latter will be dealt with in the section to follow.

The performance anxiety formulation proposes that anxiety responses may be conditioned not only to external, situational cues but also to the individual's own behaviour or performance. Fear of a performance failure (and of the anticipated negative consequences of that failure) is often described as performance anxiety. The victim of this anxiety predictably attempts to immediately control and correct the deviant performance through deliberate coping efforts, but these efforts in

turn may only contribute to longer term exacerbation of the original difficulty. The development of a vicious circle of performance fear and failure is particularly evident when related to the maintenance of control over physiological processes (Ascher, 1979). According to the model sleep is, similar to other autonomic responses, considered to be a member of a class of behaviours which cannot fully be placed under voluntary control. The individual can arrange conditions which are conducive to sleep (eg. darkened room, restful quiet) but there is a point beyond which further, deliberate control cannot be exerted. At this juncture attempts at sleep are usually relinquished, allowing sleep to develop naturally, however, it is hypothesised that those who suffer from insomnia exacerbate their problem by endeavouring to maintain direct control over the sleep process itself, and the resultant "effort to sleep" inhibits sleep-onset, either directly, or through increased physiological and/or cognitive arousal. The paradoxical instruction designed to obviate this performance anxiety is to attempt to remain awake, yet relaxed, in an environment optimal for sleep development. The preclusion of attempts to fall asleep, therefore, is thought to enable the subject to sleep naturally.

To date, there have been no systematic investigations of this model, although once again it can be seen to have some intuitive appeal. Patients commonly report frustration and self-recrimination regarding their inability to sleep, and are often very concerned about the daytime sequelae of inadequate sleep (Espie and Lindsay, 1985). The research literature is limited to clinical reports, although two recent studies have highlighted some practical and theoretical problems with the procedure. Firstly, Fogle and Dyal (1983) found that simply telling subjects to "give up" trying to sleep and to stop worrying about the negative consequences of sleep loss was as effective in improving sleep estimates as the paradoxical directive to remain awake. Interestingly,

however, only the "giving up" strategy produced a reduction in a self-report measure of performance anxiety. This study, therefore, raises the question of the critical ingredient in paradox. It also suggests that paradoxical intention may function irrespective of reduction in performance anxiety levels. It may be, for example, that patients modify their expectations of sleep and redefine sleeplessness as less aversive or problematic. For some, therefore, a reduction in anxiety concerning sleep may be a sufficient therapeutic outcome, regardless of changes in sleep pattern, whereas for others improved sleep efficiency may be satisfactory irrespective of performance fear. Secondly, it has been suggested that the instruction to remain awake may be followed literally by some patients to the point where "effort to sleep" is replaced by "effort to remain awake", in order to comply with therapeutic instructions. In other words, performance anxiety may not be obviated but refocused (Espie and Lindsay, 1985). These workers reported on several cases where paradox exacerbated the initial sleep problem, and they suggested that "effort to remain awake" could be construed as an active, arousing process compared with the more passive avoidance of effort to sleep.

There is need for research to investigate carefully the mechanism(s) by which paradoxical procedures achieve their therapeutic effect. Strong (1984), in a recent review article, has suggested that investigations should work towards an understanding of "the relations of voluntary and involuntary responses to psychological symptoms". The role of performance anxiety in paradoxical treatment is unclear. It may be that the performance-based model is a better explanation for the maintenance (rather than causation) of insomnia since an extended period of disturbed sleep could conceivably condition an apprehensive expectation of insomnia rather than representing a primary aetiological mechanism (Kales, Caldwell, Soldatos, Bixler and Kales, 1983).

Reference has been made to a number of reports which have suggested that cognitive mediational variables may account for the effectiveness of psychological treatments. In addition to the generally heightened level of anxiety characteristic of the insomniac group, a number of workers have described insomniacs as obsessive, repetitive worriers (Coursey, Buchsbaum and Frankel, 1975; Roth et al, 1976) who have difficulty in clearing their minds of anxious mentation, and more specifically, suffer from persistent cognitive intrusion prior to sleep-onset (Haynes et al, 1974; Borkovec et al, 1979; Mitchell,1979; Lichstein and Rosenthal, 1980). This state of cognitive hyperarousal at bedtime is, according to the model, responsible for inhibiting the de-arousal and relaxation necessary for sleep induction, and may become an habitual mechanism for problem-solving and goal planning. Other reports,however, have obtained contrary results where pre-sleep mental stress was not significantly associated with onset insomnia (Haynes, Adams and Franzen, 1981; Freedman and Sattler, 1982) although the latter study was conducted during a single night's recording in a sleep laboratory where worrying would perhaps be less likely owing to the novelty of the environment and its associated distraction potential.

Further investigation of the role of mental events has been undertaken by Hauri (1975) and Borkovec (1979) who have developed what is known as the "sleep system model", which hypothesises that the Reticular Activating System (RAS) regulates wakefulness while the serotonergic sleep system controls sleep. Low RAS activity can produce quiet wakefulness (and possibly stage 1 non-REM sleep), however, the active induction of the sleep system may be required to produce stage 2, 3 and 4 sleep. Hauri (1975) has proposed that insomnia may, therefore, result from high RAS activity and/or a weak sleep system. Borkovec (1979) has

suggested that the balance of the wakefulness/sleep systems differs between two sub-groups of insomniacs, ie. objective (EEG-confirmed) and subjective (experiential only) insomnia, in that the latter is a function of a normal sleep system in combination with overly active wakefulness, whereas objective insomnia is hypothesised to be the result of a weak serotonergic system. Evidence for this analysis is presented in Borkovec et al (1979) who found that their group of experiential insomniacs retrospectively reported a significantly greater amount of pre-sleep mentation than did their psychophysiological insomniacs. On the basis of this finding, these workers hypothesised that subjective insomnia may be the result of affect-laden pre-sleep cognitions which are difficult for subjects to differentiate from early sleep mentation, whereas mental correlates of psychophysiological insomnia are less affect-laden. This theoretical model goes some way towards explaining both the common reports of cognitive intrusion in the insomniac population, and also the recognised difficulty which many poor sleepers have in distinguishing early sleep from wakefulness (see Chapter 4).

A more recent study has provided further, moderate support for a relationship between cognitive factors and the subjective experience of onset insomnia (Van Egeren, Haynes, Franzen and Hamilton, 1983), although Borkovec's theory regarding a differential causal mechanism, incorporating the role of affect, was not substantiated. Van Egeren et al employed a multiple regression paradigm, the results of which suggested that the anxious content of pre-sleep cognitions and the attributions of sleep difficulties were significantly associated with sleep-onset latency. They concluded that "although at an early stage of conceptual and methodological development, research designs to identify specific pre-sleep cognitive activities and beliefs can provide the basis for the development of effective cognitive interventions for the

treatment of sleep-onset insomnia". However, their findings are also limited, by their own admission, that "a significant proportion of the variance of the sleep quality variables (eg.restedness) remains unaccounted for and requires additional investigation". The relationship between sleep satisfaction and mental stress at bedtime has yet to be investigated.

## SLEEP PATTERN VARIABILITY AND IMPOVERISHED SELF EFFICACY

There is some evidence to suggest that insomniacs exhibit greater night to night variability on most sleep parameters compared with good sleepers (study by Coates, Strossen, Rosekind and Thoresen referred to in Killen and Coates, 1979). It would seem possible, therefore, that due to this inherent unpredictability of what sleep will be like on any given night, the insomniac might consider his sleep pattern (and his sleep problem) to be uncontrollable. Killen and Coates (1979) proposed that the uncertainty which results from a variable sleep pattern might interact with other worries and concerns to inhibit sleep, or foster the perception that sleep is much worse than it really is. According to this model, therefore, insomnia develops as an anxiety response to uncertainty regarding sleep and is associated with a reduction in self efficacy; that is the perception of one's own ability to manage and modify a problem. A study by Evans (1977) attempted to identify the characteristics of sleep efficiency which are of principal concern to poor sleepers. Interestingly, the factor of "voluntary control of sleep" emerged to account for the greatest proportion of variance, indicating the perceived importance of the "decision process" in sleep onset (cf. paradoxical intention). If this process is disturbed, or rendered ambiguous, because of a highly variable sleep pattern, it would seem probable that anticipatory anxiety would increase and positive perceptions of self efficacy would reduce. As Bandura (1977)

has observed, however, the cognitive processing of efficacy information is likely to differ from individual to individual with differing expectations and anticipations arising from similar experiences of mastery and failure.

One difficulty emerging from the available literature has been the persistent use, in both descriptive and statistical analyses, of weekly mean values to quantify sleep pattern [eg. mean score (over 7 nights) for sleep latency (minutes)]. Average values, however, convey little impression of the night to night variability of sleep which may be typical of insomnia. A concomitant measure of variance would appear, therefore, to be necessary.

## DYSFUNCTION OF THE SLEEP-WAKE SYSTEM

It is a common experience for shift workers and airline personnel to develop sleep problems when they are forced to obtain sleep at times when circadian cycles favour wakefulness (Rutenfranz, Colquhoun, Knauth and Ghata, 1977). Irregular non-24 hour schedules, in which sleep and wake times are shifted frequently, have been found to decrease sleep efficiency and total sleep time and to increase time awake during the night and daytime sleepiness (Webb and Cartwright, 1978; Webb and Agnew, 1978). A number of studies have demonstrated that advancing, delaying, extending or reducing established sleep periods produces measurable deficits during waking behaviour (Taub and Berger, 1973; Taub and Hawkins, 1977), and chronically irregular sleepers have been found to have demonstrably lower physiological arousal, poorer psychomotor performance and a greater frequency of negative mood states than sleepers who maintain regular sleep-wake cycles (Johnson, 1973; Webb and Agnew, 1974, 1978). Reference has already been made to data from Monroe's (1967) study, suggesting that poor sleepers are autonomically overaroused, but Killen and Coates (1979) have proposed

that such physiological disturbances in sleep could represent abnormal fluctuations in rhythmic processes which then lead to disturbed sleep. Miles, Raynal and Wilson (1977) have also suggested that poor sleep may result from anomalies within the circadian cycle although they were unsuccessful in their attempt to modify the abnormal circadian rhythm (24.9 hours) in their single case study.

In spite of some evidence indicating the potentially disruptive influence of unsettled sleep-wake cycles, it seems unlikely that this model can explain adequately the aetiology of typical cases of insomnia. However, it may be particularly appropriate for the type of sleep problem often experienced by shift workers. Interestingly, the notion that there may be an optimal period for sleep is also incorporated in other theoretical approaches such as the stimulus control paradigm and the sleep variability model, where the principal aim of therapy is to establish a predictable nightly sleep routine. Investigation of an individual's functioning from a 24-hour perspective would appear, therefore, to be useful especially if psychological correlates are considered.

# CHAPTER 3

## OTHER COVARIATES OF INSOMNIA

This chapter will consider a number of factors which bear some important, but not necessarily causal, relationship to insomnia. The areas under consideration will be presented in the following order:-

1. Personality variables in insomnia
2. The role of life events
3. The effects of monotonous stimulation
4. The effects of physical exercise
5. The effects of diet
6. The effects of insomnia upon daytime functioning

### PERSONALITY VARIABLES IN INSOMNIA

Numerous studies have documented differences between good and poor sleepers on measures of psychopathology and personality. Free-floating anxiety, phobic anxiety, somatic concomitants of anxiety and neurotic depression have all been elevated within the poor sleep group (Kumar and Vaidya, 1984), and several studies have found that the Taylor Manifest Anxiety Scale is a useful discriminator of insomniacs and non-insomniacs (Haynes et al, 1974; Hicks and Pellegrini, 1977; Kumar and Vaidya, 1984). Only one study has suggested the opposite relationship, conceptualising short sleepers as "non-worriers" (Hartmann, 1973). The most commonly adopted measure, however, has been the MMPI (Minnesota Multiphasic Personality Inventory) where research results have been consistent over a wide range of insomniacs in terms of age, chronicity, presence or absence of medication and source of sample. There is some evidence, however, that psychopathology is more prominent in younger insomniac subjects (Roehrs, Lineback, Zorick and Roth, 1982) with the older agegroups being more likely to suffer sleep loss as part of the natural ageing process. In general, insomniacs have exhibited neurotic profiles on the MMPI, with elevations on the depression, hypochondriasis, psychopathic deviance, psychasthenia, and hysteria

scales (Monroe, 1967; Johns et al, 1971; Coursey et al, 1975; Carskadon, Dement, Mitler, Guilleminault, Zarcone and Spiegel, 1976; Freedman, 1976; Kales, Caldwell, Preston, Healey and Kales, 1976; Monroe and Marks, 1977; Shealy ,Lowe and Ritzler, 1980; Kales et al, 1983a; Levin, Bertelson and Lacks, 1984). MMPI reports have been further corroborated by studies reporting higher rates of dysphoric mood (Beutler, Thornby and Karacan, 1978; Johnson, Church, Seales and Rossiter, 1979), depression (Coursey et al, 1975) and general medical complaint (Monroe, 1967; Roth et al, 1976). Thus, the insomniac can be characterised as a person who is mildly depressed, anxious, hypochondriacal and overly worrisome.

Borkovec (1982) has pointed out that there are interpretative problems with personality study data since these psychological features may not be causatively related to disturbed sleep, and few significant correlations have been found between personality variables and objective sleep parameters. He also refers to studies which failed to establish personality differences between insomniacs and good sleepers (Rechtschaffen, 1968; Gering and Mahrer, 1972). Kales et al (1983a), however, suggest that the homogeneity of the MMPI profiles amongst their (and other) chronic insomniacs should be interpretated as strong evidence of their psychopathology being primary to their sleep disorder. They propose that, during the day, the insomniac typically inhibits, denies and represses conflicts which at night, in the relative absence of external stimulation, re-emerge, resulting in an internalised focusing of attention (Kales, Soldatos and Kales, 1982). This process of internalisation is hypothesised to lead to chronic emotional arousal, in turn also provoking physiological arousal, and rendering the subject unable to sleep.

Whether or not these psychological characteristics prove to be a cause

of insomnia, or simply correlates of disturbed sleep, the available evidence does point towards the appropriateness of considering insomnia from a cognitive-behavioural perspective, since, whether before or after the development of poor sleep, there develops a consistent clinical picture of neurosis.

## THE ROLE OF LIFE EVENTS

There is now considerable evidence that physical illness and psychological problems are more likely to develop when there is an increase in life events which effect change in the relatively stable pattern of an individual's life (eg. Holmes and Masuda, 1974; Paykel, 1974). The person's ability to cope appears also to be related to the magnitude and nature of these stressful events, and to the manner in which they are perceived and interpreted. Personal vulnerability is thought to be mediated by a combination of biological predisposition, learning history, current life situation and personality characteristics and attitudes (eg. Rahe, 1974; Hinkle and Wolff, 1958).

A study by Healey, Kales, Monroe, Bixler, Chamberlain and Soldatos (1981) compared groups of good and poor sleepers on various life experience questionnaires within the context of a structured interview. Although the onset of insomnia was regarded as gradual by the majority of poor sleepers, major life events during the year in which insomnia started were evident in 70% of cases. In addition, insomniacs exhibited a greater degree of personal vulnerability (more emotional upset) during childhood and were less content with their parents and their family lives than were the good sleepers. Consistent with previous MMPI studies, poor self-concept and frequent health complaints were noted, and the authors proposed that their poor sleepers seemed prone to internalise stress reactions. Healey et al concluded by stating that "one of the early goals of treatment should be to help insomniacs

acknowledge their sleep disturbance as inextricably related to their waking lives, and not a disease entity for which a magical cure can be found".

This study is the only one available which has investigated the association between life events and insomnia. The findings, however, may run in parallel with the research on psychopathology since obsessively worrying people might be prone to react adversely to major life change. Further investigation is required to replicate these results, particularly within the context of a prospective study. As with all retrospective analyses there is the problem of attribution where subjects may make false causal connections between current problems and previous circumstances.

## THE EFFECTS OF MONOTONOUS STIMULATION

A series of investigations by Bohlin (1971, 1972, 1973) has indicated a relationship between orienting response habituation and the development of sleep-onset. In each of these experiments subjects achieved more rapid sleep-onset after monotonous stimulation (repetition of an audible tone every 20-40 seconds) compared with the no stimulation control procedures, suggesting that orienting response habituation is an actively de-arousing process. Bohlin (1973) proposed that the rate of this habituation and subsequent sleep development interact with initial arousal level such that rapid habituation occurs when the subject is in a low state of arousal and slower habituation accompanies high arousal.

Borkovec (1979) has raised the possibility that these findings may have some implications for the traditional psychological approaches to the management of insomnia and their underlying theoretical models. It is possible, for example, that repeated tension-release cycles in

relaxation, and the repetition of a "mantra" in meditation function as soporific stimuli within this habituation model. Similarly, techniques aimed at blocking out external stimulation and the preclusion of intrusive thinking might significantly reduce initial arousal levels to facilitate habituation. Since attentional processes are of paramount importance in this theoretical analysis, any therapeutic procedure which overcomes the problem of dishabituating stimuli (overt or covert) might be of considerable value, and worthy of further investigation.

## THE EFFECTS OF PHYSICAL EXERCISE

The possibility of there being a significant relationship between physical exercise, subsequent fatigue, and night-time sleep has considerable intuitive appeal. Insomniacs frequently report that they try to "tire themselves out", and the restorative hypothesis of sleep would predict that such physical activity would increase the total time slept, particularly in "deep sleep". The earliest evidence for this view was provided by Baekeland and Lasky (1966) who obtained significant increases in SWS sleep especially following afternoon exercise in their sample of 10 athletes. By comparison, evening exercise was associated with more disturbed sleep characterised by more frequent, brief wakenings and increased stage 1 sleep. These authors, therefore, identified the importance of the timing of exercise, and suggested that the later the exercise the greater its potential as a "stressor producing CNS activation". Horne and Porter (1976) have also provided tentative evidence that late daytime exercise intrudes upon sleep. A comprehensive review by Torsvall (1983) considered 20 investigations of the association between sleep and exercise. Half of these studies were found to support the hypothesis that activity facilitates deeper and longer sleep, and three of these also found reductions in sleep-onset latency. However, the remaining studies were

categorised as either inconclusive or unsupportive of the theory. Torsvall concluded that the strongest weight of evidence for the positive impact of exercise upon sleep is in physically fit subjects. The optimal level of this activity has yet to be investigated. He also stressed the need for longitudinal studies, incorporating sufficient numbers to compare trained and untrained cases during an exercise programme.

The impact of exercise within the insomniac population has not been systematically examined. Marchini, Coates, Magistad and Waldum (1983) did find that the daytime behaviour of insomniacs and good sleepers differed in that the good sleepers had busier and more active daytime lives than their insomniac counterparts, and these workers proposed that "increased activity, especially in the mornings and early evenings may be helpful in treating insomniacs". Although such an instruction is frequently included in "self-help" programmes, controlled research is clearly required to establish firstly, whether significant changes in insomniac sleep pattern can be obtained by this method alone; and secondly, whether or not such improvements are of clinical importance. The most recent review of psychological management issues in insomnia (Lichstein and Fischer, 1985) strongly supports Paxton, Trinder and Montgomery's (1983) conclusion that "the relationship (between sleep and exercise) is unreliable and subject to alternative explanations. Clinical prudence demands that the sleep effects of exercise be judged on an individual basis".

## THE EFFECTS OF DIET

Bootzin and Engle-Friedman (1981) have stressed the importance of considering all aspects of drug use and diet in the assessment of insomnia (see Chapter 5 for information on the effects upon sleep of hypnotic drugs, including alcohol). Probably the most commonly taken

drug in daily use is caffeine, which is contained in coffee, tea, "cola" drinks and certain analgesics and weight-control agents. Research has suggested that this stimulant drug can disrupt sleep and be associated with racing, worrisome thoughts (Goodman and Gilman, 1969). Such sleep disturbance has been found to be dose-related since, in one study, the equivalent of four cups of coffee induced a reduction in total sleep time of 0.4 hours and increased sleep latency by 13 minutes, whereas one cup of coffee taken 30 minutes prior to bedtime had no significant effects. A report by Brezinova, Oswald and Loudon (1975), however, found that caffeine 15 minutes prior to bedtime produced more frequent and longer awakenings in their subjects. In addition, Bolton and Null (1981) have suggested that heavy, regular consumers are more likely to develop a tolerance to caffeine and, therefore, suffer less ill-effects on their sleep than sporadic consumers. Despite the fact that patients are often advised to avoid drinking coffee in the late evening (eg. Hauri, 1979) there is no direct evidence to suggest that such dietary control is an effective remedy for even mild insomnia. Similarly, the ingestion of Ovaltine, Horlicks or warm milk has been found, in laboratory studies, to improve sleep, especially during the last third of the night (Brezinova and Oswald, 1972) but it seems unlikely that this would offer much potential as a treatment for clinical insomnia. Although sleep parameter changes have been found to be statistically significant, providing some evidence for the value of emphasising dietary change as a piece of useful advice, such procedures have not been evaluated in comparison with other, medical or psychological treatments.

## THE EFFECTS OF INSOMNIA UPON DAYTIME FUNCTIONING

The definition of insomnia as "a persistent inability to obtain adequate sleep", highlights the importance of establishing the point at

which sleep becomes inadequate for the purposes of daytime functioning. Research has generally considered this issue by examining three main categories of "next day effects" which are consequent upon an impoverished sleep.

Firstly, Bootzin and Engle-Friedman (1981) have provided a helpful review of "performance measures" which appear to be sensitive to the effects of sleep loss. Sleep deprivation studies on normal samples have shown that subjects perform poorly on vigilance, reaction time, and arithmetic tasks making greater numbers of detection errors due to poorer attentional functioning, being slower to respond to both visual and auditory stimuli, and getting fewer problems correct in mental arithmetic exercises (Glenville, Broughton, Wing and Wilkinson, 1978; Poulton, Edwards and Colquhoun, 1974; Williams and Lubin, 1967). More recent work on insomniac patients has also revealed similar deficits on a variety of psychomotor and cognitive tasks, along with specific difficulties in semantic memory (Mendelson, Garnett and Linnoila, 1984; Mendelson, Garnett, Gillin and Weingartner, 1984). Inadequate sleep has been associated, therefore, with impairment of certain aspects of cognitive function.

A second area of concern is that of daytime fatigue. Dement, Seidel and Carskadon (1984) have reviewed the available evidence on the association between such fatigue and night-time wakefulness. They reported the results of some of their own work (Carskadon and Dement, 1982) which demonstrated a linear relationship between daytime sleepiness and sleep, in that fatigue increased as sleep was systematically reduced in experimental subjects. They recommend the use of the Multiple Sleep Latency Test (MSLT) (Richardson, Carskadon, Flagg, Van den Hoed, Dement and Mittler, 1978) as an objective measure of daytime tiredness since it is sensitive to the effects of even the

relatively small reductions in sleep which have been found to produce substantial, and highly significant increases in daytime sleepiness (Carskadon and Dement, 1981; Roehrs, Zorick, Sicklesteel, Wittig and Roth, 1983). Dement et al (1984) have proposed that, where there are no symptoms of fatigue, but there is substantial evidence of sleep disruption, the most obvious explanation is that too much time is being spent in bed. In such cases, a didactic approach may be indicated, and indeed, Lichstein (1980) has reported the successful "treatment" of such an individual by simply advancing bedtime and backing up the time of morning rising.

A third group of studies have reported disturbances in daytime mood in poor sleepers. Nicassio and Bootzin (1974) found that psychological therapy was associated with a reduction in ratings of irritability, and Marchini et al (1983) reported that their insomniacs differed from normal controls in that the former rated themselves as significantly more worried, upset, self-critical, hostile, and depressed, and significantly less energetic and physically active, and also considered that they "enjoyed themselves" less. It is of course possible that such features of insomniac mood state are a reflection of neurotic disposition, as previously described. The study by Mendelson et al (1984a), however, did not find any difference between insomniacs and controls on a mood rating scale which comprised a series of analogue measures.

In conclusion, the research literature provides conflicting evidence on the effects of insomnia upon daytime behaviour, mood and performance. Certainly, it would seem that when subjects are deprived of their usual sleep requirement, deficits become evident. However, few studies have been completed upon severe insomniacs, and it seems possible that these patients fall into a number of sub-groups; some having marked

impairment of daytime activities, others mild to moderate impairment, and yet others who exhibit no ill-effects whatsoever. For a proportion of poor sleepers, therefore, a didactic approach, highlighting the normality of their own sleep patterns, and the identification of actual sleep needs might enable them to re-define their complaints. A rescheduling of night-time routine would seem appropriate in such cases. Clearly, considerable research is still required to establish the association between subjective and objective parameters of sleepiness, and the question of whether or not the absence of daytime disturbance invalidates treatment for primary insomnia remains to be adequately addressed.

# CHAPTER 4

## THE ASSESSMENT OF SLEEP PATTERNS

Recent reviews (Coates and Thoresen, 1980; Bootzin and Engle-Friedman, 1981; Kales and Kales, 1984) have indicated that a thorough evaluation of sleep pattern should incorporate measures from each of the following areas:-

1. The taking of a sleep history
2. A medical examination
3. Electroencephalographic assessment
4. A comprehensive behavioural analysis

This chapter will describe each of these aspects of sleep measurement with reference to the assessment of insomnia. Particular attention will be paid to the practical usefulness of the information obtained. The chapter will conclude with a comparison of self-report and EEG measures and an overview of sleep assessment within the natural environment.

### THE SLEEP HISTORY

In clinical practice the sleep history and medical examination are usually completed during a brief consultation, the outcome of which is often the prescription of sedative-hypnotic medication. The main emphases of this interview are upon the confirmation of a clinically significant sleep problem and the exclusion of aetiological medical problems and transient personal/situational upsets. As with any intake interview, personal details and other demographic information are recorded followed by the eliciting of a verbal description of the nature and development of the sleep disturbance itself. The importance of considering insomnia within a developmental context, especially with elderly patients, and of distinguishing between "natural" sleep pattern changes and clinically important sleep difficulties has been stressed (eg. Bootzin and Engle-Friedman, 1981). Since individual sleep needs are known to vary considerably even within age-groups, the interviewer

must also consider the patient's perception of his/her sleep needs, and the associated expectations and attributions which result therefrom. The evaluation of the degree of "intrusiveness" experienced, in terms of next day effects such as fatigue and irritability, is also central to the diagnostic process (Dement et al, 1984). In addition, the numerous studies which characterise the insomniac as an overly anxious, mildly depressed individual point to the importance of including a careful history of such factors. Cases of clinical depression are likely to present with secondary sleep disorder. An evaluation of individual coping styles may also reveal psychological factors of aetiological significance, and although no standard measures have as yet been developed, prudent questioning can often yield a preliminary formulation in terms of the models of causation and maintenance already outlined.

MEDICAL EXAMINATION

There are three principal reasons for including a medical examination in the assessment of insomnia. Firstly, insomnia can result from physical pathology, either directly (ie. through central nervous system pathology), or indirectly as a result of pain/discomfort caused by physical illness. Secondly, the clinical implications of pharmacological intervention require consideration since the use of both stimulant and hypnotic drugs can lead to tolerance, dependance and poor sleep (Kales et al, 1983b); Kales and Kales, 1984). Patients may have to be stabilised on medication, have it withdrawn, or be safeguarded from the potentially deleterious effects of certain drug interactions. Thirdly, there is now strong evidence that a normal physical examination is adequate for differential diagnosis amongst various sub-types of insomnia. Guilleminault and Dement (1977) reported that 85% of cases of narcolepsy, sleep apnea, nocturnal myoclonus and

restless legs syndrome were diagnosable via a physician's normal physical examination. Given the relative rarity of these disorders compared with insomnia (Borkovec, 1982) it is estimated that this would result in less than two sleep-disordered patients per 100 being wrongly classified. Other workers have also supported this view (Coates and Thoresen, 1980). Furthermore, there has developed recently a reaction to what some believe to have been an over-emphasis upon physiological factors in sleep laboratory studies of insomnia. For example, Kales and Kales (1984), two of the most respected research scientists working in this field, quoted from a pertinent editorial that "it is time to re-assess the value of clinical judgement. It is time for the pendulum to swing the other way, not only because of economic pressure but because of another lost art - common sense" (Scott, 1979). Kales and Kales go on to say that the general practitioner in the context of the office setting is best able to assess all aspects of the patient's functioning in order to balance all of the contributory factors towards an understanding of the patient's problems. Similarly, Oswald (1981) concluded that the GP is the best person to diagnose and manage most sleep disorders.

## ELECTROENCEPHALOGRAPHIC METHODS

Interest in all-night polygraphic recording of sleep was initially aroused by the discovery of changes in the EEG during sleep (Loomis, Harvey and Hobart, 1937), and the subsequent definition of sleep stages (Dement and Kleitman, 1957). Over the past 30 years, a vast research literature has developed describing "objectively-defined" sleep patterns, stages and disorders. Much of this work has been the product of numerous "Sleep Disorders Centres" set up during the 1960's in various parts of the USA. Researchers have (rightly) made a science of the study of sleep physiology, but there have been problems inherent in

the evaluation of insomnia for two, related reasons. Firstly, insomnia is primarily a subjectively experienced and reported phenomenon (dissatisfaction with sleep); and secondly, objective indices of insomnia have frequently correlated poorly with self-report. The relationship between subjective and objective measures will be dealt with in some detail later in this chapter.

Although the term EEG refers only to electrophysiological recording of brain activity, via the standard positioning of electrodes, in practice, EEG is generally recorded through a polygraph with an attached printer, other channels of which are used to measure eye movement and muscle activity. These two latter measures are known as the elecro-oculogram (EOG) and the electromyogram (EMG). EEG, EOG and EMG are recorded concurrently throughout the night and the resulting polygraphic trace is usually scored visually in segments of 20 or 30 seconds, which can be assigned to stages of sleep according to recognised criteria (Rechtschaffen and Kales, 1968). Objective data are generated, therefore, on the various stages within sleep, and the EEG is uniquely appropriate for detailed descriptive analysis, and for the definitive descriptive diagnosis of difficult cases. EEG recording is not only a highly technical procedure but it is also very expensive, not least in terms of the sheer volume of recorder paper (300 to 700 metres per subject night). There are, however, two rather more serious methodological difficulties in the measurment of insomniac sleep.

Firstly, during the first night in the sleep laboratory, subjects take longer to fall asleep, awaken more often and have more total wake time than on subsequent nights when they have adjusted to their new environment (Rechtschaffen and Verdone, 1964; Agnew, Webb and Williams, 1966). For this reason, an adaptation night is now routinely included in sleep studies in order to obviate such "first night effects".

Although subsequent nights may be less reactive, it should be remembered that the stimulus control hypothesis would predict rather less transitory alterations in the sleep pattern of chronic insomniacs, and the directionality of any change might also vary (sleep pattern might improve rather than deteriorate). The removal of the subject from the home environment is, therefore, a confounding variable in psychological research. Potential changes in physical and mental relaxation may also modify the problem under observation eg. the attachment of 12 electrodes and the novel environment may occupy thinking and preclude intrusive worry.

A second methodological problem concerns the fact that researchers frequently require subjects to sleep only within the limits of predetermined recording periods, in order to cater for between group comparison. This experimental manipulation is certainly invasive of the sleep process itself on the recording nights, but might also modify the sleep pattern on subsequent nights by rescheduling wakening time. The rather more flexible "ad lib" design, however, has the advantage of permitting subjects to remain in bed for as long as they wish, but proves problematic in data comparison.

The use of home polysomnography (Coates, Killen, George, Marchini, Silverman, Hamilton and Thoresen, 1982), where polygraphic data are transmitted over a single telephone line, may be one solution to some of these problems. It is still at a relatively experimental stage, and although it allows for recording within the natural environment, it does remain considerably intrusive. In addition, in spite of the technological advances involved, one study reported the loss of 37% of EEG recordings through technical failure (Ancoli-Israel, Kripke, Mason and Messin, 1981).

## THE BEHAVIOURAL ASSESSMENT

Within the psychological literature, sleep has been construed as an observable behaviour, or set of behaviours, which conforms to the principles of learning theory. The insomniac is seen as having acquired a maladaptive sleep pattern, which may be initiated by certain antecedent conditions (for example tension or sleep-incompatible stimuli), and maintained by reinforcing consequences (eg. perception of self as a "poor sleeper", performance failure). Behavioural analysis is able, therefore, to provide information on the situational correlates of sleep and their interactions with actual sleep pattern parameters. Importantly, however, the experiential and qualitative data which form the basis of routine clinical presentation may also be usefully addressed. In common with most psychological research, behavioural assessment has become a logical extension of the initial interview, thus providing baseline data against which treatment process and treatment outcome may be measured. The methods employed in behavioural analysis of sleep problems will be reviewed under three headings. These are a) self-report, b) observer report and c) recording devices.

### Self Report

The two most commonly used self-report measures are sleep questionnaires and sleep diaries. The questionnaire is particularly useful where a large number of subjects are to be surveyed or screened (eg. McGhie and Russell, 1962) and has the advantages of being quick, easy and inexpensive to administer. Johns (1975) and Evans (1977) have reported factor analyses and normative data on sleep questionnaires, and Bootzin and Engle-Friedman (1981) have suggested that such measures may be usefully supplemented by the use of rating scales, which provide process data (eg. Parrot and Hindmarch, 1978). Most of the influential treatment studies, however, have preferred the diary to the

questionnaire because the latter is particularly vulnerable to criticism based upon the problem of reporting bias. When patients are requested retrospectively to summarise their sleep, they may be unduly influenced by recent experiences of sleep and may be prone to inaccurate attributions and generalisations. In those situations where the possibility of treatment is envisaged by the patient, there may be bias towards exaggerated reporting. Global reports, nevertheless, may serve as useful adjuncts to continuous sleep records.

The general use of daily sleep logs, based upon the revised daily sleep questionnaire (DSQ) (Monroe, 1967), has led Bootzin and Nicassio (1978) to describe these as the "staple of assessment procedures in insomnia treatment outcome research". This questionnaire is completed each morning upon wakening and includes variables such as : number of minutes to sleep-onset, total time slept (hours and minutes), frequency of intermittent wakenings, and several qualitative ratings of, for example, sleep satisfaction and restedness. The DSQ makes the subject's task more specific and less ambiguous than the sleep questionnaire and, therefore, diminishes the problem of response bias, although it does so incompletely. Some researchers have required subjects to return questionnaires each day by mail to counteract the tendency of some retrospectively to complete the logs on the day prior to their next appointment (Lick and Heffler, 1977; Lacks, Bertelson, Gans and Kunkel, 1983). Steinmark and Borkovec (1974) have also developed an experimental design which controls for therapy-induced expectancy effects and demand characteristics, thus improving reliability of self-report (A description of this procedure is contained in the methods section of this study - Chapter 7).

An investigation by Coates et al is quoted in Bootzin and Engle-Friedman (1981) as evidence for the reliability of sleep diary

measures. These workers obtained an average test-retest correlation, for daily sleep-onset latency, of 0.93 for poor sleepers and 0.58 for good sleepers. The comparable test-retest figures for EEG assessment were only 0.70 and 0.58 respectively. Overall sleep diary reliabilities were 0.69 and 0.35, while EEG reliabilities were 0.66 and 0.60 for poor and good sleepers. Bootzin and Engle-Friedman concluded, therefore, that "sleep diary reliabilities are equivalent to EEG reliabilities, especially for insomniacs". Measures of validity are equally encouraging. Sleep log estimates have been found to correlate highly with observer estimates of the same night's sleep ($r = 0.84$, Turner and Ascher, 1979a). Of equal importance, however, is the self-evident fact that, since it is verbal complaint of insomnia which initiates treatment, verbal statements of sleep pattern must, likewise, be of primary evaluative importance. Notwithstanding these results on reliability and validity there has been considerable debate concerning the accuracy and usefulness of self-reporting of insomnia. These matters will be addressed again shortly.

Observer Report

A number of workers have made use of nurses' (Kupfer, Wyatt and Snyder, 1970; Erwin and Zung, 1970) and spouse's or room-mate's (Nicassio and Bootzin, 1974; Turner and Ascher, 1979ab) observations to provide a more objective measure of sleep. The observer has been asked to attend to criteria such as : subject's eyes closed, absence of voluntary movement, deep respiration, and failure to respond to the question "are you asleep"?, and to make a decision as to whether he/she is awake or asleep. In the ward setting, time sampling procedures have been used from which estimates of sleep latency, sleep time and frequency of wakenings have been derived, although the results from the two available studies have produced very different results for the

reliability of these observations. Erwin and Zung consistently obtained reliability coefficients greater than 0.90, whereas Kupfer et al reported that less than 25% of estimates were "accurately determined" compared with EEG criteria. It should be noted, however, that the subjects sampled in the latter study were psychiatric in-patients, many of whom had depressive illnesses, and could not be considered as similar to primary insomniacs. In addition, Kupfer et al reported that almost one third of the observations themselves precipitated sleep stage changes suggesting that the monitoring process may have been unacceptably intrusive. A further serious limitation to nurse observation is that subjects need to be hospitalised. Similar to the sleep laboratory studies, therefore, the question is also raised of whether data gathered in a controlled environment can be taken as a reasonable sample of typical sleep at home.

The possibility of engaging spouses or room-mates as home-based observers certainly solves some of these difficulties, but has the disadvantage that these observers are often unable to provide consistent data, unless they too are poor sleepers, or are willing and able deliberately to remain awake and vigilant. For the assessment of moderate to severe insomnia this solution appears to be impractical. Indeed the available literature is confined to recruited populations (Nicassio and Bootzin, 1974; Turner and Ascher, 1979a,b).

Recording Devices

The importance of obtaining accurate information to corroborate the self-report of insomniac patients has led to the development of a number of recording devices. Most of these are based upon the premise that the subject's response to external stimuli reduces as sleep commences and deepens, ie. awakening threshold increases and greater intensity stimulation is required to produce arousal. The detection (or

not) of a cue stimulus, therefore, should reveal the presence or absence of sleep. For the purposes of assessment, of course, it is important that the cue is perceptible, but not intrusive.

The most useful device currently available, and for which there is most support in the research literature, is the "Somtrak" Sleep Assessment Device (SAD) (Kelley and Lichstein, 1980). The SAD generates a brief, soft tone at pre-set intervals throughout the night and tape records verbal responses to these cues. The tone generator is linked to a cassette recorder for this purpose. Clearly, if the subject is awake, the tone will be heard and the criterion response of "I am awake" will be recorded. Conversely, if no response is recorded the interpretation is made that the subject was sleeping. The volume and pitch of the tone are adjusted for the individual to ensure that it is perceptible while awake but not intrusive upon sleep. The usual inter-tone interval of 10 minutes is not sufficiently frequent to render it soporific or to cause habituation (Lichstein, Hoelscher, Eakin and Nickel, 1983). Comparisons with EEG recordings have demonstrated levels of agreement greater than 90% with no significant differences on measures of sleep latency, total sleep time and sleep efficiency (Lichstein, Nickel, Hoelscher and Kelley, 1981). The number of awakenings measure, however, has proven less valid due to the time-sampling procedure utilised by the SAD wherein some data are inevitably forfeited. The SAD would appear to be the best available objective measure of sleep for use within the natural environment. It fulfils all the criteria, proposed by Lichstein and Kelley (1979), to be important for accurate, valid and practical measurement. These criteria are as follows:- a) portable to the natural environment, b) self-administered, c) non-intrusive, d) relatively inexpensive, and e) very accurate.

It is important now to return to the key issue raised earlier, regarding the association between objective and subjective measures of insomnia. Over the past 15 years researchers have attempted to validate self-report by comparison with EEG criteria. The inherent assumption, therefore, has been that EEG identifies "true insomnia" and that the substantial sub-group of patients who complain of significant sleep disturbance, not ratified by polysomnography, suffer from "pseudo-insomnia". More recently, however, there has been some recognition of the possibility that EEG may not be sensitive to all the crucial variables which predict clinical reports of insomnia, and the two groups have been re-labelled "objective" and "experiential" insomnia respectively. Studies have also begun to reconsider the appropriateness of the EEG criteria used to establish the point of sleep-onset. In order to understand the development and complexity of current opinion, the relevant literature will be reviewed in some detail.

A number of studies have examined the subjectively and objectively defined sleep patterns of normal subjects. Johns (1971) provided the earliest review paper and quoted evidence for the satisfactory test-retest reliability of sleep questionnaires. He suggested that, although subjects were not accurate in their estimates, their reports did shift in the same direction as objective measures (eg. Lewis, 1969). Baekeland and Hoy (1971) reported greater accuracy than Lewis in sleep diary measures of sleep latency and frequency of wakenings in normal adult men. In his own work, Johns found that normal subjects estimated the time of falling asleep to within two minutes, and the total duration of sleep to within eight minutes. He concluded that no single method of assessment could give sufficient information to permit neglect of the others, and that greater attention should be paid to

information concerning sleep quality which cannot be inferred from EEG records.

A study by Frankel et al (1976) compared the recorded and reported sleep of chronic primary insomniacs with matched controls. Eighteen insomniacs, with an average age of 44.5 years, and a mean duration of insomnia of 19 years, were included in the study. Frankel et al found significant discrepancies between the insomniacs' and controls' subjective assessment of their sleep and the sleep polygraph data, with insomniacs over-estimating sleep latency and under-estimating total sleep time and sleep efficiency. Insomniacs took an average of 54 minutes to fall asleep (estimated at 81 minutes), slept a total of 340 minutes (estimated at 306) and woke up 1.9 times (estimated at 2.7). Polygraph data showed sleep efficiency to be 76.5% while questionnaire data yielded a figure of 67.1%. Control subjects' reports were not significantly different from EEG measures but they tended to rate in the opposite direction to the above errors. Frankel et al recommended that the focus of interest should be upon how much insomniacs are not sleeping, ie. their sleep efficiency, since its calculation takes into account both sleep latency and wake-time after sleep-onset in relation to total time in bed. They suggested the figure of 85% sleep efficiency as a useful diagnostic criterion to differentiate between insomniacs and non-insomniacs.

Carskadon et al (1976) compared the EEG records and subjective estimates of 122 drug-free patients who complained of chronic insomnia. Unfortunately, however, the length of the drug-free period, preceding the laboratory sleep nights, was described as "typically more than 2 weeks" which falls short of the generally accepted 5-6 weeks required for drug withdrawal effects to have ceased (Oswald and Priest, 1965). Subjective reports of sleep time averaged well below objective

measurement, and were consistently underestimated by a margin of more than one hour in one third of the cases. Similarly, there was a significant difference between the average recorded sleep latency of 26 minutes and the estimated latency of 62 minutes. Significant correlations were, however, obtained between self-report and EEG for both women ($r = 0.64$, $p < .001$) and men ($r = 0.60$, $p < .001$). Frequency of arousal demonstrated a significant difference across age-groups with, as expected, older subjects wakening more often. Importantly, Carskadon et al also found that only around one half of the insomniacs could be distinguished from normal subjects by EEG measures of total sleep time. This is a finding which has been replicated by Borkovec and his colleagues (Borkovec, Grayson, O'Brien and Weerts, 1979; Borkovec, 1979) who have emphasised the importance of differentiating between the two sub-types of primary insomniacs (objective and experiential), as previously defined.

It would seem , therefore, that both electro-physiological and self-report data are useful in a detailed descriptive analysis of insomnia. The substantial and consistently reported concordance rates indicate that verbal report may be a useful predictor of objective indices.

The discussion of EEG and self-report in terms of "agreement" does, of course, presuppose that the same question is being asked of both measures. In other words if, according to EEG criteria, the subject is objectively (factually) awake, then he should if asked at that time report the self-perception of wakefulness. Conversely, if objectively asleep he should corroborate this upon being awakened. It is only if these things are found to apply that verbal report can be considered unreliable compared with this EEG "standard".

The study by Campbell and Webb (1981) was the first to investigate the relationship between EEG-defined awakenings and subjects' awareness of

them. As a prologue, these authors commented on research examining "mental activity at sleep-onset" which showed that people often report that they are "drifting off to sleep" or "awake but drowsy" during the early stages of EEG-defined sleep. They quoted one study where 44% of subjects who reached EEG stage 1 or stage 2 stated that they had not gone to sleep, and 2 of the 13 subjects, even within SWS (stages 3 and 4), also replied in the negative (Agnew and Webb, 1972). In their own study, subjects were asked to signal periods of wakefulness by pressing a button while being recorded during a night of laboratory sleep. Campbell and Webb found that a significant proportion of the failures to signal wakefulness occurred during EEG wakefulness, and also that wakefulness was commonly perceived in the absence of EEG evidence.

In a replication study, Borkovec, Lane and Van Oot (1981) awakened 25 insomniacs out of stage 2 sleep and asked them to report on their experience of sleep/wakefulness. They found that sleep was reported by only 4-12% of the insomniac group, and that even in a "good sleep" control group only 30% stated that they had been asleep. Ratings of "certainty" of being awake were much higher in the insomniac group. The results of a study by Slama (1979), quoted in Bootzin and Engle-Friedman (1981), are also suggestive of perceptual differences between good and poor sleepers. Slama found that 8 out of 10 insomniacs who were aroused after 4.5 minutes of stage 2 sleep reported that they had been awake when roused, compared with good sleepers who all reported having been asleep. These studies raise the question of what cues the insomniac uses to determine whether or not he is asleep, and how these cues differ from those used by normal sleepers. The possibility that the sleep mentation of insomniacs is similar to waking mentation, making it difficult for them to distinguish sleep from wakefulness, is at present the most promising explanation for these research observations (Rechtschaffen and Monroe, 1969; Borkovec, 1979).

A study by Coates, Killen, George, Marchini, Silverman and Thoresen (1983) provided a powerful psychometric test of the validity and reliability of self-report compared with EEG measures. These workers pointed out that the correspondence between the two measures had largely been investigated via simple correlational methods and that more robust and sophisticated analysis was required. In their study, therefore, they applied the multi-trait multi-method matrix approach (Campbell and Fiske, 1959); a technique which permits the simultaneous exploration of reliability and convergent validity of variables (in this case physiological and self-report measures of the main parameters of sleep). Their findings indicated that although insomniacs reported significantly more minutes to sleep-onset when compared with EEG-defined non-REM stage 1 sleep, there were no significant discrepancies when the comparison was made at the onset of EEG-defined stage 2 sleep. Significant differences were found between the recorded and reported awakenings for both good and poor sleepers, and disagreements between measures of time awake after sleep-onset appeared to be related to the degree of sleep difficulty. Coates et al concluded that "if the EEG is to be regarded as the criterion of sleep, then self-reports of minutes to sleep-onset are reliable and valid measures for good sleepers. Self-reports of minutes to sleep-onset and minutes awake after sleep-onset provide a reliable and valid relative index for insomniacs".

Some work by Hauri and Olmstead (1983) has provided further support for the use of stage 2 onset as the EEG equivalent of perceived sleep in insomniacs, since "these insomniacs were about as accurate in their sleep latency estimates using this criterion, as the good sleepers were using the traditional one". They also pointed out that consideration should be given to the fact that the insomniac's task is a harder one. Insomniacs often have to estimate sleep latency lasting anywhere from 30 minutes to several hours. In Hauri and Olmstead's study they did so

to within a few minutes. The good sleepers on average had to estimate latencies of only several minutes; a much simpler task. These workers went on to say that they could see no advantage in accepting, as fact, the statement that insomniacs habitually overestimate sleep latency, but rather the issue of importance is the establishment of appropriate criteria, suitable for them. They suggested, therefore, that the beginning of the first 15 minutes of uninterrupted sleep, of stage 2 or better, may be such a criterion. Another recent study by Birrell (1983), using a student population, has reached the same principal conclusion.

Finally, further evidence from Sewitch (1984) extends the controversy by suggesting that EEG may not accurately represent the sleep reports of normal sleepers. She employed a signal detection theory (SDT) framework systematically to examine the verbal report/polygraph discrepancy in 11 volunteer subjects. She found that all of the decisions made by these normal sleepers demonstrated a strong bias towards signalling "awake" regardless of polygraphic criteria. She concluded that "what the subject attends to in making a sleep/awake decision is to a significant extent independent of what is presently defined polygraphically as sleep. In terms of SDT, the polygraphic data in current use are only a weak index of the underlying internal signals, detected and used by normal sleepers in making an awake or asleep decision."

Clearly, further research is required before definitive statements can be made regarding the precise relationship between the experience of sleep and wakening and its objectively measurable concomitants. Certainly, it is possible that a number of the early studies which doubted the accuracy of self-report may have provided less critical results had alternative criteria been used for scoring the EEG traces.

At the very least, there is the consistent finding of a significant correlation between EEG and verbal report, and the strong possibility that concordance may be further strengthened when appropriate EEG indices are finally established.

## AN OVERVIEW OF SLEEP ASSESSMENT IN THE NATURAL ENVIRONMENT

A thorough assessment of insomnia should include firstly, the taking of a comprehensive sleep history, complemented by an examination of physical and psychological status. Cases of physical illness or functional disturbances such as depression which would render insomnia a secondary symptom, can be identified through prudent clinical interviewing and a routine medical examination.

Secondly, a comprehensive behavioural analysis permits the investigation of situational factors and/or stressors which may have an aetiological role. Some of these may be transient or readily remedied. Close attention to the behavioural, cognitive and physiological correlates of the presenting insomnia will often yield information which is of both theoretical and practical importance, and details of sleep covariates such as drug use (for reasons which will become evident in the next chapter), exercise, diet, and daytime coping should be gathered routinely. It may be useful also to interview the patient's spouse to obtain adjunctive information, especially upon daytime effects, although he/she is unlikely to be able to provide reliable data on the actual parameters of sleep.

The third and most adaptable source of data comes from daily sleep charts, kept by the subject and completed each morning upon rising. Every effort should be made to ensure that recordings are not made in retrospect since reliability decreases, and experimental demand increases in such circumstances. Diaries permit the monitoring of day-

to-day sleep variations, and can be used to evaluate the process of sleep pattern change, both in terms of the sleep variables themselves, but also in tems of perceived sleep quality. Since it is often this latter element which precipitates and maintains help-seeking behaviour, adequate assessment is fundamental to treatment outcome evaluation.

Fourthly, there are now available a number of simple time-sampling devices which can provide an inexpensive, reliable and non-intrusive approach to the objective assessment of insomnia. These may be particularly appropriate for the within-therapy assessment of sleep, where corroborative evidence of the validity of verbal report is required, but without unduly disturbing the sleep pattern itself. Although EEG assessment is pre-eminent within the general sleep research literature, it is, apart from the considerable technical and financial considerations, likely to prove unacceptably intrusive in the investigation of psychological processes in insomnia. Some recent developments in the use of home-based polysomnography do, however, suggest that this problem may be at least partially surmounted in time to come. The recent evidence which has reinstated the credibility of self-report as a relative index of sleep parameters has redressed the hitherto and perhaps unequal balance, which was in favour of EEG assessment. Nevertheless, the polygraph remains the only completely reliable method of establishing diagnosis in the case of some of the rare sleep disorders. Kales, Kales, Bixler and Soldatos (1979) have best expressed these sentiments in stating that "the use of highly sophisticated and highly expensive procedures, and the close attention to polygraphic sleep patterns, often render little information of clinical importance."

# CHAPTER 5

## THE MANAGEMENT OF INSOMNIA

Reference was made in Chapter 1 to the prevalence of sleep disorders within the general population and to the clinical and financial burden which they place upon the Health Services. This chapter will provide a detailed analysis of these issues, with particular respect to establishing a case for psychological intervention in chronic insomnia. The merits and drawbacks of pharmacological treatment will be considered firstly, followed by a review of the literature on each of the major psychological approaches to therapy. The chapter will conclude with a synthesis of this literature based upon available comparative treatment studies. Methodological issues will be referred to throughout, but will be dealt with in more detail in Chapter 6 since they are salient to the development of the present study.

### PHARMACOLOGICAL TREATMENTS

Sedative-hypnotics, at first barbiturates and more recently the benzodiazepine drugs have been the medical treatment of choice for insomnia. It is beyond the scope of this thesis to explain in detail the effects of sleeping pills upon sleep, but the following information has been drawn from review papers.

Hartmann (1978) reported on more than 150 pharmacological studies covering 10 barbiturate and 25 non-barbiturate sleep medications and concluded that, when used with insomniacs at the usual clinical doses, the barbiturates and benzodiazepines "do generally reduce sleep latency and increase sleep time. ——— However, they also clearly produce distortions in normal sleep patterns." The most striking distortion effect, consistently found, was that of REM sleep suppression, although several drugs appear to be free from this phenomenon at low doses

(chloral hydrate, flurazepam, methaqualone : Kales, Allen, Scharf and Kales, 1970). Hartmann also referred to the common finding that sleeping pills suppress stage 4 sleep, ie. the "deepest" portion of non-REM sleep. Over the past 15 years, the restrictions placed upon the prescription of barbiturate drugs have led to a focusing of attention upon the benzodiazepine group. These drugs are usually, but somewhat arbitrarily, divided into two sub-groups on the basis of their anxiolytic versus sedative effects. Greenblatt, Divoll, Abernethy and Shader (1982) have highlighted the incorrect assumption that hypnotics and anxiolytics have important neuropharmacological differences, and stated that drug effects are entirely dose-related. That is, at low doses they act as anti-anxiety agents and at high doses as sleeping pills. These workers also reviewed the available range of medications and came to the conclusion that "although benzodiazepines are clearly superior to other classes of hypnotic agents in safety, and possibly also in efficacy, clinically meaningful differences among the various benzodiazepines are often subtle." (For a detailed analysis of the neurochemical action of these drugs, the reader is referred to Van Oot, Lane and Borkovec, 1982).

Although EEG studies have demonstrated that hypnotic drugs reduce sleep latency and increase total sleep time, and there is evidence that they may also have anxiety-reducing effects (Dement, Seidel and Carskadon, 1984), the key question remains - do hypnotics make insomniacs good sleepers? This most important issue was addressed by Adam (1984) who reported on some of the work undertaken by a research group in Edinburgh which found that poor sleepers reported qualitative improvement in their sleep, which was maintained for a few months of continued use (Adam and Oswald, 1982; Oswald, French, Adam and Gilham, 1982). Unfortunately, however, it has been known for some time that hypnotic drugs become very ineffective in longer-term administration,

and often render their users not only persistingly poor sleepers, but also drug-dependent (Kales et al, 1974). The prevailing opinion and advice offered to medical practitioners is, therefore, to administer sleep medication only as a short-term course of therapy, to carefully evaluate factors of aetiological significance, and to exercise care in the withdrawal of the sleeping pills, paying due attention to likely withdrawal effects (Institute of Medicine, 1979, Council on Scientific Affairs, 1981; Kales et al, 1983b). The drawbacks of using sleep medication for any extended period require further consideration since chronic insomnia is frequently associated with a history of drug use. There appear to be five main areas of concern.

Firstly, Hartmann (1978) has made the simple but important observation that pressure of work upon the GP often facilitates prescription as a fortuitous expedient, thereby undermining the exploration of potential aetiological factors. He states that "patients with sleep problems are far more likely to have a sleeping pill prescribed if their physician has only 10 minutes to spend with them than if he has 30 or 40 minutes". It is often within the context of restricted consultation time, or alternatively, through the ready availability of sleeping tablets to medical and surgical patients in hospital that patients are first introduced to benzodiazepines. There would appear, therefore, to be some dysharmony between the attested applicability of medications for sleep and routine medical practice.

A second reason for caution stems from evidence that tolerance develops to hypnotic drugs, and it often does so rapidly. Tolerance has been found to occur with most hypnotics with a diminution of effect over a period of 2 to 6 weeks of nightly administration (Kales et al, 1974; Kales, Kales, Bixler and Scharf, 1975). Such habituation proves less problematic when medication is used only occasionally, but after

regular use the drugs tend not to have a substantial potentiating effect upon sleep. They do, however, continue to suppress REM sleep and stage 4 sleep, and percentage REM is reduced relative to total sleep time (Kales et al, 1975). Thus, within just a few weeks, the drug has a consistent but largely unbeneficial effect and the patient continues to complain of insomnia. It is because of these relatively short periods of effectiveness that many chronic insomniacs have been on a wide variety of hypnotic preparations. For persistent cases of sleep disorder, therefore, there is evident danger of the development of a vicious circle involving prescription and regular nightly use, followed by an increased dosage requirement, followed in turn by the substitute prescription of an alternative sleeping tablet.

Thirdly, it has been known for some time that certain hypnotics produce "carry-over" effects including morning drowsiness, nausea and headache (Oswald, 1968). These are caused by drug accumulation during chronic use, determined primarily by the elimination half-life and metabolic clearance rate of the drug (Greenblatt and Koch-Weser, 1975). If a drug's half-life is short there will be minimal accumulation, and conversely if the half-life is long, some portion of the prior dose will remain in the body when the next is given. Long half-life implies, therefore, that the medication will continue to promote drowsiness and fatigue during the daytime.Clearly, in as far as an hypnotic is given to improve sleep, and thereby improve subsequent daytime functioning, the impairment of such functioning proves to be a serious shortcoming. A recent study by Dement et al (1984) has replicated a considerable volume of previous work in finding changes in daytime alertness after bedtime administration of long-acting hypnotics in normal subjects, chronic insomniacs and in the elderly. These workers stated that "patients and subjects generally show microsleeps and fall asleep involuntarily, with a corresponding interruption of ongoing behaviour

and performance. ——— (This) simply means that individuals are more likely to fall asleep while driving a car, while listening to a lecture, and so forth". Other reports by Hindmarch (1984) and Hindmarch and Ott (1984) have also identified specific impairments in psychomotor performance, reaction time and arousal levels after regular drug use.

Fourthly, Kales et al (1983b) have extensively reviewed the literature on withdrawal from hypnotic drugs. They have pointed out that physicians are generally concerned with issues of efficacy and side-effect during administration, but neglect to give due attention to possible changes which follow drug withdrawal. Since sedative-hypnotics are basically CNS depressants, they are capable of producing dependence and a withdrawal syndrome. Alcohol should also be included in discussion of these matters since it too is a CNS depressant, and a commonly self-prescribed hypnotic agent. Evidence from Pokorny (1978) substantiates the similarities in effects of alcohol and prescribed medications. The withdrawal syndrome typically includes nausea, excitation, agitation, insomnia and nightmares and, of course, with more severe reactions (especially from barbiturates and alcohol), gross behavioural and perceptual disturbances may be found. In addition to this general abstinence syndrome, drug withdrawal insomnia may present, consisting of severe difficulty in initiating sleep, and thereafter within sleep, a fragmentation and disruption of sleep pattern associated with marked increase in REM sleep above baseline levels. The term "rebound insomnia" is a specific type of withdrawal sleep disturbance identified with benzodiazepine drugs (Kales, Scharf and Kales, 1978). Rebound effects occur particularly with the short elimination half-life drugs and in some instances are observable after a single night-time dose (Kales et al, 1983b). Increased frequency of REM periods is also related to a number of other sleep disturbances

including intense dreams and nightmares, frequent arousals, and with the very short-acting drugs, earlier morning waking. The clinical problem posed by these phenomena is even more apparent when two other factors are taken into consideration. Firstly, withdrawal effects have been observed to endure for up to 5 weeks after total drug withdrawal (Oswald and Priest, 1965; Nicholson, 1980). This perhaps explains why many insomniacs fail to persevere with abstinence programmes. Secondly, rebound effects have been associated with increased anxiety levels (Kales et, al 1978; 1983b) and even with anti-social behaviour (Salzman, 1974; Oswald, 1982).

Finally, there are also psychological factors implicated in the protracted use of sleeping pills. Ribordy and Denney (1978), in their consideration of behavioural treatments as an alternative to chemotherapy, stressed the importance of attributional effects during drug use. They suggested that the insomniac taking sleep medication is likely to attribute the sleep which he does get to the drug and to attribute to himself little capacity for falling asleep. Correspondingly, the removal of the drug not only introduces physical rebound effects, but also gives rise to apprehensions concerning ability to fall asleep on one's own. These apprehensions are then largely confirmed by the withdrawal syndrome itself. An experimental study by Davison, Tsujimoto and Glaros (1973) investigated the causal attributions of insomniacs who had responded to a treatment package consisting of a hypnotic drug, a relaxation procedure and the scheduling of bedtime behaviours. Treatment responders were divided into two groups, half being told that they had received an optimal dosage of the drug, and half being told that they had received a minimal dosage which research had proven to be ineffective. The drug was then discontinued while subjects practised the other elements of the programme. Those subjects who had been in the minimal dosage group

and been led to attribute improvement to their own resources were found to maintain improved sleep compared with the other group who returned to their pre-treatment sleep latencies. Clearly, therefore, the attributional effects of drug-taking (psychological dependence) may interact with the physical withdrawal effects to produce an even stronger dependency syndrome.

In conclusion, there are a number of serious shortcomings associated with the prescription of sedative-hypnotics. What then are the general recommendations which can be drawn from the research literature?

Flurazepam is usually listed as the drug of choice since it appears to promote maximum improvement for most people and does not appear to lead to tolerance and dependence (Kales et al, 1974). Coates and Thoresen (1980) and Dement et al (1984), however, urge caution even in the administration of flurazepam, since long-term evaluation has included only two studies which have monitored progress for more than 28 days (Kales et al, 1975; Dement, Zarcone, Hoddes, Smythe and Carskadon, 1973). Researchers concur in advising of the potential benefits of selective use of hypnotics in cases of transient insomnia, but recommend that any such "coping strategy" should occur in the context of strengthening other adaptive coping mechanisms (Dement et al, 1984; Kales and Kales, 1984). Oswald (1979) has reiterated an earlier view (Clift, 1972) that patients should be educated into regarding any hypnotic drug as a temporary expedient, in order to minimise long-term usage. Dement et al have stated, in this connection, that "the major issue is whether or not a course of hypnotic therapy could induce a remission". Similarly, Kales and Kales have proposed that "the primary goal of using hypnotic medication in the treatment of chronic insomnia should be to alleviate the symptom of sleeplessness so that psychotherapy can proceed effectively". For cases of severe and long-

standing sleep disturbance, the regular use of medication offers, therefore, an incomplete therapeutic approach and represents poor clinical practice because of the liklihood of ultimate ineffectiveness and the possible deleterious consequences of both prolonged use and withdrawal.

## PSYCHOLOGICAL TREATMENTS

The emphasis in this review is necessarily upon sleep-onset insomnia, for two reasons. Firstly, there are only six published reports on the management of sleep-maintenance difficulties (Coates and Thoresen, 1979; 1984; Thoresen, Coates, Kirmil-Gray and Rosekind, 1981; Lacks, Bertelson, Sugerman and Kunkel, 1983; Hohenberger-Sieber, Mueller and Schindler, 1986; Espie and Lindsay, 1987). Secondly, the present study is primarily concerned with chronic sleep-onset insomnia (with or without maintenance difficulty). The order of presentation of the literature reflects the historical development and application of the various treatment strategies. The reader is referred to Chapter 2 for theoretical background. The structure of the chapter is as follows:-

1. Progressive relaxation and other anxiety-reduction techniques
2. Treatments based upon stimulus control principles
3. Treatments based upon paradoxical intention
4. Comparative studies of treatment outcome

### Progressive Relaxation and other Anxiety-Reduction Techniques

The bulk of this section concerns the application of progressive relaxation therapy (Jacobsen, 1929; Bernstein and Borkovec, 1973), however, other methods of anxiety management such as desensitisation, and variants of relaxation such as autogenic training and meditation, will be addressed as they become relevant to the review. Biofeedback techniques will be included also since these have generally appeared either in conjunction with or in comparison with relaxation training.

The early development of progressive relaxation is credited to Jacobsen (1929) who found that the sequential tensing and relaxing of the main muscle groups was associated with considerable decreases in muscle activity, blood pressure and heart rate. The advent of behaviour therapy, and particularly the proposition that relaxation could provide an effective competing response with phobic anxiety (Wolpe, 1958), however, was largely responsible for continued interest in the technique, after many years of relative obscurity. Over the past 30 years relaxation has become one of the mainstays of clinical behaviour therapy and various workers have produced abbreviated forms of Jacobsen's procedures; the best known of which being the manual by Bernstein and Borkovec (1973). Researchers have investigated the individual components of the relaxation procedure, in some detail (eg. Benson, Beary and Carrol, 1974; Davidson and Schwartz, 1976; Borkovec and Hennings, 1978; Borkovec and Sides, 1979), but, in spite of the wide applicability of the technique, and the fact that it is positively perceived by most subjects, King (1980) has recently concluded that "the critical procedural variables —— are not well understood, although certain antecedent conditions and the muscle-tension release exercises and physiological attention-focusing appear to be necessary, depending upon the processes maintaining the presenting problem". It is beyond the scope of the present report further to discuss the very extensive literature on relaxation training, but the reader is referred to King's review, which is an excellent resource.

Table 1 provides a summary of the experimental designs and principal findings of 33 treatment studies which have appeared in the major journals over the past 20 years. The table does not include studies comparing outcomes across diverse therapeutic strategies since these will be dealt with at a later point. Not every report will be referred to in the text, but an overview of the main results will be provided.

| Authors | Sample | n | Age | Duration | Treatment(s) | SOL pre | SOL post | SOL Fup | Fup mth. |
|---|---|---|---|---|---|---|---|---|---|
| Geer & Katkin (1966) | P | 1 | 29 | 1 | Systematic desensitisation | NA | NA | NA | NA |
| Kahn, Baker & Weiss (1968) | S | 13 | NA | NA | Autogenic training | 52 | 22 (median) | 15 | 11 |
| Evans & Bond (1969) | P | 1 | 45 | 7 | Systematic desensitisation | 2.25 (hours of sleep) | 5.5 | NA | NA |
| Hinkle & Lutker (1972) | S | 7 | NA | 2 | In vivo desensitisation/relaxation | 70 | 34 | 23 | NA |
| Borkovec & Fowles(1973) | S | 37 | NA | NA | Progressive relaxation | 46 | 25 | 21 | ? |
| | | | | | Hypnotic relaxation | 43 | 24 | 39 | ? |
| | | | | | Self-relaxation placebo | 42 | 24 | NA | NA |
| | | | | | No treatment | 44 | 44 | NA | NA |
| Borkovec, Steinmark & Nau (1973) | R | 23 | NA | NA | Desensitisation v. relaxation v. both | 41 (global reporting) | 25 | NA | NA |
| Budzynski (1973) | P | 11 | NA | NA | EMG + EEG Biofeedback | 6 "improved" | | | |
| Traub, Jencks & Bliss (1973) | P | 7 | 42 | NA | Autogenic training | 27 | 11 | NA | NA |
| Weil & Goldfried(1973) | P | 1 | 11 | NA | Tape-recorded relaxation | 120 | 15 | NA | NA |
| Gershman & Clouser (1974) | S | 20 | NA | NA | Group desensitisation | 75 | 30 | 18 | 12 |
| | | | | | Group relaxation | 55 | 37 | 15 | 12 |

TABLE 1 Treatment studies employing Relaxation Therapies

(including desensitisation and biofeedback)

| Authors | Sample | n | Age | Duration | Treatment(s) | SOL pre | SOL post | SOL Fup | Fup mth. |
|---|---|---|---|---|---|---|---|---|---|
| Haynes,Moran,Woodward & Alexander (1974) | S | 14 | 20 | 5 | Progressive relaxation | 61 | 34 | NA | NA |
| | | | | | Discussion placebo | 53 | 40 | NA | NA |
| Nicassio & Bootzin (1974) | R | 30 | 45 | NA | Progressive relaxation | 131 | 73 | 47 | 6 |
| | | | | | Autogenic training | 109 | 46 | 47 | 6 |
| | | | | | Self-relaxation placebo | 119 | 117 | 112 | 6 |
| | | | | | No treatment | 122 | 99 | NA | NA |
| Steinmark & Borkovec (1974) | S | 52 | NA | >.5 | Progressive relaxation | 39 | 27 | 19 | 5 |
| | | | | | Systematic desensitisation | 36 | 24 | 18 | 5 |
| | | | | | Desensitisation placebo | 42 | 24 | 30 | 5 |
| | | | | | No treatment | 32 | 42 | NA | NA |
| Borkovec, Kaloupek & Slama (1975) | S | 56 | NA | NA | Progressive relaxation | 46 | 23 | 16 | 5 |
| | | | | | Prog. Rel. (Attention focusing only) | 38 | 26 | 23 | 5 |
| | | | | | Desensitisation placebo | 42 | 25 | 32 | 5 |
| | | | | | No treatment | 35 | 33 | NA | NA |
| Graham, Wright, Toman & Mark (1975) | S | 22 | NA | NA | Progressive relaxation | 5.9 | 3.1 | NA | NA |
| | | | | | Hypnosis | 5.9 | 3.1 | NA | NA |
| | | | | | | (rating of severity) | | | |
| Borkovec & Weerts(1976) | S | 36 | NA | NA | Progressive relaxation | 40 | 28 | 27 | 12 |
| | | | | | Desensitisation placebo | 53 | 39 | 58 | 12 |
| | | | | | No treatment | 47 | 37 | NA | NA |
| Freedman & Papsdorf (1976) | R | 18 | 23 | >.5 | EMG biofeedback | 42 | 13 | NA | NA |
| | | | | | Progressive relaxation | 43 | 20 | NA | NA |
| | | | | | Exercise control | 43 | 40 | NA | NA |

TABLE 1 Cont'd

| Authors | Sample | n | Age | Duration | Treatment(s) | SOL pre | SOL post | SOL Fup | Fup mth. |
|---|---|---|---|---|---|---|---|---|---|
| Pendleton & Tasto(1976) | S | 29 | NA | >1 | Metronome-conditioned relaxation | 49 | 34 | 32 | 6 |
| | | | | | Progressive relaxation | 55 | 25 | 11 | 6 |
| | | | | | Metronome-induced relaxation | 51 | 27 | 27 | 6 |
| | | | | | No treatment | 60 | 79 | NA | NA |
| Woolfolk,C-Kaffashan, McNulty, & Lehrer (1976) | R | 24 | 44 | 14 | Progressive relaxation | 65 | 29 | 27 | 6 |
| | | | | | Meditation | 74 | 34 | 25 | 6 |
| | | | | | No treatment | 60 | 63 | NA | NA |
| Haynes, Sides & Lockwood (1977) | R | 24 | 29 | 7 | Passive relaxation | 51 | 26 | 17 | 3 |
| | | | | | EMG biofeedback | 49 | 23 | 19 | 3 |
| | | | | | Self-relaxation control | 48 | 45 | 51 | 3 |
| Lick & Heffler (1977) | R | 40 | 48 | 12 | Progressive relaxation | 63 | 30 | NA | NA |
| | | | | | Progressive relaxation + tape | 62 | 38 | NA | NA |
| | | | | | False biofeedback placebo | 69 | 66 | NA | NA |
| | | | | | No treatment | 60 | 63 | NA | NA |
| Mitchell & White (1977) | S | 13 | 23 | 2 | Self-management 1 (PR then mental Rel) | 81 | 19 | 15 | 4 |
| | | | | | Self-management 2 (PR + mental rel) | 80 | 14 | 9 | 4 |
| | | | | | Self-monitoring/attention placebo | 78 | 47 | 31 | 4 |
| Borkovec & Hennings (1978) | S | 44 | NA | NA | Progressive relaxation | 34 | 19 | NA | NA |
| | | | | | Progressive rel.(tension release only) | 47 | 26 | NA | NA |
| | | | | | No treatment | 37 | 35 | NA | NA |
| Hauri (1978) | P | 37 | NA | NA | Frontalis EMG biofeedback | 94 | 64 | NA | NA |
| | | | | | Frontalis + theta EEG biofeedback | 64 | 45 | NA | NA |
| | | | | | SMR biofeedback | 103 | 70 | NA | NA |

TABLE 1 Cont'd

| Authors | Sample | n | Age | Duration | Treatment(s) | SOL pre | SOL post | SOL Fup | Fup mth. |
|---|---|---|---|---|---|---|---|---|---|
| Toler (1978) | R | 24 | 27 | NA | Progressive relaxation | 120 | 78 | 113 | 2 |
| | | | | | Progressive rel. + stimulus control | 108 | 69 | 101 | 2 |
| | | | | | No treatment | 111 | 104 | NA | NA |
| Borkovec, Grayson, O'Brien & Weerts (1979) | S | 29 | NA | NA | Progressive relaxation | 52 | 30 | 30 | 12 |
| | | | | | Prog.rel. (attention focusing only) | 49 | 41 | 35 | 12 |
| | | | | | No treatment | 53 | 55 | NA | NA |
| Carr-Kaffashan & Woolfolk (1979) | R | 30 | 40 | 11 | Progressive rel. (moderate insomnia) | 59 | 40 | 49 | 6 |
| | | | | | Progressive rel. (severe insomnia) | 150 | 80 | 49 | 6 |
| | | | | | Attention placebo(moderate insomnia) | 50 | 35 | 66 | 6 |
| | | | | | Attention placebo(severe insomnia) | 112 | 62 | 66 | 6 |
| Shealy (1979) | S | 70 | 20 | NA | Passive relaxation | 35 | 23 | 20 | 6 |
| | | | | | Passive relaxation + stimulus control | 37 | 24 | 22 | 6 |
| | | | | | Self-monitoring | 37 | 32 | 35 | 6 |
| | | | | | Discussion placebo | 38 | 29 | 21 | 6 |
| | | | | | No treatment | 45 | 36 | 41 | 6 |
| Coursey, Frankel, Gaarder & Mott (1980) | P | 22 | 38 | 14 | EMG biofeedback | 30 | 28 | NA | NA |
| | | | | | Autogenic training | 33 | 28 | NA | NA |
| | | | | | Electrosleep | 57 | 59 | NA | NA |
| Hauri (1981) | P/R | 48 | 41 | 8 | Frontalis EMG biofeedback | 91 | 63 | 52 | 9 |
| | | | | | EEG theta biofeedback | 48 | 30 | 40 | 9 |
| | | | | | SMR biofeedback | 64 | 41 | 26 | 9 |
| | | | | | No treatment | 94 | NA | 76 | 9 |

TABLE 1 cont'd

| Authors | Sample | n | Age | Duration | Treatment(s) | SOL pre | SOL post | SOL Fup | Fup mth. |
|---|---|---|---|---|---|---|---|---|---|
| Hauri, Percy, Hellekson, Hartmann & Russ (1982) | P/R | 16 | 49 | NA | EEG theta biofeedback | 27 | 24 | 12 | 9 |
| | | | | | SMR biofeedback | 35 | 32 | 18 | 9 |
| Nicassio, Boylan & McCabe (1982) | R | 40 | 44 | 11 | Progressive relaxation | 98 | 42 | 30 | 6 |
| | | | | | EMG biofeedback | 82 | 30 | 53 | 6 |
| | | | | | Biofeedback placebo | 99 | 60 | 37 | 6 |
| | | | | | No treatment | 92 | 85 | NA | NA |
| Woolfolk & McNulty (1983) | R | 44 | 43 | 15 | Progressive relaxation | 98 | 73 | 90 | 6 |
| | | | | | Imagery training | 108 | 50 | 35 | 6 |
| | | | | | Imagery training + tension release | 101 | 55 | 35 | 6 |
| | | | | | Somatic focusing | 104 | 68 | 84 | 6 |
| | | | | | No treatment | 110 | 109 | NA | NA |

TABLE 1 Cont'd

Only 8 studies have controlled adequately for both placebo and passage of time effects (Borkovec and Fowles, 1973; Nicassio and Bootzin, 1974; Steinmark and Borkovec, 1974; Borkovec et al, 1975; Borkovec and Weerts, 1976; Lick and Heffler, 1977; Shealy, 1979; Nicassio et al, 1982), with a further 10 studies (Haynes et al, 1974; Freedman and Papsdorf, 1976; Pendleton and Tasto, 1976; Woolfolk et al, 1976; Haynes et al, 1977; Mitchell and White, 1977; Borkovec and Hennings, 1978; Toler, 1978; Borkovec et al, 1979; Carr-Kaffashan and Woolfolk, 1979; Hauri, 1981; Woolfolk and McNulty, 1983) incorporating controls for one or other of these factors. Unfortunately, not one of these 18 at least partially controlled investigations was conducted on a clinically-presenting patient sample, and only one (Hauri, 1981) included referred cases of insomnia. Nevertheless, a number of consistent findings can be highlighted.

Studies are in general agreement that relaxation procedures are superior to no treatment in reducing self-reported sleep-onset time. Untreated subjects have exhibited little therapeutic improvement whereas SOL has reduced from an average (across studies) of 69 minutes at baseline to 40 minutes at post-treatment; a mean reduction of approximately 42% with relaxation training. Those studies which incorporated EEG measures have confirmed the usefulness of relaxation, though the magnitude of treatment effect has been less than that indicated by the subjective data alone (Borkovec and Weerts, 1976; Freedman and Papsdorf, 1976; Borkovec et al, 1979). Placebo treatments have produced variable results, with some proving very ineffective (eg. Nicassio and Bootzin, 1974; Haynes et al, 1977). The majority, however, have produced at least some improvement. At times, post-treatment improvement with placebo has been equivalent to active therapy (Steinmark and Borkovec, 1974, Borkovec et al , 1975; Carr-Kaffashan and Woolfolk, 1979; Shealy, 1979). The studies by Borkovec and his

associates introduced an imaginative, experimental procedure to control for experimenter demand and expectancy effects (Steinmark and Borkovec, 1974), and their work has generally found that active treatments operate effectively, even under conditions of negative expectation (counterdemand) whereas placebo instructions are effective only in combination with induced positive demand (Borkovec et al, 1975; Borkovec and Weerts, 1976). For the purposes of comparison, the average reduction in SOL, after placebo intervention, has been around 25%.

A number of authors have questioned the clinical significance of improvements via relaxation. Given that training involves at least 20 minutes practice per evening, a mean reduction of only 30 minutes in sleep latency renders these results clinically unimpressive (Freedman and Papsdorf, 1976). In addition, the concentration upon SOL as the major, and frequently the only, outcome measure makes it impossible to ascertain whether or not there is overall sleep pattern change. Also the relationship of SOL change to perception of sleep quality and sleep-complaints is unknown. Steinmark and Borkovec (1974) did report improvements on subjective ratings of difficulty in falling asleep and morning restedness, although only the former correlated significantly with SOL, and Borkovec et al (1975) were unable to replicate this improvement on the restedness rating. Borkovec et al (1979) have also produced some tentative evidence for increases in SWS after successful treatment.

Borkovec has conducted a number of investigations into the various components of the progressive relaxation programme (Borkovec et al, 1975; Borkovec and Hennings, 1978; Borkovec et al, 1979) which tentatively suggest the superiority of the total relaxation programme over the effects of its individual parts. The results of Borkovec et al (1979) were supportive of the hypothesis that tension-release is a

critical ingredient, however, the study by Woolfolk and McNulty (1983) reported a superior response with "imagery training" (a visualisation training procedure where subjects learned to call to imagination a number of common objects). Of interest in this last study was the achievement of a reduction in sleep-latency variance over time, as well as mean SOL (ie. sleep latency reduced and became less variable from night to night). In addition, it is also noteworthy that training procedures in relaxation therapy have been administered in a variety of formats. Relaxation training has been conducted "in vivo" in most cases, although a case study by Weil and Goldfried (1973) achieved significant improvement using tape-recorded instructions, and Gershman and Clouser (1974) made quite effective use of group relaxation.

Table 1 also permits the analysis of comparative effectiveness amongst the various types of relaxation procedure. Briefly, it seems that there is little to choose between them. Borkovec and Fowles (1973) found that hypnotic relaxation and a self-administered relaxation programme were as effective as progressive relaxation, and a number of reports have indicated the utility of autogenic training, originally developed by Schultz and Luthe (1959) (Kahn et al, 1968; Traub et al, 1973; Nicassio and Bootzin, 1974; Coursey et al, 1980). Other workers have made effective use of similar, passive relaxation and meditation techniques (Shealy, 1979; Haynes et al, 1977). These procedures generally involved sequentially focusing attention on pleasant bodily feelings without muscle tension-release cycles.

Before leaving the consideration of treatment comparisons, it would be useful to deal, briefly, with biofeedback techniques. There are two current explanations forwarded to account for the mechanism of effect in biofeedback. The first stems from the common use of EMG biofeedback where muscular relaxation is presumed to help prepare the individual

for sleep; and the second hypothesis, associated with EEG biofeedback, is that specific alterations in brain activity can be made in order to closely resemble EEG sleep and, thereby, to enhance relaxation more "centrally". Table 1 contains a number of studies which have used feedback techniques, with varying degrees of success. EMG feedback has been found to be as effective as relaxation (Freedman and Papsdorf, 1976; Haynes et al, 1977; Nicassio et al, 1982). There may be, however, a particularly strong placebo element in biofeedback, since the use of elaborate equipment may magnify non-specific effects (Nicassio et al, 1982). A recent series of studies by Hauri and his colleagues (Hauri, 1981; Hauri et al, 1982) trained insomniacs firstly in EMG and then in EEG feedback and found that brain activity feedback added little to the effects of EMG, except in the case of subjects who could be regarded as "non-tense". This is a developing area of research, but for the present the techniques are of unproven efficacy.

In concluding this section on the relaxation therapies, it is worthy of note that the approximate 50% of reports which have included follow up, have revealed maintenance of treatment gains after active therapy and, in a number of cases, these have been extended beyond the post-treatment level.

Treatments Based upon Stimulus Control Principles

Bootzin (1972) was the first to present an operant analysis of insomnia when he reported the case study of a 25 year old male with initial insomnia. He found that his stimulus control approach successfully reduced SOL, increased total sleep by more than two hours, and dramatically reduced the frequency of nightly risings from bed. It was this last variable which he presented as his main outcome measure and improvements were maintained at 2 month follow-up. Data from this and other studies employing stimulus control are contained in Table 2.

| Authors | Sample | n | Age | Duration | Treatment(s) | SOL pre | SOL post | SOL Fup | Fup mth. |
|---|---|---|---|---|---|---|---|---|---|
| Bootzin (1972) | S | 1 | 25 | 5 | Stimulus control | 4/5 (risings | 0 from | 0 bed) | 2 |
| Tokarz & Lawrence (1974) | S | * | * | * | Stimulus control | 51 | 6 | 7 | * |
| | | | | | Temporal control | 46 | 8 | 13 | * |
| | | | | | Stimulus control w'out temporal cont. | 56 | 7 | 11 | * |
| | | | | | Relaxation placebo | 49 | 47 | NA | NA |
| | | | | | No treatment | 45 | 56 | NA | NA |
| Haynes, Price & Simons (1975) | R | 4 | 41 | 12 | Stimulus control (ABAB designs) | 56 | 16 | 16 | 9 |
| Turner & Ascher (1979b) | C | 6 | 47 | 15 | Stimulus control | 87 | 27 | NA | NA |
| Alperson & Biglan (1979) | R | 29 | 37 | NA | Stimulus control + relaxation (young) | 59 | 29 | 25 | 2 |
| | | | | | Stimulus control + relaxation (old) | 52 | 47 | 49 | 2 |
| | | | | | Stimulus control + active relaxation | 73 | 53 | 37 | 2 |
| | | | | | No treatment | 54 | 79 | NA | NA |
| Norton & DeLuca (1979) | P | 1 | 22 | 6 | Stimulus control + relaxation | 30 | 10 | NA | NA |
| Zwart & Lisman (1979) | S | 47 | NA | NA | Stimulus control | 46 | 26 | 25 | 1 |
| | | | | | Non-contingent control | 43 | 34 | 27 | 1 |
| | | | | | Counter-control | 49 | 25 | 18 | 1 |
| | | | | | Temporal control | 41 | 19 | 23 | 1 |
| | | | | | No treatment | 46 | 39 | 42 | 1 |
| Puder,Lacks,Bertelson & Storandt (1983) | R | 16 | 67 | 15 | Stimulus control | 66 | 33 | 33 | 2 |

* Original reports unobtainable. Data reported are from Bootzin and Nicassio (1978).

TABLE 2 Treatment studies employing Stimulus Control

Since Bootzin's pioneering work a number of researchers have published case report papers. Haynes et al (1975) reported four single case experimental designs of subjects who had longstanding sleep difficulties, but had not benefitted from hypnotic medication. After a two week baseline, stimulus control therapy was initiated and maintained until sleep pattern stability was evident. The subsequent return to baseline conditions revealed the expected reversal effect, but in only two of the four cases. For all subjects, however, treatment was effective in reducing SOL by approximately 40 minutes and improvement was maintained at follow-up 9 months later. Although the absence of a reversal could be viewed as evidence against the specific impact of stimulus control, the fact that treatment gains persisted, at lengthy follow-up, at least suggests that therapy produced some form of habit change.

Turner and Ascher (1979b) criticised the Haynes et al study on the grounds that ABAB designs are ill-advised with behavioural procedures, because of the "carry-over" effect upon cessation of treatment (Hersen and Barlow, 1976). Instead they proposed the use of a multiple baseline approach more adequately to test cause-effect relationships. They presented a series of six self-referred out-patients with initial insomnia. All subjects completed a baseline phase after which half were allocated to stimulus control and half to a quasi-desensitisation placebo, which has commonly been used in psychological research (Steinmark and Borkovec, 1974). After 4 weeks, clients in the second group were also given active therapy which the first group received throughout. Stimulus control was found to have a specific impact upon sleep latency, in each case reducing SOL by two-thirds or more. Placebo treatment did not produce this effect but when these patients were introduced to stimulus control significant change was observed. Reductions in medication also followed the introduction of stimulus

control. The slight increment in SOL in one case could have been caused by rebound insomnia. The major advantage of this study was that it reported cases of severe and long-term sleep disturbance. The rapid elimination of these difficulties provides strong evidence for the potency of stimulus control. A more recent case study by Norton and De Luca (1979) found a combination of relaxation and stimulus control procedures effective in reducing sleep latency and intermittent wakenings in a mild case.

A number of workers have attempted to investigate the effectiveness of component parts of the stimulus control "package", and some of this work was introduced in Chapter 2. Bootzin and Nicassio (1978) referred to an unpublished study by Tokarz and Lawrence (1974) which compared the efficacy of the complete stimulus control procedure with two different constituent parts. These were, a temporal control strategy, including only instructions aimed at regularising the sleeping pattern according to the constraints of time, and an alternative approach based solely upon improvement of the bed and bedroom environment's function as discriminative stimuli for falling asleep. Relaxation placebo and no treatment control groups were also included. These workers found that all three active interventions resulted in highly significant reductions in SOL, and that there were no differences between them. Zwart and Lisman (1979), however, have criticised this study suggesting that the temporal control procedure did not closely approximate to that element in Bootzin's conception of stimulus control. They conducted their own analysis comparing stimulus control with temporal control, counter-control (subjects were to sit up in bed, read, watch TV etc. at bedtime, and to deliberately engage in sleep-incompatible behaviours in the bedroom during the daytime), non-contingent control (the theoretically critical contingency of rising after 20 minutes was removed by instructing subjects to rise a fixed number of times within

20 minutes of retiring), and a waiting-list control group. All patients were given the counterdemand instruction that improvement would not occur until the fourth week of therapy. Results indicated that those receiving stimulus control and counter-control improved significantly compared with non-contingent control and waiting-list cases, during the counterdemand period, although, after positive demand the temporal control group became as effective as either of these procedures. At follow-up one month later treatment gains were maintained and there were no significant differences amongst any of the treatment groups on any measure.

Zwart and Lisman provided some useful indications as to the effective elements of stimulus control. Firstly, the study by Tokarz and Lawrence was corroborated in the finding that the complete "package" was not superior in effectiveness to some of its elements. Secondly, the efficacy of the counter-control procedures implied that supposedly sleep-incompatible behaviors may not be particularly relevant cues for arousal in insomniacs. It may be that any disruption of usual sleep pattern might produce improvement. Turner and Ascher (1979b) have reported that subjects regarded stimulus control as helpful in breaking up the usual chain of worrying and anxiety. Thirdly, there is the unexplored possibility that rising from bed may act as an aversive consequence of continued wakefulness, and this is an area which could be usefully examined in further research. Finally, the fact that Zwart and Lisman's student subjects did not improve during counterdemand, when treated with temporal control alone, may simply reflect their relatively mild insomnia difficulties. It has already been suggested that a didactic approach, involving the rescheduling of bedtime and rising time, may be particularly appropriate for older patients who have "developmental" insomnia in the absence of daytime impairment.

Alperson and Biglan (1979) have reported a comparison study where stimulus control plus passive relaxation was given to groups of young and old subjects. Interestingly, they found that therapy was statistically and clinically more beneficial for their younger group. The older subjects showed little change. Puder et al (1983) have also provided a report of stimulus control treatment with older adults. These workers randomly assigned a final sample of 16 clients (mean age of 67 years) to either immediate or delayed treatment (the latter involving a 10 week waiting period). Data on SOL revealed considerable reductions for both groups when active intervention was applied. Overall reductions of approximately 50% were maintained at 2 month follow-up. By comparison with Alperson and Biglan's study, Puder et al have demonstrated that age alone may not be a sufficient factor to preclude treatment gain. Unfortunately, however, neither study included placebo groups and it is possible that the differential effectiveness was simply related to uncontrolled demand characteristics or expectancy effects.

Treatments Based Upon Paradoxical Intention Therapy

Frankl (1955) was the first to describe the two related therapeutic techniques of paradoxical intention and dereflection. These procedures were used in situations where patients were concerned about the frequency of a response occurring either too often (eg. perspiring) or too seldom (eg. falling asleep). Since the dereflection procedure differs from paradoxical intention, only in the direction of change which the client desires, the term paradoxical intention is now employed to denote both the prescription to intentionally increase uncomfortably high frequency behaviours, and to intentionally decrease uncomfortably low frequency behaviours (Ascher, 1979). It is only recently that paradox has been adopted into the behavioural literature,

and its initial incorporation was based upon a rather eclectic view of therapy (eg. Lazarus, 1971; Fay, 1973). The development of the performance anxiety-based rationale, hypothesised to account for its mode of action (Ascher, 1979; Ascher and Turner, 1979), however, has afforded the technique more general acceptance.

Ascher and Efran (1978) provided the first case reports on poor sleepers, although only three of their five subjects had a primary problem of sleep difficulty. Nevertheless, their demonstration of the effectiveness of paradox was impressive since these were patients who had previously failed to respond to more conventional treatment (relaxation and desensitisation). For one subject who responded to paradoxical intention the original programme was reintroduced and the demonstration of a reversal effect, followed by improvement once again when paradox was readministered, provided further evidence for the specific impact of the procedure. Ascher and Efran reported only sleep latency measures, although they stated in a footnote that a reduction in frequency of night-time wakening was also evident, and that improvements in ratings of sleep quality paralleled changes on sleep parameters. Other case study material has been reported by Relinger et al (1978), Relinger and Bornstein (1979) and Espie and Lindsay (1985). The results of all of these studies are summarised in Table 3.

Relinger et al, employing a time series analysis on a single case, found that the paradoxical instruction produced and sustained substantial SOL reduction which was also associated with significant positive change in ratings of difficulty in falling asleep, restfulness of sleep, and daytime functioning. The validity of their subject's sleep complaint must, however, be called into question since, in spite of a reported 20 year duration of insomnia, the client had never previously sought professional help. These workers published a further

| Authors | Sample | n | Age | Duration | Treatment(s) | SOL pre | SOL post | SOL Fup | Fup mth. |
|---|---|---|---|---|---|---|---|---|---|
| Ascher & Efran (1978) | C | 5 | 30 | 9 | Paradoxical intention + Behav.prog. | 48 | 10 | NA | NA |
| Ascher & Turner(1979) | R | 25 | 39 | 8 | Paradoxical intention | 62 | 29 | NA | NA |
| | | | | | Desensitisation placebo | 63 | 51 | NA | NA |
| | | | | | No treatment | 71 | 62 | NA | NA |
| Relinger,Bornstein & Mungas (1978) | R | 1 | 31 | 20 | Paradoxical intention | 64 | 10 | 10 | 12 |
| Relinger & Bornstein (1979) | R | 4 | NA | 23 | Paradoxical intention | 110 | 47 | 20 | 3 |
| Ascher & Turner(1980) | R | 40 | 37 | 9 | Paradoxical intention (type A) | 63 | 29 | NA | NA |
| | | | | | Paradoxical intention (type B) | 68 | 45 | NA | NA |
| | | | | | Desensitisation placebo | 57 | 44 | NA | NA |
| | | | | | No treatment | 64 | 60 | NA | NA |
| Fogle & Dyal (1983) | R | 35 | 41 | 12 | Paradoxical intention (type A) | 75% | 78% | NA | NA |
| | | | | | Paradoxical intention (type B) | 76% | 82% | NA | NA |
| | | | | | Self-monitoring control | 73% | 80% | NA | NA |
| Ott, Levine & Ascher (1983) | R | 56 | NA | NA | Feedback (on sleep) | 53 | 35 | NA | NA |
| | | | | | Paradoxical intention | 55 | 33 | NA | NA |
| | | | | | Paradoxical intention + feedback | 53 | 72 | NA | NA |
| | | | | | No treatment | 54 | 52 | NA | NA |
| Espie & Lindsay (1985) | P | 6 | 43 | 7 | Paradoxical intention | 123 (Alternative treatment) | 129 | 34 | 3 |

TABLE 3 Treatment studies employing Paradoxical Intention

four case studies (Relinger and Bornstein, 1979) for whom insomnia constituted a more substantial problem. Employing a multiple baseline design, the average scores for SOL and frequency of wakening were significantly reduced at post-treatment, as were ratings of difficulty in falling asleep. Subjects also felt more rested upon wakening but there were no significant changes in total sleep time or on measures of daytime functioning. At follow-up 3 months later, all of the improved variables evidenced continuing gains.

Espie and Lindsay (1985) have recently published six cases of chronic insomniacs who were treated by means of paradoxical intention. These workers found a variable response to therapy in that the sleep problems of two patients were significantly exacerbated by the treatment, to the point where therapy had to be discontinued, and a third patient suffered a temporary increment in SOL. The three remaining subjects did, however, improve in a manner similar to previous reports. Changes in total sleep time appeared to reflect changes in sleep latency. Espie and Lindsay suggested that these variable outcomes may reflect the tendency of some individuals with performance anxiety to redirect their anxieties into trying to remain awake, that is an active avoidance of sleep, in order to achieve the therapist's criteria of success. This may be a mentally arousing process whereas, for the insomniac who benefits from paradox, more "passive" avoidance of performance effort may account for treatment gains.

Ascher and Turner (1979) conducted a controlled experimental investigation in which they compared paradox with placebo and no treatment controls. Subjects in the paradoxical intention group reported significant reductions in sleep-latency, had fewer problematic wakenings in terms of returning to sleep, and exhibited significant increases in "restedness" after sleep compared with either control

condition. [These results replicated those of Turner and Ascher (1979a) - to be reported in the next section]. Ascher and Turner (1980) again repeated their experimental design, but this time considered alternative methods for the administration of paradox. Type A administration provided clients with the standard performance anxiety reduction rationale; whereas type B involved the use of a "reframing" procedure where clients were instructed to remain awake for as long as possible, in order to become aware of the anxiety-provoking thoughts which they experienced while awaiting sleep-onset. They were told that these thoughts would later be desensitised. The results of this study indicated that the conventional procedure was superior in efficacy, perhaps because the reframing approach implied that therapy would not begin until after preliminary data collection. The authors did suggest, however, that reframing may be particularly useful in the case of the client who failed to understand the performance anxiety model.

Ott et al (1983) conducted an experimental study to test further the explicit demand characteristics of the reframing procedure. The design of their experiment was a 2 (feedback/no feedback) by 2 (paradoxical instruction/no paradoxical instruction) factorial, where "objective" feedback was provided by means of the "sleep-monitoring unit" (Ott et al, 1982). The results of the study confirmed their working hypothesis that patients' self-reports would be influenced by experimental demand, when these were made explicit and when access was given to actual, objective sleep data. Unfortunately, however, these workers discussed their results only with reference to reporting bias, and did not offer any explanation of the mechanism by which subjects ensured that they remained awake. A major problem with the reframing procedure is that it may simply encourage patients in some form of self-administered sleep deprivation.

Fogle and Dyall (1983) also have considered variations on the instruction which subjects can be given. They allocated their sample to either conventional paradoxical intention or to an alternative version where individuals were told simply to "give up" trying to fall asleep (ie. no direct paradoxical instruction to stay awake). They argued that sleep efficiency was the best overall measure of outcome, and reported significant improvements on this variable for each of the experimental groups. In addition, they found that it was only the "giving up" group which improved on a self-report measure of sleep performance anxiety. In spite of the statistical changes which were demonstrated, however, the improvements in actual sleep parameters were relatively small and of limited clinical significance. This may reflect the fact that both treatments were conducted as bibliotherapy. Also, the reporting of only sleep efficiency changes, obscures the locus of improvement. It is unclear whether improvements were due to changes in SOL, wakening frequency, total sleep time, or a combination of these factors. Furthermore, the relative contributions of such variables to the treatment outcomes for each therapy were left undetermined.

## Comparative Studies of Treatment Outcome

Only four studies comparing the effectiveness of different psychological approaches have been published in research journals (Hughes and Hughes, 1979; Turner and Ascher, 1979a; Lacks, Bertelson, Gans and Kunkel, 1983; Turner, DiTomasso and Giles, 1984). An additional three earlier studies (Bootzin, 1975; Slama, 1975; Lawrence and Tokarz, 1976), however, were reviewed in Bootzin and Nicassio's (1978) paper and are worthy of consideration. Unfortunately, some of the original data from these reports are missing and the source manuscripts are unobtainable. The summary results of all 7 studies are presented in Table 4. The reports will be reviewed chronologically.

| Authors | Sample | n | Age | Duration | Treatment(s) | SOL pre | SOL post | SOL Fup | Fup mth. |
|---|---|---|---|---|---|---|---|---|---|
| Bootzin (1975) | R | 66 | ** | ** | Stimulus control | 96 | 29 | NA | NA |
| | | | | | Progressive relaxation | 105 | 76 | NA | NA |
| | | | | | Relaxation placebo | 81 | 63 | NA | NA |
| | | | | | No treatment | 63 | 39 | NA | NA |
| Slama (1975) | S | * | * | * | Stimulus control | 46 | 13 | 19 | * |
| | | | | | Progressive relaxation | 36 | 16 | 18 | * |
| | | | | | No treatment | 33 | 33 | NA | NA |
| Lawrence & Tokarz (1976) | S | * | * | * | Stimulus control (individual) | 65 | 8 | NA | NA |
| | | | | | Stimulus control (group) | 65 | 17 | NA | NA |
| | | | | | Progressive relaxation | 65 | 45 | NA | NA |
| | | | | | Desensitisation placebo | 65 | 66 | NA | NA |
| Hughes & Hughes (1979) | R | 36 | 34 | NA | Stimulus control | 57 | 28 | | |
| | | | | | Progressive relaxation | 55 | 40 | 27 | 12 |
| | | | | | EMG biofeedback | 44 | 27 | | |
| | | | | | Pseudo-EMG biofeedback | 45 | 17 | | |
| Turner & Ascher(1979a) | R | 50 | 39 | 11 | Stimulus control | 64 | 21 | NA | NA |
| | | | | | Progressive relaxation | 63 | 28 | NA | NA |
| | | | | | Paradoxical intention | 63 | 29 | NA | NA |
| | | | | | Desensitisation placebo | 57 | 44 | NA | NA |
| | | | | | No treatment | 64 | 60 | NA | NA |

* Original reports unobtainable. Data reported are from Bootzin and Nicassio (1978)

TABLE 4 Treatment studies comparing different psychological therapies

| Authors | Sample | n | Age | Duration | Treatment(s) | SOL pre | SOL post | SOL Fup | Fup mth. |
|---|---|---|---|---|---|---|---|---|---|
| Lacks, Bertelson, Gans & Kunkel (1983) | R | 64 | 41 | 14 | Stimulus control | 75 | 33 | 23 | 3 |
| | | | | | Progressive relaxation | 61 | 49 | 33 | 3 |
| | | | | | Paradoxical intention | 64 | 54 | 34 | 3 |
| | | | | | Desensitisation placebo | 70 | 54 | 31 | 3 |
| Turner, DiTomasso, & Giles (1984) | P | 40 | 31 | NA | Stimulus control | 70 | 48 | NA | NA |
| | | | | | Progressive relaxation | 64 | 49 | NA | NA |
| | | | | | Stimulus control + progressive rel. | 65 | 69 | NA | NA |
| | | | | | No treatment | 64 | 59 | NA | NA |

TABLE 4 Cont'd

Bootzin (1975) randomly allocated a sample of 66 recruited subjects to either stimulus control, progressive relaxation, self-relaxation placebo or waiting-list control groups. Although self-reports of sleep latency suggest that these were severe initial insomniacs, they did not present through clinical channels, and the chronicity of the presenting problem is not known. Inspection of the data on SOL in Table 4, indicates that stimulus control produced a mean reduction of 67 minutes, compared with the active relaxation group where improvements of around 30 minutes were obtained. Bootzin and Nicassio (1978) reported that in absolute terms after treatment, 57% of those who received stimulus control averaged less than 25 minutes to fall asleep, as contrasted with 29% of those who were trained in progressive relaxation. Importantly, however, 27% of the self-relaxation group and 22% of untreated cases also achieved this criterion of improvement. Indeed, the post-treatment mean scores for stimulus control were a mere 10 minutes superior to the waiting-list procedure. In spite of the evident ambiguity of these results, Bootzin and Nicassio, and Borkovec (1982) have cited Bootzin's study as evidence for the superior effectiveness of stimulus control treatment. It seems possible, however, that the apparently therapeutic effect of the passage of time may reflect the transient nature of insomnia in at least a fair proportion of these subjects.

The study by Slama (1975) was conducted in Borkovec's laboratory using groups of undergraduate students who complained of significant sleep disturbance. It appears from baseline measures that insomnia was relatively mild (mean SOL < 40 mins). Indeed, the pre-treatment levels of sleep disturbance are similar to post-treatment scores in some of the better controlled studies (Lacks et al, 1983; Turner et al, 1984). Slama included a counterdemand instruction which revealed that both stimulus control and relaxation were superior to no treatment, even

under conditions of negative expectancy, although stimulus control was also superior to relaxation. At post-treatment, however, and after positive demand, subjects in both therapy conditions were sleeping similarly well, and improvements were maintained at unspecified follow-up.

Bootzin and Nicassio reported a third unpublished study, by Lawrence and Tokarz (1976), where a more severely sleep disturbed student population was selected. Subjects were randomly assigned to individual stimulus control, group stimulus control (3 clients seen together), progressive relaxation and desensitisation placebo. Both stimulus control approaches were found to be highly effective in reducing mean sleep latencies to less than 20 minutes. This improvement rate of 75-90% was considerably higher than the 31% exhibited after relaxation, although the durability of change was not reported. Taking these first three studies together, therefore, the preliminary evidence consistently suggests a preference for stimulus control over relaxation therapy.

Hughes and Hughes (1979) recruited 36 volunteers (12 male, 24 female) through newspaper advertisements. The mean age of their sample was 34.2 years, and although no figures for chronicity of complaint are available, one of the selection criteria specified a minimum of 4 months duration. Subjects were randomly assigned to one of the four treatment groups described in Table 4 and submitted a refundable monetary deposit, before being finally accepted into therapy. Sleep measures were recorded in daily diaries for a two week baseline period, and for a further fortnight at post-treatment. Unfortunately, time in treatment appears to have varied considerably. Both biofeedback conditions comprised 8 therapy sessions, compared with relaxation training which involved 4 sessions, and stimulus control which had only

2 sessions. (Hughes and Hughes explained that these inconsistencies were due to the "complexity of the tasks"). Stimulus control and progressive relaxation were along conventional lines, as was EMG biofeedback where subjects attended to actual feedback through headphones attached to a myograph. The pseudo-biofeedback group, however, listened to false feedback from a cassette recorder.

Results indicated that all four groups improved significantly with no significant between group differences. Stimulus control did, however, produce this improvement very rapidly. Interestingly, biofeedback had no effect upon EMG values. Indeed, even the lowest post-treatment EMG levels achieved were not typical of a state of low physiological arousal. It seems, therefore, that treatment outcome was independent of a reduction in muscle tension. Hughes and Hughes proposed that a cognitively-mediated process, similar for all treatments, might explain their results. This process, it was suggested, might function to reduce apprehension about sleep and other forms of intrusive worry. A more adequate evaluation of the placebo treatment would have been possible had the authors included a simple counterdemand instruction. Other workers have found that placebo treatments become effective under conditions of positive expectancy, but are otherwise not superior to no treatment (Borkovec et al, 1975; Borkovec and Weerts, 1976). The follow-up data in the Hughes and Hughes study were also unsatisfactory. Verbal estimates of sleep latency were available for only 12 cases, and these were summarised as a single follow-up cohort. It is, therefore, impossible to differentiate between the treatments.

The most influential outcome study to appear in the literature has been that of Turner and Ascher (1979a). This study represented a creditable response to the methodological and analytical shortcomings of previous work. These authors advised caution regarding suggestions that stimulus

control might be superior to relaxation. Turner and Ascher also brought the technique of paradoxical intention within the fold of experimental investigation. Five treatment conditions were investigated ie. stimulus control, progressive relaxation, paradoxical intention, desensitisation placebo and waiting-list control. Twenty-five men and 25 women (mean age 39 years) were recruited via newspaper articles and, following a 10 day baseline period, ten clients were randomly allocated to each of the experimental conditions. Individual sessions (of uniform length) were conducted, once weekly for four weeks, and pre and post-treatment measures were recorded on a modified version of the DSQ. Assessments of social aquiescence and therapy credibility were also included as part of the methodological controls. Treatments did not differ significantly on either of these measures. Unfortunately, the credibility assessment was completed at post-therapy and was not, therefore, a true reflection of perceived credibility of the therapy rationales, but rather is likely to have been contaminated by subjects' actual treatment responses.

Turner and Ascher employed multivariate assessments for examining outcome, arguing that MANOVA provided a more conservative approach to significance testing ( compared with numerous univariate analyses), by reducing the liklihood of Type 1 error. Their results revealed no significant between group differences at baseline, on any measure, however post-treatment comparisons demonstrated significant differences between the treatment groups and the control groups, although there were no differences amongst the three active therapies. SOL, ratings of difficulty falling asleep, and restedness, were the best discriminators of treatment outcome, whereas total sleep time contributed little discriminant weight. In addition to these statistical results, inspection of Table 4 reveals that SOL reductions were of clinical benefit, being in the range 54-67%. It would appear from this study

that the three psychological therapies are equally effective.

However, some important issues were not dealt with in Turner and Ascher's study. Non-clinical subjects were again selected, and it may be that such clients will readily respond to most therapeutic endeavours. In addition, Turner and Ascher permitted the uncontrolled use of sleep medication, and even cited drug intake as a useful dependent variable. This is unsatisfactory because of the contaminating effects of varied usage upon sleep pattern. The major criticism, however, concerns the lack of any data during the process of treatment or during follow-up. It was not possible to tell at what point therapeutic effects emerged, or whether treatment gains persisted beyond the termination of therapy.

Lacks et al (1983a) completed a thorough replication of Turner and Ascher's work on a more clinically-based sample. Their 64 participants (48 female, 16 male) were obtained from both newspaper (and other media) announcements and letters to referring physicians. In spite of the liklihood that these modes of selection might constitute two discrepant samples, Lacks et al made no attempt to compare on this basis, or even to report the relative proportions which the sub-groups comprised. Similar to a number of previous reports, all subjects were required to submit a monetary deposit, prior to completing a 7 day baseline period. On the basis of this pre-treatment assessment, subjects were labelled as either mild (SOL 15-44 mins), moderate (SOL 45-75 mins) or severe insomniacs (SOL 76-152 mins) and were randomly allocated, within severity blocks, to each of the treatments outlined in Table 4. Therapy was conducted in four, weekly, small group sessions, of approximately 1 hour duration. The counterdemand procedure was applied to the effect that improvement would not occur until the final week.

Lacks et al reported outcome data on only the measure of sleep-onset latency. The SOL of the stimulus control group was clearly, and consistently, the lowest at each assessment point after week one, and this was true for all degrees of insomnia. Overall, subjects receiving this intervention reduced sleep latency by an average of 52% at Week 3, and even during the positive demand phase no other treatment exceeded this level of success. The authors pointed out that, although all treatment groups continued to decrease their level of initial insomnia during follow-up, "the major advantage of stimulus control was that high levels of treatment success were accomplished very quickly". They concluded that "regardless of the severity of onset insomnia, the treatment of choice is stimulus control". In consideration of the issue of severity on a broader basis, their strongest evidence was against the existence of a negative relationship between severity and outcome, ie. a severe presenting problem does not contra-indicate therapy.

Lacks et al discussed their results with reference to the study by Turner and Ascher (1979a). By way of contrast, they pointed out that paradoxical intention did not appear to be a recommended treatment, since some of their patients demonstrated increases in mean SOL after therapy (cf. Espie and Lindsay, 1985). Criticisms of the Lacks et al study rest upon the subject selection procedures, and the choice of a single sleep pattern variable as the outcome measure. By their own admission, these workers referred to the skewed distribution of their sample, stating that they were largely white, middle class, well educated, financially able to afford to deposit $40 for four months, and were all drug-free. At the very least, they were clearly a motivated sample, since less enthusiastic subjects would be likely to hesitate at the financial contract. Their study does, however, provide an influential controlled investigation into the management of insomnia and has challenged Turner and Ascher's finding of equivalent outcomes

across therapies. Stimulus control did once again emerge favourably, at least for SOL reduction.

Finally, Turner et al (1984) have reported a comparative study of 40 referred patients who were treated by either stimulus control, relaxation, a combination of the two procedures, or waiting-list control. The mean age of this sample was relatively young (31 years), and patients had suffered from insomnia for an average of 4 years. The data in Table 4 reveal that both stimulus control and progressive relaxation produced a treatment response superior to both the combined therapy programme and waiting-list control. The average reductions in SOL for the relaxation and stimulus control groups were, however, only 15 minutes and 21 minutes respectively (a non-significant difference) and such improvements may be of limited clinical value. Turner et al also examined a number of other sleep parameters such as total sleep time, number of wakenings, and ratings of restedness and difficulty falling asleep, but found no significant changes. They also, again inappropriately, included drug intake as a dependent variable.

One interesting finding from this study, however, was the failure of the combined "package" treatment. It might reasonably be expected that such an approach would prove more powerful than either of the components alone, but conversely, Turner et al reported an 82% failure rate. They interpreted these results in terms of De la Pena's (1978) theory that treating insomniacs with an antagonistic, arousal-reducing procedure (relaxation) and an (arguably) arousal-increasing one (stimulus control) would lead to therapeutic failure. De la Pena regarded the aetiology of primary insomnia as a state of "chronic stimulus underload", with insomnia functioning to balance this lack of equilibrium through increments in sensory input. This theory, however, has attracted little interest in the research literature and has very

limited empirical support. Perhaps the provision of two sets of therapeutic instructions, and also the associated rationales, simply rendered the subjects' task ambiguous with a consequent weakening of therapeutic response.

Lichstein (1985) has summarised very succinctly the effects of psychological treatments upon insomnia in stating that "most psychological treatments are helpful, and no one treatment clearly emerges as superior to others, although stimulus control has an edge". The present study was designed to provide further evaluation of a number of psychological approaches with clinically-presenting cases.

# CHAPTER 6

## OVERVIEW OF THE TREATMENT OUTCOME LITERATURE

## SOME METHODOLOGICAL ISSUES

Chapter 5 provided an extensive review of the available psychological treatment studies. Interventions, of whatever type, have generally been positive in their impact, although the degree of measurable change has been found to vary considerably. Relaxation methods have undoubted face validity and popular appeal, but appear to be equally effective in whatsoever form of administration, making it difficult to determine what is/are the critical ingredient(s), and indeed the mechanism by which relaxation obtains its effect. Reductions in SOL have been modest in proportion. Paradoxical intention may be viewed as another anxiety-reduction procedure focusing, however, more upon cognitive than physiological state. By comparison with relaxation training, the literature on paradox is very small and contains no clear statement of its usefulness. Of all the procedures reviewed, it appears to have been uniquely capable of exacerbating insomnia for some people. Of the three major treatments, stimulus control perhaps emerges as the most promising. Similar to relaxation training, the means by which stimulus control achieves its effects remain uncertain. As a "package" of instructions, however, it does seem to offer the greatest potential in the management of insomnia, regardless of severity. Such a conclusion, however, must be tempered by the fact that such effects have been reasonably demonstrated within only one outcome variable, that is SOL.

From a methodological point of view, treatment studies have varied from the grossly inadequate to the imaginative and well-controlled. Such a spread of experimental method is inevitable in the historical review of any clinical condition. More recent reports have identified and remedied a number of weaknesses in methodology which were previously

evident. Nevertheless, there remain some serious shortcomings, and it was partly through a concern to address these that the present study was undertaken. For the purposes of clarity it will be useful to consider design features under several headings.

SUBJECT SELECTION

Published reports on insomnia have been largely on non-clinical subjects and are, therefore, subject to the criticisms commonly levelled against analogue research. An analogue approach does of course have its place: being of an exploratory nature, key issues have been identified and methodological, practical and conceptual questions raised for further enquiry, but subsequent experimental refinements have failed to deal with the major issue of the generalizability of these findings to the patient population. The mere passage of time appears, rather, to have afforded a status and clinical efficacy to procedures which may not be empirically justified. Approximately half of the available reports have been conducted on student populations, with most of the remainder being the product of "recruitment drives" through media advertisements, with subjects being partly selected on the basis of ensured compliance. Importantly, most studies have given no indication as to whether or not subjects were actively seeking help for insomnia at the time of selection. Three recent studies utilised a combination of media announcements and physician referrals (Lacks et al 1983a,b; Puder et al, 1983) and the study by Turner et al (1984) employed referred clients. These are, therefore, indicative of some awareness concerning the need more closely to approximate the clinical population. The greatest part of the literature is, however, based upon convenient populations.

The practising clinician cannot be satisfied until results are replicated in studies using unsolicited subjects. Killen and Coates

(1979) have made the point that "college students ——— do not respond to treatments in the same way as do persons from the community", but this argument might also be extended by questioning the assumption that "adults in the community" equate readily with adults who present at the clinic requiring treatment. Coates and Thoresen (1980) refer to the "modest to negative outcomes obtained when (behavioural) strategies are evaluated with subjects who are older, who have experienced insomnia for two years or more, who present themselves at clinics for treatment, or who are referred by physicians". It is estimated that 10% of all NHS prescriptions in the UK are for hypnotic drugs, which indicates that it is hardly necessary to solicit candidates.

Patients on Hypnotic Drugs

Past research has generally excluded subjects currently taking hypnotic drugs. This has the major methodological advantage of eliminating a potentially confounding variable. The case for the inclusion of such subjects, however, may be a valid one, for several reasons.

Firstly, a substantial proportion of out-patients attending sleep clinics make regular use of night-time medication (82% in a study by Roth, Kramer and Lutz, 1976). There is, therefore, the danger of selecting out of research studies a large proportion of the clinically presenting group. Studies may have problems in the generalizability of results. Secondly, there appears to be sufficient understanding of hypnotic drug effects to make complete exclusion of such patients unnecessary. The rebound phenomenon is sufficiently understood to permit the presentation of accurate information regarding likely changes in sleep pattern subsequent to withdrawal. Supportive and educative input may enable patients to tolerate the initial few weeks more readily, and help to counteract the problem of misattribution which reinforces the patient's perception of himself as a poor sleeper.

The literature indicates that a minimum 5 week withdrawal period is required (see Chapter 5), and research studies should follow this period with the collection of a stable baseline. In some instances withdrawal may need to be gradual at the recommended reduction rate of one therapeutic dose per week (Kales et al, 1974). Such a systematic programme would be preferable to simply encouraging patients to discontinue medication, and including them only if they are successful.

Thirdly, an alternative model is also a realistic possibility for those patients who are unable at first to cease hypnotics. Habituation quickly develops to most hypnotics, after several weeks of nightly administration (Chapter 5). It should be possible, therefore, to maintain such patients on drugs at the habituated level, gather baseline data, and assuming stability can thereby be demonstrated, to institute psychological treatment to promote sleep pattern improvement. Thereafter, greater success may be achieved in "weaning" patients off drugs at the prescribed rate. This would seem to be a valid procedure, both methodologically and clinically, since the effects of hypnotics can be justifiably considered constant if taken at the same dosage after habituation. Observed treatment effects can then be attributed to the experimental manipulation per se. This model seeks to ensure that drug intake becomes a controlled variable. Clearly if pre-treatment withdrawal proves feasible in a given case then that would be the option of choice, but both procedures are preferable to either the total exclusion of drug-using patients or their inclusion on the basis of a statement that they will not take hypnotics during the experimental period. Drug level cannot be employed as a dependent variable (patients self-medicate as required) because of the greater long-term effectiveness of intermittent use and the contaminating effects upon data of rebound insomnia.

One of the outstanding questions to be addressed by the literature is the extent to which psychological treatments are effective in routine clinical work. The above drug-management protocols were, therefore, adopted in this study in order to answer this question (see Chapter 7).

## MEASURES OF CHANGE

There have been a number of problems associated with data collection in previous studies.

Firstly, the Daily Sleep Questionnaire (DSQ) (Monroe, 1967) has been the most commonly employed measure in the psychological literature. Comparisons between EEG and DSQ parameters were made in Chapter 4, from which it would seem acceptable to view self-report as relatively reliable and valid for most sleep parameters. Nevertheless, greater confidence can be placed in those studies which have established DSQ validity within their reported populations. Unfortunately, this has been the exception rather than the rule.

Secondly, an equally important shortcoming has been the general, and sometimes exclusive, reporting of only one outcome variable, namely SOL. It cannot be assumed that general alterations in sleep pattern occur as an inevitable effect of SOL change. Furthermore, it is unclear to what extent quantitative change correlates with changed perception of sleep quality, restedness, and the insomniac's consideration of her/himself as a poor or good sleeper.

Thirdly, it is also helpful to be able to predict the point at which a given therapeutic procedure is likely to exhibit significant change. The evidence so far suggests that stimulus control operates rather more quickly than other interventions, but no systematic attempt has been made to gather the process data which would help to characterise treatments in terms of within therapy change.

Fourthly, adequate follow-up is of considerable importance. For the purposes of most psychological research it appears that follow-up at 3 or 6 months is viewed as reasonable and studies on insomnia seem to have aimed at this criterion. However, clinical insomnia should be regarded as a chronic disorder, the natural course of which dictates that lengthy follow-ups be completed.

One final area of neglect has been the failure to investigate the generalised changes in day to day functioning which result from therapy. Some of the psychological treatments used are commonly regarded as anti-anxiety strategies, and measures of symptomatic anxiety and depression would prove informative. The daytime consequences of improved sleep might also be considered. Measures of concentration level, irritability, daytime coping etc., would be useful.

The present study was designed to make it possible to address these matters.

DESIGN ISSUES

Two important methodological developments have improved substantially the quality of experimental control. The first of these is the imaginative, counterdemand instruction (Steinmark and Borkovec, 1974) which controls for expectancy effects. It has proven useful in the discrimination of active versus placebo treatment effects, where the latter have been demonstrated only under conditions of positive demand. The adequate control of demand characteristics is of fundamental importance given that social acquiescence and other non-specific factors may be responsible for treatment effects. The second creditable development has been the use of Borkovec and Nau's (1972) Credibility Evaluation Questionnaire (CEQ). However, this is most useful as a post-rationale evaluation of expected treatment benefit, rather than the

sometimes reported mid or even post-therapy evaluation, when perceived credibility may be contaminated by actual treatment response. An ideal point at which to administer the CEQ would be during the first week of therapy, after the programme has been put into practice but before benefit can be systematically monitored by the patient.

The use of a single therapist across treatment conditions can limit the external validity of results. A number of studies, however, failed to find any effects of "therapist factor" in the relaxation and stimulus control treatment of insomnia (Carr-Kaffashan and Woolfolk, 1979; Nicassio and Bootzin, 1974; Steinmark and Borkovec, 1974; Tokarz and Lawrence, 1974). The only study to do so has been that of Turner and Ascher (1982) who, not surprisingly, found that the level of improvement achieved by trainee therapists was less than that obtained by experienced therapists. Of greater concern is the potential for differing skills and/or enthusiasms to be applied to the various treatments by a single therapist. In studies where a suitable number of similarly experienced and skilled therapists is not available, external validity checks, perhaps in the form of independent ratings of treatment sessions, would be a useful to ensure that any differences in treatment outcome are not due to systematic bias in the part of the therapist.

Most researchers have randomly allocated subjects across the experimental conditions to provide a strong test of between group difference. There have not been any controlled studies which have addressed the important clinical issue of selecting treatments to suit individual needs. Such a "tailored" approach could be usefully compared with randomly assigned psychological treatment.

The present study seeks to address these design issues.

ANALYSIS OF TREATMENT OUTCOME DATA

The most useful outcome data are those which are both statistically and clinically meaningful. Analyses may be statistically robust and significant without necessarily being of great clinical importance. The traditional comparison of group mean values across experimental conditions may be particularly suspect since no information is provided on the variability of treatment outcome amongst subjects in each group. This information is of considerable interest as clinical research should drive clinical practice, but may do so only when research findings are known reliably to predict outcome for the individual case. A number of workers have made recommendations as to how testing for clinical significance might be achieved. For example, Kazdin (1977) has suggested that a return to normal functioning may be a standard against which to determine the significance of clinical change. This approach requires that some normative information is available with which to compare post-treatment scores. The proportion of patients achieving normal scores after treatment can then be deduced. Other workers have recommended a 50% change of symptomatic level (Jansson and Ost, 1982; Lichstein and Fischer, 1985) as a useful criterion. Jacobsen, Follette and Revenstorf (1984) have also discussed these issues and have stressed the need for an agreed standard convention which is "applicable to a wide variety of clinical problems, ——— objective, relatively free of bias, and sound from both a psychometric and clinical perspective". Although no such standard has as yet been determined, some analysis of this type is clearly required.

Analysis of outcome, conducted upon mean values, may be incomplete also where the mean score is taken to be a representative statistic descriptive of "typical" sleep pattern. An average weekly SOL of 60 minutes for a given patient suggests that this individual takes around

one hour to fall asleep each night. Raw score variance, however, may be considerable (on occasions patients fall asleep very quickly; at other times SOL may be very lengthy). Indeed, the inherent unpredictability of what sleep will be like on a given night may have a bearing upon the complaint of insomnia itself (see Chapter 2). A simple measure of range or standard deviation would appear to be useful as an index of night to night variability in sleep. It may be that effective treatment should improve not only the average sleep pattern but also increase the predictability of that pattern on a night to night basis. The employment of both mean and SD scores would make it possible to report treatment effects in terms of changes in the typical pattern of sleep.

The present study addresses both of the analytical issues outlined above. Both mean and SD scores for sleep pattern variables will be analysed in the Results section and the clinical significance of the results obtained will form a major focus of the Discussion to follow.

In summary, this investigation was designed to determine the usefulness of various psychological treatments with patients presenting clinically as insomniac. Drug-taking patients were not excluded, but care was taken adequately to control the effects of drugs. Both the range of outcome measures and the summary statistics entered in analyses were extended to ensure valid hypothesis-testing, and the clinical impact of treatment changes were assessed. In these ways some of the shortcomings of past research were overcome.

# CHAPTER 7

## METHODOLOGY OF THE STUDY

### SUBJECTS

The subjects were a series of consecutively referred adult out-patients described, in the terms of a referral letter, as suffering from "chronic sleep-onset insomnia". All of the patients were GP referred, and the GP practices in north Lanarkshire and the west end of Glasgow involved in the study were familiar with making direct referral to clinical psychologists (Espie and White, 1986a,b; Storrar, 1984). All GPs had received correspondence explaining the selection criteria and were encouraged to refer suitable cases.

During the period of data collection 141 consecutive referrals were received of whom 130 attended initial interview. A further 29 were excluded by the author as failing to meet the selection criteria outlined below. This excluded group comprised 11 patients considered to have sub-clinical sleep problems; 14 who demonstrated significant psychopathology (9 depressed, 3 alcohol/drug abusers, 1 impotent, and 1 marital problem); and 4 who had clinically diagnosable other sleep disorders (2 restless legs syndrome, 1 nocturnal myoclonus, 1 sleep apnea). There remained, therefore, 101 available and suitable cases of whom 17 were regarded as drop-outs from the experimental design. Eight of these furnished data for four weeks or less of the treatment period, 6 failed to achieve adequate control over medication and were excluded, and the remaining 3 repeatedly failed satisfactorily to complete the Daily Sleep Questionnaire. Drop-outs were evenly distributed across the treatment conditions and did not represent any form of systematic bias.

This study describes the final sample of 84 patients (57 females, 27 males) who completed the study. Thirty-one subjects were from

Lanarkshire and 53 were from Glasgow. The mean age of the sample was 45.5 years (range 17-82, SD 15.9), and insomnia had presented clinically as a problem for an average of 11.8 years (SD 11.6). Further confirmation of the chronicity of insomnia can be found in that mean time since first prescription of sleep medication was 9.0 years (SD 10.6). Independent sample T-TESTs revealed that there were no significant differences on any demographic variable between males and females, or between the two geographical cohorts. Similarly, ONEWAY ANOVAs revealed no differences across treatment groups in terms of sex, age, duration of insomnia, or time since first prescription (see Table 5 for demographic characteristics).

Selection Criteria

1. Sleep-onset latency greater than 30 minutes on average per night. That is a total latency of at least 3.5 hours over the week.

2. Chronic initial insomnia present for minimum of one year.

3. Previous advice-seeking as evidence of clinical relevance of insomnia. In practice this constituted written physician referral.

4. Legitimate to treat insomnia in isolation as the main presenting or primary problem.

5. Able to ensure the withdrawal of all drugs which might interfere with the experimental design, or to maintain the patient on the same doseage of the same drugs throughout the period of study.

6. No other ongoing therapy for insomnia, anxiety or depresson.

7. Exclusion of patients presenting as clinically depressed at initial interview or with scores of 60 or higher on the Zung Depression Scale (Zung, 1965). Exclusion also if anti-depressant medication had been prescribed at any time during the 6 months preceeding referral.

8. Exclusion of patients considered to have drink problems.

9. Exclusion of insomnia problems possibly related to medical conditions, and of sleep disorders not conforming to categories 1,2 or 9 of the Diagnostic Classification of Sleep and Arousal Disorders (Sleep, 1979, 2(1) - see Chapter 2).

10. Exclusion of patients non-compliant with either treatment instruction or adequate record-keeping.

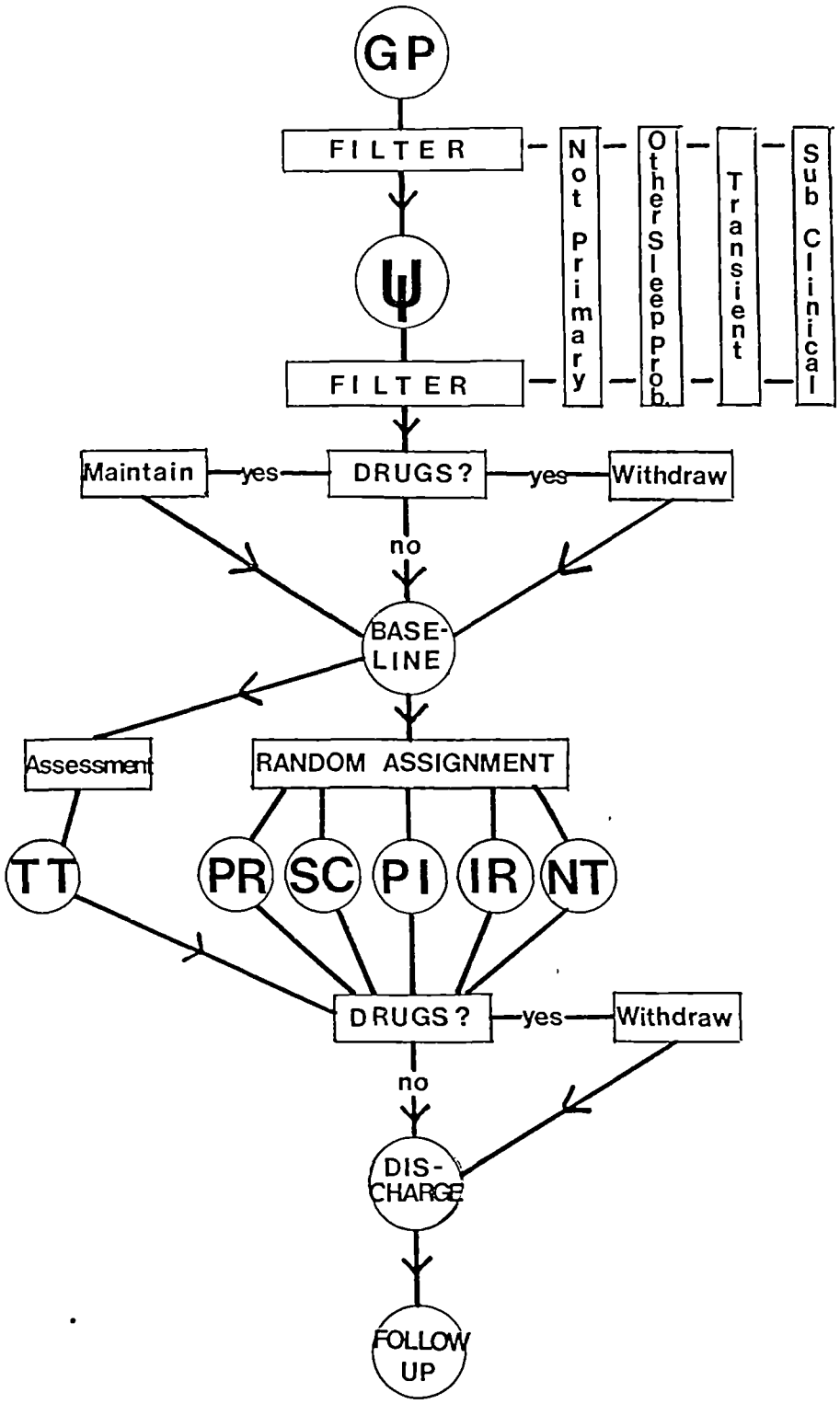



FIGURE 1: Design of the treatment outcome study.

PR Progressive Relaxation
SC Stimulus Control
PI Paradoxical Intention
IR Imagery Relief (placebo)
NT No Treatment
TT Tailored Therapy

DESIGN AND PROCEDURE

The overall design of the study from referral to follow-up is outlined in Fig 1. Following screening and final selection a decision was made concerning patients who were habituated to hypnotic medication, either to withdraw that medication pre-treatment or to maintain its use during psychological therapy and withdraw thereafter. This decision was largely a pragmatic one, withdrawing where possible at the outset, but if patients were unable or unwilling to persevere with the standardised 5-week withdrawal regime they were assigned to the maintenance design. The use of these alternative experimental models, therefore, afforded flexibility which in turn permitted the inclusion of "difficult" cases. The rationale for these procedures was detailed in Chapter 6. Summary flow-charts of the two protocols are, however, presented in Fig 2. The reader should note that the experimental phases are identical in the two models apart from the timing of the withdrawal regime.

| Baseline | Withdrawal | Baseline | Treatment | Follow-up |
|---|---|---|---|---|
| 1/52 | 5/52 | 2/52 | 4/52 | 6/52 |
| | | | + | 3/12 |
| | | | 4/52 | 6/12 |
| SELECTION | | | | 17/12 |

| Baseline | Treatment | Withdrawal | Follow-up |
|---|---|---|---|
| 2/52 | 4/52 | 5/52 | 6/52 |
| | + | | 3/12 |
| | 4/52 | | 6/12 |
| | | | 17/12 |

Fig 2 Outline of the two experimental models for pre and post-treatment drug withdrawal

Of the 84 patients, 58 (69%) were stated, by their GPs, to be "drug-free". Only 14 of these patients, however, had never been on hypnotic medication, and it transpired upon further assessment, that approximately one third of the remainder had not entirely discontinued medication. In most cases drug use was occasional and low dose. The author supervised the final cessation of hypnotics for these patients

and the full pre-treatment withdrawal protocol was not required. Twenty-six patients were referred with persisting sleep difficulties who were also on nightly sleep medication. Eleven of these (13% of total reference group) were allocated to the pre-treatment withdrawal protocol, and 15 (18% of total) were maintained at habituated levels while psychological therapy proceeded. The GP of each patient was contacted by letter in order to secure cooperation with the demands of the programme being followed.

Prior to psychological treatment, baseline assessments were completed on the DSQ, in order to provide information on untreated sleep quantity, pattern and quality. The DSQ functioned as a process measure and was completed for every night's sleep throughout the experimental period. The other rating scale measures were included to assess the generalised effects of sleep loss and other symptomatology and were used in a pre-post format. The original experimental design allowed for three psychological treatment groups and two experimental controls (see Fig 1). Subjects were allocated, according to a predetermined random list, to either progressive relaxation (PR), stimulus control (SC), paradoxical intention (PI), imagery relief placebo (IR) or no treatment (NT). After completion of the main study, however, it was felt desirable also to include a tailored therapy group (TT) where subjects received treatment tailored to suit their individual needs. A Sleep Disturbance Questionnaire was devised for use with this group to provide a more structured method of evaluating the therapeutic ingredients appropriate for each individual. Allocation to the tailored group was by consecutive referral since the randomisation design had already been completed. Since the tailored therapy condition was not part of the original study design it will be dealt with as a separate chapter in the Results section. Descriptive information on the treatment groups is available in Table 5.

| | n | M/F | Age | Duration | First prescription* |
|---|---|---|---|---|---|
| Relaxation | 14 | 7/7 | 48.1 (10.6) | 16.2 (11.6) | 10.4 (11.5) |
| Stimulus control | 14 | 4/10 | 44.9 (14.4) | 7.8 (13.1) | 8.4 (10.1) |
| Paradoxical intention | 15 | 3/12 | 43.7 (15.8) | 13.1 (10.6) | 8.6 (8.6) |
| Imagery relief placebo | 14 | 4/10 | 42.4 (15.3) | 11.6 (12.9) | 9.1 (11.9) |
| No treatment | 13 | 5/8 | 45.3 (20.9) | 13.5 (16.1) | 13.0 (15.9) |
| Tailored therapy | 14 | 4/10 | 48.6 (18.8) | 8.9 (7.0) | 5.2 (4.5) |

* Of the 84 patients only 14 (17%) had never been on sleep medication

TABLE 5 Demographic characteristics of the six experimental groups. (Figures in brackets are standard deviation scores of the mean values)

Treatment Conditions

All active treatments and the placebo intervention comprised a series of six individual out-patient appointments (of approximately 40 minutes duration) during the eight-week therapy period, comprising 4 weeks under counterdemand, and 4 weeks under positive demand.

Counterdemand Instruction

All patients were given counterdemand instruction (Steinmark and Borkovec, 1974) during the first four weeks of therapy. They were told that treatment would take some time to have an effect upon sleep pattern and that patience and perseverance were essential. They were informed that experience had shown that fully four weeks of treatment would pass without benefit, but that thereafter dramatic improvement would take place. Positive demand instructions were issued from the fifth week onwards encouraging expectation of change. In order to reinforce these demand manipulations an "Important Note" was included in all treatment hand-outs, restating the instructions. The counterdemand period was extended to four weeks, compared with previous research, in order to allow for the possibility that with severe

insomnia therapeutic effects might take longer to achieve.

The rationale and treatment procedures employed were in close agreement with those applied in previous studies. A brief resume of the therapeutic instructions for each condition will be provided.

Progressive Relaxation

Subjects were instructed individually in the application of an abbreviated progressive relaxation procedure, similar to that of Bernstein and Borkovec (1973). The exercises comprised tension-release cycles covering major muscle groups in the arms and hands, neck and shoulders, face and mouth, back and abdomen, and legs and feet. Regulation of breathing pattern was also an integral part of the training procedure, and the cue word "relax" was trained latterly as a discriminative stimulus according to the cue-control technique (see Marchetti, McGlynn and Patterson, 1977).

Two in vivo training sessions were administered. Subjects were instructed while lying down. A relaxation training cassette was recorded during one of these sessions and was subsequently used for home practice. Patients were required to practise the exercises daily and to apply them in bed with the light out. The set of exercises lasted approximately 20 minutes, but patients were told to continue to apply the technique until they fell asleep. After the in vivo sessions, clinic appointments were used to monitor progress, to tailor the exercises to suit individual needs, and to foster the development of cue control.

Stimulus Control

Standard directions were given to each subject, although care was taken to identify for each individual those behaviours thought to be sleep-incompatible, and those conducive to sleeping well. The Sleep Behaviour

Rating Scale (see section on Measures to follow) was helpful in this respect. The therapeutic instructions were as follows (see also Bootzin, 1972):-

a) Lie down intending to go to sleep only when you feel sleepy

b) Do not read, watch TV, listen to the radio, etc. in the bedroom

c) If you do not fall asleep within 20 minutes, get up and go into another room, returning to bed only when you feel sleepy

d) If you cannot fall asleep repeat step c, and do this as often as is necessary throughout the night

e) Set the alarm and rise at the same time each morning

f) Do not nap during the daytime

Paradoxical Intention

The paradoxical intention programme was explained using illustrative examples to help patients understanding of the relationship between performance anxiety and sleep-onset. They were told that their paradoxical intention was to remain awake for as long as possible rather than continuing the effort to fall asleep. They were told, however, that the environment should be conducive to sleep ie. they were to lie in bed with the lights out, but to keep awake by having their eyes open. They were not to actively prevent sleep through moving about or through other overt activities. Patients were instructed to remind themselves that it was their intention to remain awake whenever they felt their eyelids closing. Clinic sessions were used to rehearse these procedures and the rationale given, and to deal with any practical problems arising from implementation.

Imagery Relief Placebo

Patients in this group were treated in accordance with the quasi-desensitisation placebo instructions commonly used in past research (Steinmark and Borkovec, 1974). The term "imagery relief" was, however, coined by the author. During the first training session a hierarchy of

chronologically ordered evening and bedtime activities was constructed, in such a way as to be typical of the patient's usual routine. The patient was then informed that these items were an integral part of the sleep problem itself and that sleep onset would be facilitated by the desensitisation process and the relief of tensions presumed to be associated with the nightly routine. A list of mental images was then elicited from the subject, to be paired off with the hierarchy items in a deconditioning paradigm. The therapist ensured that the images selected were pleasant, but on the whole neutral in connotation for the subject. (A typical example of the hierarchy and list of images used is provided in Appendix A). It was stressed to the subject that time should be spent on daily practice of the training programme, but that under no circumstances should a session occur later than 8.00 pm. Subjects were trained to concentrate upon the first item of the chronological list, and once visualisation was achieved, to switch attention to one of the mental images. This procedure was then repeated with the next item, and so on until 20 minutes practice had elapsed. The programme was, therefore, analagous to relaxation therapy/desensitisation but with the important omission of any known active ingredient, either theoretically or practically. Patients received no instruction in dealing with sleeplessness per se.

No Treatment

This group functioned as a waiting list control and had minimal therapist contact. Patients were seen after referral for the purposes of training in the use of the DSQ. This typically involved two appointments. Occasional contact by telephone was also made to ensure that sleep diaries were being completed as required, but at no time was advice or treatment offered. Subjects were seen again at the end of the ten week data collection period having thus provided data for the entire duration of the experimental period. These subjects were told

that, at the end of the 10 weeks, they would be put on to a therapy programme, and that gathering data in the meantime would greatly assist in the assessment of their sleep difficulties. Patients were not re-allocated within the randomisation design since it was felt unethical to, by chance, allocate some of them to placebo therapy. In addition, it was felt unreasonable to require these patients to complete a further ten weeks of data collection to conform to the study design. Waiting-list subjects were, therefore, treated on an ad hoc individualised basis.

In addition to the verbal instructions given during treatment sessions, each subject was also given a hand-out which summarised the main points of rationale and procedure for his/her therapy programme. These were simply take home summaries of the programme as outlined in the consulting room. The hand-outs were subjected to analysis of readability, according to the Flesch (1948) formula, and were standardised for "reading ease" and "human interest" at a level approximating to that of the "popular" press. A copy of each hand-out is contained in Appendix B.

At the final therapy session, post-treatment assessments were completed, and follow-up procedures explained. Follow-up data were collected on the DSQ for 7 consecutive nights, at 6 weeks, 3 months and 6 months, and 17 months.

Investigation of Therapist Factor

The author conducted all therapy sessions across treatment conditions. In order to confirm that there was no systematic bias resulting from the application of differential therapeutic skill across treatments, external ratings of tape-recorded therapy sessions were provided by an experienced therapist who was not involved in the study.

Audio cassettes were made of treatment sessions on a random sample of 9 patients. This group comprised 2 from relaxation training (Cases 5 and 6 in Fig. 3), 3 from stimulus control (Cases 2, 4 and 8), 1 from paradoxical intention (Case 9), 2 from tailored therapy (Cases 3 and 7), and 1 from placebo therapy (Case 1). A rating instrument was derived by selecting and adapting items from the Cognitive Therapy Rating Scale (Young and Beck, 1980). The Young and Beck scale was designed specifically for the purpose of independently evaluating therapist skills. Four items were included as ratings for the present study. These were "interpersonal effectiveness", "collaboration", "strategy for change" and "application of techniques". Each was rated on a 7 point scale from 0 through 6. Detailed descriptive criteria were provided for scoring each item (as in Young and Beck - see Appendix C). The rater randomly selected the order in which to listen to the tapes and made his ratings, separately for each patient, immediately after hearing the relevant material.

The results of this investigation are presented graphically in Fig. 3. The data points plotted in each section of the graph represent the ratings made of that particular therapist skill in relation to each of the 9 cases. Because of the small numbers in each experimental condition it was not possible to analyse the data statistically. Visual inspection, however, clearly indicates that there was marked consistency of skill across patients and across treatments. For "interpersonal effectiveness" the median score of 4 applied to 6 of the 9 subjects. On the assumption that a score +/- 1 scale point represents reasonable consistency only Case 4 deviates from the "norm". Similarly for "collaboration", ratings were consistently within the same range with this single exception. Case 4 was treated by stimulus control but consistently failed to comply with certain aspects of the programme. The audio-tapes, therefore, reflected a degree of antipathy.

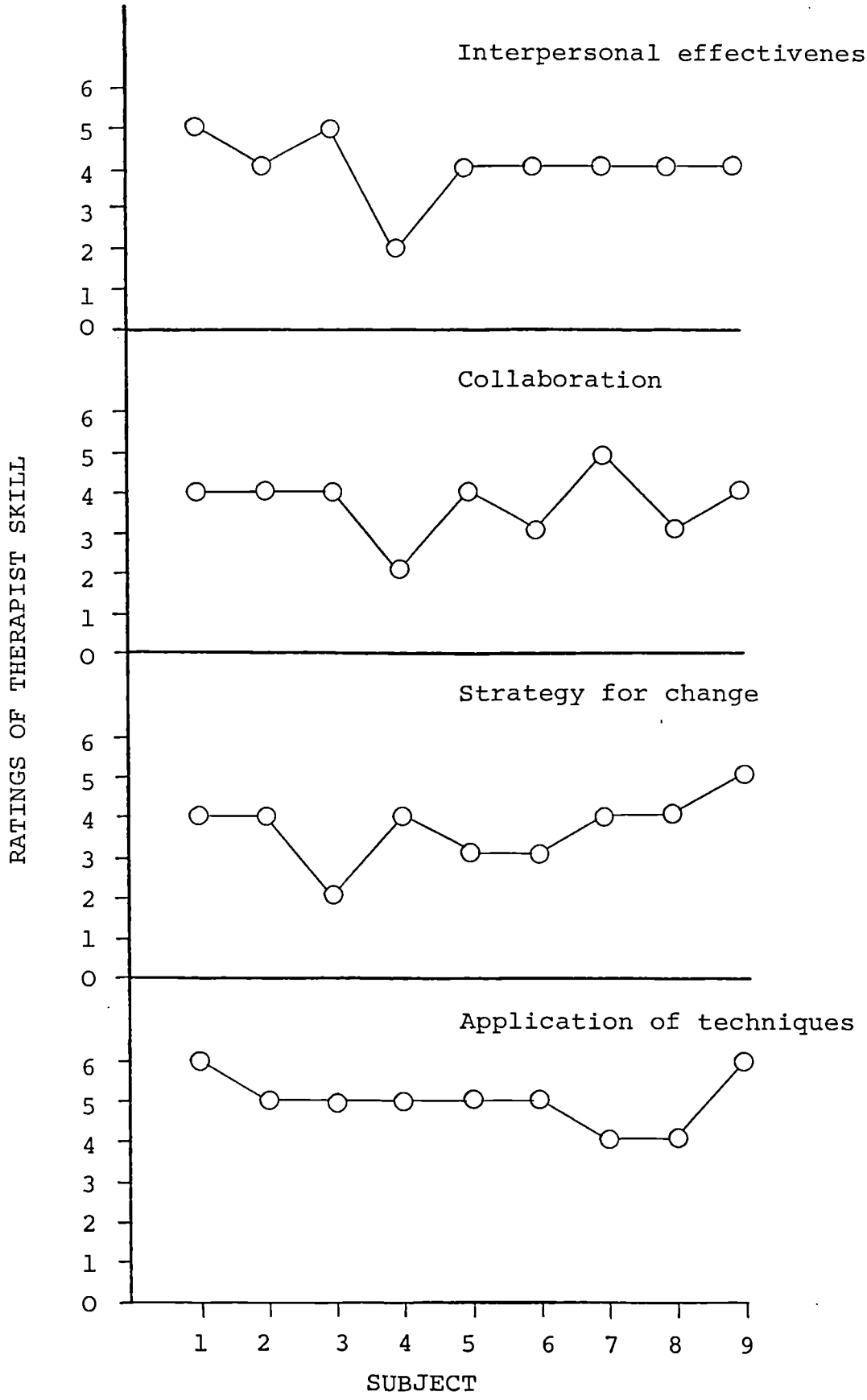



FIGURE 3: Independent ratings of therapist skills across subjects on four domains of skill derived from the Cognitive Therapy Rating Scale (Young and Beck, 1980).

For "strategy for change", ratings of therapist skill fell within 1 point of the median score for 8 of the 9 patients. It seems that again skill was being equally applied. A score of 2 was given in relation to Case 3 (tailored therapy). This patient had completed drug withdrawal successfully. Unfortunately, tape recordings represented only the first session of treatment when various therapeutic options were discussed.

Ratings of "application of techniques" were distributed evenly around a median score of 5. The treatment intervention for five patients was rated at the median, two other cases were rated as 6, and the remaining two as 4. There was, therefore, no significant deviation on this rating according to the above criterion.

Since only 3 out of the 36 ratings made could be considered as evidence of inconsistency, it can be concluded that the range of therapist skills investigated were equally evident in each case studied. The use of a single therapist across treatments is unlikely, therefore, to account for any systematic difference between groups.

## MEASURES

The outcome measures can be sub-divided into two groups. Firstly, measures of therapeutic process were taken in order to evaluate changes in the main dependent variables over time; and secondly, "before and after" testing was conducted to evaluate more general changes in psychological status associated with the interventions. Assessment procedures will be described under these headings.

### Process Measures

Self-report measures of nightly sleep were recorded throughout the experimental period on a Daily Sleep Questionnaire (DSQ) based upon that of Monroe (1967) and subsequent workers (eg. Turner and Ascher,

1979a). The DSQ comprised nine items completed each morning upon rising, and a further two items, referring to subsequent daytime functioning, completed upon retiring to bed (see Appendix D). Subjects provided estimates of the three main parameters of sleep latency (mins), total sleep time (hrs/mins) and wakening frequency, and also recorded the time at which they had gone to bed, their terminal waking time, and their time of rising. In addition to these quantitative variables, the DSQ also incorporated rating scale measures of intrusive anxious thoughts at bedtime, overall sleep enjoyment and restedness upon rising. These ratings were made on 5-point Likert scales where 0 represented "not at all" and 4 "very much". Subjects generally found the ratings simple and straightforward to use. Occasional difficulties, however, did arise with the semantics of the scales. In particular, some patients asked what was meant by "enjoyment" of sleep, and to a lesser extent, "restedness" in the morning. In order to control for the possibility that different interpretations might be made, the author routinely explained the intended meanings of the items and relabelled them, if necessary, with appropriate synonyms. Therefore, and for example, an "enjoyable" sleep was considered to be a "good" sleep, and a sleep with which one was "pleased" or "satisfied". A similar 5-point scale was employed as a measure of daytime irritability which is a frequently reported correlate of impoverished sleep. Subjects also kept a record of time spent napping each day. In addition, patients who were continuing to make use of sleep medication were required to enter each dose taken on the DSQ so that control of drug intake could be maintained.

In recent years there has been considerable debate regarding the merits and demerits of sleep diaries, particularly in relation to objective descriptors of sleep. Chapter 4 considered these issues in detail. At present, the arguments for and against both subjective and objective

assessment continue thereby necessitating; firstly, careful control over all assessment procedures; and secondly, the appraisal of validity within each subject population. In this investigation care was taken to train patients in the use of the DSQ forms, and a validity study was conducted to evaluate the accuracy of self-reporting.

DSQ Training Procedures

All patients were given detailed instructions on DSQ recording at the initial interview. In addition, however, each subject was required to complete the DSQ for one week in order to become familiar with the procedures involved in self-monitoring. A second appointment was arranged to discuss any problems which had arisen and to examine the DSQ. Where inconsistencies in records were evident, these were pointed out and further instruction given to rectify errors. For example, if estimates of one sleep measure did not "tally" with other measures of sleep pattern, further training was provided to improve the quality of record-keeping. Subjects were required to estimate sleep parameters to within 10 minutes, in order to avoid the possibility that "rough guesses" would be entered to the nearest hour or half hour. It was also stressed that the record for any given night should be left blank if it had not been completed within one hour of rising. It was felt that retrospective guesses would be highly unreliable. This procedure proved to be permissive, in that it was not regarded as a "failure" to have blank spaces on the response sheet. Subjects were told to keep the DSQ and a pencil at the bedside for completion each morning, and were advised that late entries would confuse the picture and would be liable to lead to ineffective management.

For 34 patients (40%), data from this first week were regarded as unsatisfactory and a further two weeks baseline assessment was completed until satisfactory compliance, according to these criteria,

was established. At least two weeks of pre-treatment recording was, therefore, obtained for each subject in the study. Unreliable data collected during the training period were discarded. Throughout the remainder of the experimental period patients continued to monitor their sleep pattern on the DSQ, and were frequently reminded to observe the above guidelines.

Validity Assessment

It was decided to employ the "Somtrak" Sleep Assessment Device (SAD) (Kelley and Lichstein, 1980) as an objective measure of sleep pattern against which to evaluate self-report. Previous studies have found the SAD to be a useful measure of both insomniac and non-insomniac sleep when compared with EEG assessment (see Chapter 4).

The SAD generates a brief, soft tone at pre-set intervals throughout the night, and simultaneously activates a cassette recorder. The subject is instructed to respond to the cue tone by stating "I am awake", thus providing time-sampled data readily convertible to measures of sleep latency, total sleep time, wakening frequency and duration, and sleep efficiency. In this study the inter-tone interval was set at 12 minutes (ie. 5 per hour), the tone duration at 1 second, and tape recording was for 10 seconds at each assessment point. The volume and pitch of the tone were set individually for each subject, within acoustic range yet without being intrusive. Subjects were instructed to reduce the volume slightly if awakened from sleep by the tone.

Twenty patients were random sampled (24% of the total group) and asked to complete a number of nights of home assessment using the SAD. The number of nights recorded per patient ranged from 2 to 10 with a median of 6. In this way 110 subject-nights were available for comparison with DSQ data. SAD recordings were also made randomly across the three

phases of baseline, counterdemand and positive demand. Decoding of the SAD cassette tapes was performed using prepared scoring sheets (Appendix E), which required the rater to enter a tick in response to each tone depending upon the presence or absence of the criterion response of "I am awake". A simple categorical decision could thus be readily made. Nevertheless, an inter-rater reliability check was conducted on a 20% sample of the subject nights which yielded a 99.2% agreement rate on the presence/absence of criterion responses.

The 20 patient sample comprised 8 men and 12 women, with a mean age of 43.2 years (SD 13.9) and a mean duration of insomnia of 11.4 years (SD 10.7). Comparison with descriptive data for the total patient group (Table 5) indicates that this was a representative sample. Table 6 presents the breakdown of DSQ and SAD scores across experimental phases along with paired T-TESTs which were conducted to evaluate the significance of differences between the objective and subjective measures. PEARSON correlations were also computed for each pair of measures. Inspection of the Table reveals that DSQ estimates of sleep-onset latency (SOL) were significantly higher than SAD records of the same nights' sleep during baseline and counterdemand phases. In both cases the average overestimation was approximately 18 minutes. Under positive demand, however, the means were not significantly different, perhaps reflecting the much simpler task which patients had in estimating reduced sleep latencies within the range 13-52 minutes (ie. +/- 1 SD). PEARSON coefficients revealed very strong positive associations between the two measures, consistent across the experimental period. In each case significance was at the .001 level or greater. These findings are in keeping with the view that insomniacs overestimate SOL but tend to do so consistently. The pattern of improvement evident in SAD scores was also paralleled by self-report, thus confirming DSQ estimates as a reliable relative index of objective

| | | Subject nights | Mean | SD | T | df | prob | Pearson r | prob |
|---|---|---|---|---|---|---|---|---|---|
| SOL | | | | | | | | | |
| Baseline | DSQ | 40 | 111.6 | 75.4 | 2.95 | 39 | .005** | .872 | .0001*** |
| | SAD | | 94.1 | 74.5 | | | | | |
| Counter-demand | DSQ | 31 | 63.2 | 53.3 | 2.27 | 30 | .031* | .563 | .001*** |
| | SAD | | 45.3 | 32.4 | | | | | |
| Positive Demand | DSQ | 38 | 30.6 | 16.9 | -1.71 | 37 | .096 | .913 | .0001*** |
| | SAD | | 32.8 | 19.7 | | | | | |
| TOT | | | | | | | | | |
| Baseline | DSQ | 41 | 4.95 | 1.64 | 1.26 | 40 | .215 | .833 | .0001*** |
| | SAD | | 4.76 | 1.70 | | | | | |
| Counter-demand | DSQ | 31 | 5.78 | 1.65 | 0.60 | 30 | .553 | .793 | .0001*** |
| | SAD | | 5.66 | 1.64 | | | | | |
| Positive Demand | DSQ | 38 | 6.57 | 1.19 | 2.47 | 37 | .018* | .926 | .0001*** |
| | SAD | | 6.39 | 1.16 | | | | | |
| WAKE | | | | | | | | | |
| Baseline | DSQ | 40 | 0.85 | 1.17 | -4.32 | 39 | .0001*** | .620 | .0001*** |
| | SAD | | 1.93 | 2.01 | | | | | |
| Counter-demand | DSQ | 30 | 0.83 | 1.34 | -5.25 | 29 | .0001*** | .429 | .018* |
| | SAD | | 2.67 | 2.06 | | | | | |
| Positive Demand | DSQ | 38 | 0.13 | 0.34 | -3.02 | 37 | .005** | .053 | .752 |
| | SAD | | 0.82 | 1.37 | | | | | |

TABLE 6 Comparison of DSQ and "Somtrak" SAD scores for sleep latency (SOL), total sleep time (TOT) and frequency of wakening (WAKE) on paired T-TEST and PEARSON correlation measures
* $p \leq .05$ ** $p \leq .01$ *** $p \leq .001$

sleep latency.

Interestingly, previous reports stating that insomniacs underestimate the duration of sleep (TOT) were not confirmed. Across the experimental period there was, in fact, a slight tendency towards overestimation of TOT. This, however, reached modest significance only during positive demand. In general, therefore, these patients were accurate in their DSQ estimates of total sleep time. Correlational data substantiate this, with coefficients ranging from 0.79 to 0.93, all highly significant at the .0001 level of probability.

Comparisons between DSQ and SAD for wakening frequency (WAKE) yielded highly significant between measure differences during each experimental phase. Patients markedly underestimated their number of wakenings. In addition, correlational assessment revealed an inconsistent relationship between self-report and SAD. DSQ was particularly unpredictive of SAD records in the final stages of therapy when wakenings were less frequent. It is clear, therefore, that concordance was poor for this variable. Two ready explanations are available to account for this result. Firstly, patients were asked to record only those wakenings which were associated with "difficulty in returning to sleep". In practice, subjects generally considered arousals of 15 minutes or greater to be disruptive of sleep. It is not surprising, therefore, that DSQ records should underestimate actual arousal frequency. Secondly, the time-sampling method employed by the SAD necessitates the forfeiting of data, particularly for the WAKE variable, and thus no true objective "standard" was available for useful comparison. It is worth noting, however, that SAD records are likely also to be underestimates. It is possible that subjects' imperception (apart from non-reporting) of relatively brief intra-night arousals contributed to the tendency towards report of excess TOT

mentioned above. The evident limitations of the WAKE variable will be reconsidered at a later point.

In summary, both SOL and TOT were validly estimated by insomniac patients after training in use of the diary form. Direct interpolation from the DSQ appears justified for TOT, whereas for SOL (especially lengthy SOL) the relativity of the self-report data should be borne in mind. It was not the purpose of this investigation to consider treatment benefit, although the data are encouraging in this respect.

Before leaving the question of validity it is important to consider how typical the sampled nights were of subjects' sleep, both immediately prior to and subsequent to SAD recording. The possibility that subjects might estimate sleep parameters more carefully and more conservatively while being monitored is a potential limiting factor to the above conclusions. The DSQ data on SOL and TOT, provided by the six patients upon whom inter-rater reliability checks had been conducted, were selected for this analysis. Individual mean scores for these variables were computed separately for the 7 nights preceding SAD assessment and the 7 nights subsequent to assessment, and these values were compared with mean DSQ scores reported for the period of objective monitoring. Twelve pairs of DSQ scores were, therefore, available for each variable. The WILCOXON matched-pairs signed-ranks test was applied to evaluate the significance of observed differences. For both SOL and TOT, the critical value of T at $p < .05$ ($n=12$) is 14. The observed T scores of 23.5 and 44.5 respectively, being greater than 14, failed to reach significance, indicating that mean scores over time could be regarded as belonging to the same population. In conclusion then, the sampled nights were representative of "usual" sleep at that time, and appear not to have been influenced by the monitoring procedure per se.

Before and After Measures

Four measures were administered at pre and post-treatment. These were included to consider any differential impact of the therapies upon symptomatic anxiety, depression, day to day functioning and behaviour patterns at or near bedtime. The measures were, respectively, the Zung Self-Rating Anxiety Scale (ZAS) (Zung, 1971), the Zung Self-Rating Depression Scale (ZDS) (Zung, 1965), a 10 item Analogue Rating Scale (ARS) developed for the study, and the Sleep Behaviour Self-Rating Scale (SBRS) (Kazarian, Howe and Csapo, 1979). Each of the scales is reproduced in Appendix F.

Zung Self-Rating Anxiety Scale

The ZAS is commonly used in research and clinical practice to provide a measure of anxiety state. It is a 20 item, 4 point rating scale covering largely somatic and affective symptomatology. The items were originally selected from verbatim records of patient interviews and are, therefore, representative of frequently reported symptoms. Zung (1971) reported a substantial and statistically significant correlation between ZAS scores and a clinician rated instrument ($r = .74$, $p < .01$), and split-half correlations were also found to be significant at the same probability level. The validity and reliability of the ZAS appear, therefore, to be reasonably attested.

During pilot work for the present study the Taylor Manifest Anxiety Scale (TMAS) (Taylor, 1953) was also considered but the Zung scale was finally preferred. There were several reasons for this. Firstly, for reasons of economy and patient compliance, it was felt that the 20 item ZAS would be more readily administered compared with the 50 item TMAS without loss of sensitivity. Secondly, the TMAS is scored only on a true/false basis rather than the more discriminating four-point Likert scale in the ZAS. Thirdly, it was noted that errors in scoring of the

TMAS were more frequent, especially due to the employment of "double negative" phrasing.

Zung Self-Rating Depression Scale

Similar to the ZAS, the ZDS is an established scale, comprising 20 items rated on a four point scale. The identical scaling properties of the two Zung scales were found to ease administration, hence one practical reason for selection of the ZDS. The ZDS represents an early attempt to provide brief quantification of depressive state, and in his own work Zung found that his scale was a useful index of clinically diagnosed depression. He also reported that it was a measure sensitive to mood change and, therefore, useful as a treatment outcome device (Zung, 1965). More recently, the reliability and validity of self-report measures of depression (the ZDS included along with six other scales) have been criticised by Boyle (1985), who questioned their psychometric adequacy and recommended the development of multivariate measures of depression. Unfortunately, no such scale has as yet been published. Another recent report, however, has supported the ZDS in preference to the Beck Depression Scale (Beck, Ward, Mendelson, Mock and Erbaugh, 1961), and the MMPI Depression Scale (Hathaway and McKinlay, 1942). Schaefer, Brown, Watson, Plemel, DeMotts, Howard, Petrik, Balleweg and Anderson (1985) compared the validities of these three measures in relation to a number of clinical ratings, including psychiatric diagnosis based upon DSM III criteria. The ZDS emerged with the highest validity coefficients (range 0.71 to 0.79) and correlations with DSM criteria were significantly greater than for either of the other scales ($p < .005$, in each case).

Analogue Rating Scale

The ARS was developed during pilot investigations. It was felt desirable to include a measure to evaluate changes in daytime

functioning associated with treatment of insomnia. Since no such measure was available, ten commonly reported daytime correlates of impoverished sleep were selected for inclusion in the scale. Items included assessments of daytime mood, concentration and general coping, and these were rated by patients placing a cross at any point along a 10 cm line, anchored at either end by "not at all" and "extremely". A global score (out of 100) and 10 item sub-scores were, therefore, available for treatment outcome analysis. The author has also used similar analogue scales in other research studies (Espie, 1986; Semple, Gray, Borland, Espie and Beastall, 1987) and has found them to be readily administered and discriminating instruments.

Sleep Behaviour Self-Rating Scale

The SBRS was developed by Kazarian et al (1979) as an assessment of sleep incompatible behavior. It was felt to be a useful instrument for the present study to survey the bedtime behaviour of patients allocated to receive stimulus control, but also to assess the degree of habit change associated with therapy of whatever type. Kazarian et al reported satisfactory internal consistency for their scale (0.75), and test-retest reliability was found to be high at 0.88. They also found that the SBRS reliably differentiated between insomniacs and non-insomniacs on the basis of latency to sleep-onset scores. The SBRS is rated on a 5-point scale of frequency of engagement in each of 20 targetted behaviours. There were, however, a number of shortcomings in the original SBRS, particularly in the questionable appropriateness of some of the items. In this study, therefore, the scale was revised. It was decided to exclude nine items pertaining to daytime activities since these appeared to have little validity for the purposes of evaluating sleep incompatible behaviour. Examples of these are "eating during the day", "reading during the day", "having sex during the day", and "talking on the phone during the day". The item of "having sex at

bedtime" was also omitted. Eight more salient items were then added to the scale to assess such areas as exercise levels, and mental reflection and planning at bedtime. The revised scale is presented in Appendix F, with the additional items asterisked.

Credibility Evaluation Questionnaire

Borkovec and Nau's (1972) CEQ has already been discussed as an important methodological control. It was included in the present study to investigate any perceived differences in the credibility of treatment rationales. Clearly any such non-specific "treatment" effects would be of importance in the appraisal of comparative treatment gains. The CEQ was administered at the end of the first week of intervention to minimise contamination by emerging therapeutic change. The questionnaire is reproduced in Appendix G.

## STATISTICAL ANALYSIS

All statistical analyses were conducted by means of SPSSX programmes using mainframe computing facilities. In the Results section SPSSX commands and (where appropriate) sub-commands will be printed in block capital script in order to identify the procedures selected. The design of the study required analysis of two main classes of data, as previously outlined, ie. process data and pre-post test data. The reasoning behind the selection of statistical analyses was as follows.

### Process Measures

The DSQ generated a considerable quantity of data for each patient ie. 2 baseline weeks, 8 treatment weeks and 4 separate follow-up weeks for each of 11 DSQ variables. These 11 variables comprised 6 which described sleep pattern and 5 which referred to aspects of sleep quality. For each variable, therefore, repeated measures were available across the time course of the experimental period. There being 5

treatment conditions in the main study design, the appropriate statistical analysis was a repeated measures analysis of variance with treatment type as the grouping factor. This form of analysis generated main effects for treatment group, for time, and for treatment x time interaction. The sub-effects of treatment group within time (ie. which between group differences were significant at which time points) and of time within treatment group (ie. at which time points did significant change occur within each treatment) were then examined to explore further those main effects found to attain significance. This design is usually termed "split-plot factorial" (SPF); in this study having 5 levels of the between group factor (treatments) and 9 levels of the within group factor (time points - taking 1 baseline measure and 8 measures during treatment). The reader is referred to Kirk (1968) for detailed information on the design; identified as SPF-2.4 (p 248 ff).

Repeated Measures Analysis using MANOVA

The required repeated measures model was generated by the SPSSX MANOVA programme following the detailed recommendations and computing instructions outlined by O'Brien and Kaiser (1985). The procedures involved for obtaining main effects will be described in the Results chapter. At this juncture, however, the reasoning behind the selection of the MANOVA rather than the ANOVA approach will be summarised. There are four principal points.

Firstly, one effect of performing separate univariate analyses of multiple measures on the same subjects may be to inflate the Type 1 error, ie. reach a conclusion to reject the null hypothesis when it should not be rejected (Bock, 1975; Turner, 1978). This is particularly problematic in therapy outcome research since it amounts to concluding that there are true differences in therapy methods when in fact there are none. One option is to correct conservatively the repeated measures

ANOVA by adjusting alpha levels. Alternatively, MANOVA may be preferred and has been regarded as more likely to produce reliable treatment effects (Hummel and Sligo, 1971).

Secondly, a number of workers have pointed out the statistical constraints upon repeated measures ANOVA. One such constraint is that in regular ANOVA it is assumed that repeated observations are independent. Applications in clinical psychology are, however, unlikely to meet this "sphericity" condition (Jaccard and Ackerman, 1985). Successive or adjacent measurements are often more highly correlated than non-adjacent measures. In addition to equality in correlation between pairs of repeated measures, the ANOVA model assumes that all variances of the repeated measurements are equal. This is another precondition for sphericity. O'Brien and Kaiser (1985) have stated, however, that "in general, sphericity is unnatural for most repeated measures data, and we believe that it is commonly violated in most designs with more than 2 repeated measurements". They quote work by Box (1954) which established that nonsphericity artificially inflates F values for omnibus tests of main effects, again resulting in an inflated Type 1 error rate. Systematic reduction of of the degrees of freedom for the sampling distribution of the F statistic may be one solution for the ANOVA approach [eg. Huynh and Feldt's (1976) best estimator modification is now included in BMDP2v]. In the MANOVA approach, however, there are no difficulties regarding proper error terms or modifications to the degrees of freedom.

Thirdly, R.M. Turner (Temple University Medical School) has been influential in the evaluation of psychological treatments for insomnia (see Chapter 5) and has argued strongly for the use of multivariate tests in therapy outcome research (Turner, 1978). Indeed, he chose as his working example, of the applicability of MANOVA, sleep pattern data

collected by means of DSQ forms.

Finally, in summarising the available studies comparing modified traditional tests with MANOVA O'Brien and Kaiser (1985) concluded that "because no clear-cut power differences exist, the best strategy is to choose a single method and seek to master it. We believe that method should be the MANOVA approach". In this study the MANOVA method was preferred because the author was familiar with SPSSX procedures and SPSSX uses MANOVA to perform repeated measures analysis. SPSSX does not generate univariate repeated measures models except for a within group design (without between group factor) ie. RELIABILITIES procedure. By default, therefore, SPSSX requires the MANOVA approach, largely for the mathematical reasons outlined earlier.

Spector (1977) and others have addressed the important issue of how to follow up significant multivariate effects in order to understand the data set and identify the source(s) of main effects. Some workers have argued for the use of discriminant function analysis (eg. Borgen and Selling, 1978), while others have advocated further exploration via univariate ANOVAs especially where hypothesis-testing is required (Spector, 1977; O'Brien and Kaiser, 1985). In this study significant multivariate effects were investigated using the univariate approach. The SPSSX MANOVA programme proved very adaptable in this respect (cf. Table 6 in O'Brien and Kaiser).

Before and After Measures

These measures were administered to patients on 2 occasions, ie. at pre and post-treatment. Appropriate tests and procedures for paired data were, therefore, selected to investigate the significance of change over time within each of the treatment groups. In order to investigate between group differences, change scores were computed (gain scores

over pre-test) and subjected to ONEWAY ANOVA. This procedure was selected in preference to ANCOVA with pre-test score as the covariate (Huck and McLean, 1975) since the SPSSX ONEWAY programme permits the use of supplementary range-testing procedures (eg. Scheffe) to identify the loci of significant effect.

# CHAPTER 8

## THE EFFECTS OF TREATMENT UPON SLEEP PATTERN AND SLEEP QUALITY

Three important matters must be dealt with prior to embarking upon treatment outcome analyses. Firstly, the stability of baseline scores over time and their similarity across conditions must be investigated. Secondly, it is necessary to determine whether or not the demand characteristics manipulation per se significantly affected response to treatment. Thirdly, the credibility of the treatments applied must be established. The reader should note that all of the analyses contained in Chapter 8 exclude the tailored therapy condition since it will be evaluated in Chapter 10.

### SLEEP MEASURES AT BASELINE

Each subject completed sleep diaries for 14 pre-treatment nights. Individual mean and standard deviation (SD) scores were computed for each of the two baseline weeks for the variables sleep-onset latency (SOL), total sleep time (TOT) and wakening frequency (WAKE) [See Appendix H, Table (i) for breakdown across treatments]. Mean weekly scores were also computed for the remaining five items from the sleep diary. These latter measures are best regarded as "sleep quality" variables since they provided information on sleep satisfaction and daytime correlates of sleep. They were as follows - repetitive thoughts at bedtime (REPTH), feeling of restedness after sleep (RESTED), sleep enjoyment (ENJOY), daytime irritability (IRRIT) and daytime napping (NAPDAY) [See Appendix H, Table (ii)]. Analyses of baseline data were conducted to test the following hypotheses.

1) Neither baseline sleep pattern nor ratings of sleep quality would differ significantly across conditions due to random allocation.

2) Sleep diary reports of these variables would reveal stability over time and no significant differences across conditions over time during baseline.

In order to investigate these hypotheses a repeated measures analysis was required for each of the 11 variables. The SPSSX MANOVA programme was applied to generate the repeated measures model utilising the procedures outlined by O'Brien and Kaiser (1985). These authors recommend the computation of change scores as time contrast variables for the within subjects part of the effect of interest, and the use of these as dependent variables in a regular MANOVA with the grouping factor acting as the between subjects test. This approach provides multivariate tests of significance with associated F ratios and probabilities for the time main effect and the treatment x time interaction. The available tests are those of Pillai, Hotelling and Wilks which have similar roots and distributional assumptions and when converted to F ratios generally reveal similar result (see Srivastava and Khatri, 1979). For the purposes of this thesis the Pillai's statistic only will be quoted as standard. Finally O'Brien and Kaiser recommend that the treatment group main effect is obtained by conducting ONEWAY ANOVA upon the average score of all data points.

Inspection of the results of these analyses in Table 7 indicates that neither the treatment nor the treatment x time main effects reached significance for any variable. It can be concluded, therefore, that strong similarities existed between the treatment groups across the baseline period. The main effects for time reached significance at the .05 level of probability for three of the analyses, namely TOT, REPTH and RESTED. Thus for these variables there was a tendency for some change over time which was independent of treatment group allocation. Inspection of the data in Tables (i) and (ii) in Appendix H reveals a slight general shift towards higher TOT and RESTED and lower REPTH at baseline week two. This may best be regarded as a minor adjustment to reporting practice, although it will be of interest to see if any trend continues during the first weeks of intervention. With this proviso the

| Source of Variation | MS Between | MS Within | F Ratio | F Prob | Sig Level |
|---|---|---|---|---|---|
| SOL mean | | | | | |
| Treatment | 650.57 | 2454.37 | 0.265 | 0.899 | NS |
| Time | 125.92 | 1807.65 | 0.069 | 0.793 | NS |
| Treatment x Time | 577.32 | 1807.65 | 0.319 | 0.864 | NS |
| SOL SD | | | | | |
| Treatment | 293.47 | 817.47 | 0.359 | 0.837 | NS |
| Time | 897.91 | 900.01 | 0.998 | 0.322 | NS |
| Treatment x Time | 681.21 | 900.01 | 0.757 | 0.557 | NS |
| TOT mean | | | | | |
| Treatment | 1.072 | 1.378 | 0.778 | 0.544 | NS |
| Time | 0.245 | 0.054 | 4.505 | 0.038 | * |
| Treatment x Time | 0.055 | 0.054 | 1.016 | 0.406 | NS |
| TOT SD | | | | | |
| Treatment | 0.121 | 0.271 | 0.446 | 0.775 | NS |
| Time | 0.133 | 0.510 | 0.260 | 0.612 | NS |
| Treatment x Time | 0.301 | 0.510 | 0.589 | 0.672 | NS |
| WAKE mean | | | | | |
| Treatment | 1.774 | 1.195 | 1.485 | 0.217 | NS |
| Time | 0.251 | 0.643 | 0.390 | 0.534 | NS |
| Treatment x Time | 0.731 | 0.643 | 1.136 | 0.347 | NS |
| WAKE SD | | | | | |
| Treatment | 0.263 | 0.274 | 0.961 | 0.435 | NS |
| Time | 0.015 | 0.421 | 0.036 | 0.850 | NS |
| Treatment x Time | 0.022 | 0.421 | 0.052 | 0.995 | NS |
| REPTH | | | | | |
| Treatment | 0.762 | 0.871 | 0.874 | 0.484 | NS |
| Time | 2.532 | 0.495 | 5.115 | 0.027 | * |
| Treatment x Time | 0.716 | 0.495 | 1.446 | 0.229 | NS |
| RESTED | | | | | |
| Treatment | 0.337 | 0.351 | 0.959 | 0.436 | NS |
| Time | 0.340 | 0.452 | 4.141 | 0.046 | * |
| Treatment x Time | 0.340 | 0.452 | 0.751 | 0.561 | NS |
| ENJOY | | | | | |
| Treatment | 0.869 | 0.417 | 2.084 | 0.093 | NS |
| Time | 0.708 | 0.401 | 1.764 | 0.189 | NS |
| Treatment x Time | 0.206 | 0.401 | 0.513 | 0.727 | NS |
| IRRIT | | | | | |
| Treatment | 0.481 | 0.844 | 0.570 | 0.685 | NS |
| Time | 0.190 | 0.352 | 0.054 | 0.817 | NS |
| Treatment x Time | 0.131 | 0.352 | 0.371 | 0.829 | NS |
| NAPDAY | | | | | |
| Treatment | 529.28 | 267.05 | 1.982 | 0.108 | NS |
| Time | 437.04 | 247.65 | 1.765 | 0.189 | NS |
| Treatment x Time | 37.03 | 247.65 | 0.149 | 0.963 | NS |

TABLE 7 MANOVAs at baseline for the eleven sleep diary variables. F values based upon Pillai's statistic. Time main effects df= (1,65), treatment and interaction effects df = (4,65). * $p \leq .05$

hypotheses can be accepted. It is clear that no systematic between group differences emerged during baseline. The random allocation procedure appears to have been successful.

## THE EFFECT OF THE DEMAND CHARACTERISTICS MANIPULATION

After baseline assessment each subject received eight consecutive weeks of treatment under one of the experimental conditions. With the exception of the waiting-list group this treatment period was sub-divided into counterdemand and positive demand phases, each of four week duration. The data can be analysed, therefore, either as a whole series or as two separate series. The latter possibility, however, would be appropriate only if the reversal in demand characteristics produced demonstrable change in the overall pattern of scores over time (ie. treatment gains observable during positive demand which were not evident during counterdemand). In order to test these possibilities the simple effects of time within treatment group were obtained for each variable. This was achieved via the CONSPLUS sub-command of the SPSSX MANOVA programme which generates an overall F ratio for the entire treatment period, and univariate F ratios for the sub-effects at each data point. Once again the scores entered for each time point were change scores over baseline. Included in the results displayed in Table 8 are those analyses where the multivariate F probability was less than .10.

Inspection of the Table clearly reveals that for the vast majority of variables, change scores first became significant during the counterdemand phase when negative expectancies were engendered. The introduction of positive demand had no apparent effect beginning at Week 5 on any variable as would have been expected had demand characteristics markedly affected response to therapy. Only 4 of the 20 significant F values refer to treatment changes emerging under positive

demand. Sufficient explanation of these may be in terms of delayed treatment response and/or possible false positive results due to F values in the range .05 to .10. Furthermore, the specific purpose of including the demand manipulation was to monitor the effects of demand upon placebo treatment. The data in Table 8 clearly indicate that little change took place at any time for this group.

| Variable | Group | F Prob | Counterdemand Week 1 | 2 | 3 | 4 | Positive Demand Week 5 | 6 | 7 | 8 |
|---|---|---|---|---|---|---|---|---|---|---|
| SOL mean | Relaxation | .016 | | * | * | | * | * | * | * |
| | Stim Control | .000 | * | * | * | * | * | * | * | * |
| | Paradox | .016 | | | | * | * | * | * | * |
| SOL SD | Relaxation | .012 | | * | * | * | * | * | * | |
| | Stim Control | .001 | * | * | * | | * | * | * | * |
| | Paradox | .000 | | * | * | * | * | * | * | * |
| TOT mean | Relaxation | .010 | | | * | | | * | * | * |
| | Stim Control | .014 | | | | | | | * | * |
| TOT SD | Relaxation | .003 | * | * | | | | * | * | * |
| | Stim Control | .003 | | | | * | | | * | |
| | Paradox | .078 | | * | | * | * | * | | * |
| WAKE mean | Paradox | .011 | | * | | * | * | * | * | * |
| WAKE SD | Relaxation | .097 | | * | * | | * | * | * | |
| | Stim Control | .030 | | | * | | | * | * | * |
| | Placebo | .093 | | | * | | | | | |
| RESTED | Relaxation | .003 | | * | * | * | | * | * | * |
| | Paradox | .033 | | | | | | * | * | * |
| ENJOY | Relaxation | .002 | | * | * | * | * | * | * | * |
| | Placebo | .074 | | | | | | | | * |
| NAPDAY | Paradox | .084 | | | | | | | | * |

TABLE 8 Time within treatment group simple effects and sub-effects at each week during the treatment period. Overall F probabilities are based upon Pillai's statistic. (* represents a time sub-effect $p < .05$)

These results indicated that it was appropriate to consider the eight week treatment period as a whole in further data analysis. The alteration in demand characteristics appears to have had little impact.

## ASSESSMENT OF TREATMENT CREDIBILITY

In order to investigate the credibility of treatment rationales, the Credibility Evaluation Questionnaire (CEQ) was completed by subjects in each experimental condition, excluding the waiting-list group. The maximum score obtainable on the CEQ was 30. Since mean scores for each condition fell within the range 24.4 to 25.2 [Appendix H, Table (iii)], it is clear that each of the treatments was considered highly credible, and that there was little variation across conditions. ONEWAY ANOVAs on CEQ scores confirmed that there were no significant between group differences [$F = 0.056$; df (3,40); $p = 0.983$]. Of particular importance is the fact that patients regarded the active treatments and the placebo treatment as equally logical and credible.

## THE EFFECTS OF DIFFERENT TREATMENTS UPON SLEEP PATTERN

Treatment effects upon DSQ sleep pattern variables will be considered prior to effects upon sleep quality.

### The Hypotheses

3) Each of the three active treatments would be superior to no treatment during the course of therapy. It was expected that the development of therapeutic effects might vary amongst treatments.

4) Similarly, each of the active treatments would be superior to placebo control.

5) No significant differences were predicted amongst the active treatments.

6) These hypotheses were expected to apply to SOL and TOT variables. Effects upon WAKE variables were regarded as uncertain since treatment was primarily aimed at reducing initial insomnia.

### Descriptive Analysis

A simple descriptive analysis was conducted first of all to provide a preliminary examination of the data. Table 9 presents mean values for each sleep pattern variable at baseline and the final experimental week

across conditions.

Both stimulus control and paradoxical intention produced considerable reduction in SOL mean and SOL SD with changes in the range 50-70%. By comparison relaxation training was less effective and more similar to placebo. For both of these conditions therapeutic change was in the range 25-35%. Interestingly, untreated patients exhibited exacerbation of their sleep-onset problem. Sleep duration (TOT) increased by a modest 10% across conditions with the exception of waiting-list control. It seems likely, therefore, that this variable will reveal few between group differences in subsequent analyses. TOT SD, however, was rather more discriminating. Paradox produced a greater than 40% reduction compared with minimal change in the other active treatments. Placebo control also effected a substantial reduction

| | | Relaxation | Stimulus Control | Paradoxical Intention | Placebo | No Treatment |
|---|---|---|---|---|---|---|
| SOL mean | pre | 90.5 | 82.7 | 72.4 | 85.5 | 84.5 |
| | post | 57.4 | 31.1 | 35.8 | 63.5 | 96.5 |
| | % change | -36.6 | -62.4 | -50.6 | -25.7 | +14.2 |
| SOLSD | pre | 50.0 | 41.9 | 47.9 | 44.2 | 53.6 |
| | post | 37.4 | 19.0 | 14.2 | 30.6 | 63.5 |
| | % change | -25.2 | -54.7 | -70.4 | -30.8 | +18.5 |
| TOT mean | pre | 5.79 | 6.10 | 5.86 | 5.34 | 5.84 |
| | post | 6.41 | 6.66 | 6.55 | 5.82 | 5.84 |
| | % change | +10.7 | + 9.2 | +11.8 | + 9.0 | 0.00 |
| TOT SD | pre | 1.41 | 1.17 | 1.33 | 1.23 | 1.25 |
| | post | 1.32 | 1.07 | 0.78 | 0.94 | 1.28 |
| | % change | - 6.4 | - 8.5 | -41.4 | -23.6 | + 2.4 |
| WAKE mean | pre | 1.56 | 0.93 | 1.16 | 1.76 | 1.01 |
| | post | 1.23 | 0.44 | 0.53 | 1.34 | 0.85 |
| | % change | -21.2 | -52.4 | -54.3 | -23.9 | -15.8 |
| WAKE SD | pre | 0.96 | 0.67 | 0.82 | 0.95 | 0.68 |
| | post | 0.75 | 0.16 | 0.60 | 0.94 | 0.63 |
| | % change | -21.9 | -76.2 | -26.8 | - 1.5 | - 7.4 |

TABLE 9 Comparison across experimental conditions of mean scores during baseline (pre) and the final week of treatment (post) along with percentage change scores for each of the sleep pattern variables

on this measure. Interpretation of change scores for the WAKE variables is rather problematic due to the relatively low frequency of nightly wakenings occurring at baseline. Nevertheless, stimulus control and paradox, as for the SOL variables, emerged as the most effective interventions. Both placebo and no treatment patients, however, also reported considerable reduction in wakening frequency.

Since Table 9 considers only pre-post data, more detailed descriptive information is included in Figs 4 to 9. These graphs illustrate changes in sleep variables across the eight treatment weeks and permit visual comparison of the rate of change across conditions. It is of interest to note that for both SOL mean and SOL SD (Figs 4-5) the profile of scores for relaxation and placebo conditions is similar, whereas stimulus control and paradox demonstrated a superior response. Stimulus control produced immediate changes in these variables and paradox yielded improvement from the fourth week onwards. For TOT mean (Fig 6), progressive increases in duration of sleep are evident with relaxation and paradox, whereas stimulus control appears to have reduced TOT initially but was associated with improvements similar to the other active treatments in the latter stages. There was a comparable gradient of improvement also with placebo treatment. By comparison, no treatment produced erratic sleep. For TOT SD there was considerable overlap in treatments over time (Fig 7). Paradox alone produced consistent improvement over time although all of the other active treatments and the placebo group improved initially. Placebo and stimulus control had similar absolute values at post-treatment. Both WAKE mean and WAKE SD exhibited marked similarity in pattern over time (Figs 8-9) and visual inspection of the former graph suggests little systematic between group difference. For WAKE SD, however, greater variability was evident in placebo and stimulus control interventions, with stimulus control gradually improving compared with placebo.

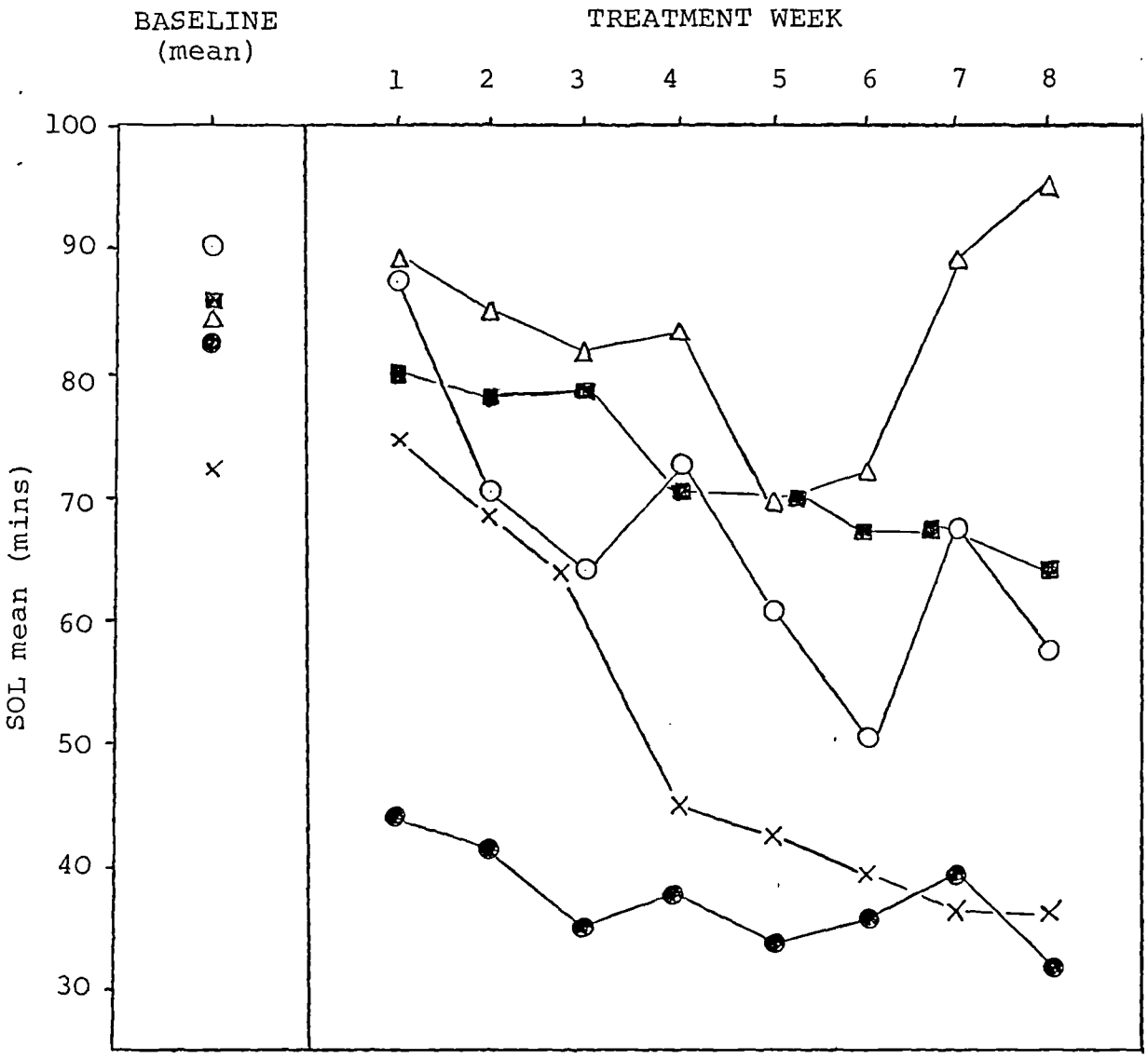



FIGURE 4: Mean sleep latency scores for each of the experimental groups across each week of the experimental period.

○ Relaxation
● Stimulus Control
× Paradoxical Intention
■ Placebo
△ No Treatment

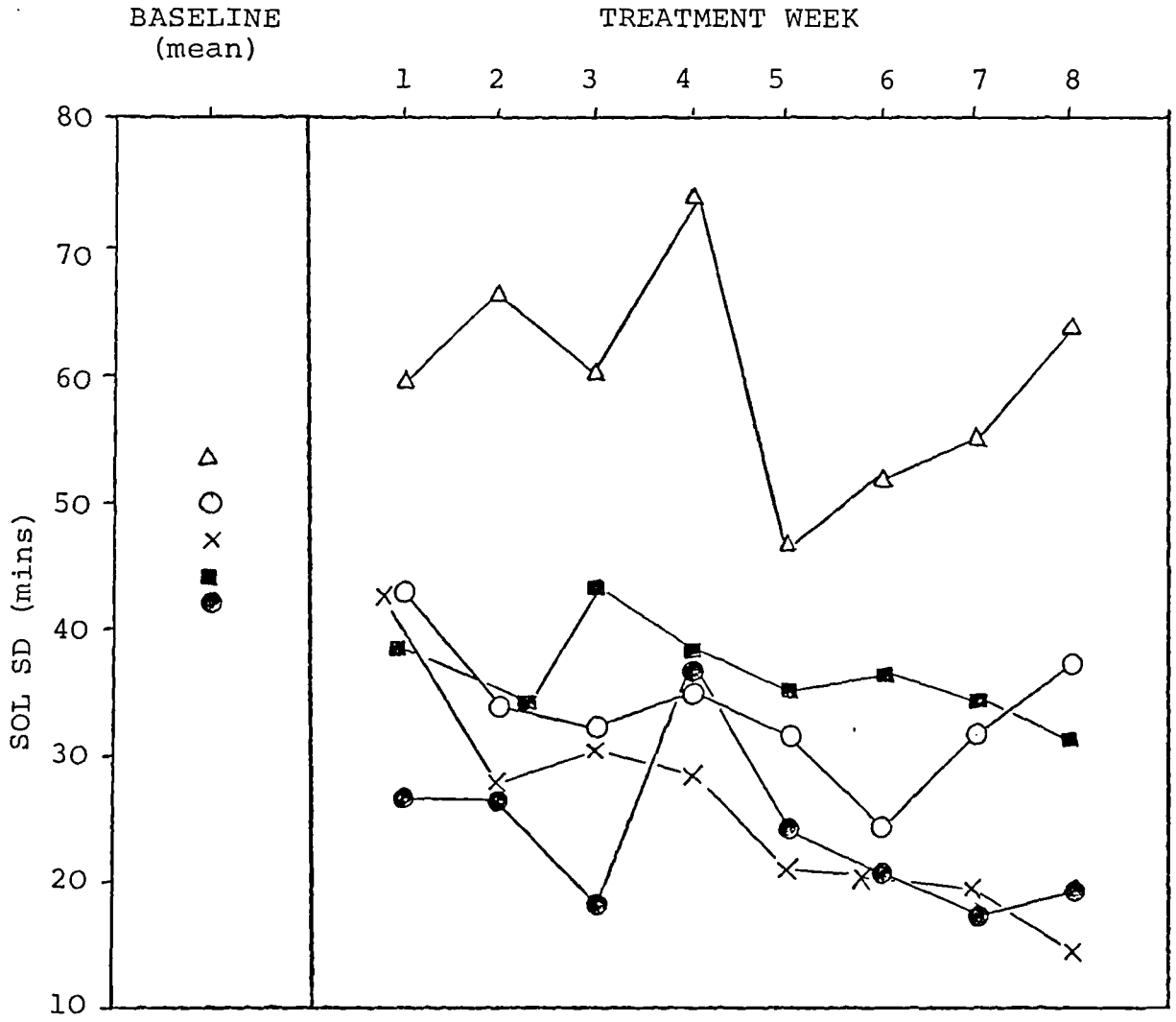
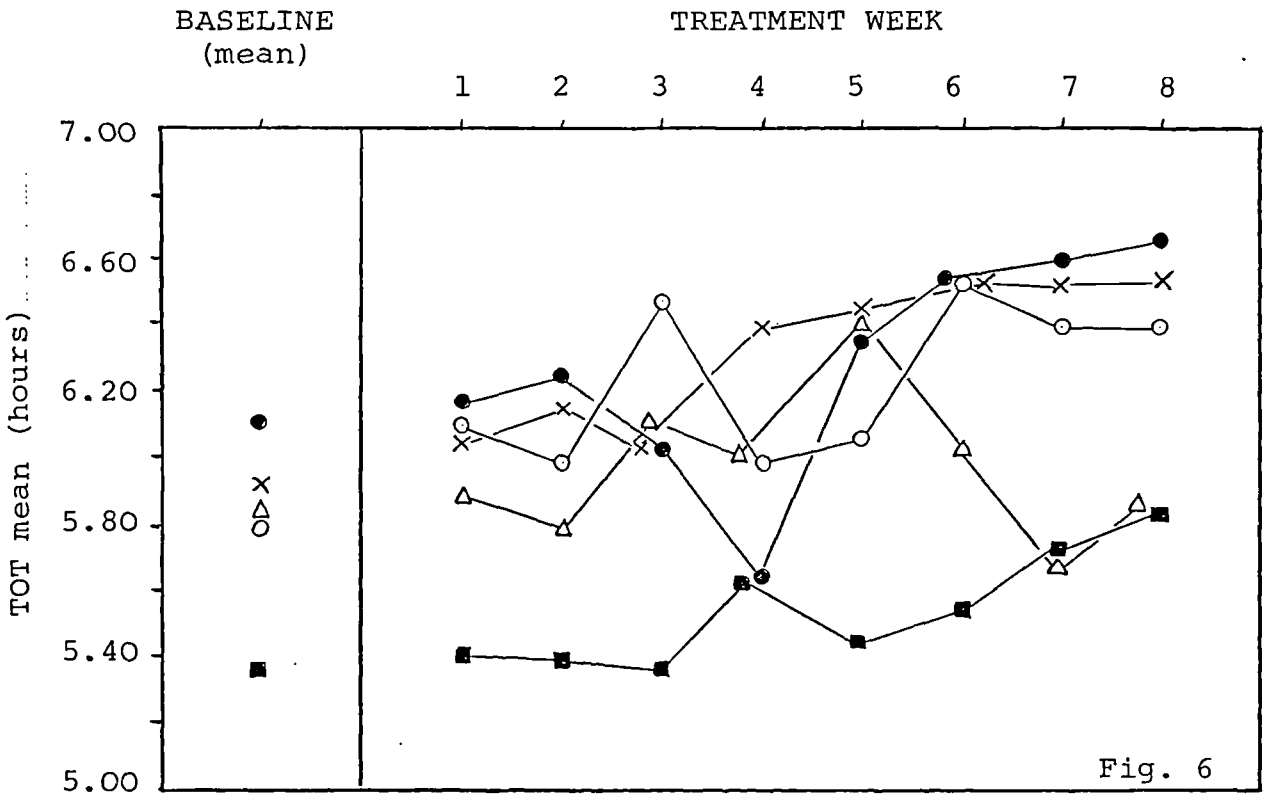



FIGURE 5: Mean scores for sleep-onset variability for each of the experimental groups across each week of the experimental period.

○ Relaxation
● Stimulus Control
X Paradoxical Intention
■ Placebo
△ No Treatment



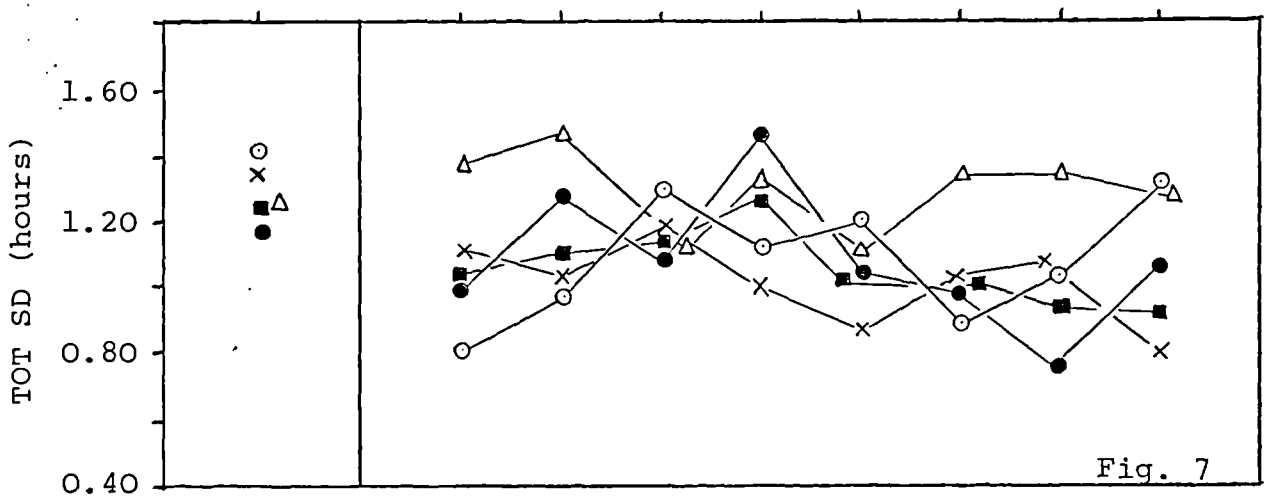
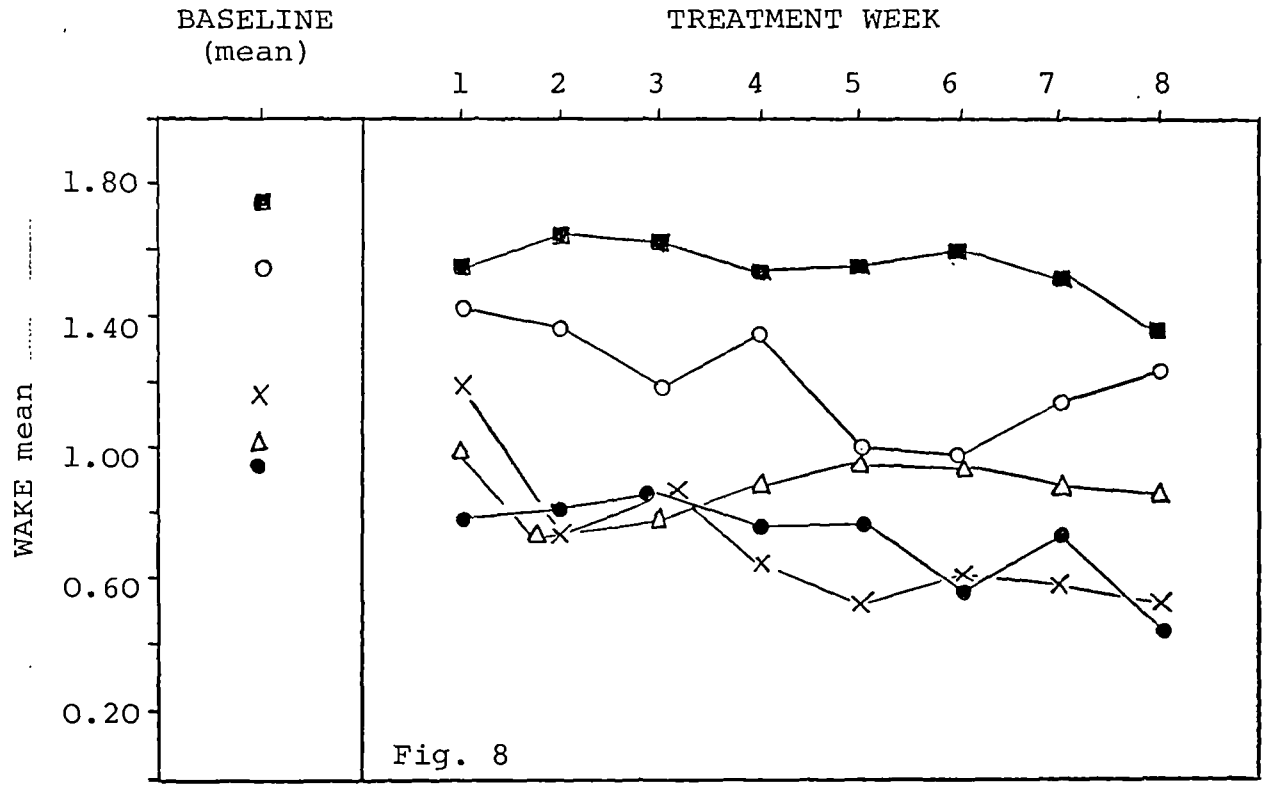




FIGURE 6: Mean scores for sleep duration for each of the experimental groups across each week of the experimental period.

FIGURE 7: As Fig. 6, but for variability of sleep duration.

○ Relaxation
● Stimulus Control
× Paradoxical Intention
■ Placebo
△ No Treatment



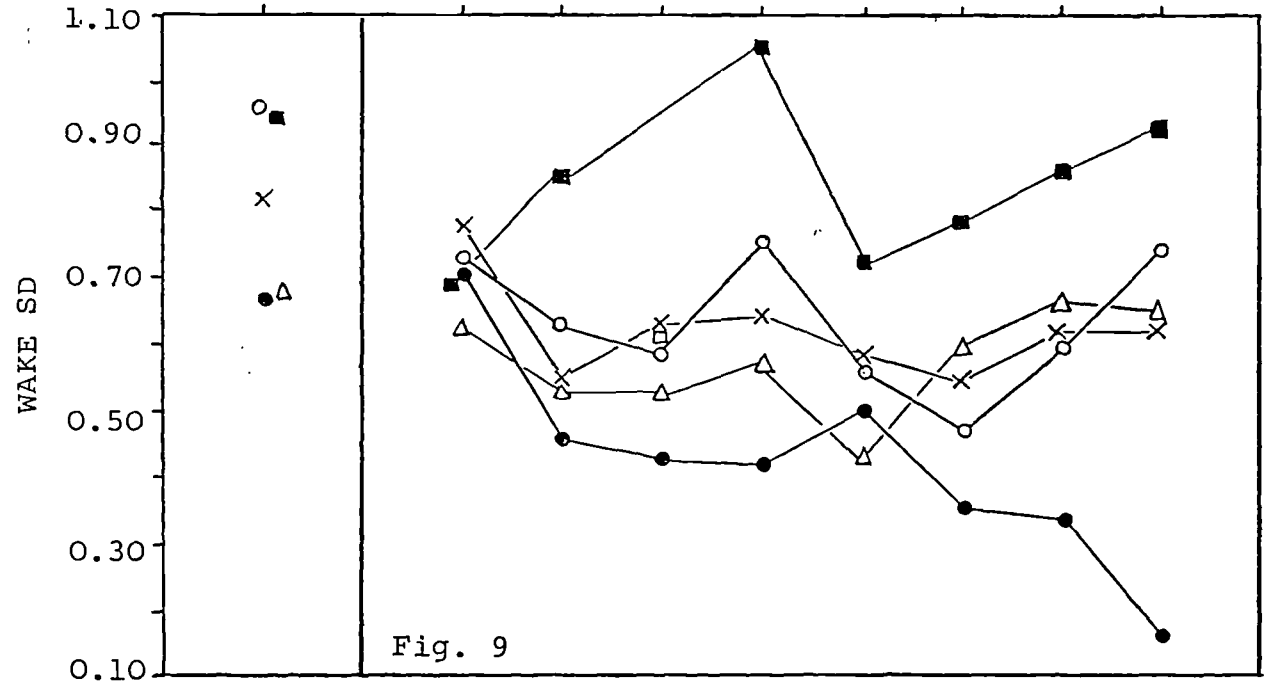




FIGURE 8: Mean scores for wakening frequency for each of the experimental group across each week of the experimental period.

FIGURE 9: As Fig. 8, but for variability of wakening.

○ Relaxation
● Stimulus Control
× Paradoxical Intention
■ Placebo
△ No Treatment

Since only tentative suggestions could be made regarding Hypotheses 3-6 on the basis of these descriptive analyses, matters will be left until the completion of statistical appraisal. Statistical analysis of the outcome data across time and between treatments required a repeated measures design which was again conducted by means of the SPSSX MANOVA programme. As previously described this procedure involved the computation of change scores as time contrast variables. For the following analyses, therefore, change scores were derived using the formula; Baseline Mean - Score at Week n. The baseline mean was selected as the summary statistic most descriptive of untreated sleep pattern.

Statistical Analysis

1) Main Effects

Table 10 presents the results of MANOVAs for the sleep pattern variables. Both treatment main effects and the treatment x time interactions were significant for the variables SOL mean and SOL SD. The former effect indicated overall between group differences in terms of average treatment response and the latter pointed to the systematic nature of these differences over time. TOT SD also exhibited a significant interaction effect. Further treatment within time analyses were conducted, therefore, on these three variables only, in order to establish which were the critical between group differences and at which point(s) during therapy these differences emerged. Since every variable demonstrated significant time main effects, it was of interest also to consider thereafter the simple effects of time within treatment group for all six variables.

2) Treatment within Time Sub-effects

In order to investigate the treatment within time sub-effects, ONEWAY ANOVAs across the five experimental conditions were conducted at each

| Source of variation | Pillai | Hyp df | Error df | F Ratio | F Prob | Sig Level |
|---|---|---|---|---|---|---|
| SOL mean | | | | | | |
| Treatment | - | 4 | 65 | 3.696 | 0.009 | ** |
| Time | 0.437 | 8 | 58 | 5.636 | 0.000 | *** |
| Treatment x Time | 0.758 | 32 | 244 | 1.783 | 0.008 | ** |
| SOL SD | | | | | | |
| Treatment | - | 4 | 65 | 5.339 | 0.001 | *** |
| Time | 0.507 | 8 | 58 | 7.449 | 0.000 | *** |
| Treatment x Time | 0.709 | 32 | 244 | 1.643 | 0.020 | * |
| TOT mean | | | | | | |
| Treatment | - | 4 | 65 | 1.779 | 0.144 | NS |
| Time | 0.324 | 8 | 58 | 3.477 | 0.002 | ** |
| Treatment x Time | 0.635 | 32 | 244 | 1.440 | 0.067 | NS |
| TOT SD | | | | | | |
| Treatment | - | 4 | 65 | 1.050 | 0.388 | NS |
| Time | 0.315 | 8 | 58 | 3.338 | 0.003 | ** |
| Treatment x Time | 0.730 | 32 | 244 | 1.701 | 0.014 | * |
| WAKE mean | | | | | | |
| Treatment | - | 4 | 65 | 2.147 | 0.085 | NS |
| Time | 0.294 | 8 | 58 | 3.015 | 0.007 | ** |
| Treatment x Time | 0.403 | 32 | 244 | 0.854 | 0.695 | NS |
| WAKE SD | | | | | | |
| Treatment | - | 4 | 65 | 0.640 | 0.636 | NS |
| Time | 0.307 | 8 | 58 | 3.211 | 0.004 | ** |
| Treatment x Time | 0.464 | 32 | 244 | 1.001 | 0.471 | NS |

TABLE 10 Repeated measures analysis of the sleep pattern variables across the experimental period using MANOVA
(* $p \leq .05$ ** $p \leq .01$ *** $p \leq .001$)

of the eight data points. Only those sleep variables which had demonstrated significant interaction effects were included (ie. SOL mean, SOL SD, TOT SD). Both Scheffe range tests and planned contrast t-tests were applied to identify the loci of significant difference. The inclusion of both procedures was to maximise the interpretability of the results through the use of both "conservative" and "sensitive" multiple comparison tests. The Scheffe test is a post hoc procedure which has been developed for "data snooping", that is to make all possible comparisons among mean values to determine the source of an already demonstrated effect (Scheffe, 1953). It is regarded as conservative in that it will minimise the false positive "Type 1" error rate for pairwise comparisons and in this respect may be preferred to

other a posteriori tests such as that of Tukey [see Kirk (1968) for extensive review]. It does so, however, at the expense of sensitivity to potentially important differences. As Kerlinger (1976) puts it "to attain significance, differences have to be rather substantial —— The Scheffe test makes things precise in a conservative way (but) —— one must examine one's data in detail; one rummages for insights and clues" (p 235). In recognition of his test's conservatism Scheffe himself has recommended that screening for significant between group differences be conducted at both the 5% and 10% levels (Scheffe, 1959).

The (a priori) planned CONTRAST procedure of the SPSSX ONEWAY programme was also employed further to increase the sensitivity of between group testing. This approach involves multiple independent t-test comparisons, a procedure which raises the probability of spuriously significant results being obtained (Kirk, 1968). The alpha level was, therefore, set at .01 to control for heightened error rate and the possibility of overinterpretation of results.

The results of these analyses are presented in Table 11. For SOL mean, sleep latency reduction during the first two weeks was significantly greater with stimulus control treatment compared with both no treatment and paradox (Scheffe $p < .05$). Smaller effects were also evident in comparison with relaxation and placebo conditions indicating that stimulus control was the most effective intervention for rapid sleep latency reduction. During Weeks 3 and 4, however, these effects were much reduced as evidenced by the smaller F ratios, and the lack of significance on Scheffe testing at the .05 level. The extended range of Scheffe sensitivity at $p < .10$, along with planned contrasts suggested, however, that the pattern of treatment effect was similar. From the fifth to the final week, stimulus control remained the most effective intervention, with highly significant F ratios again emerging

| | df | F Ratio | F Prob | Pair | Scheffe Range .05 | Scheffe Range .10 | Planned t .01 |
|---|---|---|---|---|---|---|---|
| SOL mean | | | | | | | |
| Week 1 | (4,63) | 4.125 | 0.005 | 2 v 5 | * | * | * |
| | | | | 2 v 3 | * | * | * |
| | | | | 2 v 1 | | * | * |
| | | | | 2 v 4 | | | * |
| Week 2 | (4,62) | 4.269 | 0.004 | 2 v 5 | * | * | * |
| | | | | 2 v 3 | * | * | * |
| | | | | 2 v 4 | | * | * |
| Week 3 | (4,64) | 3.379 | 0.014 | 2 v 5 | | * | * |
| | | | | 2 v 4 | | | * |
| | | | | 2 v 3 | | | * |
| Week 4 | (4,59) | 1.643 | 0.177 | (2 v 5) | | | (.02) |
| Week 5 | (4,61) | 3.863 | 0.007 | 2 v 5 | * | * | * |
| | | | | 2 v 4 | | * | * |
| | | | | 2 v 3 | | | * |
| Week 6 | (4,60) | 2.471 | 0.054 | 2 v 5 | | | * |
| Week 7 | (4,61) | 4.371 | 0.004 | 2 v 5 | * | * | * |
| Week 8 | (4,58) | 4.701 | 0.003 | 2 v 5 | * | * | * |
| | | | | 1 v 5 | * | * | * |
| SOL SD | | | | | | | |
| Week 1 | (4,63) | 1.172 | 0.332 | - | | | |
| Week 2 | (4,62) | 4.054 | 0.005 | 3 v 5 | * | * | * |
| | | | | 2 v 5 | | * | * |
| | | | | 1 v 5 | | * | * |
| Week 3 | (4,64) | 3.954 | 0.006 | 2 v 5 | * | * | * |
| | | | | 3 v 5 | | | * |
| Week 4 | (4,59) | 4.434 | 0.004 | 3 v 5 | * | * | * |
| | | | | 1 v 5 | | * | * |
| Week 5 | (4,62) | 1.590 | 0.188 | - | | | |
| Week 6 | (4,61) | 3.409 | 0.014 | 3 v 5 | | * | * |
| | | | | 1 v 5 | | * | * |
| | | | | 2 v 5 | | | * |
| Week 7 | (4,61) | 3.087 | 0.022 | 3 v 5 | | * | * |
| | | | | 2 v 5 | | * | * |
| Week 8 | (4,58) | 2.702 | 0.040 | 3 v 5 | | * | * |
| | | | | 2 v 5 | | | * |
| TOT SD | | | | | | | |
| Week 1 | (4,63) | 2.598 | 0.044 | 1 v 5 | * | * | * |
| Week 2 | (4,62) | 3.518 | 0.012 | 1 v 5 | * | * | * |
| | | | | 1 v 2 | | | * |
| Week 3 | (4,63) | 0.050 | 0.995 | - | | | |
| Week 4 | (4,58) | 1.452 | 0.230 | - | | | |
| Week 5 | (4,61) | 0.515 | 0.725 | - | | | |
| Week 6 | (4,61) | 2.261 | (0.073) | 1 v 5 | | | * |
| Week 7 | (4,61) | 1.613 | 0.183 | - | | | |
| Week 8 | (4,58) | 1.343 | 0.266 | - | | | |

TABLE 11 ANOVAs conducted upon each sleep variable during the experimental period across the five experimental conditions (Relaxation=1, Stimulus control=2, Paradoxical intention=3, Placebo=4, Waiting list=5). The results of Scheffe and Planned Contrast procedures are also recorded

during Weeks 5, 7 and 8. Range tests indicated that differences between stimulus control and no treatment were largely responsible for these effects, although placebo and paradox were also inferior at Week 5. In the final week of treatment relaxation training also emerged as superior to waiting-list control.

Summarising the data on SOL mean leaves little doubt that the strongest and most consistently beneficial treatment was stimulus control. Indeed, range testing on the significant main effects for treatment group (average response over time) revealed significant differences between stimulus control and no treatment (Scheffe $p < .05$) and a weaker but significant effect for stimulus control compared with placebo (Scheffe $p < .10$).

The data on SOL SD were also supportive of stimulus control as superior to waiting-list during Weeks 2, 3, 6, 7 and 8. Even stronger effects, however, were generally evident over time for the advantages of paradoxical intention over no treatment. On three occasions during the middle weeks, relaxation also appeared to be better than no treatment. Superiority over placebo, however, was not demonstrated by any of the active treatments on SOL SD and there were no significant differences amongst the treatments. Consistent with the above results, the overall treatment group main effect was accounted for by the superiority of both paradox and stimulus control over no treatment (Scheffe $p < .05$).

Summarising so far, it seems that stimulus control reduced both sleep onset latency and the variability of that latency compared with no treatment. Paradox appeared to make sleep-onset more predictable (ie. reduced variability) but it did not necessarily make sleep latency less than no treatment.

The third variable of interest was TOT SD. Significant F ratios were

achieved during Weeks 1 and 2, with a near significant effect also at Week 6. The consistent explanation for these results was a reduction in TOT SD under relaxation therapy compared with no treatment. It is perhaps surprising that an initially strong effect on Scheffe range testing should disappear. It may be that the advantages of relaxation were short-lived and did not foster maintained habit change.

3) Time within Treatment Group Sub-effects

Having considered the treatment within time sub-effects which permitted consideration of important between group differences, it was appropriate next to consider the complementary information provided by the time within treatment sub-effects. These analyses provided information, within each condition, on significant variation from baseline values across the experimental period. Time within treatment effects are essentially a series of within subjects repeated measures ANOVAs, and these were generated via the CONSPLUS sub-command of the MANOVA programme. These data were presented in simplified form in Table 8 in consideration of the effects of the demand change. Table 12, however, provides more detailed information, separately for each of the experimental conditions and illustrates the magnititude of effect of treatment upon each of the sleep pattern variables.

The immediate visual impact of the Table leaves little doubt that the three active treatments were associated with significant change over baseline on the majority of variables compared with the minimal changes obtained with either waiting-list or placebo conditions. Indeed, for the waiting-list group the only significant change was in terms of exacerbation of the SOL SD variable (cf. Fig 5). SOL mean and SOL SD were the variables most responsive to active treatment. Significant improvement on both variables occurred immediately at Week 1 with stimulus control and at Week 2 with relaxation. Paradox produced

| Treatment/ Variable | Pillai | F Prob | Weeks of Therapy 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 |
|---|---|---|---|---|---|---|---|---|---|---|
| RELAXATION | | | | | | | | | | |
| SOL mean | 0.266 | 0.016 | | * | * | | ** | *** | * | ** |
| SOL SD | 0.275 | 0.012 | | * | ** | * | * | *** | ** | |
| TOT mean | 0.280 | 0.010 | | | ** | | | ** | * | ** |
| TOT SD | 0.320 | 0.003 | *** | ** | | | | *** | ** | * |
| WAKE mean | 0.191 | 0.116 | | | | | | | | |
| WAKE SD | 0.198 | 0.097 | | * | ** | | ** | ** | * | |
| STIM CONTROL | | | | | | | | | | |
| SOL mean | 0.438 | 0.000 | *** | *** | *** | *** | *** | *** | *** | *** |
| SOL SD | 0.343 | 0.001 | * | * | *** | | * | ** | *** | ** |
| TOT mean | 0.269 | 0.014 | | | | | | | * | * |
| TOT SD | 0.321 | 0.003 | | | | * | | | ** | |
| WAKE mean | 0.109 | 0.536 | | | | | | | | |
| WAKE SD | 0.243 | 0.030 | | | * | | | * | * | *** |
| PARADOX INT | | | | | | | | | | |
| SOL mean | 0.265 | 0.016 | | | | ** | ** | ** | *** | *** |
| SOL SD | 0.405 | 0.000 | | ** | ** | ** | *** | *** | *** | *** |
| TOT mean | 0.215 | 0.162 | | | | | | | | |
| TOT SD | 0.207 | 0.078 | | * | | * | ** | * | | *** |
| WAKE mean | 0.277 | 0.011 | | ** | | ** | *** | ** | ** | *** |
| WAKE SD | 0.112 | 0.511 | | | | | | | | |
| PLACEBO | | | | | | | | | | |
| SOL mean | 0.070 | 0.816 | | | | | | | | |
| SOL SD | 0.086 | 0.713 | | | | | | | | |
| TOT mean | 0.097 | 0.626 | | | | | | | | |
| TOT SD | 0.118 | 0.468 | | | | | | | | |
| WAKE mean | 0.077 | 0.767 | | | | | | | | |
| WAKE SD | 0.200 | 0.093 | | | ** | | | | | |
| WAITING LIST | | | | | | | | | | |
| SOL mean | 0.225 | 0.049 | | | | | (0.14) | | | |
| SOL SD | 0.263 | 0.017 | | * | | ** | | | | |
| TOT mean | 0.192 | 0.112 | | | | | | | | |
| TOT SD | 0.115 | 0.490 | | | | | | | | |
| WAKE mean | 0.079 | 0.753 | | | | | | | | |
| WAKE SD | 0.073 | 0.796 | | | | | | | | |

TABLE 12 Time within treatment group simple effects and sub-effects at each week during treatment presented separately for each experimental condition (* $p < .05$ ** $p < .01$ *** $P < .001$)

change at Weeks 4 and 2 for SOL mean and SOL SD respectively. Consistency of improvement over baseline was particularly evident for SOL mean under both stimulus control and paradox, and SOL SD was also consistently reduced by the paradoxical treatment. Some variability over time was evident on both SOL variables during relaxation training, and also on SOL SD for the stimulus control group.

In terms of TOT mean and TOT SD, stimulus control had relatively little impact, except perhaps during the final two weeks. With relaxation, treatment benefit emerged initially and disappeared during the middle three weeks of therapy, on both variables, thereafter reemerging fairly strongly. Paradoxical intention did not achieve a significant F ratio for TOT mean although it should be noted that percentage improvement from pre to post-treatment was in fact greatest under paradox (Table 9). TOT SD was generally reduced from Week 2 onwards with paradox though the overall F ratio was of limited significance.

Frequency of night-time wakening was affected only by paradoxical treatment, being consistently reduced below baseline from the fourth week of treatment. For WAKE SD, which is the variability of nightly arousals over time, both relaxation and stimulus control demonstrated some change. The overall multivariate test was, however, of greater significance for stimulus control; perhaps accounted for by the large change over baseline observed during the final week of intervention.

## Addressing the Hypotheses

Having completed all the necessary analyses it is now possible to address the hypotheses which were outlined earlier. Hypothesis 3 stated that each of the three active treatments would be superior to no treatment during the course of therapy. This important hypothesis has received considerable support. Table 10 revealed that SOL mean, SOL SD and TOT SD responded differentially to treatments over time and Table 11 identified the superiority of an active treatment over no treatment as the responsible between group difference. More specifically, stimulus control was superior to waiting-list on both the SOL variables, paradox was superior on SOL SD only, and there was some limited evidence for the advantages of relaxation over no treatment on TOT SD. These findings were confirmed also by the results presented in

Table 12, since the strongest within subjects effects were evident on these three variables. It is noteworthy that stimulus control produced improvement on both SOL variables at Week 1. Paradox reduced SOL SD scores from Week 2, but it was not until Week 4 that change over baseline was obtained for SOL mean. Indeed, stimulus control was found to be superior to paradox during the first few weeks (cf. Hypothesis 5). The time within treatments analyses also suggested improvements over baseline on the other sleep pattern variables which appeared not to be due to the effects of the passage of time alone. These nevertheless failed to demonstrate significance on between group tests.

In terms of Hypothesis 4, stimulus control was the only treatment to demonstrate superiority over placebo, doing so consistently on the variable SOL mean. Placebo patients were found, however, from the time within treatments analysis, to present with a markedly stable sleep pattern over time with only one significant improvement over baseline being achieved out of the 48 comparisons made (6 variables times 8 data points). Again, Table 12 suggests that the active treatments were generally superior to placebo on most variables. Between group comparisons, however, failed to reach significance apart from the superiority of stimulus control over placebo on SOL mean.

Only one significant difference was found amongst the active treatments thus Hypothesis 5 was largely confirmed. The initial superiority of stimulus control over paradox is, however, of some interest (see Espie and Lindsay, 1985) and will be addressed later. Hypothesis 6 was broadly confirmed in that SOL and TOT variables were more sensitive to changes over time and to between group differences than were WAKE variables. Only the time within treatment effects yielded significant F ratios for the WAKE variables, the strongest being for the reduction of WAKE mean via paradox. No significant between group difference emerged.

## THE EFFECTS OF DIFFERENT TREATMENTS UPON SLEEP QUALITY

### The Hypotheses

The hypotheses concerning the sleep quality variables were similar to those proffered regarding sleep pattern.

7) Each of the active treatments would be superior to no treatment during the course of therapy.

8) Each of the treatments would also be superior to placebo control during therapy.

9) No significant differences would be found amongst the active treatments.

10) The above hypotheses were expected to apply to each of the variables REPTH, RESTED, ENJOY, IRRIT and NAPDAY.

### Descriptive Analysis

Initial inspection of the raw data for each variable suggested that there were few significant differences across treatments throughout the experimental period. For this reason simple descriptive analysis will not be presented in the text. Tabulated data are, however, available in Appendix H [Tables (iv) to (viii)]. Statistical analyses of the sleep quality data were identical to those described in the previous section. It should be noted, however, that four of the five variables (all excluding NAPDAY) comprised rating scale data rather than continuous scales. It was decided, nevertheless, to use MANOVA and associated procedures rather than non-parametric techniques because of the detailed information on sub-effects which could be readily obtained by this method. Consequently, it was decided to regard as tentative any conclusions based upon relatively weak statistical effects.

### Statistical Analysis

1) Main Effects

Table 13 presents the MANOVA repeated measures analyses with main

effects for each of the sleep quality variables. Treatment main effects yielded modestly significant between group differences for the variables REPTH and NAPDAY, but neither of the interactions approached significance for these variables. To investigate these treatment main effects, planned comparisons tests were conducted which revealed that relaxation training produced a greater average reduction in REPTH than did placebo control (Scheffe $p < .10$), and stimulus control was associated with less daytime napping than the waiting-list condition (Scheffe $p < .10$). The only treatment x time effect to come close to significance was for the variable ENJOY ($p = .065$). No other F ratio yielded a probability value < .10.

| Source of Variation | Pillai | Hyp df | Error df | F Ratio | F Prob | Sig Level |
|---|---|---|---|---|---|---|
| REPTH | | | | | | |
| Treatment | - | 4 | 65 | 2.654 | 0.041 | * |
| Time | 0.224 | 8 | 58 | 2.096 | 0.051 | NS |
| Treatment x Time | 0.373 | 32 | 244 | 0.784 | 0.794 | NS |
| RESTED | | | | | | |
| Treatment | - | 4 | 65 | 1.151 | 0.341 | NS |
| Time | 0.382 | 8 | 58 | 4.490 | 0.000 | *** |
| Treatment x Time | 0.570 | 32 | 244 | 1.268 | 0.162 | NS |
| ENJOY | | | | | | |
| Treatment | - | 4 | 65 | 1.868 | 0.127 | NS |
| Time | 0.371 | 8 | 58 | 4.362 | 0.000 | *** |
| Treatment x Time | 0.637 | 32 | 244 | 1.445 | 0.065 | NS |
| IRRIT | | | | | | |
| Treatment | - | 4 | 65 | 0.762 | 0.554 | NS |
| Time | 0.201 | 8 | 58 | 1.831 | 0.089 | NS |
| Treatment x Time | 0.411 | 32 | 244 | 0.886 | 0.647 | NS |
| NAPDAY | | | | | | |
| Treatment | - | 4 | 65 | 2.559 | 0.047 | * |
| Time | 0.141 | 8 | 58 | 1.188 | 0.322 | NS |
| Treatment x Time | 0.477 | 32 | 244 | 1.033 | 0.424 | NS |

TABLE 13 Repeated measures analysis of the sleep quality variables across the experimental period using MANOVA (* $p \leq .05$ *** $p \leq .001$)

2) Treatment within Time Sub-effects

ONEWAY ANOVAs across treatments were conducted, therefore, at each week of the experimental period for the variable ENJOY only, in order to

identify any loci of significant effect. The only significant between group effect to emerge from this analysis was at Week 6 where relaxation was superior to placebo on Scheffe testing at the .05 level [F = 3.123, F Prob =.021, df (4,61)].

3) Time within Treatment Group Sub-effects

Table 13 also revealed highly significant time main effects for RESTED and ENJOY. Time within treatment group effects were, therefore, investigated by means of the CONSPLUS sub-routine for each of these variables. The results of those analyses achieving significance $p < .10$ are available in Table 14 which is a development of part of Table 8 presented earlier in the chapter. Relaxation therapy produced highly significant improvement over time in restedness after sleep and sleep enjoyment. The time sub-effects indicated a similar pattern of change in the variables commencing at Week 2. Paradoxical intention was associated with weaker overall effects than relaxation. During the final three weeks of therapy restedness increased and there was a very small effect for NAPDAY at Week 8. The only other minor effect was for the placebo group where a small increase in sleep satisfaction was evident but again only during the final week.

| Treatment/ Variable | Pillai | F Prob | Weeks of Therapy 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 |
|---|---|---|---|---|---|---|---|---|---|---|
| RELAXATION | | | | | | | | | | |
| RESTED | 0.321 | 0.003 | | * | ** | * | | *** | * | *** |
| ENJOY | 0.326 | 0.002 | | ** | *** | * | * | *** | ** | *** |
| PARADOX | | | | | | | | | | |
| RESTED | 0.240 | 0.033 | | | | | | ** | ** | ** |
| NAPDAY | 0.204 | 0.084 | | | | | | | | * |
| PLACEBO | | | | | | | | | | |
| ENJOY | 0.209 | 0.074 | | | | | | | | * |

TABLE 14 Time within treatment group simple effects and sub-effects for the sleep quality variables where overall F probability < .10 (* $p \leq .05$ ** $p \leq .01$ *** $p \leq .001$)

Addressing the Hypotheses

In summary, it can be concluded that the five sleep quality variables demonstrated little differential response to the treatments over time. In terms of Hypothesis 7, none of the active treatments demonstrated systematic superiority over no treatment on any variable. Stimulus control did, however, produce greater average reduction in daily napping than did waiting-list (treatment main effect), and changes over time within treatments were evident with relaxation and to a lesser extent with paradox for measures of sleep satisfaction. These were not paralleled in the untreated group. Relaxation therapy appears to be the best treatment for improving reports of sleep quality.

Comparisons with placebo yielded similarly limited significant findings (Hypothesis 8). Relaxation significantly improved sleep enjoyment relative to placebo but only at Week 6, and relaxation was again responsible for the significant treatment main effect for REPTH, being modestly superior to placebo. Interaction effects did not reach significance for any variable.

No significant differences were found amongst active treatments (Hypothesis 9). It was, however, of interest to find that stimulus control, which emerged favourably from the analysis of sleep pattern variables, appeared to produce no complementary sleep quality changes. Finally, it is clear that only RESTED and ENJOY were useful in discriminating across and within treatments (Hypothesis 10). Treatments made little if any reliable impact upon anxious thoughts at bedtime, daytime irritability and daytime napping.

# CHAPTER 9

## MAINTENANCE AND GENERALISATION EFFECTS AND PREDICTORS OF OUTCOME

This chapter begins with analyses of sleep diary follow-up data in order to investigate the maintenance of treatment effects. Only those variables which exhibited significant change during therapy are, therefore, relevant for consideration. Symptomatic and behavioural ratings gathered pre and post-treatment will then be analysed to investigate generalised changes associated with the treatment programmes. Outcome will then be considered as a function of demographic and clinical factors, to investigate possible predictor variables. Finally, the effectiveness of the drug withdrawal programmes will be considered.

### THE MAINTENANCE OF TREATMENT EFFECTS

Patients in the three active treatments were contacted on four separate occasions to provide follow-up data by completion of one week's sleep diary. Routine follow-ups were at 6 weeks, 3 months and 6 months, and all subjects were also contacted at some point from the ninth month onwards for "long-term" follow-up. The mean time since treatment for this final assessment was 16.8 months (SD 9.2; range 9-41). Return rates were satisfactory across treatments with 77% of patients returning forms at 6 weeks, 58% at 3 months, 56% at 6 months and 70% at 17 months. The increased return rate for the final follow-up was due to repeat contacts stressing the importance of making a return. It was hypothesised that:-

11) Follow-up assessments would reveal durability of post-treatment improvements for each of the psychological therapies.

Table 15 presents summary data during baseline, the final therapy week, and at each of the follow-up times. Only sleep diary variables which

had demonstrated significant treatment response were included [see Appendix I, Table (ix) for complete print-out]. Inspection of the Table reveals that not only were treatment effects maintained during follow-up, but often they were extended. Continued improvement was particularly evident with relaxation training across all follow-up points, although each of the treatments was associated with further gain on some variable. Paired T-TEST comparisons were conducted (from post-treatment to follow-up point) within each condition for each variable and these confirmed the general pattern of maintenance. No significant deterioration over post-treatment values was found for any variable. The only significant changes to emerge were with paradoxical intention where total sleep time increased during the first three follow-ups and remained higher than post-treatment at 17 months. Ratings of sleep restedness were also significantly higher at 6 month

| Treatment/ Variable | Baseline | Final Week | Follow-up 6 Week | 3 Month | 6 Month | 17 Month |
|---|---|---|---|---|---|---|
| RELAXATION (n = | 14 | 13 | 11 | 8 | 10 | 9 ) |
| SOL mean | 90.5 | 57.4 | 48.1 | 45.3 | 37.5 | 45.1 |
| SOL SD | 50.0 | 37.4 | 27.9 | 30.2 | 16.3 | 30.6 |
| TOT mean | 5.79 | 6.41 | 6.73 | 6.65 | 6.81 | 6.61 |
| TOT SD | 1.41 | 1.32 | 1.02 | 1.07 | 0.92 | 1.45 |
| RESTED | 1.57 | 2.21 | 2.32 | 2.34 | 2.12 | 2.32 |
| ENJOY | 1.55 | 2.13 | 2.37 | 2.24 | 2.24 | 2.24 |
| STIM CONTROL (n = | 14 | 12 | 10 | 7 | 6 | 9 ) |
| SOL mean | 82.7 | 31.1 | 25.6 | 29.9 | 37.3 | 53.3 |
| SOL SD | 41.9 | 19.0 | 15.9 | 19.0 | 22.3 | 32.0 |
| TOT mean | 6.10 | 6.66 | 7.05 | 7.05 | 6.98 | 6.76 |
| WAKE SD | 0.67 | 0.16 | 0.34 | 0.54 | 0.29 | 0.35 |
| PARADOX (n = | 15 | 13 | 12 | 10 | 8 | 12 ) |
| SOL mean | 72.4 | 35.8 | 36.4 | 28.7 | 40.7 | 37.9 |
| SOL SD | 47.9 | 14.2 | 21.8 | 14.7 | 23.9 | 23.7 |
| TOT mean | 5.86 | 6.54 | 7.00 * | 7.10 * | 7.14 * | 6.83 |
| TOT SD | 1.33 | 0.78 | 0.94 | 1.22 | 0.98 | 1.08 |
| WAKE | 1.16 | 0.53 | 0.62 | 0.43 | 0.73 | 0.51 |
| RESTED | 1.92 | 2.33 | 2.37 | 2.50 | 2.61 * | 2.30 |

TABLE 15 Mean scores on sleep diary variables during baseline, the final therapy week, and at each of the follow-up assessments, presented separately within each of the active treatments (* $p \leq .05$ T-TEST)

follow-up. Given the multiple T-TEST comparisons involved in the overall analysis, however, the significance of these increments should be regarded with caution. Nevertheless, inclusion is useful to provide sensitivity to potentially important trends.

Significant between group differences were thought to be unlikely given the high levels of maintained improvement in each condition. Nevertheless, ONEWAY ANOVAs across treatments were conducted on change scores from final week to each follow-up point, using baseline scores as covariates. The only significant effect was for TOT SD where, at 3 month follow-up, relaxation was modestly superior to paradox [F = 3.63, F Prob = .048, df(2,18); Scheffe $p < .10$]. Examination of the data in Table 15 reveals that TOT SD was continuing to reduce in the relaxation group compared with a temporary relapse towards baseline under paradox. It seems likely that this is a spurious result.

In summary then, it is encouraging to note the strong continuing benefits of treatment during follow-up. Hypothesis 11 was, therefore, confirmed. There was a general trend towards further improvement, especially during the first months after treatment, but no significant relapse occured even 17 months after treatment.

GENERALISED CHANGES ASSOCIATED WITH TREATMENT

In addition to DSQ measures subjects completed rating scale assessments before and after treatment. Several tentative predictions were made:-

12) Relaxation and paradoxical intention, being recognised treatments for anxiety, would reduce state anxiety (ZAS) relative to other interventions.

13) Improvements in mood (ZDS) were expected after each of the active therapies, but not after the control procedures.

14) Only stimulus control would significantly reduce a measure of sleep-incompatible behaviour (SBRS).

15) Active treatments would be associated with improvements in a wide range of daytime functions (ARS). Control groups would not.

Prior to the consideration of pre-post change, pretest values on the ZAS, ZDS, SBRS, ARS and ARS items were subjected to ONEWAY ANOVA across treatments to investigate potential baseline differences between groups. The results of these analyses indicated that the experimental groups were functioning similarly at pre-treatment on these measures. None of the F ratios approached significance [Appendix I, Table (x)]. The possibility of sex differences in these symptomatic and behavioural measures was also considered, but independent sample T-TESTs again produced no significant effect [Appendix I, Table (xi)].

Table 16 presents mean scores for pre and post-treatment along with results of paired T-TESTs within each of the experimental conditions. The significance of each test result is presented in relation to three probability criteria in order to provide both conservative and sensitive consideration of the effects obtained.

Inspection of the Table reveals that each active treatment was associated with significant reduction in Zung Depression Scale (ZDS) scores, thus confirming Hypothesis 13. In each case improvement was of around six scale points, although the strongest effect was for the relaxation group. Significant changes on the Zung Anxiety Scale (ZAS) were also obtained with paradoxical intention and with placebo although the latter group averaged a score reduction of less than three points. Even the paradoxical intention group failed to achieve significant score reduction according to the more conservative criteria. Nevertheless, this result partly confirms Hypothesis 12 ie. improvement via paradox. Relaxation subjects did generally reduce anxiety level but not significantly.

The total score from the Analogue Rating Scale (ARS), as expected, also reduced significantly under each active treatment (Hypothesis 15) with paradox achieving the most reliable change. In addition, each

| Treatment/ Variable | pre | post | SE diff | T | df | Prob | Sig Level |
|---|---|---|---|---|---|---|---|
| RELAXATION | | | | | | | |
| ZAS | 36.5 | 32.6 | 2.72 | 1.44 | 12 | .175 | NS |
| ZDS | 42.2 | 35.5 | 1.62 | 4.09 | 12 | .002 | ** |
| SBRS | 42.1 | 40.4 | 1.51 | 1.17 | 12 | .264 | NS |
| ARS | 51.0 | 40.9 | 3.01 | 3.36 | 12 | .006 | ** |
| ARS 2 | 5.83 | 4.56 | 0.42 | 3.04 | 12 | .010 | ** |
| ARS 3 | 4.55 | 6.35 | 0.64 | -2.82 | 12 | .016 | * |
| ARS 7 | 3.98 | 5.64 | 0.35 | -4.77 | 12 | .000 | *** |
| ARS 9 | 2.65 | 5.81 | 0.76 | -4.19 | 12 | .001 | *** |
| STIM CONTROL | | | | | | | |
| ZAS | 37.4 | 34.2 | 2.20 | 1.48 | 11 | .168 | NS |
| ZDS | 45.3 | 38.9 | 2.05 | 3.08 | 11 | .010 | ** |
| SBRS | 41.6 | 33.0 | 2.74 | 3.14 | 11 | .009 | ** |
| ARS | 53.4 | 41.8 | 4.85 | 2.39 | 12 | .033 | * |
| ARS 9 | 1.92 | 4.74 | 0.73 | -3.88 | 12 | .002 | ** |
| PARADOX | | | | | | | |
| ZAS | 36.0 | 30.7 | 1.96 | 2.70 | 13 | .018 | * |
| ZDS | 39.8 | 34.6 | 1.61 | 3.23 | 13 | .007 | ** |
| SBRS | 45.4 | 43.9 | 1.60 | 0.94 | 13 | .365 | NS |
| ARS | 47.4 | 37.3 | 2.93 | 3.43 | 13 | .004 | ** |
| ARS 9 | 2.59 | 5.76 | 0.73 | -4.34 | 13 | .001 | *** |
| ARS 10 | 4.53 | 6.26 | 0.65 | -2.68 | 13 | .019 | * |
| PLACEBO | | | | | | | |
| ZAS | 39.9 | 37.0 | 1.26 | 2.30 | 10 | .047 | * |
| ZDS | 39.1 | 39.1 | 2.39 | 0.00 | 10 | .999 | NS |
| SBRS | 52.2 | 52.3 | 2.36 | -0.04 | 10 | .967 | NS |
| ARS | 46.6 | 39.2 | 5.49 | 1.35 | 10 | .211 | NS |
| ARS 9 | 1.36 | 4.49 | 1.27 | -2.46 | 10 | .036 | * |
| WAITING LIST | | | | | | | |
| ZAS | 41.5 | 41.8 | 1.40 | -0.19 | 10 | .850 | NS |
| ZDS | 42.3 | 43.1 | 1.18 | -0.71 | 11 | .494 | NS |
| SBRS | 45.4 | 44.2 | 0.55 | 2.17 | 11 | .058 | NS |
| ARS | 49.7 | 52.1 | 2.20 | -1.09 | 12 | .300 | NS |
| ARS 1 | 5.05 | 4.62 | 0.19 | 2.25 | 12 | .048 | * |

TABLE 16 Mean scores for pre and post-treatment rating scale assessments and results of paired T-TEST comparisons for each of the experimental conditions (* $p \leq .05$ ** $p \leq .01$ *** $p \leq .001$)

experimental condition produced some change on at least one ARS item [only significant effects are reproduced in the Table - see Appendix I, Table (xii) for all ARS item scores]. Relaxation reduced "worrying" (ARS 2) and improved daytime function in terms of "coping with work" and "concentration" (ARS 3 and 7). The latter improvement was highly significant. Ratings of "sleeping well" (ARS 9) also improved very significantly with relaxation, but also with stimulus

control and paradox. Placebo subjects, however, improved only modestly on this variable. Paradoxical intention was associated with some increase in "confidence" (ARS 10) and the waiting-list group were slightly less "energetic" (ARS 1) at post-treatment. The remaining four items from the ARS exhibited no significant change with any intervention.

The only group to exhibit significant improvement on the Sleep Behaviour Rating Scale (SBRS) was the stimulus control group. This was as expected (Hypothesis 14). Analysis of SBRS item scores for the stimulus control group demonstrated significant change (WILCOXON; $p < .05$) on only three items, ie. Item 1 "reading in bed", Item 17 "light off immediately in bed" and Item 18 "light on until almost asleep". Since the latter two were perfectly inversely correlated the most significant alteration from pre-bedtime routine was not to read in bed and to go to sleep immediately [see Appendix I, Table (xiii) for full analysis].

In order to consider the possibility of differential response to therapy on these pre-post measures, ONEWAY ANOVAs were conducted across treatments. Computed change scores (before - after scores) were used in this analysis to control for any minor variation in pre-treatment values. The results of those analyses yielding F ratios $p < .10$ are presented in Table 17, along with the results of range testing procedures. Inspection of the Table reveals that both relaxation and stimulus control were superior to no treatment in reducing ZDS scores, with the difference between relaxation and waiting-list contributing more strongly to the overall effect. Each of the active treatments and also the placebo demonstrated significant improvement over no treatment on "sleeping well" (ARS 9). The only other analogue item with F probability $< .10$ was "coping with work" (ARS 3) but no significant

between group differences emerged. Stimulus control demonstrated modest but consistent superiority over each of the other conditions on SBRS. Results of the remaining, non-significant ANOVAs can be found in Appendix I [Table (xiv)].

| Variable | df | F Ratio | F Prob | Pair | Scheffe .05 | Range .10 | Planned t .01 |
|---|---|---|---|---|---|---|---|
| ZDS | (4,57) | 3.994 | 0.006 | 1 v 5 | | * | * |
| | | | | 2 v 5 | | | * |
| SBRS | (4,57) | 3.158 | 0.021 | 2 v 4 | | * | * |
| | | | | 2 v 3 | | | * |
| | | | | 2 v 5 | | | * |
| | | | | 2 v 1 | | | * |
| ARS 3 | (4,53) | 2.259 | 0.076 | - | | | |
| ARS 9 | (4,59) | 4.320 | 0.004 | 1 v 5 | * | * | * |
| | | | | 3 v 5 | * | * | * |
| | | | | 4 v 5 | * | * | * |
| | | | | 2 v 5 | | * | * |

TABLE 17 ANOVAs on rating scale change scores across the experimental conditions (Relaxation=1, Stimulus control=2, Paradoxical intention=3, Placebo=4, Waiting-list=5). The results of range tests are also presented

In terms of the hypotheses, the between group effects were supportive of the specific impact of stimulus control upon SBRS scores (Hypothesis 14). There were no differential responses on ZAS (Hypothesis 12) and ZDS revealed relative improvements in mood, only when relaxation and stimulus control were compared with no treatment (Hypothesis 13). ARS total scores did not discriminate between groups and the only item score which did so referred to "sleeping well". This was not a measure of generalised function (Hypothesis 15).

## POSSIBLE PREDICTOR VARIABLES

It was of interest to identify any variables which might be predictive either of initial clinical presentation or of treatment response. Both demographic and clinical variables were considered in an attempt to evaluate such potential predictive factors. Since this was a

retrospective investigation, no hypotheses were forwarded. A correlational analysis was conducted, calculating a PEARSON co-efficient for the relationship between each potential predictor variable and the sleep diary measures. Both baseline and change (baseline - final week) scores were considered. Table 18 presents results for those variables achieving significance $p < .01$. Relationships significant at the 5% level only were considered to contain an unacceptably high error variance component [coefficients generally less than 0.25; see Appendix I, Table (xv) for complete analysis].

Out of the greater than 100 co-efficients computed, inspection of the Table reveals that only 14 achieved significance. Age and duration of insomnia bore little relationship either to baseline presentation or to outcome. The number of years since hypnotics were first prescribed (MEDPAST), however, was inversely related to several baseline measures. There was a significant tendency for TOT SD, WAKE SD and IRRIT to reduce with lengthier drug histories, and correspondingly, for RESTED to increase. This may suggest that sleep pattern becomes more predictable and evokes less emotional reaction in patients who have for longer made use of drugs as a coping strategy. Outcome measures of WAKE SD and REPTH, however, revealed less improvement for patients with longer drug histories. Therefore, no clear pattern emerged.

| | TOT SD Base | WAKESD Base | WAKESD Change | REPTH Base | REPTH Change | RESTED Base | IRRIT Base |
|---|---|---|---|---|---|---|---|
| AGE | | | | | | | -.281 * |
| DURATION | | | | | -.302 * | | |
| MEDPAST | -.296 * | -.304 * | -.338 * | | -.357 * | .286 * | -.359 * |
| ZAS pre | | | | .444 ** | | | .650 ** |
| ZDS pre | | | | .311 ** | .299 * | -.323 * | .641 ** |

TABLE 18 Predictor variables and their relationship with baseline and outcome sleep diary measures. Pearson co-efficients and probability values (* $p \leq .01$ ** $p \leq .001$) are displayed

It is of particular interest to note that no predictive relationships were demonstrated with any of the mean values for SOL, TOT and WAKE. These measures, which have been regarded as the standard summary descriptors of sleep pattern, appear not to vary systematically with age, chronicity of insomnia or symptomatic anxiety and depression.

The strongest and most consistent predictor variables were the clinical rating scales, although these were correlated only with sleep quality variables. Pre-treatment scores on both Zung scales were positively correlated with REPTH and IRRIT ie. the greater the symptomatology the greater the pre-bedtime worry and daytime irritability. Additionally, the ZDS was inversely correlated with RESTED (greater mood disturbance predicted less restedness after sleep). These findings would appear to make clinical sense. Greater ZDS score was, however, also modestly associated with greater reduction in pre-sleep anxiety (REPTH).

In summary, none of the demographic or clinical variables were good outcome predictors. Affective measures were consistently related to qualitative report at baseline but did not predict sleep pattern. The relationship between drug history and sleep was indeterminate. Most sleep pattern variables were not associated with any predictor variable.

The presence or absence of ongoing drug therapy was also considered as a potential predictor. The methodology chapter described the programmes for drug withdrawal. Fifteen patients were maintained on medication throughout the experimental period, with the remainder being finally drug-free (11 after structured pre-treatment withdrawal and many others after less formal but supervised withdrawal). A categorical analysis was conducted involving chi-square testing via the CROSSTABS procedure for the variable sex, and independent T-TESTs with drug status as the grouping factor for the remaining demographic and all of the sleep

diary variables. No significant between group differences emerged on any baseline sleep parameter or gain score at post-treatment. Similarly, drug status was not significantly different across sex, age and duration of insomnia [results available in Appendix I, Table (xvi)]. These results indicate that subjects who remained upon hypnotics had sleep difficulties and treatment responses similar to drug-free patients.

Although drug status has not proven to be a predictor variable, it would be appropriate to present data on the management of drug-using patients at this point.

THE DRUG WITHDRAWAL PROGRAMME

Twenty-six patients participated in a formal programme of withdrawal; 11 pre-treatment and 15 post-treatment. Unfortunately complete data are not available for every case. Five cases of pre and five cases of post-treatment withdrawal will, therefore, be presented to demonstrate the two models (Espie, Lindsay and Brooks, 1987).

Allocation to withdrawal programme was based solely upon compliance. In practice, subjects unwilling or unable to cease medication were allocated to the post-therapy regime. Description of each subject along with SOL mean data across experimental phases are presented in Tables 19 and 20. Inspection of the Tables confirms that these 10 patients were similar to the total study sample in terms of sex and age distribution, and mean duration of insomnia. Baseline SOL was substantially higher for this sample of post compared with pre-treatment withdrawal subjects, but scores for these patients as a whole fall comfortably within the range for the main study population.

Pre-treatment Drug Withdrawal

Patients in the pre-therapy group had a mean baseline SOL of 64 minutes which increased by greater than 50% during the first two withdrawal weeks. Thereafter, there was a rapid and consistent return to baseline levels. Psychological therapy produced an average 47 minute reduction in SOL, and patients completed therapy taking an average of 25 minutes to fall asleep. Improvements were maintained at both F1 (6 months) and F2 (mean 13 months). These summary statements appear to be fairly accurate also for individual responses.

Statistical analyses were conducted, firstly, by means of the SPSSX RELIABILITIES procedure to generate a Friedman's non-parametric ANOVA across the time points from baseline to the final withdrawal week. This approach computes the Chi-square statistic and revealed a near significant overall effect [Chi-square = 9.43; df = 5; $p = .090$]. WILCOXON tests for paired samples were then applied to test for significant differences between baseline and scores at each withdrawal week. The only significant effect was at Week 2 where SOL was significantly higher than at baseline ($z = -2.02$; $p = .043$). This confirms that there was a significant rebound effect during the second week, but not thereafter. Comparison between pre and post-treatment scores revealed significant SOL reduction (WILCOXON $z = -2.02$; $p < .05$). No significant relapse was found between post-therapy and either of the follow-up weeks. It should be noted, however, that subject 2 had resumed medication at F2.

Post-treatment Drug Withdrawal

The post-treatment group received psychological intervention prior to drug withdrawal. Therapy was associated with an approximate 50% reduction in SOL which just failed to reach significance at the 5%

Table 19

| Case | Sex | Age | Insomnia Duration | Drug (dose) | Drug History | Base B1 | W1 | Drug Withdrawal W2 | W3 | W4 | W5 | Pre-T B2 | Post-T T | Follow Up F1 | F2 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | F | 53 | 22 | Temazepam (80mg) | 20 | 61 | 121 | 103 | 58 | 99 | 69 | 103 | 21 | 19 | 21 |
| 2 | M | 48 | 13 | Flurazepam (30mg) | 7 | 59 | 51 | 108 | 79 | 54 | 52 | 59 | 34 | 34 | 60 + |
| 3 | M | 41 | 8 | Triazolam (0.25mg) | 4 | 98 | 120 | 135 | 71 | 47 | 62 | 58 | 8 | 9 | 8 |
| 4 | F | 66 | 5 | Nitrazepam (20mg) | 5 | 39 | 32 | 76 | 46 | 35 | 45 | 45 | 19 | 13 | 20 |
| 5 | F | 54 | 6 | Temazepam (30mg) | 6 | 64 | 155 | 107 | 90 | 90 | 94 | 94 | 43 | 61 | 63 |
| Mean | | 52.3 | 10.8 | | 8.5 | 64 | 96 | 106 | 69 | 65 | 64 | 72 | 25 | 27 | 34 |

Table 20

| | | | | | | Pre-T B | Post-T T | W1 | Drug Withdrawal W2 | W3 | W4 | W5 | Follow Up F1 | F2 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 6 | F | 68 | 18 | Temazepam (40mg) | 15 | 90 | 39 | 219 | 118 | 195 | 49 | 60 | - | 91 + |
| 7 | M | 51 | 8 | Triazolam (0.25mg) | 8 | 85 | 15 | 48 | 60 | 8 | 6 | 15 | 7 | 88 + |
| 8 | F | 46 | 11 | Triazolam (0.50) | 9 | 179 | 96 | 315 | 480 | 385 | 320 | 120 | 60 + | 73 + |
| 9 | F | 45 | 5 | Temazepam (60mg) | 5 | 86 | 60 | 141 | 124 | 90 | 90 | 84 | 72 | 49 |
| 10 | F | 49 | 14 | Lormetazepam (2mg) | 10 | 33 | 34 | 60 | 51 | 107 | 48 | 42 | 25 | 34 |
| Mean | | 51.8 | 9.6 | | 9.4 | 95 | 49 | 157 | 167 | 157 | 103 | 64 | 41 | 67 |

TABLES 19 and 20 Clinical characteristics and sleep latency scores of the five patients following the pre-treatment (Table 19) and the five patients following the post-treatment (Table 20) withdrawal programmes Treatment was by relaxation (cases 1,8,9), stimulus control (cases 2,4,6,7) and paradox (cases 3,5,10)
+ patients resuming medication

level ($z = -1.75$; $p = .079$). Since the Wilcoxon test is a signed-rank procedure it is likely that the lack of improvement in one subject (case 10) was responsible for this result. Nevertheless, substantial improvements were recorded for the other patients. Friedman's ANOVA from post-treatment across drug withdrawal produced a significant overall effect [Chi-square = 13.28; df = 5; $p = .021$]. Inspection of the group mean data suggested that rebound effects were present during the first four weeks of withdrawal, with a return to near baseline at Week 5. WILCOXON tests were applied to identify the loci of significant effect. Significant increments in SOL were found at Weeks 1 and 2 (in both cases $z = -2.02$; $p = .043$), with lesser effects evident at Weeks 3 and 4 (in both cases $z = -1.75$; $p < .08$) and at Week 5 ($z = -1.83$; $p < .07$). The result for Week 5 is surprising since mean SOL was only 15 minutes greater than the post-treatment value. The Wilcoxon test, however, does not take into account the degree of difference between measures, and since four of the five W5 scores were higher than post-treatment, an inflated z score was perhaps obtained. Statistical analyses were not conducted at follow-up because of missing data in one case, but more importantly, the resumption of medication in three cases at F2.

The results from both withdrawal programmes are encouraging. Effective pre-treatment withdrawal was supplemented by psychological therapy to establish an improved sleep pattern, and the five cases presented suggest that treatment benefits were largely retained for this group. Those who were unable to stop hypnotics initially presented with lengthier sleep latency difficulties and experienced rebound SOL effects for a longer period. The type and doseage of drug taken, however, did not appear to discriminate between groups. Psychological treatment enabled these patients successfully to stop medication but in three cases relapse occurred and medication was resumed within

approximately 1 year. It is noteworthy that SOL scores at the final withdrawal week, for the latter group, were higher than baseline sleep problems for the pre-treatment withdrawal group. Greater clinical problems may be expected, therefore, when patients experience protracted withdrawal reactions. Booster therapy may be one option to maintain treatment effectiveness for this group. The implications of these drug-management models will be discussed later.

# CHAPTER 10

## AN EVALUATION OF TAILORED THERAPY

This chapter presents, firstly, information on the assessment and treatment of the 14 consecutively allocated patients in the tailored therapy condition; and secondly, evaluation of this approach compared with the randomly allocated treatment groups.

### THE DEVELOPMENT OF THE SLEEP DISTURBANCE QUESTIONNAIRE

On the basis of individual presenting characteristics of sleep pattern, bedtime habits, and personal coping style, an attempt was made to predict which form of intervention would be most appropriate. To formalise clinical judgement in this matter, a Sleep Disturbance Questionnaire (SDQ) was compiled as a brief, structured interview assessment of potential contributory factors. Three items were selected from clinical experience to assess each of the areas of physical tension, sleep incompatible behaviour, anxious "effort to sleep", and general cognitive intrusion. Patients were allocated to relaxation training, stimulus control, paradoxical intention, cognitive restructuring therapy (Beck and Emery, 1979) or a combination of these procedures, depending upon the profile of scores obtained on the SDQ. The cognitive therapy option was included since it had been the author's experience that problems with mental control and reasoning were frequent insomniac complaints. Each of the 12 statements in the questionnaire was rated on a 5-point scale from "never true" to "very often true" to indicate how characteristic the item was of the subject's typical sleep pattern. The maximum subscale score was 15, and the total score maximum was 60. In order to clarify further the allocation to treatment, patients were asked to select the single item which was most relevant to their sleep problem and to comment on any

other factors which were not included in the assessment (The SDQ is presented in Appendix J). The SDQ was, therefore, primarily a clinical tool to aid the consistent appraisal of aetiological factors.

Completed questionnaires were available from a group of 42 insomniacs (11 male, 31 female), comprising both those in the tailored therapy group and other patients attending their GP for management of insomnia. The mean age of the sample was 46.8 years (SD 18.0). Comparison with demographic data for the main study suggested that this was a representative group. The most commonly selected single items were Item 5 "I find it hard to let go and relax" (26%), Items 2 "My mind keeps turning things over" and 3 "I can't get my sleep into a proper routine" (each 17%), and Item 10 "I am unable to empty my mind" (14%). These four items, therefore, accounted for 74% of all choices.

In order to investigate the presumed factor structure of the SDQ, the data were submitted to FACTOR ANALYSIS (FA) using Principal Components Analysis and Varimax rotation. In spite of the relatively small sample size (n = 42) the Kaiser-Meyer-Olkin measure of sampling adequacy (MSA) (see Kaiser and Rice, 1974) yielded a value of 0.701 which is greater than the critical value of 0.500. Thus the correlation matrix was acceptable for factor-analytic purposes according to the MSA approach.

Three factors emerged from FA satisfying the SPSSX default criterion of having eigenvalues greater than or equal to 1.000. These factors, together accounting for 68.3% of total variance (Table 21) were

| Factor | Eigenvalue | % Variance | Cumulative % |
|---|---|---|---|
| 1 | 4.780 | 39.7 | 39.7 |
| 2 | 2.162 | 18.0 | 57.7 |
| 3 | 1.278 | 10.7 | 68.3 |

TABLE 21 Factors extracted from the SDQ under Principal Components Analysis

accepted for further analysis. Inspection of the complete factor extraction table [Appendix K, Table (xvii)] indicates that the remaining factors not only had eigenvalues less than 1.000 but each also explained less than 10% of total variance. Thus the decision to restrict extraction to three factors was confirmed.

The rotated factor matrix is presented in Table 22. Given the relatively small n of cases, a correlation of 0.50 was selected as a minimum criterion for significant factor loading. It was hoped that this would be a reasonably conservative procedure.

| | Factor 1 | Factor 2 | Factor 3 |
|---|---|---|---|
| Item 2 | .90537* | .09700 | -.01654 |
| Item 10 | .88744* | .03683 | .07730 |
| Item 6 | .84749* | .18342 | -.10656 |
| Item 12 | .73933* | -.44722 | -.03357 |
| Item 5 | .69872* | .05278 | .55866* |
| Item 4 | .63777* | .11913 | .27770 |
| Item 8 | .56147* | .45514 | .17058 |
| Item 7 | .07275 | .81256* | -.25665 |
| Item 3 | .02013 | .75622* | .22923 |
| Item 11 | .07772 | .52823* | .26732 |
| Item 1 | -.11789 | .32289 | .78737* |
| Item 9 | .57879* | -.15500 | .63337* |

TABLE 22 Rotated factor matrix showing SDQ item correlations and loading on each of the three principal factors. * Items loading r ≥ .50

Inspection of Table 22, with reference to the SDQ (Appendix J) reveals that Factor 1 accounted for more than one-third of total variance. It can be regarded as a "mental anxiety" factor, with all of the cognitive restructuring items (2,6,10) and paradoxical items (4,8,12) loading at

highly significant levels. Interestingly, Items 5 and 9 loaded significantly on both Factors 1 and 3. The likely explanation for this in the case of Item 5 is that ambiguity of wording ("I find it hard to let go and relax") permitted subjects to construe it either in terms of mental or physical relaxation. There is, however, no clear explanation for the significant correlation of Item 9 with Factor 1. The remaining items had minimal loadings on Factor 1 (all $p < .12$).

Items 1 and 9, along with Item 5, loaded significantly upon Factor 3 yielding a relatively "pure" factor of "physical tension". The highest of the nine other coefficients for Factor 2 was only 0.28. The wording of Item 5 has now been amended to read "I find it hard to let go and relax my body", to clarify its intended emphasis upon physical relaxation.

Factor 2 may be described as "sleep pattern problem", since all three stimulus control items (3,7,11) loaded significantly. No other items reached the criterion level of 0.50 on this factor. Items 1, 8 and 12, however, merit comment having coefficients within the range 0.30 to 0.50. It is perhaps not surprising that difficulties in attaining a "comfortable position in bed" (Item 1) should be positively associated with the stimulus control model. Being comfortable in bed could be readily regarded as a state discriminative of rapid sleep-onset. The combination of effort to sleep (Item 8) and the absence of worry about the consequences of sleep loss (inverse of Item 12) may also be in keeping with a "sleep pattern problem". The emphasis underlying Factor 2 may be that of a resistant to change habit disorder ie. behavioural pattern problem, but without concomitant affective disturbance.

The results of FA, therefore, broadly confirm the clinical judgements which were based upon the SDQ. Relaxation and stimulus control items loaded as expected on separate factors, but a third "cognitive" factor

comprised all items referring to both performance anxiety and dysfunctional thought. These findings suggest that a Three Systems Model with behavioural, physiological and cognitive elements, may be appropriate in the investigation of aetiological and maintaining factors.

PROCEDURE

Using the SDQ to allocate the 14 patients, 4 received relaxation, 4 stimulus control, 2 paradoxical intention and 4 cognitive restructuring as the principal intervention. In some cases (where appropriate and indicated by the SDQ) subjects also received instruction based upon elements of the other procedures. This was in order to approximate closely what would normally happen in clinical practice. Therefore, and for example, "cognitive restructuring" at times comprised elements of paradoxical intention , stimulus control or relaxation training where the score on another sub-scale of the SDQ was noticably elevated. The experimental phases for the tailored group were identical to those in the design of the main study. Patients were required to complete the same process and outcome measures, and they received the same pattern of out-patient appointments as did subjects in the main study.

ASSESSMENT OF TREATMENT CREDIBILITY

The Credibility Evaluation Questionnaire (CEQ) was administered to patients in the tailored therapy condition in order to permit comparison with the main group study. Eleven out of the 14 subjects completed the CEQ and the mean score obtained was 25.7 (SD 3.95). Comparison with scores for the randomly allocated groups [Appendix H, Table (iii)] suggests little difference in patients' perception of credibility. ONEWAY ANOVA across the five treatment groups (3 randomised active treatments, placebo control, and tailored

therapy) confirmed this result [F = 0.159; df(4,50); p = 0.958].

## THE EFFECTIVENESS OF TAILORED THERAPY

No specific predictions regarding the comparative effectiveness of tailored and untailored treatment were made. Clinical practice frequently assumes the superior effectiveness of individualised programmes, however, this assumption was unsupported by Turner et al's (1984) unsuccessful attempt to maximise treatment gain by combining relaxation and stimulus control instructions. Lichstein (personal communication, 1986) has also reported limited gain in the use of tailored treatments for insomnia.

### Descriptive Analysis

In evaluating outcome, the comparison of greatest interest was that between the tailored group and the three active treatments (combined) ie. tailored psychological treatment versus randomly assigned psychological treatment. Table 23 presents a descriptive summary of DSQ variables for this comparison, incorporating pre and post-treatment values and percentage change scores. Placebo and no treatment scores are also presented.

Inspection of the Table indicates that, for both SOL variables, post-treatment change was greater in the random therapy group compared with the tailored group. Indeed, the tailored condition demonstrated a response more similar to placebo on these variables. TOT mean and TOT SD revealed modest treatment effects for both tailored and untailored groups and these effects were similar to placebo. The pre-treatment mean value for TOT in the tailored group was noticeably lower than in any of the other conditions. Tailoring was associated with reductions approaching 50% on WAKE mean and WAKE SD compared with around 40% in the random treatment group, but in all conditions wakening was

infrequent at baseline.

| (n = | | Random Therapy 43 | Tailored Therapy 14 | Placebo 14 | No Treatment 13 ) |
|---|---|---|---|---|---|
| SOL mean | pre | 81.7 | 85.3 | 85.5 | 84.5 |
| | post | 42.1 | 55.8 | 63.5 | 96.5 |
| | %ch | -48.5 | -34.6 | -25.7 | +14.2 |
| SOL SD | pre | 46.6 | 57.0 | 44.2 | 53.6 |
| | post | 24.0 | 40.7 | 30.6 | 63.5 |
| | %ch | -48.5 | -28.6 | -30.8 | +18.5 |
| TOT mean | pre | 5.92 | 4.50 | 5.34 | 5.84 |
| | post | 6.53 | 5.17 | 5.82 | 5.84 |
| | %ch | +10.3 | +14.9 | + 9.0 | - |
| TOT SD | pre | 1.30 | 1.32 | 1.23 | 1.25 |
| | post | 1.06 | 0.98 | 0.94 | 1.28 |
| | %ch | -18.5 | -25.8 | -23.6 | + 2.4 |
| WAKE mean | pre | 1.22 | 1.53 | 1.76 | 1.01 |
| | post | 0.75 | 0.77 | 1.34 | 0.85 |
| | %ch | -38.5 | -49.7 | -23.9 | -15.8 |
| WAKE SD | pre | 0.82 | 1.12 | 0.95 | 0.68 |
| | post | 0.52 | 0.61 | 0.94 | 1.41 |
| | %ch | -36.6 | -45.5 | - 1.5 | - 7.4 |
| REPTH | pre | 1.41 | 1.56 | 1.92 | 1.64 |
| | post | 1.11 | 1.00 | 1.90 | 1.41 |
| | %ch | -21.1 | -35.9 | - 1.0 | -14.0 |
| RESTED | pre | 1.77 | 1.66 | 1.70 | 1.93 |
| | post | 2.23 | 1.69 | 2.01 | 1.87 |
| | %ch | +26.0 | + 1.8 | +18.2 | - 3.1 |
| ENJOY | pre | 1.90 | 1.76 | 1.71 | 2.06 |
| | post | 2.33 | 1.97 | 2.12 | 2.16 |
| | %ch | +22.6 | +11.9 | +24.0 | + 4.9 |
| IRRIT | pre | 1.41 | 1.28 | 1.46 | 1.43 |
| | post | 1.12 | 1.04 | 1.14 | 1.18 |
| | %ch | -20.6 | -18.8 | -21.9 | -17.5 |
| NAPDAY | pre | 6.85 | 6.75 | 6.46 | 19.9 |
| | post | 4.44 | 0.00 | 3.82 | 16.5 |
| | %ch | -35.2 | - 100 | -40.7 | -17.1 |

TABLE 23 Comparison between randomly allocated and tailored treatments of mean scores during baseline (pre) and the final week of treatment (post) along with percentage change scores for each DSQ variable

Results for the sleep quality variables failed to demonstrate any general pattern of superiority. Tailored therapy produced greater reduction in REPTH and NAPDAY (though napping was a very minimal

problem), whereas for RESTED, ENJOY and IRRIT any superiority tended towards the random approach. Considerable changes after placebo treatment, on all but one of the sleep quality variables, further suggested that between group differences were at best modest.

There being little evidence, therefore, for the existence of important final outcome differences between tailored and randomised treatments it was felt unnecessary to conduct a complex repeated measures analysis. The following statistical analyses were, however, conducted.

## Statistical Analysis

### Baseline Measures

Examination of the data in Table 23 indicated that a significant between group difference might emerge at baseline on the variable TOT mean. In order to investigate this possibility, and to consider whether or not other variables might differ across groups, mean scores for each DSQ variable were submitted to ONEWAY ANOVA with treatment type (random, tailored, placebo, no treatent) as the grouping factor. Two of the 11 variables yielded significant F ratios. TOT mean was indeed associated with a highly significant effect [$F = 5.08$; $df(3,80)$; $p = .003$] which Scheffe testing revealed was accounted for by significant differences between tailored and random therapy ($p < .05$), and to a lesser extent between tailored therapy and no treatment ($p < .10$). In both cases tailored therapy subjects slept for shorter periods on average. The other significant baseline difference was for NAPDAY and was modest by comparison [$F = 2.72$; $df(3,80)$; $p = .050$]. No treatment subjects napped significantly more than random therapy subjects (Scheffe $p < .10$). The results of the other non-significant baseline analyses are available in Appendix K [Table (xviii)].

Treatment Outcome

In order to evaluate the comparative outcome of treatments ONEWAY ANOVAs across treatment type were conducted upon change scores (baseline value - score at final therapy week) for each DSQ variable. The results of these analyses are presented in Table 24. Inspection of the Table reveals that significant between treatment effects were achieved for only 3 of the 11 variables. Improvements in SOL mean were significantly greater in the randomised treatment group compared with no treatment and modest effects ($p < .10$) were also found for this same between group comparison on SOL SD and RESTED. Of greatest importance, however, was the failure to find any significant benefit for tailored therapy over any other condition. Tailored therapy was never superior to random therapy. Random therapy demonstrated superiority over no treatment on some variables but tailored treatment did not.

| Variable | df | F Ratio | F Prob | Pair | Scheffe .05 | Range .10 | Planned t .01 |
|---|---|---|---|---|---|---|---|
| SOL mean | (3,72) | 3.624 | 0.018 | 1 v 4 | * | * | * |
| SOL SD | (3,72) | 2.200 | 0.097 | 1 v 4 | | | * |
| TOT mean | (3,72) | 1.142 | 0.339 | - | | | |
| TOT SD | (3,72) | 0.501 | 0.679 | - | | | |
| WAKE mean | (3,72) | 0.873 | 0.460 | - | | | |
| WAKE SD | (3,72) | 1.715 | 0.173 | - | | | |
| REPTH | (3,73) | 0.643 | 0.581 | - | | | |
| RESTED | (3,73) | 2.436 | 0.073 | 1 v 4 | | | * |
| ENJOY | (3,72) | 0.685 | 0.564 | - | | | |
| IRRIT | (3,71) | 0.595 | 0.620 | - | | | |
| NAPDAY | (3,72) | 1.452 | 0.236 | - | | | |

TABLE 24 ANOVAs on change scores for each of the sleep diary variables across treatment types (Random therapy=1, Tailored therapy=2, Placebo=3, No treatment=4). Results of range tests are also presented

One final analysis was conducted to consider the impact of tailored therapy. A repeated measures ANOVA, within the tailored condition, was generated by means of the SPSSX RELIABILITIES procedure to provide data comparable with the time within treatment analyses conducted for the individual treatments in the main study (see Tables 12 and 14). The results of this analysis can be found in Table 25 [see Appendix K,

Table (xix) for DSQ data at each therapy week].

These results confirm the relatively limited impact of tailored therapy upon the sleep diary measures. Neither SOL mean nor SOL SD were significantly reduced from baseline values during the course of treatment, apart from a temporary reduction in sleep latency during Weeks 2 to 4. A modestly significant increase in TOT mean emerged at Week 2 and was maintained until the end of the treatment period. TOT SD, however, was unaffected over time by tailored treatment. Comparison of these results on sleep pattern with those obtained from relaxation, stimulus control and paradox (Table 12), suggests that the tailored approach was of limited value. Having already established that tailored therapy was not superior to the combined random therapy group, it is now evident that it compared even more unfavourably with the more powerful specific effects of individual treatments. Reductions in both WAKE and WAKE SD, however, were highly significant over time with tailored therapy and were at least comparable in magnitude with the treatment effects demonstrated in Table 12. Tailored treatment was the only treatment approach to reduce significantly both mean and SD scores for intermittent wakening.

| | Weeks of Therapy | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 |
| df | (1,13) | (2,26) | (3,39) | (4,52) | (5,65) | (6,78) | (7,91) | (8,104) |
| SOL mean | | * | ** | * | | | | |
| SOL SD | | | | | | | | |
| TOT mean | | * | * | * | * | * | * | * |
| TOT SD | | | | | | | | |
| WAKE mean | | | | * | * | ** | *** | *** |
| WAKE SD | | | * | ** | ** | *** | *** | *** |
| REPTH | * | ** | | | | | | |
| RESTED | | | | | | | | |
| ENJOY | | | | | | | | * |
| IRRIT | | | | | | | | |
| NAPDAY | * | | | | | | | |

TABLE 25 Repeated measures ANOVA providing time within treatment group sub-effects on each DSQ variable for the tailored therapy condition
* $p \leq .05$ ** $p \leq .01$ *** $p \leq .001$

In terms of sleep quality, tailored therapy yielded minimal change. Early reduction in REPTH was not maintained and significant effects at the 5% level occurred at single data points only, for the variables ENJOY and NAPDAY. Relaxation training, therefore, remains the most favoured intervention for measures of sleep quality (cf. Table 14).

In conclusion, tailored psychological therapy was not superior to randomly allocated psychological therapy. Between group testing only confirmed the benefits of the randomised group over no treatment; an effect not paralleled in the tailored therapy condition. Previous analyses have already explored the impact of the specific treatments which comprise the random group. Repeated measures analysis within the tailored condition revealed consistent improvements on only three measures of sleep pattern, namely TOT mean, WAKE mean and WAKE SD. Tailored therapy was, however, the only intervention to reduce both wakening frequency and variability of wakening. Sleep quality measures did not demonstrate any useful improvements over time.

BEFORE AND AFTER MEASURES

In consideration of the generalised changes associated with tailored therapy paired T-TEST comparisons were conducted from pre to post-treatment on the ZAS, ZDS, SBRS and ARS. The results of these analyses are presented in Table 26.

| Variable | pre | post | SE diff | T | df | Prob | Sig Level |
|---|---|---|---|---|---|---|---|
| ZAS | 36.0 | 32.3 | 1.27 | 2.89 | 10 | .020 | * |
| ZDS | 39.2 | 36.3 | 3.20 | 0.90 | 10 | .393 | NS |
| SBRS | 48.6 | 39.7 | 8.10 | 1.10 | 10 | .305 | NS |
| ARS | 52.3 | 46.6 | 3.95 | 1.45 | 10 | .186 | NS |
| ARS 9 | 1.70 | 5.34 | 1.06 | -3.45 | 10 | .009 | ** |

Table 26 Mean scores for pre and post-treatment rating scale assessments and results of paired T-TEST comparisons for the tailored therapy condition (* $p \leq .05$ ** $p \leq .01$)

Inspection of the Table reveals that tailored therapy was associated with a modest but significant reduction in anxiety level (ZAS) but had no substantial impact upon depressed mood (ZDS) or on generalised daytime functioning (ARS). The only ARS item score to improve significantly was ARS 9 "sleeping well". Tailored therapy subjects, therefore, regarded sleep as having improved on this global measure. The results of the remaining, non-significant results on ARS items are available in Appendix K [Table (xx)]. At post-treatment, scores on the SBRS had reduced by approximately 9 points on average. However, the variability of response across subjects is evident from the relatively high SE of difference between means, rendering the T-TEST comparison non-significant.

Comparison of Table 26 with Table 16 (Chapter 9) indicates that tailored therapy produced less change on pre-post assessments than any of the three other active interventions. Indeed, even placebo control was associated with significant reductions in state anxiety and with improved rating of sleep. ONEWAY ANOVAs on change scores across the six experimental conditions (5 from main study plus tailored therapy) were also conducted. The only significant effect to emerge, in addition to the between group effects reported in the previous chapter (Table 17), was for ARS 9 where Scheffe testing revealed a significant difference between tailored therapy and no treatment ($p < .05$) [$F = 6.366$; df (5,66); $p = 0.006$]. Clearly there were no particular advantages conferred upon the pre-post measures by the use of the tailoring procedure.

# CHAPTER 11

# DISCUSSION

Discussion of the results of this study will be organised around a number of important themes, and each theme will form a section of this chapter. The areas of interest are as follows:-

1. The study population
2. Psychological treatments and their effects
3. The issue of clinical versus statistical significance
4. Some important methodological issues
5. Recommendations for clinical practice
6. Suggestions for further investigation

## THE STUDY POPULATION

### General Characteristics

One of the principal criticisms levelled against previous studies was their failure to investigate treatment effects within clinically presenting groups of insomniacs (Chapter 6). Tables 1-4 clearly demonstrated that subjects had been recruited largely via advertisements, thereby imposing restrictions upon the generalizability of results. An essential part of the selection process in this study was that each subject was referred by a GP through normal clinical channels. Results are, therefore, directly applicable to clinical practice.

The final sample of 84 patients had an average age of 45.5 years and had experienced severe insomnia for a mean of 11.8 years. Both in terms of age and chronicity, therefore, these subjects differed from the great majority in previous reports who have tended to be younger and have had more recent problems with sleep. Certainly, this study comprises the largest sample of chronic insomniacs ever investigated in the psychological treatment literature.

Average SOL at baseline also reflected the more clinical presentation of insomnia in the present study. Patients took an average of around 83 minutes to fall asleep, by comparison with which, those past reports which have compared outcome across different psychological treatments (Table 4) have generally described sleep latencies some 15-45 minutes shorter on average. Only one (unpublished) study has reported on similarly lengthy SOL (Bootzin, 1975).

Only 14 patients in the final sample of 84 (17%) had never been users of hypnotic medication. The group as a whole, therefore, were typical of the insomniac out-patient poulation in making regular use of sleeping tablets. The mean period since first prescription was 9 years, and 26 subjects (31% of total) required a formal programme of drug withdrawal due to long-term dependence. Treatment effects were investigated, therefore, within a group who posed considerable management problems in general practice.

It would be of interest to compare insomniacs with other attenders at psychology clinics in primary care settings. A number of workers have found that the typical psychology caseload comprises mainly patients with anxiety/stress disorders or interpersonal problems (Koch, 1979; Jerrom, Simpson, Barber and Pemberton, 1982; Espie and White, 1986a). The Espie and White study presented an analysis of 1165 GP referrals to the clinical psychology service in Lanarkshire. Two-thirds of these cases were female, and the mean age for the total sample was 37.4 years. The sex distribution of the insomniac group in the present study was very similar to that of this large reference group; 68% being female and 32% male. Interestingly, however, insomniacs appeared to be older by an average of around 8 years (mean 45.5 years - see Table 5). This age difference was accounted for by the relative paucity of very young adults along with the much higher proportion of elderly

subjects in the insomniac group. Espie and White reported that only 8% of the total out-patient group were aged 60 or over; this compares with over 20% in the present study. It is, of course, in keeping with the literature on prevalence rates to find that insomnia was more strongly associated with the older agegroups who experience shorter and more disturbed sleep than younger adults [see Chapter 1, and McGhie and Russell (1962) for information on the local population]. Part of the normal change in sleep pattern with age is an increased frequency of night-time wakening. The typical patient in this study experienced troublesome wakenings at the rate of approximately 9 per week (mean of around 1.3 per night - see Table 9). Unfortunately it is not possible to compare this figure with other studies, which have defined differently the criteria for including wakenings/arousals. Those few psychological treatment studies which have reported on intermittent insomnia have employed WASO (wake time after sleep onset - total minutes awake during the night) as the dependent variable. These data were not collected on the DSQ in this study (methodological issues concerning the sleep diary will be dealt with later).

Psychological Disturbance

Insomniacs have been characterised as mildly depressed worriers (Chapter 3). It was the author's clinical impression that some patients suffered from anxiety and/or mild depressive symptoms. Clinically depressed subjects were, of course, excluded. The average score on the ZAS was around 38, and on the ZDS was 41. Although no formal comparison with anxious and depressed patients, respectively, has yet been conducted, reference to the original papers may be useful.

Zung (1971) reported that normal control subjects obtained an average ZAS score of 27, whereas an "anxiety disorder" group had a mean score of 47. The insomniac group's average score of 38 is, therefore,

intermediate and coincides with the point on Zung's distribution which is 1 SD below the mean for the anxious group. The author has been involved in treatment studies of anxious patients in the local population. Lindsay, Gamsu, McLaughlin, Hood and Espie (1987) found that pre-treatment ZAS scores for a group of 40 patients with generalised anxiety was around 52. It would seem reasonable to conclude, therefore, that the insomniac group experienced anxiety levels in the mild to moderate range.

As far as ZDS scores are concerned, Zung (1965) reported that a control group of 100 subjects scored 26 on average, whereas a depressed group of 56 patients had a mean score of 59 before treatment. The insomniacs in this study presented with a mean ZDS score of 41, which once again is intermediate between the normal control and depressed populations. It is also of interest to note that Espie (1986) reported an average ZDS score of 52 at pre-treatment in a small group of obsessive-compulsive patients who were habitual relapsers. Clearly, the subjects in this study were not suffering from major depressive disorder but, consistent with previous studies, they were mildly depressed relative to the general population norm.

Selection/Attrition Issues

The 84 included subjects represented 60% of the 141 referrals received. Detailed explanation concerning the loss of subjects was provided in Chapter 7. In summary, half of the subject loss was due to the operation of strict selection criteria, and half due to subjects dropping out. Of the "drop-outs", however, considerably more than one-third failed to attend even the first appointment. The true "drop-out" rate amongst assessed and suitable subjects was only 17 out of 101 patients. This attrition rate is at least comparable to clinical research studies in any field of application. As previously stated,

"drop-outs" were evenly distributed across the experimental conditions. It is particularly noteworthy that waiting-list subjects were prepared to monitor and record sleep pattern for fully 10 weeks before receiving help. Indeed, a number of individuals regarded the exercise as helpful and informative.

In conclusion, the goal of selecting-in a representative sample of the clinically presenting chronic insomniac population was achieved. It is, therefore, possible to discuss the results obtained in such a manner as to be directly applicable to routine clinical practice.

## PSYCHOLOGICAL TREATMENTS - THEIR EFFECTS UPON SLEEP PATTERN AND QUALITY

In this section, the effects of the various psychological treatments upon DSQ variables, as presented in Chapters 8 and 10, will be discussed in some detail. Treatment conditions will be dealt with separately under sub-headings and a summary/conclusion will be provided for each form of intervention.

### Relaxation Training

The literature on insomnia, reviewed in Chapter 5, revealed that relaxation approaches have been the most commonly reported, partly because relaxation preceded historically other forms of management. Relaxation has generally had a positive impact upon initial insomnia and it was concluded in the literature review that there was little to differentiate amongst the range of techniques available. In this study, perhaps the best known form of abbreviated progressive relaxation (Bernstein and Borkovec, 1973) was used.

Subjects treated with relaxation reduced initial SOL mean by around one-third on average, but completed therapy still taking approximately one hour to fall asleep; an outcome similar to placebo treatment (Table

9). Superior outcome on the SOL variable was demonstrated, however, by both stimulus control and paradox, which achieved final week SOL scores of 31 and 36 minutes respectively. Table 12 revealed that the within group repeated measures effect was nevertheless significant for relaxation (unlike placebo treatment). Treatment effects were more consistent over time with the stimulus control and paradoxical approaches. Statistical comparison of these between group effects was presented in Table 11, which revealed that in spite of the apparent advantages over relaxation of the stimulus control and paradoxical treatments, between group differences on SOL did not generally achieve levels of significance. The sole exception to this was at Week 1 where stimulus control was superior to relaxation (Scheffe $p < .10$). SOL for the waiting-list group increased by 14% at post-treatment which contributed to the superiority of relaxation over no treatment during the final therapy week (Scheffe $p < .05$).

In terms of sleep-onset variability from night to night (SOL SD), reductions under relaxation therapy (around 25%) were once again modest when compared with the two other active interventions (stimulus control 55%; paradox 70% ; Table 9). Placebo also produced changes of slightly greater magnitude than relaxation. The advantages of relaxation appear limited, therefore, to comparisons with the no treatment control group. Statistical evaluations of between group comparisons revealed the modestly significant advantages of relaxation over waiting-list at Weeks 2, 4 and 6 (Table 11), but Table 12 revealed that the untreated group deteriorated significantly at Weeks 2 and 4 which perhaps accounts for these between group effects.

As far as sleep latency is concerned, therefore, the impact of relaxation training, was comparatively limited. Improvements over time did emerge but were somewhat erratic from week to week. Changes with

relaxation were more similar in magnitude to placebo and both stimulus control and paradoxical intention appear preferable although between group effects were not statistically significant. Fluctuations towards deterioration in the untreated control group largely accounted for the apparent benefits of relaxation over waiting-list.

For total sleep time (TOT mean), relaxation was associated with an 11% increment and patients completed therapy obtaining 6.41 hours on average per night (Table 9). This outcome was comparable with each of the other treatment conditions including placebo. Within treatment analysis (Table 12) indicated that significant changes with relaxation were concentrated during the final 3 weeks of therapy, indicating that relaxation took some time to increase TOT. Comparison on this form of analysis with the other experimental conditions, however, revealed that improvements by other means were equally limited. Treatment x time interaction failed to reach significance for TOT; a finding which is supportive of the view that changes were limited and similar across treatments.

The significant interaction main effect for TOT SD was accounted for largely by superiority of relaxation over no treatment. Significant effects were demonstrated at Weeks 1, 2 and 6 (Table 11). The strength of the sub-effect at Week 6 was, however, much reduced compared with the earlier weeks and Table 9 revealed that the magnitude of reduction at final outcome was only around 6% with relaxation training. Treatment effects were, therefore, extremely short-lived. Interestingly and by comparison there was a marked reduction with paradox during Week 8 (Table 12), accounting for the 41% pre-post change score (Table 9).

As regards measures of sleep duration, therefore, relaxation-induced changes were modest. An initial reduction in the variability of the nightly sleep period was not paralleled by gains in TOT. Latterly,

however, the sleep period did increase by approximately 40 minutes. Comparisons with other experimental conditions are suggestive of marginally greater overall benefit under relaxation although differences are likely to be clinically negligible.

Relaxation was associated with reductions in WAKE mean and WAKE SD of a little more than 20% (Table 9). These changes were considerably less than those for stimulus control and paradox. Within treatment simple effects failed to reach significance for the relaxation group at the .05 level on both variables. WAKE SD, however, did achieve an F probability < .10 reflecting a modest reduction in variability of wakening during the middle weeks of treatment only (Table 12). Stronger within group effects were evident for paradox (WAKE) and stimulus control (WAKE SD). It can be concluded that relaxation did not produce any useful impact upon measures of intermittent wakening. The other psychological treatments were somewhat more effective although treatment x time interactions for both WAKE mean and WAKE SD were far from significant ($p > .40$; Table 10).

In spite of the above, relatively limited, effects of relaxation upon sleep pattern, relaxation did prove to be the best intervention for effecting change in the sleep quality variables (Table 14). The treatment group main effect for REPTH was accounted for by a modestly significant difference between relaxation and placebo. This result indicated that relaxation was associated with a greater average reduction in pre-sleep anxiety. However, neither treatment x time nor time main effects were significant for REPTH.

Highly significant time main effects for RESTED and ENJOY ($p < .001$; Table 13) were largely accounted for by significant improvements over time with relaxation training (Table 14). Increments in these measures of sleep satisfaction were observed from Week 2 onwards. Stimulus

control was not associated with any significant improvement on these variables, and paradox achieved significant change only upon RESTED, and only during the final three treatment weeks. Interaction main effects (Table 13) were again, however, non-significant, indicating that between group differences were not of sufficient magnitude to obtain statistically significant results. Relaxation did not significantly affect reports of daytime irritability or daytime napping.

Conclusions Regarding Relaxation Training

Compared with other active treatments, changes in sleep latency under relaxation were modest. Limited changes also in total sleep duration and wakening frequency suggest that relaxation did not have a dramatic impact upon presenting sleep pattern. Nevertheless, patients were more likely to be satisfied with their sleep when relaxation techniques were applied. Relaxation appears to be the best treatment for modifying subjective perceptions of sleep quality. There are a number of possible explanations for the "mechanism" of effect here. Subjects may have become less anxious about their disordered sleep and learnt to value a state of relaxation even if not sleeping. Alternatively, relaxation training may have enabled patients to "get more out" of the sleep which they obtained, either objectively [eg. greater proportion of SWS (deep sleep)] or subjectively (feeling more refreshed). Another explanation might lie in increased coping during the daytime which, in turn, might have fostered more positive evaluation of sleep. These possibilities are worthy of further investigation.

The psychological treatment literature on relaxation has generally reported its high levels of face validity. Patients with psychological problems often confess to "difficulties in relaxing" and the training procedures are aimed directly at modifying this complaint. In addition,

it is possible that the demand characteristics associated with relaxation are somewhat different to, for example, stimulus control. The latter technique, comprising strict behavioural instructions, perhaps offers little which might be regarded as "therapeutic" or "supportive". Relaxation by contrast has more general application, and it may be implicit in the process of therapy that subjective anxieties and concerns are likely to alter. Interestingly, paradoxical intention, which can also be regarded as an anxiety-reduction technique, did produce some benefits in terms of "restedness", although to a lesser degree than relaxation training. It is an interesting possibility, therefore, that relaxation techniques may have a greater capacity to reduce "complaining behaviour" even though other psychological methods are superior in improving actual sleep pattern. This is a matter to which reference will be made at a later point.

Stimulus Control

Past research has marginally favoured stimulus control over other behavioural treatments. In the present study this treatment emerged without doubt as the best approach for reducing SOL mean scores. Stimulus control was associated with a 62% reduction in sleep latency, with patients completing therapy requiring approximately 31 minutes to fall asleep. This improvement represented an absolute reduction of 51 minutes, which was substantially greater than with any other condition (Table 9). Inspection of Table 12 revealed that dramatic reduction in SOL was an immediate phenomenon and was maintained from Week 1 until the end of treatment. Indeed, of all the variables investigated under any treatment condition, changes in SOL by means of stimulus control were the most statistically powerful. The highly significant interaction effect reported in Table 10 referred, almost exclusively, to the consistent superiority over time of stimulus control relative to

other forms of intervention (Table 11). In particular, stimulus control emerged as superior to no treatment throughout the experimental period. Superiority over placebo control, and also over paradox presented strongly at first, although this waned during the middle weeks of therapy, and was absent from Week 6 onwards. The overall treatment group main effect confirmed that the major differences were between stimulus control and no treatment (Scheffe $p < .05$), and between stimulus control and placebo (Scheffe $p < .10$). There is no clear reason for the non-significant between group effect at Week 4 (Table 11).

It is evident from the results on SOL that stimulus control represents by far the most cost-effective approach to reducing sleep latency. Although 8 weeks of treatment were provided there was little additive effect on this variable from the second week onwards. By comparison, although substantial changes emerged with paradoxical intention these were not significant until Week 4.

Reductions in SOL SD were also associated with stimulus control treatment. Changes from pre to post-treatment approached 55% (Table 9), greater than any treatment other than paradox. Improvements over time on SOL SD emerged at Week 1 for the stimulus control group although strongly significant effects were obtained only at Week 3 and from Week 6 onwards (Table 12). Consideration of between treatment effects revealed that stimulus control was superior to waiting-list at various points from Week 2. The magnitude of effect, however, was generally less than that of paradox versus no treatment (Table 11).

The stimulus control instruction to refrain from going to bed until "sleepy tired" may be, however, a virtual guarantee of reduced SOL. Assuming that patients generally stayed up later rather than went to bed earlier, they would be automatically excluding from estimates of

SOL, that period of time which extended beyond "usual bedtime" at baseline. In other words patients may have been falling asleep quicker simply because they were going to bed later, at which time they were more tired. Measures of sleep latency should, therefore, be investigated in parallel with measures of the duration of the sleep period.

Inspection of Table 12 revealed that increments in TOT mean were not achieved under stimulus control until the final two weeks of therapy. Indeed Fig 6 indicated that there was an average tendency towards reduction in total sleep especially during Weeks 3 and 4, and not until Week 5 did TOT demonstrate any marked improvement over baseline. It seems possible, therefore, that the mechanism of action of stimulus control involves initial and dramatic SOL reduction in association with restriction of sleep duration. Thereafter overall sleep pattern change does develop. It may be partly through this element of sleep "deprivation" that the sleep pattern later develops a strong and predictable rhythm. The immediate effectiveness of stimulus control in terms of SOL is, therefore, limited by latent impact upon sleep time. It seems likely that patients would require to remain in therapy until these effects are demonstrated, rather than simply going by reports of SOL.

According to Table 9, stimulus control was associated with a greater than 50% reduction in WAKE mean and a reduction of 76% in WAKE SD. In spite of these apparently large improvements, however, within treatment effects (Table 12) failed to demonstrate any significant repeated measures effect for WAKE mean, and the significant result ($p = .03$) for WAKE SD was accounted for mainly in the highly significant reduction at Week 8. These results also may be suggestive of the later readjustment of sleep pattern after perseverance with stimulus control therapy.

Stimulus control made no impact upon any of the sleep quality variables and in this respect compared unfavourably with the other active treatments. This is a surprising result given the evident improvements in sleep pattern associated with the treatment. Clearly the relationship between actual sleep pattern and qualitative measures is an uncertain one, highlighted particularly by the diverse effects of relaxation and stimulus control. With relaxation, qualitative improvements were reported in the relative absence of pattern change, whereas with stimulus control, substantial sleep pattern improvement was unaccompanied by qualitative change. It would be useful at this point to develop further the tentative model introduced in the preceding section.

It seems feasible that progressive relaxation is associated with an anxiety reduction/ subjective change locus of intervention whereas the stimulus control approach fits in better with a didactic/sleep habit change model. Stimulus control offers no advice on anxiety-management. Indeed, subjects are not encouraged by the treatment to consider qualitative aspects as relevant variables for change. The advice given may be, in fact, less than neutral as regards subjective concerns since patients are required to focus entirely upon overt behaviour. Stimulus control may be envisaged, therefore, as a component of a broader didactic approach where patients are educated about normal sleep pattern, the importance of strong habit formation, and the facilitation of normal sleep pattern restructured within suitable boundaries. Of course a didactic approach should, in itself, be anxiety reducing in so far as it provides accurate information to modify inappropriate attitudes and beliefs. In the present study, however, "didactic treatment" was not specifically applied, but rather such elements of information as seemed appropriate were given to subjects along with whatsoever form of intervention. The usefulness of the didactic

approach will be further discussed later.

Conclusions Regarding Stimulus Control

The effects of stimulus control upon measures of sleep latency were impressive, both in terms of immediacy and magnitude of impact. There was some evidence to suggest, however, that gains on these measures were associated with an initial reduction in sleep duration, probably due to a delayed bedtime and to the anchored rising time affecting opportunities for "catching up" on sleep loss. Latterly, however, patients in this group did sleep significantly longer than at baseline. Habit-restructuring perhaps takes some time to achieve. Therefore, it may be premature to regard stimulus control as effective solely on the basis of, albeit dramatic, early SOL reductions. Because of the absence of change via stimulus control on measures of restedness and sleep enjoyment there may be limitations to the treatment's applicability. It appears crucial to identify predictors of help-seeking for insomnia. If complaints are based upon lack of restedness for example, then stimulus control is likely to miss the mark. A more broadly-based didactic treatment will be proposed at a later point in order to address this critical clinical issue.

Paradoxical Intention

Paradoxical intention has only recently been adopted into the behavioural literature. Available studies have produced rather conflicting evidence regarding its usefulness (see Chapter 5). It is, therefore, of particular interest to consider its effectiveness within the clinical population in the present study.

In terms of SOL mean, patients in the paradoxical intention group completed therapy taking an average of 36 minutes to fall asleep. This outcome figure is similar to that of stimulus control, although the

percentage reduction over baseline was somewhat less (51% compared with 62%) because of the lower baseline SOL scores in the paradoxical group. Nevertheless, paradox was clearly superior to relaxation and to both control groups on a simple pre-post analysis (Table 9). Inspection of Table 12 revealed that significant reductions in SOL were obtained from the fourth week of treatment. These improvements appeared both from this Table and from Fig 4 to be highly consistent during the last 5 weeks. In spite of the relatively low SOL scores during these latter weeks and the significant within group repeated measures effect ($p < .02$; Table 12) between group testing revealed that improvements via paradox never achieved levels significantly superior to waiting-list procedure. Possibly this was due to the lower baseline values in the paradoxical group leaving less room for change.

It is interesting that in the short-term paradox did not produce significant improvement. Indeed, Table 11 revealed that stimulus control was superior to paradox, particularly during the first 2 weeks of treatment. Inspection of Fig 4 suggested that the impact of paradox was delayed for a number of weeks although scores during the final weeks were similar to stimulus control. One clue to a potential explanation for the delayed effect can be gleaned from the study by Espie and Lindsay (1985). These workers reported on several case studies and commented upon the variability in therapeutic response evident with paradox, particularly during the early weeks of treatment. It seemed from the data presented that some subjects experienced initially exacerbated SOL whereas others improved rapidly. It seems possible, therefore, that there are differing modes of response to the paradoxical treatment.

In order to consider further this possibility, treatment effects during the first 2 weeks were examined for each of the active conditions. The

results of this simple analysis are presented in Appendix L [Table (xxi)]. Inspection of these data indicate that, in 6 cases treated by paradox, significant increments in SOL occurred during early therapy (an increment of at least 33% was regarded as substantial). Exacerbation of at least similar magnitude was, by comparison, found in only 2 relaxation treatment cases and there were none in the stimulus control group who deteriorated significantly. Although these data can only be considered as suggestive, it seems that paradox has the potential to exacerbate initial insomnia in a manner which the other approaches do not.

Espie and Lindsay (1985) have already outlined a number of possible explanations for the diversity of therapeutic response with paradox but these are worth reiterating here. Firstly, the instruction to remain awake may be followed literally by some patients, thereby temporarily increasing SOL. Paradox does not provide specific advice on how to avoid "effort to sleep", therefore, some subjects may refocus performance anxiety into an "effort to remain awake", perhaps employing sleep-incompatible imagery to ensure waking cognition even when physically relaxed. This may account for some subjects reportedly finding it "too easy" to remain awake.

A second explanation stems from the possibility that counterdemand instructions may be interpreted as literal demands to report no improvement, amounting therefore to a development of paradoxical strategy. Patients may wish to achieve the experimenter's apparent criterion of success.

Thirdly, some patients may be predisposed to respond adversely to paradox. Those who present with severe intrusive thinking may find that paradox encourages their state of cognitive alertness. Conversely, those patients who can readily identify with "effort to sleep" and who

can successfully avoid that effort without undue arousal will respond best. Unfortunately, the tailored group in the present study included only two patients selected to receive paradoxical instruction, therefore, comparison of random and tailored assignment, specifically in relation to paradox, was not possible.

Clearly further studies are required to address the merits and demerits of these explanations. Clinically, however, therapists ought to be aware of the potential for albeit temporary exacerbation of SOL under paradoxical intention. Furthermore, it is self evident that the more severe the initial insomnia the more troublesome will be deterioration with any therapy.

Paradoxical intention was perhaps the most effective treatment for reducing SOL SD. A 70% pre-post reduction was obtained (Table 9), greater than even that of stimulus control. The within treatment simple effects (Table 12) revealed consistent and highly significant reductions from the second week of treatment onwards. These effects were also reflected in between group testing (Table 11) where paradox was superior to no treatment across the experimental period. Paradox was not superior, however, to any other treatment group. It should be noted that reduction in sleep latency variability refers to raw score variance (night to night variability) for any one subjct and is not, therefore, at odds with the argument above of variability, across subjects, in terms of initial treatment response. The fact that SOL SD reduced prior to SOL mean hints at modification to predictability/stability in sleep latency occurring as a prelude to latency reduction per se. This may be because paradox operates more quickly upon "deviant" nights to "normalise" them before reducing that norm itself.

Similar to the other treatments the effects of paradox upon the

duration of sleep were limited. Percentage gains in TOT were comparable with other forms of intervention (11.8%; Table 9), although the within treatment effect did not achieve significance (Table 12). Nevertheless, inspection of the mean scores for TOT in Fig 6 do suggest a modest gradual increment, especially during the latter weeks. On average, patients finally slept for 42 minutes longer than at baseline.

The 41% reduction in TOT SD associated with paradox (Table 9) was clearly a final week effect (see Table 12). The repeated measures analysis failed to achieve significance at the .05 level.

Paradox was the only treatment to achieve a significant time within treatment simple effect ($p = .011$) for WAKE mean. It did so consistently from the fourth week of treatment although the post-therapy change score was little different from that of stimulus control. The demonstration of improvement over time with paradox does, however, suggest that this is a fairly reliable treatment benefit. Interestingly, however, no parallel changes in WAKE SD were achieved.

Paradoxical intention significantly improved the level of restedness associated with sleep, during Weeks 6, 7 and 8 (Table 14). Parallel improvements in ENJOY, however, were not demonstrated and there were no significant reductions in REPTH or IRRIT. Daytime napping was modestly reduced during Week 8 only, and the repeated measures effect failed to reach significance at $p < .05$. As suggested earlier, it is possible that the more closely treatments approximate to anxiety-management models, the greater will be the change in subjective/qualitative ratings. Paradox was not as effective in this respect as relaxation training, although it appeared to be superior to stimulus control. Once again, however, between group effects at no point reached significance.

Conclusions Regarding Paradoxical Intention

Paradox produced significant reduction in sleep latency when the group was considered as a whole. Initial individual responses, however, were found to vary and persistence with treatment was crucial for ultimate benefit to be obtained in some cases. It seems important to assess the potential for exacerbation of SOL with this treatment. Predictors of such a response may be extreme performance anxiety, and/or the use of counterdemand or other instructions which might occasion an overly literal understanding of the paradoxical prescription. There may be a subtle clinical skill involved in paradox; to encourage subjects to remain awake but yet not to become actively aroused. The ideal may be a "passive" avoidance of sleep-onset. Reductions in nighttime wakening with paradox are encouraging and worthy of further investigation.

In terms of the proposed model, paradoxical intention may be regarded as intermediate in that it can be construed as both anxiety-reducing and habit-restructuring in its mode of action. It was the only treatment to modify both sleep pattern and sleep quality variables to any marked extent. For the former, absolute improvements (ie. at final week) were as great as those for stimulus control although between group testing favoured stimulus control (compared with the control conditions). For sleep quality, improvements were not as great as with relaxation, although both relaxation and paradox were superior to stimulus control.

Tailored Therapy

Designing treatments to match individual presenting characteristics appears clinically to be eminently sensible. Comparative outcome studies, however, have not evaluated the tailored approach. The results from this study are, therefore, of particular interest.

Table 23 revealed that the results of tailored therapy were generally disappointing. A mean pre-post score reduction of approximately 35% for SOL mean compared unfavourably with the total random therapy group (49%), and even less favourably with the most effective randomised treatment (stimulus control; 62%; Table 9). In absolute terms tailored treatment was similar in effectiveness to placebo and relaxation, and SOL remained some 20-25 minutes greater than paradox and stimulus control at post-treatment. Pre-post change scores were not significantly greater under tailored therapy than with no treatment on a simple (non-repeated measures) ANOVA (Table 24) and time within treatment simple effects for SOL indicated little systematic change over time (Table 25).

Outcome for SOL SD was also unimpressive. Reductions of under 30% again paralleled the results obtained for placebo and relaxation, and amounted to less than half that of stimulus control and paradox (Table 23 cf. Table 9). The within treatment repeated measures analysis failed to achieve significance at any point across therapy.

Improvements in TOT mean were, however, greater in percentage terms with tailored treatment than with any other condition. A 15% increment was obtained pre to post-treatment (Table 23) compared with a range of 9-12% for the 3 individual psychological treatments (Table 9). Time within treatment analysis revealed modest gains emerging consistently from Week 2 onwards. Baseline TOT was, however, significantly lower in the tailored group, and in absolute terms the gains in sleep duration via the various approaches were similar to within a few minutes. Pre-post change was not significantly different between any pair of conditions (Table 24).

TOT SD reduced by 26% under tailored therapy (Table 23) but there was no between or within group significant effect. The above benefits in

sleep duration were not paralleled, therefore, by increased stability in sleep length over time.

Tailored therapy was the only treatment to effect significant change over time in both of the WAKE variables (Table 25 cf. Table 12). Reductions greater than 45% were achieved on both WAKE mean and WAKE SD (Table 23), with significant improvement emerging at around Week 4. Although between group effects proved to be non-significant, these results were encouraging for the tailored approach.

It was particularly surprising to find that tailoring of treatment was associated with minimal change on all measures of sleep quality. In circumstamces where intervention arguably should be regarded by patients as highly valid one might expect much greater subjective change. However, the results suggested the converse. Random allocation produced greater qualitative improvement, especially so for the relaxation and paradoxical treatments (Table 25 cf. Table 14).

Conclusions Regarding Tailored Therapy

Tailoring conferred little added benefit to random treatment comprising the same active psychological treatments. The sole exception to this statement might be in terms of wakening frequency and predictability since only tailored treatment reduced both of these variables. For the remaining variables, both quantitative and qualitative, one or other of the three active treatments emerged as preferable. Although these between group differences failed to achieve statistical significance there can be little doubt that stimulus control, for example, given at random is more likely to reduce SOL mean than is even a carefully constructed individual programme comprising less effective techniques. The same could be said for relaxation in relation to measures of sleep satisfaction. In other words, to the extent that tailoring strayed from providing the most effective treatment technique it represented a

dilution of therapeutic effect. It must be concluded, therefore, that the locus of prediction for treatment outcome lies more within the power of the techniques per se than within individual presenting characteristics. Finally, it should be noted that the tailored therapy condition included 4 patients (out of 14) who were treated with "cognitive restructuring" methods since these appeared (from SDQ analysis) to be positively indicated. Such an approach has seldom been adopted in the management of initial insomnia, and with hindsight it may have been better to limit the tailoring to the three interventions employed in the main study. Examination of the raw data indicates that stimulus control patients in the tailored group responded better than the remainder, and that relaxation and cognitive restructuring patients did relatively poorly. It seems justifiable to conclude that tailoring is simply a watering down of the best therapeutic techniques. Perhaps tailoring which is based upon stimulus control principles plus additional elements as required would present a more powerful tailoring option.

## THE ISSUE OF CLINICAL VERSUS STATISTICAL SIGNIFICANCE

Prior to dealing critically with some important methodological issues and formulating general guidelines for good clinical practice, it would be useful to consider the question of clinical significance. This section will review the evidence for the clinical utility of the results so far presented and discussed.

Most straightforwardly, the results obtained in this study are directly relevant to the clinical population. There is no problem of generalizability and the results are valid against the presenting criterion of clinical need. In this sense the results are of clinical significance. Several other sources of evidence regarding clinical significance may also be examined.

Evidence from "Somtrak" Assessment

Although the principal database (the DSQ) was of the self-report type, objective monitoring by means of the "Somtrak" SAD confirmed previous work in establishing the validity of self-report as a relative index of delayed sleep-onset. Indeed, the accuracy of self-report against the SAD standard was remarkably high given the difficulties inherent in estimating lengthy SOL (Table 6). Estimates of sleep duration were very similar to SAD recordings. Measures of intermittent wakening were, as expected, discrepant due to the differing criteria in operation for self-report and time-sampled monitoring. Since the DSQ measures were thus corroborated, the results from the study can be regarded with confidence as being relatively free from reporting artefact.

Table 6 also presented DSQ/SAD sleep pattern scores which confirmed treatment induced changes across the experimental phases in this randomly selected group of patients. SOL as measured by the SAD reduced from 94 minutes at baseline to 33 minutes in the final weeks of therapy, TOT increased from 4.76 to 6.39 hours, and WAKE reduced from 1.93 to 0.82 nightly wakenings. SAD recordings, however, were made of different subjects across phases and these results, therefore, do not reflect a repeated measures model. Nevertheless, SAD scores were consistent with the results obtained on the DSQ in the main study when psychological treatments were applied.

It was also possible to calculate from SAD records a measure of sleep efficiency for each phase of the experimental period (sleep efficiency, expressed as a percentage, refers to the proportion of time asleep in relation to total time spent in bed). At baseline, average sleep efficiency was 68% (SD 21.3). This rose to 78% (SD 17.4) during the first four weeks of treatment and to 88.5% (SD 6.8) during the final treatment phase. This steady increase in efficiency, accompanied by the

reduced variance (SD), is also indicative of clinically significant improvement. Sleep efficiency less than 85% is a widely accepted criterion of significant sleep disturbance. Patients in this study, therefore, appear to have completed therapy with sleep patterns which fell within normal limits. During the course of treatment, SAD measures of efficiency shifted from the significantly deviant/clinical into the normal/sub-clinical range.

Evidence from Follow Up Data

Data presented in Chapter 9 confirmed that treatment-induced changes in sleep pattern and quality persisted during the follow-up period without significant relapse (Table 15). Improvements were maintained, not only in the relatively short-term, but also up to 17 months post-treatment. These later follow-ups in particular are of outstanding clinical importance since they point to the enduring benefits available via psychological management. Where chronic clinical complaints are concerned early evaluations of maintained gains are likely to be of limited value, and are not necessarily predictive of longer-term prognosis. No previous insomnia treatment study has attempted to gather such a large body of follow-up data so long after the end of treatment. The encouraging long-term outcome, therefore, adds very substantially to the clinical applicability of the psychological procedures. This is especially so since, in many cases, conventional medical management had been of only transient benefit.

In presenting the evidence on behalf of prolonged effectiveness it is recognised that some patients resumed medications, particularly where dependence had been problematic and a formal withdrawal programme was required. Tables 19 and 20 recorded 4 out of 10 such patients as resuming drugs after a number of months. Patients' self-report from those who had lesser problems with dependence suggested that less than

10% resumed at least occasional use of drugs. More objective data on post-experimental period drug use were not always available to the author. Where possible, GPs were asked to corroborate patients' reports of being drug-free, but in some cases patients may have had access to stored supplies of hypnotics which had not been recently prescribed by the GP. Short of random blood-testing it is impossible to be certain about the reliability of self-report given that there may be a tendency for social acquiescence towards non-reporting of drug intake. During the experimental period itself much stricter (although probably not absolute) control was of course feasible. Regular contact and accurate information regarding the effects of sleeping-pills upon sleep, along with the precondition of control over the supply of pills to be exercised jointly by the GP and the author, probably minimised the unsanctioned use of medication.

Follow-up return rates in the range 56-77% compare well with other psychological treatment studies and permit reasonable confidence in the positive conclusions outlined above. There is of course the possibility that relapsers might have been less likely to return follow-up assessments, in which case the results obtained would be an overestimate of maintenance effects. This is a problem confronting all research studies which rely upon solicited progress reports. Taking these potential limiting factors into account, however, the consistently positive findings, over 4 follow-up times and across all variables and treatment conditions, remain impressive. The follow-up data are strongly supportive of the clinical worth of the various psychological procedures.

Measurement of Clinical Change

There has developed in recent years (since Bergin and Strupp, 1972) a considerable literature reflecting the concern of some practitioners

that strictly "scientific" analyses fail to tackle material of clinical importance. As Barlow (1981) has put it, "At present, clinical research has little or no influence on clinical practice" (p 147). Jacobsen et al (1984), in reviewing the question of statistical versus clinical significance, have made two major criticisms of psychotherapy outcome data reporting. Firstly, statistical comparisons between experimental conditions are generally based on average improvement scores which provide no information on therapy changes for individuals; and secondly, "significance tests" impose criteria for determining treatment effects which often have unestablished clinical relevance. Hence studies may either overestimate or underestimate the clinical significance of the results obtained.

As outlined in Chapter 6 a number of approaches for testing for clinical significance have been suggested. Kazdin (1977) has argued that a change in therapy is clinically significant when the patient moves from the dysfunctional to the functional range on whatever variable is used to quantify the clinical problem. With such a "distributional" model, it should be possible to deduce the relative proportion of subjects who return to normal funtioning after intervention. This approach does of course presuppose that normative data are available to establish cut-off points for the functional and dysfunctional distributions.

An alternative approach has been to determine the proportion of subjects who improved at post-treatment and to compare with the proportion who did not improve. This tackles the issue of individual variability in response. Some workers have further categorised improvement eg. "markedly", "moderately", "slightly" (Hand et al, 1974; example quoted in Jacobsen et al), and others have defined significant improvement as a change over baseline of greater than 50% (eg.

Lichstein and Fischer, 1985).

In the present study it was decided to apply some of the above criteria to evaluate clinically significant change on the variable SOL mean. This variable was selected since it is generally accepted in the literature that SOL less than or equal to 30 minutes is an appropriate cut-off point for normal functioning. For measures of TOT and WAKE there is no such accepted convention. Also these latter measures tend to be age-related making calculation both complex and spurious. Appendix L [Table (xxii)] contains raw SOL data for subjects in the relaxation, stimulus control, paradoxical intention, and tailored therapy conditions, during baseline and at the final therapy week and presents an analysis of clinical significance according to each of the three following criteria:-

1. Proportion of patients achieving absolute (ie. any) reduction in SOL magnitude at post-treatment
2. Proportion of patients achieving SOL reduction of at least 50%
3. Proportion of patients achieving final SOL less than or equal to 30 minutes

Table 27 below presents a summary of the results from Table (xxii) and permits comparison of the clinical impact of the various psychological treatments.

| Treatment | Absolute Reduction | 50% Reduction | SOL <= 30 Minutes |
|---|---|---|---|
| Relaxation | 71% | 21% | 7% |
| Stimulus control | 100% | 64% | 71% |
| Paradox | 80% | 47% | 40% |
| Tailored therapy | 71% | 50% | 43% |

TABLE 27 Proportion of subjects in each of the active treatment conditions achieving SOL reductions of clinical significance according to three criteria

Consistent with the results presented after statistical analysis (Chapter 8), stimulus control emerged as the most effective treatment according to all three criteria. Every patient improved to some extent and almost two-thirds reduced SOL by greater than half. Seventy-one percent also achieved sleep latencies of 30 minutes or less at post-treatment. Paradox by comparison was less considerably effective on each measure. Only 40% achieved the 30 minute SOL cut-off. Relaxation, however, compared even less favourably. Only around 20% of patients managed to halve baseline SOL after eight weeks of treatment, and only 1 patient (7%) achieved the 30 minute criterion. Almost 30% of patients failed to improve at all at post-treatment with relaxation. Clearly relaxation was clinically useful as a means of reducing sleep latency for only a minority of cases.

The results for the tailored group are particularly interesting. Chapter 10 presented various statistical analyses from which tailored therapy emerged as a poor treatment option. It compared unfavourably with both stimulus control and paradox and appeared to be no more effective than the relaxation and placebo groups. The results presented in Table 27, however, are rather more supportive of the tailored approach. Although only 71% of cases demonstrated improvement, a relatively high proportion of those who did improve achieved the criterion of 50% SOL reduction. For the sake of comparison, all 14 patients improved under stimulus control, 9 of whom achieved 50% SOL reduction; only 10 tailored therapy cases improved, but of these 7 achieved 50% improvement. The problem with tailored therapy appears to be that 30% (4 patients) did not improve at all, and this effect contributed to the comparative ineffectiveness of the treatment in statistical terms. Forty-three percent of tailored cases achieved final SOL of 30 minutes or less which was both absolutely and relatively superior to paradox, and again compares favourably with stimulus

control once non-responders are accounted for.

The four non-responders were treated as follows. Two received relaxation, one received paradox, and the fourth received cognitive restructuring. It seems likely, therefore, that as previously suggested, treatment response was largely dictated by the power of the treatment applied. Tailored therapy was indeed tantamount to a watering down of the most effective interventions for SOL reduction. Nevertheless, clinical outcome was good for almost half of the patients, slightly better than for paradox, but somewhat less than for stimulus control.

## The Generalised Benefits of Treatment

Chapter 9 presented analyses of various pre-post measures in order to consider the generalised changes which were associated with each treatment approach. Significant improvements on measures of anxiety, mood and daytime performance clearly contribute to the clinical value of treatments for insomnia. These generalised effects will be discussed under a number of headings.

### Anxiety State

In keeping with the hyperarousal model of aetiology it might be anticipated that successful treatment of insomnia would be associated with reduction in physical and/or cognitive measures of anxiety. In this study, the ZAS was employed to assess this area of functioning and certain items from the ARS were also relevant to the assessment of anxiety. Tables 16 and 26 presented pre-post data on the ZAS revealing that all experimental conditions (excepting waiting-list) were associated with a reduction in mean ZAS scores. Paradox produced the greatest absolute reduction, and similar to tailored therapy, change was moderately significant ($p <= .02$). Rather surprisingly, however,

relaxation training did not yield statistically significant change on the ZAS although mean score change was as great as that of the tailored approach. Greater variability around mean values rendered the overall effect non-significant. Placebo was associated with a modestly significant reduction in anxiety state on this measure.

It has been proposed that relaxation and paradox may follow an anxiety reduction/subjective model whereas stimulus control represents a habit restructuring/behavioural approach. It is in keeping with these proposals that stimulus control failed to demonstrate significant ZAS reduction and that paradox did produce significant change, not only on ZAS, but also a significant increase in "confidence" (ARS 10) at post-treatment. Relaxation, however, would also be expected to modify anxiety state. Had patients been trained in the application of relaxation as a daytime coping strategy then perhaps changes would have emerged which were more consistent across patients and more substantial in magnitude. Nevertheless, relaxation was the only treatment to reduce significantly reports of "worrying" (ARS 2). Improvements in anxiety state were also obtained with tailored therapy. Given the relatively high proportion of anti-anxiety treatments (relaxation, paradox, cognitive restructuring) compared with habit retraining (stimulus control) this result is also consistent with expectation. The small but significant reduction in ZAS score with placebo treatment may be accounted for in that the rationale underlying placebo was that of desensitisation, and although no active ingredients were included, practice sessions occurred in the early evening, perhaps explaining the small impact upon daytime anxiety state.

In terms of clinical utility, therefore, it was encouraging to note that generalised anxiety reduction may accompany psychological treatment for insomnia. The magnitude of change was, however, limited,

partly reflecting initial levels of only mild to moderate anxiety disorder in this population.

Mood Disorder

All three active treatments in the main study were associated with highly significant ($p < .01$) reductions in ZDS scores (Table 16). Neither placebo nor waiting-list control groups changed significantly. A three point reduction for tailored therapy failed to achieve significance on statistical testing (Table 26). The findings for the active treatments are encouraging. None of the patients in the study were undergoing, nor did they require, specific treatment for depressive illness. Levels of symptomatology were at the mild end of the clinical range. It seems untenable, therefore, that recovery from "depression" could be responsible for observed improvements in sleep measures. Rather it is probable that these mood improvements were consequences of the successful treatment of sleep disorder. It seems justifiable to regard the insomnia as primary and these mild depressive symptoms as secondary; the latter improving in relation to reduced sleep difficulty. The author would stress that there is no assumption being made here that, where insomnia and depression coexist, the insomnia should ordinarily be tackled as primary. Simply, in circumstances where mood disorder is mild and insomnia severe and chronic it seems clinically sensible to treat the insomnia and expect mood to somewhat improve.

In spite of the ZDS changes it is noteworthy that certain ARS items relevant to mood (ARS 5 "irritable"; ARS 6 "downhearted/sad"; ARS 8 "warm and affectionate") did not change significantly with any form of intervention. Possibly overall ZDS reductions were influenced by changes in those items bearing an obvious relationship to sleep pattern (eg. 4, 10, 13; see Appendix F). Item by item analysis, however, was

not conducted since it was not regarded as of central importance.

Daytime Functioning

Measures of daytime performance and coping were included in the ARS. Once again the three psychological treatments in the main study were associated with significant reductions in total ARS score, indicative of general improvement in global functioning. Both the tailored therapy and placebo groups also improved, but not significantly, and the no treatment group deteriorated slightly (see Tables 16 and 26). All interventions (excluding no treatment) improved ratings of "sleeping well" (ARS 9), with active treatments achieving probability levels $p < .01$, and placebo $p < .05$. Relaxation was associated with the greatest spread of improvement when specific ARS items were considered. In addition to the reduction in "worrying" already mentioned, a significant improvement in "coping with work" (ARS 3) and a highly significant increment in "concentration" (ARS 7) were achieved. These findings are in keeping with the other qualitative reports of these patients that they were more rested after their sleep. The only other positive ARS item change was the modest increase in "confidence" (ARS 10) associated with paradoxical intention. The other ARS items failed to discriminate change although the untreated control group did report being slightly less "energetic" (ARS 1) at the end of the experimental period.

The daytime improvements outlined above are of obvious practical importance. However, given that insomnia is usually defined as an inability to obtain adequate sleep (that is, adequate in relation to daytime needs), greater change might have been anticipated, and with all of the active treatments. There are two possible reasons for the failure to demonstrate more broadly-based changes. Firstly, the concept of "adequacy of sleep" is a subjective one, and perceived daytime needs

may be realistic or otherwise. Undoubtedly there are insomniacs who are dissatisfied with the sleep period per se, regardless of its relationship to the daytime. Equally, others may make unreasonable demands of their sleep in terms of resolving daytime problems for which more highly developed problem-solving skills are the appropriate solution.

Secondly, the measures of daytime functioning in the present study were perhaps not sufficiently sensitive or specific, adequately to assess changes. Global pre-post measures do not pick up the specific relationship between sleep pattern changes and daytime correlates. Also, there were no daytime performance measures included in the DSQ (eg. tiredness, concentration) and the only measure of daytime mood (irritability) did not discriminate between or within groups.

A number of methodological improvements will be suggested in the section to follow.

. Pre-bedtime Behaviour Pattern

As expected, stimulus control was the only treatment to significantly reduce SBRS scores, doing so to a greater extent than any other form of intervention (Table 17). The SBRS, therefore, proved to be a sensitive and useful clinical instrument for use in relation to habit-restructuring. The fact that other effective treatments (eg. paradox) demonstrated minimal reduction in sleep-incompatible behaviors at post-treatment does, however, challenge the assumption of the operant model that stimulus factors dictate sleep pattern. Indeed, the behavioural change evidenced on the SBRS under stimulus control by no means proves that these changes were responsible for the treatment effects.

Conclusions Regarding Clinical Significance

Results based upon self-report data were corroborated by the SAD

validity study. Objective records confirmed that significant sleep pattern changes emerged, resultant from psychological treatment. Sleep efficiency increased by around 20% and the insomniac sub-group in the "Somtrak" study obtained final sleep patterns which were within the efficiency range of the normal population.

Lengthy follow-up data established that the effects of psychological treatment were persisting and, therefore, of considerable practical clinical impact. Relapse towards drug-use was a recognised problem in only a small percentage of cases, although where withdrawal had proven particularly difficult there was a greater likelihood of post-discharge drug use. The lack of methodological control over drugs during follow-up may be a limiting factor to interpretation of the data. Long-term outcome was, nevertheless, very encouraging.

Statistical tests may not yield clinically meaningful results. Analysis of clinical significance according to three criteria confirmed the clinically significant impact of stimulus control in at least two-thirds of cases. Paradox was effective for less than 50% of subjects according to these criteria, and relaxation emerged very unfavourably at 20% or less. Tailored therapy, however, was certainly as clinically effective as paradox and considerably more so than relaxation. These results conflict with the statistical analyses of Chapter 10 where group average responses for the tailored condition were deflated by a non-responsive sub-group. It seems that tailoring therapy may indeed be useful clinically with the proviso that the procedures considered for tailoring are of attested merit.

Treatment-related improvements were not confined to sleep pattern or reports of sleep quality. Psychological treatments significantly reduced levels of depressive symptomatology, and anxiety reduction (on either ZAS or ARS scales) was a feature of treatments other than

stimulus control. The latter treatment was associated with marked behavioural pattern change prior to bedtime. Measures of daytime functioning require attention in order to evaluate adequately the correspondence between nighttime and daytime measures. Nevertheless, relaxation in particular appeared to afford patients generalised benefit in terms of concentration and work performance.

## SOME IMPORTANT METHODOLOGICAL ISSUES

### Issues of Experimental Control

Reference has already been made to the very satisfactory compliance of patients in the waiting-list group, in spite of minimal contact. Some previous studies have, however, required subjects to submit monetary deposits (refundable upon good data recording) and to send in sleep diaries by postal return in order to ensure compliance. The author would suggest that there were two critical issues in this study which were predictive of compliance. Firstly, specific training in record-keeping was essential. Patients are much more likely to want, and to be able, to comply with a task which is unambiguous. A training period helps to define for subjects what are the basic criteria for self-monitoring, and gives constructive feedback on performance. Secondly, patients who are highly motivated are much more likely to comply. This may seem very obvious, however, studies which have employed solicited subjects may have to rely heavily upon external controls (eg. deposits) to maintain motivation. By contrast, spontaneously presenting patients demonstrate motivation through their help-seeking actions. The subjects of this study being chronic insomniacs appeared capable of both patience (being long-term sufferers they did not generally expect or demand immediate "cure") and perseverance (they were prepared to follow instructions for the sake of longer-term benefit).

The results for the untreated group demonstrated unequivocally that spontaneous remission did not account for treatment effects in the present study. Such slight changes over time as did emerge in fact tended towards deterioration (Tables 9, 12, 16). Figs 4-9 suggested a degree of instability over time in this group's sleep pattern which is perhaps supportive of the view that variability is a "normal" function for sleep-disturbed groups and, as proposed in Chapter 2, this may contribute to subjective dissatisfaction with sleep.

Subjects in the waiting-list group did not re-enter the study for active treatment since it was felt unreasonable to require them to complete a further, lengthy experimental protocol. All patients did, however, receive psychological treatment on an individual basis, in many cases via stimulus control since this emerged as an effective approach. Data have not been presented on these treatments since there was no uniformity of approach. Formal analysis, therefore, was not attempted.

Patients in the quasi-desensitisation placebo group regarded the treatment rationale as a credible one, and no less so than the active psychological interventions. This finding is consistent with previous studies which have employed this placebo strategy (eg. Turner and Ascher, 1979a; Lacks et al, 1983a). Similarly, there were no significant differences amongst the active treatments on the CEQ. Results cannot, therefore, be attributed to differential expectation or motivation based upon the perception of treatment instructions. Although modest gains were achieved on most sleep pattern variables under placebo treatment (Table 9), within group repeated measures effects failed to achieve significance (Tables 12 and 14). Statistical appraisal of treatment effects therefore, supported non-improvement over time. It can be concluded then that placebo effects are unlikely

to offer a competing explanation for the impact of the psychological treatments. Between group effects, however, achieved sufficient magnitude for statistical significance only for the comparison of stimulus control with placebo.

Furthermore, it is clear from the results that placebo group patients did not improve significantly even under conditions of positive demand. This finding is in contrast to some previous work which reported that the engendering of positive expectancy produced significant improvements in the sleep pattern of placebo subjects, whereas only active treatments instituted change during counterdemand (Borkovec et al, 1975; Borkovec and Weerts, 1976). It is probably a reflection of the severity of insomniac complaints in this study that such demands or suggestions made no impact upon outcome. Table 8 demonstrated that active treatment effects generally emerged under negative demand and that on no occasion did they commence at Week 5 when the positive demand instruction was first issued. The demand manipulation was, therefore, a useful inclusion in this study in order to rule out the competing explanation of experimental demand. The use of both placebo and demand manipulations, therefore, provided a useful double methodological control over non-specific treatment effects.

One further potentially confounding explanation for the results was dealt with in the evaluation of "therapist factor". It was regarded as feasible that using a single therapist across conditions might bias outcome in favour of treatments with which the therapist was most familiar or in which he exercised greatest skill. Chapter 7 described a study conducted to investigate this possibility, the results of which, presented in Fig 3, indicated that there was no evidence to support systematic bias in terms of interpersonal and collaborative skills, or

in terms of explanation and application of treatment programmes. Certainly this study was conducted on only a small (10%) random sample of the total population, and greater confidence may have been possible via more comprehensive evaluation. Nevertheless, the study did not hint at the need for more detailed investigation. Most previous reports have failed to consider therapist effects of this type as a competing explanation for outcome. Those which have, have reported non-significant effects (see Chapter 6) with the exception of the Turner and Ascher (1981) study where experienced clinicians were found to make better therapists than trainees. This clearly did not present a difficulty in the present study.

To summarise so far, the methodological controls used in this study appear to have functioned well. The waiting-list and placebo groups respectively controlled for passage of time and non-specific treatment effects, and the demand manipulation further controlled for therapy demands to report improvement. All interventions were regarded as highly credible and the therapist applied treatments in an equally skilled manner.

Application of Techniques

In spite of the comprehensive methodology it could be argued that patients in different groups applied the treatments with differing degrees of consistency or motivation, thus accounting for the various effects demonstrated. The fact that CEQ scores were high and similar does, of course, argue against this, but nevertheless the possibility is deserving of some discussion.

Clinical experience indicates that some patients carry out therapeutic directions/homework better than others, and that where procedures are "inconvenient" there may be a greater tendency for such failure to

comply. The present study was no exception to this observation. It is, of course, part of the therapist's clinical skill to encourage and facilitate implementation of therapeutic procedures in the home environment. Working towards between session change is an indispensible aim in psychological treatment.

In this study the author had to rely upon patient self-report to determine whether or not procedures were being carried out as required. Adherence to the demands of the stimulus control programme could be checked upon by detailed examination of the DSQ (eg. bedtime, waking time, rising time, daytime napping) and some patients did require correction if they took a broad "interpretation" of the rules eg. by waiting longer than 20 minutes before rising, or sleeping beyond waking time at weekends. Similarly, some of those in the paradoxical intention group found the instruction to remain awake difficult to apply since it demanded a reversal in attitude which did not come naturally. It is also difficult to be sure that relaxation subjects practised their exercises as prescribed, and that placebo patients always did their "imagery relief" training programme in the early evening.

One possible solution might have been to ask spouses to record or report on homework practice. This would have provided useful, and (arguably) independent corroboration of self-report. However, practical difficulties concerning the fact that spouses generally fall asleep faster than their insomniac partners, and the possibility that reports would be biased towards confirming patients' report, would be likely to confound this assessment. On balance it seems unlikely that patients in any one condition would be particularly inconsistent in the application of the procedures. The fact that the patients as a group were reliable DSQ reporters in relation to the SAD external criteria suggests that other forms of reporting may also be reasonably credible.

One recent and interesting development is noteworthy before leaving this matter. Lichstein and Hoelscher (in press) have developed a relaxation assessment device (RAD) which appears to subjects to be a standard cassette player but which also, discretely, records tape playing time. Their preliminary investigation of the RAD indicated that anxious patients practised exercises on 4.4 nights on average out of the targetted 7, and that hypertensive patients practised fully up to criterion. They concluded that practice is highly correlated with motivational state. Such a device may prove to be a useful assessment option also for the insomniac group.

Measures of Outcome

Validation of the sleep pattern measures in the DSQ was discussed in an earlier section. Mean scores for SOL and TOT appeared satisfactory, however, it was noted that measures of wakening frequency required further consideration. In this study patients were required to record only the number of wakenings per night which were associated with "difficulty in returning to sleep". This measure was designed to quantify the "problem" wakenings, rather than those, perhaps frequent, brief arousals which present as a part of the normal sleep of older subjects. Kales et al (1984) in a large descriptive study found that difficulty in falling asleep either initially or during the night was descriptive of the presenting insomniac group (see Chapter 1). Nevertheless, the measure of problem wakenings alone may have been inadequate. Patients often complained of the broken quality of their sleep which is likely to be a reflection of frequent short arousals. Clearly, there are problems inherent in the accurate numerical estimation of wakenings, some of which are barely (if at all) perceptible. There are, however, a number of measures which might be useful in future studies, in addition to the one employed.

The most widely used self-report sleep-maintenance measure has been that of WASO (wake time after sleep-onset). This measure is an estimate of the total time spent awake during the night after sleep commenced. The author has recently used WASO for cases of intermittent insomnia (Espie and Lindsay, 1987) and patients appear to find it a sensible and descriptive measure. Another useful measure may be a qualitative rating of either "brokenness" or "depth" of sleep. Such a rating has seldom been applied, in spite of its obvious face validity in characterising the problem. Turner and Ascher (1979a) asked subjects to rate "difficulty in returning to sleep", but found that this measure did not demonstrate significant change after treatment. In addition, it seems unlikely that such a rating could be an improvement upon a numerical estimate of problematic wakenings. EEG assessment is the only sure method of obtaining reliable information on both the frequency and duration of nighttime arousals. Where access to such facilities is not available, measures of WASO and "brokenness" of sleep would appear to be useful.

In addition to weekly mean values for SOL, TOT and WAKE, measures of standard deviation were also calculated in this study. The objective in doing so was to describe sleep pattern, not only in terms of the (possibly artificial) "average night's sleep", but also in terms of the variability in the individual's sleep from night to night. It had been the author's observation that insomniacs often slept well for at least part of the time, but that "bad nights" occurred, often unpredictably. Variability, therefore, may be part of the norm which predicts clinical complaint. Such variability was indeed evident at baseline, especially for SOL where the typical sleep latency was around 80 minutes and typical raw score SD was +/- 45 minutes (Table 9). Interestingly, of the 3 treatment x time interactions to achieve significance, two were for measures of variability (SOL SD, TOT SD) and only one was for mean

score (SOL). Since mean values for WAKE were less than 10 per week at baseline the measure WAKE SD was perhaps irrelevant. It is suggested that, in the interests of demonstrating that a consistently improved sleep pattern has emerged after intervention, both mean and SD scores should be used as outcome measures, certainly for assessments of sleep latency and duration. The inclusion of these in this study has yielded novel information never before presented in the insomnia literature. There may be a parallel here between the clinical importance of knowing what proportion of patients improve with a given treatment (variability across persons) and the clinical importance of knowing how predictable improvements are for a given night (variability over time).

Stimulus control and paradoxical intention were particularly effective in reducing significantly both SOL mean and SOL SD (Tables 9, 11, 12). This is an important finding since previous studies, employing only mean values, have fostered conclusions along the lines that "subjects took X minutes to fall asleep at baseline which was reduced by Y% to Z minutes SOL at post-treatment". The impression thus conveyed is that sleep has changed from being predictably poor to being predictably good. A more accurate picture, however, may be that of highly unpredictable sleep pattern (often poor but not always) being stabilised and improved. In an unpublished report Espie, Lindsay and Hood (1986) examined SOL data of the 14 subjects treated by stimulus control in the present study. They found that 47% of baseline SOL values fell within the post-treatment range, suggesting that therapy may have acted to "normalise" scores from the top half of the original distribution. That is, they were brought into line with the "good" nights which existed all along.

With the possible exception of the measures of wakening frequency then, the DSQ measures of sleep pattern used in the study appear to have been

satisfactory. The inclusion of both mean and SD scores allowed for more informative analysis than is possible using mean values alone.

Relaxation therapy, and to a lesser extent paradox, produced some improvement on sleep quality variables whereas stimulus control was not associated with significant change. These results have been interpreted in terms of the specific modes of action of anxiety/subjective change treatments compared with habit/behaviour change approaches. Nevertheless, it must be concluded that sleep quality reports emerged as less sensitive outcome measures than had been anticipated. A number of comments are appropriate.

Firstly, the variables RESTED and ENJOY were synonymous with a "refreshing, satisfying, good" sleep. Patients were asked to provide a rating on a 5-point scale. Given that there may be rather more of a categorical basis to sleep pattern (ie. good and bad nights) it might have been useful to obtain reports of dichotomous choices such as satisfactory/unsatisfactory, acceptable/unacceptable or simply good/bad. Such forced choices would perhaps get round the tendency of patients to "hug" the middle ground of linear scales and to fail to use the breadth of ratings available.

Secondly, subjects were required to complete the DSQ soon after rising. This was essential for retrospective estimation of sleep parameters. However, it may be that more valid assessment of restedness would be achieved through ratings made some while later, perhaps mid-morning, when people are better able to judge the value of the previous night's sleep in relation to performance. Indeed, there is no evidence to support the view that good sleepers feel rested immediately upon rising. Clearly investigation of the timing of such assessments would be welcome.

Thirdly, REPTH did not discriminate well between or within groups. The only significant finding on this variable was the modestly significant treatment main effect accounted for by the greater average reduction in pre-bedtime repetitive thoughts associated with relaxation compared with placebo. Although this finding is consistent with the proposed model, neither interaction nor time main effects were significant, and paradox (also an anxiety reduction approach) was not associated with significant change. A number of studies were reported in Chapter 2 (section on cognitive hyperarousal model) which found that insomniacs presented with persistent cognitive intrusions prior to falling asleep. Perhaps the wrong question was asked in the present study. Items from the SDQ which were commonly selected descriptors of sleep disturbance may prove to be more useful, eg. Item 2 "mind keeps turning things over", Item 10 "unable to empty mind" (see Chapter 10). In addition, the performance anxiety based rationale for paradox perhaps demands a more specific question relating to pre-sleep mentation. This study did not include any measure of performance anxiety per se. Again the relevant items from the SDQ can now be forwarded as valid process measures.

Reference has already been made to measures of daytime functioning. Pre-post assessment (ARS) was rather insensitive and this form of evaluation does not provide useful information on the relationship between nightime sleep and subsequent daytime functioning. Although it was not the purpose of this study to consider this issue in any detail, a number of recommendations can be made.

The application of both objective and subjective process measures of daytime performance and fatigue would seem to be appropriate. It is to this area that a number of influential researchers in the USA have been turning of late. The multiple sleep latency test (MSLT) was developed

at Stanford University during the 1970's as an objective measure of daytime sleepiness. Subjects are required to spend the day at the sleep laboratory and take a series of short naps at about 2-hourly intervals. Sleepiness is measured by how long it takes them to fall asleep. Due to the impracticability of the MSLT, however, other workers at Memphis State University have been conducting pilot tests of other more convenient strategies for determining daytime sleepiness. These include auditory discrimination, abstract logic, visual focusing and mental arithmetic tasks, along with pupillometric measures (see Lichstein, 1984).

The increasing emphasis upon daytime effects is, of course, a reflection of the move towards more didactic models of treatment (eg. Bootzin, Engle-Friedman and Hazlewood, 1983; Bootzin and Engle-Friedman, in press; Lacks and Rotert, 1986) and the accompanying evidence that a proportion of insomniacs do not suffer daytime sleepiness, although they do have an unsatisfying sleep experience (Lichstein, 1984).

Where sleep laboratory methods or performance/cognitive assessments of daytime functioning are not feasible, future studies might consider the extension of diary reports to provide self-report information on sleepiness, concentration, and work performance. It may be a limiting factor to the present study that it is not possible to discriminate between the two insomniac subgroups of those with insomniac sleep pattern and daytime correlates, and those with disturbed sleep pattern but without significant daytime consequences.

## GUIDELINES FOR CLINICAL PRACTICE

Guidelines for clinical practice will now be presented based, not only upon the results of this study, but upon an integration of these

results and the most recent other research evidence. In considering the issue of good clinical practice several key questions require to be addressed:-

a) What constitutes "improvement" for this patient?

b) Is improvement in these terms a valid and achievable goal? What informational/educational input is required?

c) Which psychological intervention(s) predict the best outcome?

The first question raised concerns the individual patient's perception of his/her problem and his/her personal view on what outcome would constitute satisfactory improvement. The term "sleep-onset insomnia" is a convenient generalisation which enables discrimination of this sub-type from other categories of sleep disorder. However, within this group of patients, emphases upon and interpretations of the problem are likely to differ. Some are concerned primarily about the "abnormality" of their sleep compared with their peers, and consequently feel that sleep requires to improve. Others compare current with former pattern and hope for a reinstatement to previous norms. Another sub-group are over-anxious and find the sleep experience per se unsatisfying (or even aversive) and desire to become "good sleepers". Still others complain of excessive tiredness and regard treatment as a way of resolving daytime performance deficits. Clearly perspective varies, and this is not an exhaustive list. Another important facet of presentation concerns the focus of complaint. Some present the problem in terms of sleep parameters (latency, total time, etc.), others in terms of unacceptable sleep quality.

The first rule of clinical practice must be, therefore, to recognise that insomnia is not a unitary phenomenon. The application of a treatment technique, without re-forming the patient's perceptions of sleep, may be invalid. The absence of subjective improvement after stimulus control, for example, is likely to be a limiting factor to

applicability with patients who are demanding of qualitative change. Equally, modest reductions in sleep latency after relaxation training will possibly dissatisfy the patient who wishes to fall asleep rapidly. Intervention is not, however, simply a matter of modifying variables in line with patient demand. As any therapist knows, success with such an approach can be elusive since complaints have a habit of re-emerging in another form. Rather, there is need for thorough assessment in order that complaints and expectations may be validated, and if validated, treated.

The second question of interest, therefore, concerns this validation procedure, and the subsequent agreement between the patient and therapist of relevant and achievable targets for change. Reference has already been made to the importance of training patients in the recording of sleep pattern, and of establishing the clinical significance of the presenting complaint. In routine practice objective validation by means of EEG assessment is seldom feasible. The "Somtrak" SAD, however, appears to be a useful, non-intrusive objective device to corroborate self-report. Validation also applies, however, to three other equally important aspects of reporting.

Firstly, practitioners must employ a developmental perspective in assessment. Sleep patterns are closely related to age, and for older people, often unwelcome shifts in sleep occur. The shifts may be gradual and imperceptible or sudden and striking, however, the individual who fails to interpret these changes as part of a normal developmental process may be liable to conclude that sleep has become pathological. Subjective reports must be validated , therefore, against the criterion of "normality".

Secondly, clinicians should employ a 24-hour view rather than limiting interest to the sleep period. It may be that patients who fail to

demonstrate significant daytime dysfunction cannot validly be considered as insomniac against the criterion of "adequate sleep". Lichstein (1984) has suggested the label "insomnoid" for such people who present with a discrepancy between biological sleep needs and sleep goals.

Thirdly, consideration must be given to the role of sleep within overall functioning. Clearly some patients exhibit diminished cognitive and performance ability as a direct consequence of sleep loss. Others, however, may exhibit daytime deficits which are quite independent of sleep sufficiency and may falsely attribute causation to insomnia. Such subjects are likely to set invalid goals for their sleep pattern. Validation of complaint requires also, therefore, to be conducted against attributional criteria.

It is only through attention to these matters that agreement can be reached between patient and therapist on appropriate methods of intervention and appropriate measures of change. It is in this connection that the educational/didactic model has a crucial role.

In the last few years a number of studies have been published which have investigated the usefulness of didactic treatment (Lichstein, 1980; Bootzin et al, 1983; Bootzin, 1985; Bootzin and Engle-Friedman, in press; Lacks and Rotert, 1986). Intervention has generally involved educating patients about sleep needs in relation to age and daytime requirements, and so identifying reasonable targets for change. A further feature has been the emphasis upon sleep efficiency as a suitable, but much neglected measure (Lichstein, 1984). Perhaps too much attention has been paid to increasing the numerator of this ratio (ie. total sleep) and insufficient effort made at reducing the denominator (time spent in bed).

Bootzin and Engle-Friedman have conducted the only comprehensive controlled study incorporating the didactic approach. They compared three active treatment conditions with a waiting-list control group in a sample of 53 older insomniacs (mean age 59 years). A "sleep information" group received support, sleep hygiene advice (cf. Lacks and Rotert, 1986), and sleep education. A second group received this support and information plus progressive relaxation training, and the third treatment group received the didactic package plus stimulus control instruction. Their results showed improvement in all three treated groups in terms of improved sleep pattern, increased restedness and self-efficacy, and reduced depression scores (Beck Depression Inventory). Bootzin and Engle-Friedman regarded the increases in self-efficacy and decreased concern regarding sleep as being of particular importance since the goal of all of the treatments was to help insomniacs see themselves as people who could cope with sleep disturbance, rather than be victims of it. Some differential treatment effects did also emerge. The addition of either relaxation or stimulus control to the didactic treatment resulted in significantly increased sleep efficiency, and sleep latency reduction was significantly greater with stimulus control than with the other two interventions. This pattern of results was maintained at lengthy follow-up.

In the light of these results it would seem to be clinically useful to include routinely in therapy, a preliminary assessment and intervention along didactic lines. The qualitative/subjective improvement obtained in the Bootzin and Engle-Friedman study through this approach was also reported in earlier single case reports (Lichstein, 1980; Bootzin, 1985). It may be that the inclusion of didactic training along with stimulus control in the present study would have yielded qualitative gains which the stimulus control approach alone did not produce.

The third question raised earlier concerned the efficacy of the various specific psychological treatments. Results in the present study favoured stimulus control as the means by which the most rapid SOL reduction may be obtained, although gains in total sleep duration did not emerge until some weeks later. Reservations regarding the stimulus control approach were in terms of its insensitivity to the subjective concerns of the patient. The practising therapist, therefore, should be aware that patients may regard this treatment as a rather "clinical" and strict programme of retraining, and that they may fail to report greater satisfaction with sleep at post-treatment. However, stimulus control instruction appears to be particularly compatible with the didactic model. The two approaches may offer, therefore, a powerful combination of procedures.

Stimulus control aims to maximise the control of the "sleep system" over sleep by ensuring that biological drive cues bedtime and that sleep requirements achieve equilibrium in a regular pattern preceding a set waking time. The essence of the didactic approach is to educate patients to the possibility that such natural sleep requirement may be less than they would aim for, partly because sleep needs are age-correlated. The recognition that sleep goals are not entirely determined by biological need/capacity, but also are responsive to other demands, may foster a re-attribution process and result in greater satisfaction with sleep. Without the educational process, stimulus control may appear to subjects to be a harsh discipline which, although effective in modifying sleep pattern, does little for extant anxiety and misinformation. The therapist is, therefore, encouraged to apply didactic and stimulus control procedures in tandem to facilitate both quantitative and qualitative change.

It has been proposed that relaxation and paradoxical intention are more

likely (than stimulus control alone) to modify subjective report since they follow an anxiety-reduction model. Qualitative change may of course be enhanced for these treatments through an additional didactic process. The usefulness of paradox appears, however, to be limited by a "risk factor". That is, a proportion of patients respond unfavourably although latterly sleep pattern gains may match those obtained via stimulus control. It is recommended, therefore, that practitioners attempt to assess the individual's ability to defer sleep-onset without undue arousal as part of the selection process for paradoxical strategies. It seems that the clinical utility of the technique may rest upon the passive avoidance of falling asleep as opposed to the deliberate induction of high arousal. Assessment of this subtle difference, however, may not be easy. One possibility might be by means of biofeedback training. Patients could be trained in de-arousal of physiological functioning along conventional lines, and then trained to develop "fine control" over arousal levels in the relaxed state so that, by degrees, "avoidance training" could be achieved. Alternatively, and perhaps more simply, Fogle and Dyall's (1983) variation on paradoxical strategy might be worthy of consideration. In their approach patients were told to "give up" trying to fall asleep rather than trying to "stay awake". Interestingly, Fogle and Dyall reported that the paradoxical giving-up strategy was more effective in reducing performance anxiety than was the conventional procedure.

Where care is exercised in the selection and training of patients, therefore, the practitioner is likely to find paradox as effective as stimulus control. In addition, some reduction in wakening frequency may be expected through paradox although other techniques of "worry control" may be preferable (cf. Espie and Lindsay, 1987).

The clinician should be aware of the severe limitations of progressive

relaxation in producing clinically significant change in disturbed sleep pattern. In this study only one in five subjects achieved a reduction of 50% or more in SOL. The positive indications for the use of relaxation appear limited to those situations where sleep complaint relates to a dissatisfaction with the enjoyment of sleep. Relaxation training appears to improve patients' attitudes towards sleep rather than affecting sleep parameters per se, but it also has some advantages in terms of daytime improvement of concentration and coping. It is possible that such benefits would be matched by the use of didactic treatment in association with either stimulus control ar paradox. The simple provision of a relaxation tape for home practice is a common clinical expedient. In itself this cannot be expected to substantially modify sleep pattern, but patients will probably report feeling a bit better about their sleep and about general daytime functioning.

Tailoring treatment offers little benefit over the best conventional treatments given at random. Didactic input would, of course, need to be appropriate to the individual's presentation, and in this sense be tailored, however, beyond that stimulus control first and then paradox appear to be the treatments of choice. The usefulness of cognitive restructuring programmes remain to be evaluated adequately. These guidelines may go against clinical judgement in some cases, since individualised treatments have intuitive appeal. The evidence from this study, however, is supportive of selecting on the basis of treatment effectiveness rather than individual presentation. Clearly there are implications here for the validity of presumed aetiological factors in the individual's presentation.

Finally, it is worth reiterating that the clinician should be able to manage the drug-using patient as well as those who are drug-free. Here also the role of the therapist as educator is paramount to obtain

compliance, maintain motivation, and encourage accurate attribution. The models of drug withdrawal outlined have received preliminary support both methodologically and clinically. The practising therapist should make use of these to ensure adequate control of medication and to maximise the effectiveness of psychological treatment.

## SUGGESTIONS FOR FURTHER INVESTIGATION

Guidelines for clinical practice have been laid down, representing a synthesis of the results of this study and other recent developments. However, there remain significant gaps in the current understanding and treatment of insomnia. The following suggestions, therefore, are made to focus research attention.

Firstly, the identification of valid predictors of insomniac complaint must be a priority concern. In spite of the burgeoning literature, no clear or comprehensive "formula" can be given; although the expectation of such may be unrealistic. The phenomenology of insomnia is complex and it appears that biological, developmental, social, emotional and performance factors may all be implicated. Dysharmonies between objective and subjective criteria of evaluation are particularly problematic. It is not easy to quantify a "good sleep", even for the individual; and far less for the insomniac group as a whole. Sub-classification into objective and subjective insomnia (Borkovec et al, 1978) or insomniac and insomnoid (Lichstein, 1984) have been proposed and these are worthy of further consideration. In particular the latter categorisation is promising since it recognises the validity of sleep complaints which relate to dissatisfaction with the sleep experience itself.

An attempt was made in this study to identify variables predictive of clinical presentation and of treatment response. Rather surprisingly,

neither age nor chronicity of insomnia were significantly correlated with severity of sleep disorder or intensity of qualitative reports. This was perhaps due to the "categorical" nature of the presenting group, ie. by selection - severe, clinical insomnia. Nevertheless, a range of ages and durations of insomnia were represented. Longer use of medication did, however, appear to be predictive of somewhat more stable and restful sleep from night to night. Affective measures were also of some predictive utility. More anxious and/or depressed patients suffered greater pre-sleep cognitive intrusion and were more irritable. Increased neurotic symptomatology was not, however, associated with greater sleep pattern disturbance.

Subjects who required post-treatment drug withdrawal were on average, older, had longer histories of insomnia, and took longer to fall asleep. Between group differences, however, were not significant. Similarly, there were no significant differences in terms of treatment response although Tables 19 and 20 suggested that relapse to drug-taking was greater in the post-treatment compared with the pre-treatment withdrawal group. Booster therapy for such patients may be a practical solution and consideration could be given to this in future studies.

Detailed baseline assessment of daytime and nighttime variables, and subjective perception of these, would provide the necessary basis upon which to investigate predictors of complaint. This study did not provide data for this purpose. Nevertheless, in terms of predicting outcome from treatment, the study does makes clear the efficacy of psychological treatments in producing maintained benefit even in severe and chronic cases of insomnia. The severity of complaint does not contraindicate psychological management.

A second priority for further study concerns the mechanism(s) by which

psychological treatments achieve their effects. As outlined in Chapter 2, each treatment has a logical theoretical basis, but also has inconsistent evidence to support its hypothesised mode of action. This study did not attempt specifically to examine such aspects of treatment process. It has been suggested, however, that two theoretical perspectives might be applicable.

The stimulus control approach as an operant paradigm may operate upon behaviour patterns via the laws of learning and reinforcement, and so be effective in habit restructuring. The failure to produce qualitative (cognitive/attitudinal) change may be due to the "cognitive lag" phenomenon which has been reported in other parts of the behavioural literature (Rachman and Hodgson, 1974). It has been suggested that desynchrony can exist between behavioural and affective components of a problem such that behaviour change occurs prior to, or in the absence of subjective change. Alternatively, a re-education process may be required, ie. didactic treatment. This may contribute, directly at the informational/cognitive end, to the newly learned repertoire by modifying attitudes and self-evaluative procedures. Some preliminary evidence is supportive of the joint use of stimulus control and didactic methods (Bootzin and Engle-Friedman, in press) and it may prove a fruitful avenue of research to consider the complementary nature of these approaches. It appears conceptually sound to regard stimulus control instructions as a set of cohesive retraining rules rather than a collection of "ingredients" which require to be separately evaluated (cf. Zwart and Lisman, 1979).

The other theoretical position proposed embraces both relaxation and paradoxical treatments in so far as each are recognised anxiety-reduction procedures. Here improvement is more likely to be associated with subjective improvement, with the proviso that behaviour change

(ie. sleep pattern) may occur also, dependent upon the power of the treatment. Alternatively, behaviour change may be regarded by patients as unnecessary if anxiety reduction and other subjective improvements are sufficently extensive. Other forms of anxiety management treatment would also fall within this second theoretical perspective.

Clearly care must be exercised in future studies to investigate separately the impact of treatments upon physiological and cognitive aspects of presentation, although it seems likely that the latter is the more critical function. Indeed, it has been argued in the past that all treatments, including stimulus control, achieve their effects through the elimination of bedtime worrying. Equally, from the operant perspective, it could be argued that anxiety reduction as such is unnecessary as long as anxiety does not occur in the bedroom or around bedtime. Worry may simply be another example of sleep-incompatible activity. The work of Espie and Lindsay (1987) in using a "worry control" procedure should be further investigated in this respect.

Thirdly, and from a cost-effective clinical perspective, it would be useful to compare the efficacy of a stimulus control/didactic intervention presented as bibliotherapy, with other more labour-intensive psychological treatments. The GP remains the primary advice-giver to poor sleepers and such a minimal contact approach might be applied readily within the constraints of the GP appointments system. Interestingly, in spite of the availability to GPs of information on behavioral management approaches for insomnia (eg. Giblin, 1983), it has been the author's experience that few practise these techniques. This appears so in spite of the considerable efforts of many practitioners to reduce considerably the quantity of hypnotics given on prescription. Perhaps the health education role of the family physician requires greater emphasis, and also the role of the psychologist as

disseminator of psychological and interpersonal skills (see Espie and White, 1986a).

Finally, it is noteworthy that the multivariate repeated measures model applied in this study had both advantages and disadvantages. Firstly, the MANOVA approach permitted greater confidence in the interpretation of significant effects because of its conservatism and minimisation of error rate. Secondly, the approach followed, ie. SPSSX using the guidelines of O'Brien and Kaiser (1985), was a straightforward one, relatively free from procedural complication, and considerably simpler than that outlined in the SPSSX User's Manual. In addition, the computation of gain scores allowed for inbuilt covariance of baseline values rather than comparisons of absolute post-treatment scores. Thirdly, the examination of treatment process data has been a feature of this study and the available SPSSX MANOVA sub-commands proved excellent in this respect. The CONSPLUS sub-routine generated an overall time within treatment group multivariate statistic and sub-effects for each week of therapy. This analysis proved invaluable in treatment comparison, given the relatively few significant interaction main effects which were achieved.

By way of criticism, the main effects achieving significance using MANOVA were probably fewer than would have been the case with a univariate approach. There is, therefore, the possibility of missing potentially important between group differences due to loss of sensitivity. In this study care was taken, however, to examine treatment x time data and to highlight potential benefits of one treatment over another even when between group comparisons failed to achieve significance. It is clearly important, with whatever analysis is selected, to make use of statistical tests and the profile of results obtained to address the salient research questions. A balance

must be struck between under-interpreting and over-interpreting.

It is also important to recognise that results in terms of clinically significant outcome do not always parallel those from statistical testing (cf. Table 28). Further research is required to develop the range of assessments available to measure individual variability in response to treatment. One of the outstanding problems inherent in any group comparison study which derives general rules, is that these are not necessarily applicable to the individual who presents in the consulting room for treatment. Indices of clinical change based upon both inter-subject and intra-subject variability require greater recognition and further development.

# APPENDIX A

## Sample Imagery Relief Hierarchy and Mental Image List

## EVENTS PRECEDING BEDTIME

(in chronological sequence)

1. Coffee and read paper
2. Close curtains, lights on
3. Wash up
4. Tidy living room
5. Children's clothes laid out for the morning
6. Lock doors and put bottles out
7. Lights out, plugs out downstairs
8. Ready for bed
9. Wash and toilet
10. Tidy up in bedroom
11. Organise clothes for the morning
12. Check children OK
13. Lights out in hall
14. Wind up clock and watch
15. Main light out in bedroom
16. Into bed
17. Switch bedside lamp off
18. Go to sleep

## MENTAL IMAGES

1. Watching the TV.
2. The colours of a rainbow
3. Taking the dog to the park
4. Knitting
5. Reading a book
6. Trees

# APPENDIX B

## Handouts for Each Treatment Condition

1. Progressive relaxation
2. Stimulus control
3. Paradoxical intention
4. Imagery relief

## OVERCOMING INSOMNIA THROUGH PROGRESSIVE RELAXATION TRAINING

The treatment programme described below has proved to be effective in helping people to get a better night's sleep.

Most people who suffer from insomnia either find it hard to get off to sleep when they go to bed, or find their night's sleep is broken by numerous wakenings. Some people experience both of these difficulties. Very often the cause of these problems is an inability to relax. It is certainly true that people who are good at relaxing seldom have difficulty sleeping.

### How does relaxation work?

Relaxation is really a skill, and like other skills it requires practise in order for it to be learnt. The relaxation exercises teach you to recognise where there is tension in your body and they show you how to relax such tensions away. You will become aware of the difference between tensed and relaxed muscles, and you will be able to choose to relax parts of your body at will. Thus, since it is impossible to be both relaxed and uptight at the same time, you will have learnt a way to help yourself relax your body to sleep.

If at present you go to bed feeling anxious and wondering whether or not you will get a good night's rest, then these relaxation exercises will also help build your confidence that you can become a "good sleeper".

Here is a list of the muscles in the body that we are interested in relaxing.

Muscles in both arms and hands
Muscles in the neck and shoulder area
Muscles in the face - eyes, forehead, cheeks, jaw
Muscles in the stomach and abdomen
Muscles in the back
Muscles in the feet and legs

In between each set of tension and relaxation exercises you should concentrate on your breathing, to try and keep it regular, slow and comfortable for you. You will find that this helps you in relaxing. You will, of course, receive a period of training at the clinic in these procedures, and you may have a cassette which you can play at home to help you learn the exercises.

### Some points to remember:

1) You must practise. At the moment being relaxed does not come naturally to you, so you must be prepared to practise twice a day. Practise once during the daytime and a second time when in bed at night. You should also do some of the exercises to help you fall back to sleep again should you waken up during the night. Remember a relaxation session takes around 20 minutes.

2) Relaxation involves "letting go" so try not to rush through the exercises or tense muscles too hard so that they become painful or strained. It is only necessary that you notice the tension in the muscles, and then the relaxing of the tension as you let go. The general principle is to allow yourself to relax because you cannot make yourself relax.

3) Always make a point of focussing you attention on the changes occurring in the muscles. Notice the difference between a tensed and relaxed muscle. If you have a relaxation cassette, make sure that you have it within reach to switch off so that you don't have to get out of bed. You should, of course, put the light off before you start the exercises.

4) If you are not asleep at the end of the session, then you should continue you should continue exercising and relaxing those parts of your body which are still tense. The emphasis is on being patient, you will find that your body relaxes in time.

### IMPORTANT NOTE

The above instructions take time to have an effect on your sleep pattern. Patience and perseverance are essential. You must not expect to see any improvements during the first four weeks of the treatment programme, but by the beginning of the following week you will begin to see dramatic improvements and you will be relaxing yourself to sleep.

## OVERCOMING INSOMNIA BY CHANGING YOUR SLEEPING HABITS

The treatment programme described below has proved to be effective in helping people to get a better night's sleep and achieve a consistent sleep rhythm.

In order to be a "good sleeper" your bed and bedroom must serve as cues for successful and enjoyable sleep. At the present time, because of your insomnia, you probably experience some anxiety and frustration at the thought of going to bed and thus associate bedtime not with having a good night's sleep, but with a poor one. These worries themselves, of course, interfere with falling asleep and contribute to the insomnia problem.

In this treatment programme we want to get you to the stage where falling asleep quickly is associated with being in bed. We must therefore identify behaviours which interfere with falling asleep so that we can stop these and, instead, do things which will help us to fall asleep. We want to strengthen bed as a cue for sleep and remove all other activity which we sometimes associate with bedtime. The only exception to this being sexual activity.

Here is a list of some of the things which people are in the habit of doing in bed or in the bedroom.

Eating and drinking
Reading
Watching TV or listening to the radio
Smoking
Thinking about the past day
Planning the following day's activities
Discussing things

Although you may do some of these things because you are not falling over to sleep, they are all, in fact, unhelpful. They are all incompatible with falling asleep quickly.

The Treatment Programme:

The following set of instructions have been shown to bring about improvements in sleep patterns. You may find some of these instructions uncomfortable or unpleasant, but the treatment will not work unless you carry them out.

1) Go to your bedroom intending to sleep when you feel sleepy and stick to this even if it means that you stay up very late.

2) When you go to bed, put the light out, and try to relax. Do not use your bed for anything except sleep, that is, eating, reading, worrying, and so on. Sexual activity is the only exception. On such occasions the instructions are to be followed afterwards when you intend to sleep.

3) If you find yourself unable to fall asleep you must not lie there awake. Get up if you are not asleep within 20 minutes, and go into another room. This is the most difficult part, as it is very tempting to lie on for a while longer. Remember we are trying to associate your bed with falling asleep quickly.

4) In the other room you should do something relaxing such as reading or listening to music. Do not do anything energetic. Go back to bed again only when you feel sleepy.

5) If you still cannot fall asleep repeat step 3. You may have to get up many times in the one night, especially at first. If you waken later during the night and do not fall asleep quickly, then you should also repeat step 3 at this point.

6) Set the alarm for the same time each morning and get up then regardless of how much sleep you have had. This also is very important as it will help your body develop a consistent sleep rhythm.

7) Do not sleep during the daytime, not even for a short nap.

IMPORTANT NOTE

The above instructions take time to have an effect upon your sleep pattern. Patience and perseverance are essential. You must not expect to see any improvement during the first four weeks of the treatment programme, but by the beginning of the following week you will begin to see dramatic improvements and you will develop a consistent sleep rhythm.

## OVERCOMING INSOMNIA BY PARADOXICAL INTENTION

The treatment programme described below has proved to be effective in helping people to get a better night's sleep.

### The Problem

Many people who suffer from insomnia find that they become anxious and worried as bedtime draws near. Unpleasant memories of hours spent lying awake or tossing and turning come to mind, and the worrying prospect is that the same thing might happen again tonight. It is, therefore, not surprising that all sorts of things are tried out to combat sleeplessness. Some people take extra exercise, others a cup of warm milk, or read a boring book; others may try lying this way or that. It is the insomniac's hope that by doing such things he can make himself drowsy, and get to sleep.

Unfortunately, it seldom works, and sleeplessness continues, while frustration grows.

### The Treatment Programme

Paradoxical Intention is a difficult term to understand but the treatment programme is really very simple. If you ask a good sleeper how he manages to fall asleep, and stay asleep, you will find that he shrugs his shoulders and says that it just happens! This is a very important point - sleep is an INVOLUNTARY PROCESS. In other words we do not make it happen. It must happen naturally.

Sleep appears to be a member of a class of behaviours which cannot fully be placed under voluntary control. That is, although it is possible to arrange conditions most suitable for sleep (a bed, darkened room, constant temperature), there is a point beyond which the person cannot exert further control in a positive direction. You cannot make yourself fall asleep.

Research has shown that all effort at sleeping actually inhibits sleep - it stops it happening naturally. When you attempt to directly control an involuntary process you simply disrupt the process further. Therefore, in order to overcome the insomnia problem you must stop trying to make yourself fall asleep. You must stop worrying about getting to sleep.

You can do this by going to bed with the INTENTION of remaining awake. The paradoxical instruction then is that you should try to lie awake as long as is possible. As you do this you give up all effort at falling asleep, so sleep will come on NATURALLY. As it does so, however, and you begin to feel drowsy, you are to resist that drowsiness and try to remain awake - you will not be able to do this for all that long if you are really tired. The secret is to stop trying so hard and let it happen. Trying makes us tense instead of being relaxed.

### Some points to remember

1) There is now no need to worry at bedtime because your paradoxical intention is to stay awake, not to go to sleep. You can go to bed knowing that you will always succeed at this goal. You can see that the word paradoxical just means "the opposite" intention to what you might think.

2) Try to stay awake for as long as possible. Perseverance is required, especially since this programme is not one that is easy to follow.

3) Although you have to try to remain awake, do not move around or do things deliberately to prevent sleep. The idea is simply to remain awake, not become highly aroused. You are to resist falling over but still lie relaxed in bed with the light out.

All these points will be carefully explained to you at the clinic.

### IMPORTANT NOTE -

The above instructions take time to have an effect on your sleep pattern. Patience and perseverance are essential. You must not expect to see any improvement during the first four weeks of the treatment programme, but by the beginning of the following week you will begin to see dramatic improvements, and you will be relaxing yourself to sleep.

## OVERCOMING INSOMNIA BY IMAGERY RELIEF

The treatment programme described below has proved to be effective in helping people to get a better night's sleep.

### The Problem

Many people who suffer from insomnia find that they become anxious and worried as bedtime draws near. As the usual routine in the evening progresses and time moves on towards bedtime, anxiety increases at the thought of possibly not sleeping. Because of this, you may find yourself unsettled even before getting into bed, and, therefore, find it difficult to fall over to sleep. It is really a vicious circle starting with getting ready for going to bed, being a bit worried, not falling over, and then a dissatisfaction with the amount of sleep you've had when morning comes. As a result you worry even more the next evening during the run up to bedtime. This whole process should be seen as part of the insomnia problem. Evening activities become as much a part of it as does being in bed itself.

### The Treatment Programme

The purpose of the imagery relief programme is to stop you developing this vicious circle of anxiety and poor sleep.

The first stage is the construction of a list of bedtime activities. This list will be made up of the various things you do in the evening leading up to bedtime. You will do this at the clinic with the help of the therapist. It requires careful thought so as to achieve a list of activities in sequence, which is descriptive of what normally happens in your case. Eighteen items are included in this list which covers all important events leading up to bedtime and then being in bed.

The second stage is to develop a set of six mental images which you can readily call to your imagination. Again the therapist will help you select these and train you to visualise them.

The third stage involves pairing items from the list with these mental images. The purpose here is to dislodge the anxiety normally associated with the various events. With training and practice at the clinic, and at home, you will then be able to rehearse your bedtime routine in a frame of mind more compatible with sleep, and, therefore, your sleep pattern will improve.

### Some points to remember

1) You must practise. Imagery relief therapy is easier for some people to learn than for others. Individual abilities to visualise differ, but few derive benefit without regular practise. You are recommended to practise twice daily and the average session will last 20 minutes.

2) The best times for you to practise would be once early in the day, with a second session immediately after teatime. Always try to have your day's training completed by early evening.

3) Patience is required. Remember you are learning a skill.

### IMPORTANT NOTE

The above instructions take time to have an effect upon your sleep pattern. Patience and perseverance are essential. You must not expect to see any improvement during the first four weeks of the treatment programme, but by the beginning of the following week you will begin to see dramatic improvements and you will develop a satisfactory sleep pattern.

# APPENDIX C

## Rating Instrument Derived from Young and Beck's (1980) Cognitive Therapy Rating Scale

INDEPENDENT ASSESSMENTS OF AUDIO TAPES

1. Inter-Personal Effectiveness

0 - Therapist had poor inter-personal skills. Seemed hostile, demeaning, or in some other way destructive to the patient.

2 - Therapist did not seem destructive, but had significant inter-personal problems. At times, therapist appeared unnecessarily impatient, aloof, insincere, or had difficulty conveying confidence and competence.

4 - Therapist displayed a satisfactory degree of warmth, concern, confidence, genuineness and professionalism. No significant inter-personal problems.

6 - Therapist displayed optimal levels of warmth, concern, confidence, genuineness and professionalism, appropriate for this particular patient in this session.

2. Collaboration

0 - Therapist did not attempt to set up a collaboration with patient.

2 - Therapist attempted to collaborate with patient but had difficulty either defining a problem that the patient considered important or establishing rapport.

4 - Therapist was able to collaborate with patient, focus on a problem that both patient and therapist considered important, and establish rapport.

6 - Collaboration seemed excellent; therapist encouraged patient as much as possible to take an active role during the session so that they could function as a "team".

3. Strategy for Change

(Note: For this item, focus on the quality of the therapist's strategy for change, not on how effectively the strategy was implemented.)

0 - Therapist did not select appropriate techniques.

2 - Therapist selected appropriate techniques; however, either the overall strategy for bringing about change seemed vague or did not seem promising in helping the patient.

4 - Therapist seemed to have a generally coherent strategy for change that showed reasonable promise and incorporated appropriate techniques.

6 - Therapist followed a consistent strategy for change that seemed very promising and incorporated the most appropriate techniques.

4. Application of Techniques

(Note: For this item, focus on how skillfully the techniques were applied, not on how appropriate they were for the target problem.)

0 - Therapist did not apply any appropriate techniques.

2 - Therapist used appropriate techniques, but there were significant flaws in the way they were applied.

4 - Therapist applied appropriate techniques with moderate skill.

6 - Therapist very skillfully and resourcefully applied appropriate techniques.

All items to be rated on a 7 point scale, i.e.

0 1 2 3 4 5 6

If you believe the therapist falls between two of the descriptors (0 2 4 6), select the intervening odd number (1 3 5).

# APPENDIX D

## Daily Sleep Questionnaire

| | DAY 1 | DAY 2 | DAY 3 | DAY 4 | DAY 5 | DAY 6 | DAY 7 |
|---|---|---|---|---|---|---|---|
| Estimate how long it took you to fall asleep(hours/mins) | | | | | | | |
| About how long did you sleep altogether?(hours/mins) | | | | | | | |
| How many times did you wake up during last night and didn't fall back asleep easily? | | | | | | | |
| Were you bothered by repetitive thoughts running through your mind before falling asleep? Enter either 0,1,2,3 or 4 as follows.<br>0 not at all 1 2 moderately 3 4 very | | | | | | | |
| Rate how rested you feel after your sleep.<br>0 not at all 1 2 moderately 3 4 very | | | | | | | |
| Rate how enjoyable your sleep was.<br>0 not at all 1 2 moderately 3 4 very | | | | | | | |
| About what time did you go to bed? | | | | | | | |
| About what time did you waken (the last time)? | | | | | | | |
| About what time did you rise? | | | | | | | |
| *About how long did you spend napping during the day? (hours/mins) | | | | | | | |
| *Rate how irritable you felt during the day.<br>0 not at all 1 2 moderately 3 4 very | | | | | | | |

# APPENDIX E

## Sleep Assessment Device Scoring Sheet

NAME _____ DATE _____ REF. _____

TIME _____

| INT. | AWAKE | ASLEEP | TIME |
|---|---|---|---|
| 1 | | | |
| 2 | | | |
| 3 | | | |
| 4 | | | |
| 5 | | | 1HR |
| 6 | | | |
| 7 | | | |
| 8 | | | |
| 9 | | | |
| 10 | | | 2HR |
| 11 | | | |
| 12 | | | |
| 13 | | | |
| 14 | | | |
| 15 | | | 3HR |
| 16 | | | |
| 17 | | | |
| 18 | | | |
| 19 | | | |
| 20 | | | 4HR |
| 21 | | | |
| 22 | | | |
| 23 | | | |
| 24 | | | |
| 25 | | | 5HR |

| INT. | AWAKE | ASLEEP | TIME |
|---|---|---|---|
| 26 | | | |
| 27 | | | |
| 28 | | | |
| 29 | | | |
| 30 | | | 6HR |
| 31 | | | |
| 32 | | | |
| 33 | | | |
| 34 | | | |
| 35 | | | 7HR |
| 36 | | | |
| 37 | | | |
| 38 | | | |
| 39 | | | |
| 40 | | | 8HR |
| 41 | | | |
| 42 | | | |
| 43 | | | |
| 44 | | | |
| 45 | | | 9HR |
| 46 | | | |
| 47 | | | |

TIME _____

SOL _____ TOT _____

WAKE _____ % _____

## APPENDIX F

### Pre-Post Rating Scale Assessment Measures

1. Zung Anxiety Scale
2. Zung Depression Scale
3. Sleep Behaviour Rating Scale
4. Analogue Rating Scale

ZUNG SAS

| | Not at all or A little of the Time | Some of the time | Good Part of the time | Most of the time |
|---|---|---|---|---|
| 1. I feel more nervous and anxious than usual. | | | | |
| 2. I feel afraid for no reason at all. | | | | |
| 3. I get upset easily or feel panicky. | | | | |
| 4. I feel like I'm falling apart and going to pieces. | | | | |
| 5. I feel that everything is alright and nothing bad will happen. | | | | |
| 6. My arms and legs shake and tremble. | | | | |
| 7. I am bothered by headaches, neck and back pains. | | | | |
| 8. I feel weak and get tired easily. | | | | |
| 9. I feel calm and can sit still easily. | | | | |
| 10. I can feel my heart beating fast. | | | | |
| 11. I am bothered by dizzy spells. | | | | |
| 12. I have fainting spells or feel like it. | | | | |
| 13. I can breathe in and out easily. | | | | |
| 14. I get feelings of numbness and tingling in my fingers, toes. | | | | |
| 15. I am bothered by stomach aches or indigestion. | | | | |
| 16. I have to empty my bladder often. | | | | |
| 17. My hands are usually dry and warm. | | | | |
| 18. My face gets hot and blushes. | | | | |
| 19. I fall asleep easily and get a good night's rest. | | | | |
| 20. I have nightmares. | | | | |

ZUNG SDS

| | Not At All or A little of the Time | Some of the time | Good Part of the time | Most of the time |
|---|---|---|---|---|
| 1. I feel down-hearted and blue | | | | |
| 2. Morning is when I feel the best | | | | |
| 3. I have crying spells or feel like it | | | | |
| 4. I have trouble sleeping at night | | | | |
| 5. I eat as much as I used to | | | | |
| 6. I still enjoy sex | | | | |
| 7. I notice that I am losing weight | | | | |
| 8. I have trouble with constipation | | | | |
| 9. My heart beats faster than usual | | | | |
| 10. I get tired for no reason | | | | |
| 11. My mind is as clear as it used to be | | | | |
| 12. I find it easy to do the things I used to | | | | |
| 13. I am restless and cant keep still | | | | |
| 14. I feel hopeful about the future | | | | |
| 15. I am more irritable than usual | | | | |
| 16. I find it easy to make decisions | | | | |
| 17. I feel that I am useful and needed | | | | |
| 18. My life is pretty full | | | | |
| 19. I feel others would be better off if I were dead | | | | |
| 20. I still enjoy the things I used to do | | | | |

SLEEP BEHAVIOUR SBS

Please indicate how often you do the following things at bedtime and while in your bedroom by placing an X in the appropriate box.

Complete the form by considering what you would do in an average normal week.

| | Bedtime Behaviour | Never | Rarely | Sometimes | Often | Very Often. |
|---|---|---|---|---|---|---|
| | Reading a book or magazine | | | | | |
| | Watching T.V. | | | | | |
| | Listening to the radio | | | | | |
| | Speaking on the telephone | | | | | |
| * | Drinking tea/coffee etc. | | | | | |
| | Eating | | | | | |
| | Smoking | | | | | |
| | Talking about the day past | | | | | |
| | Talking about the following day | | | | | |
| * | Thinking about the day past | | | | | |
| * | Thinking about the following day | | | | | |
| | Thinking pleasant thoughts | | | | | |
| | Thinking unpleasant thoughts | | | | | |
| * | Drinking alcohol | | | | | |
| | Please also answer the following | | | | | |
| * | I go for a walk before retiring to bed | | | | | |
| * | I do some other form of exercise near bedtime | | | | | |
| * | I switch the light off as soon as I get into bed | | | | | |
| * | I leave the light on until I am almost asleep | | | | | |

ANALOGUE RATING SCALE

Below you will see a list of words which describe how a person feels or acts. You have to consider each description carefully and decide how true it could be of you during the past week.

Simply place a cross (X) anywhere along the line opposite each description to indicate how true a description it would be from "not at all" at one end to "extremely" at the other end. Obviously the closer you put the cross to the "extremely" end the more true you feel the description is and the closer to the "not at all" end the less true, and so on.

Go ahead and remember you can put your cross at any point along the line between "not at all" and "extremely".

| | | | |
|---|---|---|---|
| Energetic | not at all | ——————————————— | extremely |
| Worrying | not at all | ——————————————— | extremely |
| Coping well | not at all | ——————————————— | extremely |
| Healthy | not at all | ——————————————— | extremely |
| Irritable | not at all | ——————————————— | extremely |
| Downhearted/ sad | not at all | ——————————————— | extremely |
| Concentrating well | not at all | ——————————————— | extremely |
| Warm and affectionate | not at all | ——————————————— | extremely |
| Sleeping well | not at all | ——————————————— | extremely |
| Confident | not at all | ——————————————— | extremely |

PLEASE COMPLETE THIS FORM ON YOUR OWN

# APPENDIX G

## Credibility Evaluation Questionnaire

BORKOVEC AND NAU CEQ

| | not at all 0 | 1 | 2 | 3 | 4 | 5 | extremely 6 |
|---|---|---|---|---|---|---|---|
| 1. How sensible does this treatment seem to you? | | | | | | | |
| 2. How confident are you that this treatment will work for you? | | | | | | | |
| 3. How confident would you be in recommending this treatment to a friend who suffers from insomnia? | | | | | | | |
| 4. Do you feel satisfied that the therapist is giving you enough personal attention? | | | | | | | |
| 5. Do you feel that the therapist is warm and accepting of your problems? | | | | | | | |

# APPENDIX H

## Additional Tables for Chapter 8

| Variable/ Week | Relaxation | Stimulus Control | Paradox | Placebo | No Treatment |
|---|---|---|---|---|---|
| SOL mean | | | | | |
| B1 | 88.0(59.3) | 86.8(50.4) | 70.3(43.5) | 89.5(41.6) | 84.5(55.1) |
| B2 | 93.1(77.6) | 78.6(49.1) | 74.5(61.2) | 81.6(35.3) | 84.6(54.7) |
| SOL SD | | | | | |
| B1 | 56.6(37.7) | 38.8(19.4) | 49.1(32.8) | 46.8(27.8) | 56.2(45.4) |
| B2 | 43.3(33.8) | 44.9(28.2) | 46.8(33.7) | 42.6(29.7) | 51.0(28.2) |
| TOT mean | | | | | |
| B1 | 5.79(1.12) | 6.26(1.59) | 5.72(1.18) | 5.24(1.15) | 5.77(0.89) |
| B2 | 5.80(1.20) | 5.94(1.38) | 6.00(1.36) | 5.44(1.15) | 5.92(1.29) |
| TOT SD | | | | | |
| B1 | 1.32(0.53) | 1.16(0.36) | 1.25(0.53) | 1.20(0.63) | 1.35(0.54) |
| B2 | 1.50(0.79) | 1.18(0.56) | 1.41(0.77) | 1.26(0.82) | 1.15(0.63) |
| WAKE mean | | | | | |
| B1 | 1.61(1.28) | 0.97(1.14) | 1.27(1.05) | 1.59(1.22) | 1.13(1.32) |
| B2 | 1.51(1.37) | 0.89(0.90) | 1.05(0.74) | 1.92(1.52) | 0.89(0.88) |
| WAKE SD | | | | | |
| B1 | 0.96(0.64) | 0.68(0.59) | 0.81(0.51) | 0.94(0.54) | 0.72(0.51) |
| B2 | 0.95(0.78) | 0.67(0.59) | 0.83(0.43) | 0.95(0.82) | 0.64(0.62) |

TABLE (i) Mean scores (with SD scores bracketted) during the two pre-treatment baseline weeks (B1, B2) for the DSQ sleep pattern variables under each of the experimental conditions in the main study. SOL scores are in minutes, TOT scores are in hours, and WAKE scores are simple frequency counts

| Variable/ Week | Relaxation | Stimulus Control | Paradox | Placebo | No Treatment |
|---|---|---|---|---|---|
| REPTH | | | | | |
| B1 | 1.42(1.20) | 1.63(0.90) | 1.69(0.80) | 1.88(1.05) | 1.60(0.89) |
| B2 | 1.23(1.16) | 1.34(0.97) | 1.17(0.91) | 1.95(0.86) | 1.63(1.20) |
| RESTED | | | | | |
| B1 | 1.44(0.82) | 1.67(0.73) | 1.80(0.68) | 1.74(0.44) | 1.89(0.55) |
| B2 | 1.70(0.77) | 1.98(0.52) | 2.03(0.81) | 1.66(0.76) | 1.98(0.59) |
| ENJOY | | | | | |
| B1 | 1.43(0.82) | 2.07(0.74) | 1.94(0.87) | 1.73(0.55) | 2.05(0.58) |
| B2 | 1.68(0.75) | 2.15(0.73) | 2.13(0.83) | 1.68(0.74) | 2.08(0.40) |
| IRRIT | | | | | |
| B1 | 1.29(0.94) | 1.68(0.95) | 1.39(0.88) | 1.46(1.04) | 1.39(0.90) |
| B2 | 1.13(0.95) | 1.74(0.85) | 1.30(0.87) | 1.46(1.11) | 1.47(1.16) |
| NAPDAY | | | | | |
| B1 | 8.4(12.0) | 5.3( 8.8) | 10.8(20.9) | 7.0( 9.5) | 21.5(40.6) |
| B2 | 3.4( 5.6) | 3.0( 6.7) | 9.7(14.9) | 5.9(10.2) | 18.3(24.8) |

TABLE (ii) Mean scores (with SD scores bracketted) during the two pre-treatment baseline weeks (B1, B2) for the DSQ sleep quality variables under each of the experimental conditions in the main study. For NAPDAY, scores are in minutes; for the remaining variables scores refer to the 5-point rating scale (see Chapter 7)

| Treatment Group | n | Mean | SD |
|---|---|---|---|
| Relaxation | 10 | 25.17 | 2.93 |
| Stimulus control | 12 | 24.40 | 3.75 |
| Paradoxical intention | 11 | 24.89 | 3.89 |
| Placebo | 11 | 25.11 | 4.43 |

TABLE (iii) Mean and SD scores obtained within each treatment group in the main study on the Credibility Evaluation Questionnaire (CEQ)

| Treatment Group | Baseline Mean | Treatment Week 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 |
|---|---|---|---|---|---|---|---|---|---|
| Relaxation | 1.33(1.14) | 0.86(0.79) | 1.02(1.13) | 0.77(0.75) | 1.13(1.18) | 0.96(1.04) | 0.86(0.73) | 0.84(0.83) | 1.05(0.94) |
| Stimulus control | 1.48(0.83) | 1.35(1.02) | 1.16(0.77) | 1.44(0.95) | 1.04(0.88) | 1.10(0.70) | 1.24(0.90) | 1.16(0.99) | 1.20(0.86) |
| Paradoxical intention | 1.43(0.82) | 1.47(0.70) | 1.42(0.78) | 1.27(0.86) | 1.12(0.59) | 1.20(0.64) | 0.92(0.65) | 0.99(0.61) | 1.10(1.04) |
| Placebo | 1.92(0.86) | 1.61(0.76) | 1.78(0.83) | 1.61(0.87) | 1.86(0.78) | 1.66(0.97) | 1.78(0.96) | 1.75(0.90) | 1.90(0.81) |
| No treatment | 1.64(0.98) | 1.52(1.21) | 1.44(0.86) | 1.41(1.15) | 1.16(0.93) | 1.33(0.93) | 1.42(0.88) | 1.56(1.06) | 1.41(1.02) |

TABLE (iv) Mean and SD scores (bracketed) on the DSQ variable REPTH during each week of the experimental period, under each of the treatment conditions in the main study. Scores are on the 5-point rating scale (see Chapter 7)

| Treatment Group | Baseline Mean | Treatment Week 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 |
|---|---|---|---|---|---|---|---|---|---|
| Relaxation | 1.57(0.70) | 1.81(0.56) | 1.80(0.74) | 1.95(0.71) | 1.91(0.88) | 1.83(0.71) | 2.13(0.86) | 1.97(0.96) | 2.21(0.93) |
| Stimulus control | 1.82(0.52) | 2.06(0.54) | 2.14(0.57) | 1.91(0.42) | 2.13(0.42) | 1.96(0.64) | 2.14(0.59) | 2.15(0.77) | 2.15(0.52) |
| Paradoxical intention | 1.91(0.66) | 1.95(0.71) | 1.99(0.89) | 1.85(0.65) | 2.15(0.61) | 2.13(0.62) | 2.33(0.74) | 2.37(0.69) | 2.33(0.66) |
| Placebo | 1.69(0.58) | 1.59(0.76) | 1.68(0.71) | 1.64(0.92) | 1.61(0.60) | 1.72(0.80) | 1.71(0.75) | 1.85(0.82) | 2.01(0.84) |
| No treatment | 1.93(0.45) | 2.10(0.66) | 2.10(0.64) | 2.11(0.75) | 1.98(0.76) | 2.15(0.78) | 2.16(0.54) | 1.71(0.68) | 1.87(0.71) |

TABLE (v) Mean and SD scores (bracketted) for the DSQ variable RESTED during each week of the experimental period under each of the treatment conditions in the main study. Scores are on the 5-point rating scale (see Chapter 7)

| Treatment Group | Baseline Mean | Treatment Week 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 |
|---|---|---|---|---|---|---|---|---|---|
| Relaxation | 1.55(0.67) | 1.79(0.67) | 1.88(0.78) | 2.03(0.72) | 1.96(0.97) | 1.86(0.79) | 2.24(1.02) | 2.02(0.93) | 2.13(0.95) |
| Stimulus control | 2.11(0.67) | 2.39(0.54) | 2.37(0.67) | 2.23(0.70) | 2.47(0.71) | 2.37(0.82) | 2.28(0.82) | 2.36(0.90) | 2.52(0.82) |
| Paradoxical intention | 2.03(0.81) | 2.03(0.77) | 2.10(0.88) | 2.04(0.81) | 2.41(0.60) | 2.34(0.68) | 2.32(0.73) | 2.37(0.76) | 2.36(0.58) |
| Placebo | 1.70(0.54) | 1.52(0.58) | 1.72(0.71) | 1.67(0.84) | 1.91(0.64) | 1.84(0.90) | 1.73(0.78) | 1.95(0.82) | 2.12(0.93) |
| No treatment | 2.06(0.36) | 2.04(0.61) | 2.30(0.51) | 2.24(0.68) | 2.23(0.92) | 2.30(0.75) | 2.34(0.53) | 1.94(0.75) | 2.16(0.66) |

TABLE (vi) Mean and Sd scores (bracketted) for the DSQ variable ENJOY during each week of the experimental period, under each of the treatment conditionsin the main study. Scores are on the 5-point rating scale (see Chapter 7)

| Treatment Group | Baseline Mean | Treatment Week 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 |
|---|---|---|---|---|---|---|---|---|---|
| Relaxation | 1.21(0.91) | 0.96(0.80) | 1.16(1.17) | 0.94(0.84) | 1.13(1.13) | 0.88(1.03) | 0.73(0.80) | 0.81(0.84) | 0.92(1.09) |
| Stimulus control | 1.70(0.83) | 1.48(0.81) | 1.52(0.78) | 1.42(0.93) | 1.34(0.78) | 1.33(1.08) | 1.31(1.05) | 1.22(1.19) | 1.42(1.14) |
| Paradoxical intention | 1.33(0.82) | 1.22(0.81) | 1.30(0.94) | 1.06(0.72) | 0.76(0.72) | 1.14(0.85) | 1.09(0.91) | 1.02(0.83) | 1.06(0.79) |
| Placebo | 1.46(1.04) | 1.47(1.15) | 1.33(1.25) | 1.29(1.03) | 1.36(0.99) | 1.46(1.22) | 1.32(1.02) | 1.14(0.95) | 1.14(0.94) |
| No treatment | 1.43(0.98) | 1.36(0.98) | 1.37(1.14) | 1.48(0.96) | 1.38(0.94) | 1.11(0.68) | 1.27(0.81) | 1.44(0.98) | 1.18(0.96) |

TABLE (vii) Mean and SD scores (bracketted) for the DSQ variable IRRIT during each week of the experimental period, under each of the treatment conditions in the main study. Scores are on the 5-point rating scale (see Chapter 7)

| Treatment Group | Baseline Mean | Treatment Week 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 |
|---|---|---|---|---|---|---|---|---|---|
| Relaxation | 5.9( 8.1) | 7.0(10.6) | 7.4(18.4) | 1.5( 3.7) | 10.5(23.3) | 5.5(14.8) | 9.1(20.2) | 6.7(17.6) | 9.3(18.2) |
| Stimulus control | 4.1( 5.3) | 2.6( 5.6) | 0.5( 1.0) | 1.8( 4.6) | 0.2( 0.6) | 0.8( 3.1) | 0.9( 1.9) | 1.8( 4.7) | 0.0( 0.0) |
| Paradoxical intention | 10.1(17.3) | 8.0(16.4) | 8.9(17.3) | 12.5(21.5) | 4.4( 6.0) | 9.5(11.5) | 9.6(14.9) | 6.2( 9.8) | 3.3( 6.1) |
| Placebo | 6.5( 7.6) | 7.6(11.0) | 3.8( 6.1) | 6.8(12.5) | 2.8( 5.1) | 5.3(10.3) | 5.3(11.8) | 5.2( 9.5) | 3.8( 8.5) |
| No treatment | 19.9(30.5) | 18.4(31.7) | 15.5(26.0) | 14.7(26.7) | 19.2(32.1) | 21.6(34.0) | 25.4(46.8) | 22.5(32.9) | 16.5(31.1) |

TABLE (viii) Mean and SD scores (bracketted) for the DSQ variable NAPDAY during each week of the experimental period, under each of the treatment conditions in the main study. Scores are on the 5-point rating scale (see Chapter 7)

# APPENDIX I

## Additional Tables for Chapter 9

| Treatment/ Variable | Baseline Mean | Final Therapy Week | Follow Up 6 Week | 3 Month | 6 Month | 17 Month |
|---|---|---|---|---|---|---|
| RELAXATION | (n= 14 | 13 | 11 | 8 | 10 | 9 ) |
| SOL mean | 90.5(57.8) | 57.4(25.0) | 48.1(31.1) | 45.3(22.8) | 37.5(27.9) | 45.1(24.5) |
| SOL SD | 50.0(32.6) | 37.4(30.8) | 27.9(21.9) | 30.2(30.4) | 16.3(21.6) | 30.6(29.4) |
| TOT mean | 5.79(0.99) | 6.41(0.93) | 6.73(1.07) | 6.65(1.31) | 6.81(0.80) | 6.61(1.35) |
| TOT SD | 1.41(0.52) | 1.32(0.83) | 1.02(0.65) | 1.07(0.67) | 0.92(0.69) | 1.45(0.93) |
| WAKE mean | 1.56(1.30) | 1.23(1.01) | 0.81(0.58) | 0.80(0.95) | 0.57(0.50) | 1.00(1.09) |
| WAKE SD | 0.96(0.66) | 0.75(0.50) | 0.58(0.32) | 0.68(0.40) | 0.51(0.36) | 0.73(0.66) |
| REPTH | 1.33(1.14) | 1.05(0.94) | 1.05(1.08) | 0.79(1.06) | 0.90(0.85) | 1.14(1.05) |
| RESTED | 1.57(0.70) | 2.21(0.95) | 2.32(0.72) | 2.34(1.02) | 2.21(1.03) | 2.32(0.68) |
| ENJOY | 1.55(0.67) | 2.13(0.95) | 2.37(0.67) | 2.24(0.99) | 2.24(0.91) | 2.24(0.68) |
| IRRIT | 1.21(0.91) | 0.92(1.09) | 0.75(0.86) | 0.53(0.84) | 0.98(0.89) | 0.67(0.70) |
| NAPDAY | 5.9( 8.1) | 9.3(18.1) | 18.3(44.4) | 0.5( 1.4) | 23.4(56.0) | 8.0(14.4) |
| STIMULUS CONTROL | (n= 14 | 12 | 10 | 7 | 6 | 9 ) |
| SOL mean | 82.7(48.5) | 31.1(27.1) | 25.6(13.9) | 29.9(29.6) | 37.3(32.1) | 57.3(44.2) |
| SOL SD | 41.9(21.0) | 19.0(18.2) | 15.9(19.5) | 19.0(27.2) | 22.3(38.7) | 32.0(34.1) |
| TOT mean | 6.10(1.44) | 6.56(1.14) | 7.05(1.13) | 7.05(0.84) | 6.98(0.66) | 6.76(1.09) |
| TOT SD | 1.17(0.41) | 1.07(0.70) | 1.06(0.31) | 0.66(0.48) | 1.05(1.01) | 1.05(0.90) |
| WAKE mean | 0.93(0.97) | 0.44(0.90) | 0.55(0.87) | 0.65(1.07) | 0.79(0.83) | 0.63(0.87) |
| WAKE SD | 0.67(0.48) | 0.16(0.22) | 0.34(0.40) | 0.54(0.77) | 0.29(0.24) | 0.35(0.39) |
| REPTH | 1.49(0.83) | 1.20(0.86) | 0.70(0.43) | 0.26(0.43) | 1.55(1.02) | 1.84(1.13) |
| RESTED | 1.83(0.65) | 2.15(0.52) | 2.38(0.72) | 2.26(0.74) | 2.33(0.84) | 2.22(0.90) |
| ENJOY | 2.11(1.08) | 2.52(0.82) | 2.66(0.70) | 2.64(0.74) | 2.39(0.82) | 2.57(0.68) |
| IRRIT | 1.71(0.83) | 1.42(1.14) | 0.93(0.92) | 1.13(1.10) | 1.67(1.07) | 1.68(0.92) |
| NAPDAY | 4.1( 5.3) | 0.0( 0.0) | 0.8( 1.6) | 0.0( 0.0) | 2.5( 6.1) | 3.3( 8.6) |
| PARADOX | (n= 15 | 13 | 12 | 10 | 8 | 12 ) |
| SOL mean | 72.4(49.9) | 35.8(39.3) | 36.4(29.9) | 28.7(40.7) | 40.7(41.4) | 37.9(24.7) |
| SOL SD | 47.9(27.2) | 14.2(15.8) | 21.8(12.1) | 14.7(11.1) | 23.9(21.5) | 23.7(22.4) |
| TOT mean | 5.86(1.23) | 6.54(0.77) | 7.00(0.94) | 7.10(0.79) | 7.14(0.49) | 6.83(0.95) |
| TOT SD | 1.33(0.49) | 0.78(0.35) | 0.94(0.36) | 1.22(0.51) | 0.98(0.37) | 1.08(0.63) |
| WAKE mean | 1.16(0.86) | 0.53(0.42) | 0.62(0.63) | 0.43(0.47) | 0.73(0.79) | 0.51(0.35) |
| WAKE SD | 0.82(0.44) | 0.61(0.42) | 0.62(0.57) | 0.59(0.55) | 0.78(0.65) | 0.76(0.78) |
| REPTH | 1.43(0.43) | 1.10(1.04) | 0.98(0.58) | 1.00(0.72) | 0.97(0.55) | 1.52(0.85) |
| RESTED | 1.92(0.67) | 2.33(0.66) | 2.37(0.64) | 2.50(0.58) | 2.61(0.80) | 2.30(0.66) |
| ENJOY | 2.03(0.81) | 2.36(0.66) | 2.51(0.61) | 2.59(0.53) | 2.70(0.88) | 2.43(0.76) |
| IRRIT | 1.33(0.82) | 1.06(0.79) | 0.90(0.88) | 0.97(0.80) | 1.11(0.94) | 1.10(0.91) |
| NAPDAY | 10.3(17.3) | 3.3( 6.1) | 7.4(10.0) | 2.7( 4.8) | 5.9(11.5) | 5.3( 6.2) |

TABLE (ix) Mean and SD scores (bracketted) for each of the DSQ variables at baseline, final therapy week, and at each follow-up assessment. Results are presented separately for each of the three active treatment conditions

| Pre-treatment Measure | df | F Ratio | F Prob | Significance Level |
|---|---|---|---|---|
| ZAS | (4,61) | 1.245 | 0.302 | NS |
| ZDS | (4,62) | 0.578 | 0.680 | NS |
| SBRS | (4,61) | 1.451 | 0.228 | NS |
| ARS | (4,62) | 0.430 | 0.787 | NS |
| ARS 1 | (4,62) | 0.501 | 0.735 | NS |
| ARS 2 | (4,62) | 0.407 | 0.803 | NS |
| ARS 3 | (4,58) | 0.845 | 0.502 | NS |
| ARS 4 | (4,62) | 1.130 | 0.351 | NS |
| ARS 5 | (4,62) | 0.583 | 0.676 | NS |
| ARS 6 | (4,62) | 0.080 | 0.988 | NS |
| ARS 7 | (4,62) | 0.545 | 0.704 | NS |
| ARS 8 | (4,61) | 0.453 | 0.770 | NS |
| ARS 9 | (4,62) | 0.800 | 0.530 | NS |
| ARS 10 | (4,62) | 0.180 | 0.948 | NS |

TABLE (x) ONEWAY ANOVAs across the five experimental groups in the main study on each rating scale assessment measures at pre-treatment

| Pre-treatment Measure | Male | Female | T | df | Prob | Sig Level |
|---|---|---|---|---|---|---|
| ZAS | 37.3 | 39.3 | -0.72 | 64 | 0.475 | NS |
| ZDS | 41.6 | 43.0 | -0.44 | 65 | 0.662 | NS |
| SBRS | 44.6 | 46.0 | -0.57 | 64 | 0.573 | NS |
| ARS | 51.2 | 50.9 | 0.07 | 65 | 0.942 | NS |
| ARS 1 | 4.18 | 4.50 | -0.46 | 65 | 0.646 | NS |
| ARS 2 | 5.25 | 5.40 | -0.20 | 65 | 0.844 | NS |
| ARS 3 | 5.43 | 5.34 | 0.12 | 61 | 0.908 | NS |
| ARS 4 | 5.75 | 5.78 | -0.05 | 65 | 0.963 | NS |
| ARS 5 | 4.29 | 4.18 | 0.13 | 65 | 0.901 | NS |
| ARS 6 | 4.30 | 4.02 | 0.32 | 65 | 0.750 | NS |
| ARS 7 | 4.30 | 4.84 | -0.74 | 65 | 0.404 | NS |
| ARS 8 | 5.78 | 5.68 | 0.14 | 64 | 0.889 | NS |
| ARS 9 | 2.40 | 2.00 | 0.72 | 65 | 0.477 | NS |
| ARS 10 | 4.52 | 4.50 | 0.03 | 65 | 0.974 | NS |

TABLE (xi) Independent T-TESTS of sex differences on each of the rating scale assessment measures at pre-treatment

| Treatment/ Variable | pre | post | SE diff | T | df | Prob | Sig Level |
|---|---|---|---|---|---|---|---|
| RELAXATION | | | | | | | |
| ARS 1 | 4.88 | 5.58 | 0.52 | -1.34 | 12 | .206 | NS |
| ARS 2 | 5.83 | 4.56 | 0.42 | 3.04 | 12 | .010 | ** |
| ARS 3 | 4.55 | 6.35 | 0.64 | -2.82 | 12 | .016 | * |
| ARS 4 | 6.70 | 6.88 | 0.44 | -0.40 | 12 | .697 | NS |
| ARS 5 | 3.76 | 3.59 | 0.73 | 0.23 | 12 | .820 | NS |
| ARS 6 | 4.26 | 4.23 | 1.42 | 0.02 | 12 | .983 | NS |
| ARS 7 | 3.98 | 5.64 | 0.35 | -4.77 | 12 | .000 | *** |
| ARS 8 | 6.95 | 7.36 | 0.37 | -1.07 | 12 | .306 | NS |
| ARS 9 | 2.65 | 5.81 | 0.76 | -4.19 | 12 | .001 | ** |
| ARS 10 | 4.27 | 5.32 | 0.62 | -1.68 | 12 | .119 | NS |
| STIMULUS CONTROL | | | | | | | |
| ARS 1 | 3.92 | 4.37 | 0.77 | -0.58 | 12 | .571 | NS |
| ARS 2 | 4.99 | 4.29 | 0.95 | 0.74 | 11 | .476 | NS |
| ARS 3 | 6.10 | 5.73 | 0.54 | 0.69 | 10 | .508 | NS |
| ARS 4 | 5.26 | 5.62 | 0.79 | -0.45 | 12 | .661 | NS |
| ARS 5 | 5.45 | 4.32 | 1.14 | 1.00 | 12 | .336 | NS |
| ARS 6 | 4.21 | 2.85 | 0.93 | 1.47 | 12 | .167 | NS |
| ARS 7 | 4.67 | 4.71 | 0.68 | -0.06 | 12 | .956 | NS |
| ARS 8 | 5.32 | 6.17 | 0.56 | -1.52 | 12 | .155 | NS |
| ARS 9 | 1.92 | 4.74 | 0.73 | -3.88 | 12 | .002 | ** |
| ARS 10 | 5.08 | 4.86 | 0.80 | 0.27 | 12 | .793 | NS |
| PARADOX | | | | | | | |
| ARS 1 | 4.51 | 4.63 | 0.49 | -0.60 | 13 | .840 | NS |
| ARS 2 | 4.45 | 3.65 | 0.60 | 1.34 | 13 | .202 | NS |
| ARS 3 | 6.00 | 6.49 | 0.49 | -1.00 | 12 | .337 | NS |
| ARS 4 | 6.04 | 6.92 | 0.70 | -1.26 | 13 | .229 | NS |
| ARS 5 | 3.62 | 3.36 | 0.82 | 0.31 | 13 | .769 | NS |
| ARS 6 | 4.22 | 3.05 | 0.83 | 1.42 | 13 | .179 | NS |
| ARS 7 | 5.36 | 5.87 | 0.64 | -0.81 | 13 | .434 | NS |
| ARS 8 | 5.57 | 5.78 | 0.70 | -0.30 | 12 | .771 | NS |
| ARS 9 | 2.59 | 5.76 | 0.73 | -4.34 | 13 | .001 | *** |
| ARS 10 | 4.53 | 6.26 | 0.65 | -2.68 | 13 | .019 | * |
| PLACEBO | | | | | | | |
| ARS 1 | 4.63 | 4.71 | 0.68 | -0.14 | 10 | .894 | NS |
| ARS 2 | 5.09 | 3.94 | 0.71 | 1.62 | 10 | .139 | NS |
| ARS 3 | 6.07 | 5.72 | 0.98 | 0.26 | 9 | .800 | NS |
| ARS 4 | 6.14 | 6.87 | 0.68 | -1.07 | 10 | .312 | NS |
| ARS 5 | 3.59 | 2.77 | 1.02 | 0.80 | 10 | .442 | NS |
| ARS 6 | 2.95 | 2.50 | 0.88 | 0.51 | 10 | .622 | NS |
| ARS 7 | 5.44 | 6.03 | 0.58 | -1.02 | 10 | .336 | NS |
| ARS 8 | 7.05 | 6.19 | 0.78 | 1.11 | 10 | .296 | NS |
| ARS 9 | 1.36 | 4.49 | 1.27 | -2.46 | 10 | .036 | * |
| ARS 10 | 4.92 | 5.50 | 1.08 | -0.54 | 10 | .605 | NS |
| WAITING LIST | | | | | | | |
| ARS 1 | 5.05 | 4.62 | 0.19 | 2.25 | 12 | .048 | * |
| ARS 2 | 5.37 | 5.39 | 0.54 | -0.03 | 12 | .974 | NS |
| ARS 3 | 5.47 | 5.23 | 0.52 | 0.46 | 10 | .656 | NS |
| ARS 4 | 5.57 | 5.83 | 0.42 | -0.61 | 12 | .556 | NS |
| ARS 5 | 3.90 | 4.18 | 0.95 | -0.30 | 12 | .773 | NS |
| ARS 6 | 4.57 | 4.17 | 0.64 | 0.63 | 12 | .546 | NS |
| ARS 7 | 5.00 | 4.78 | 0.28 | 0.78 | 12 | .452 | NS |
| ARS 8 | 6.08 | 5.57 | 0.29 | 1.75 | 12 | .111 | NS |
| ARS 9 | 2.40 | 1.56 | 0.56 | 1.49 | 12 | .167 | NS |
| ARS 10 | 4.77 | 4.13 | 0.70 | 0.77 | 12 | .461 | NS |

TABLE (xii) Paired T-TESTs on pre and post-treatment Analogue Rating Scale assessments presented separately for each experimental condition

| Sleep Behaviour Rating Scale | Z | 2-tailed Prob | Sig Level |
|---|---|---|---|
| Item 1 | -2.37 | .018 | * |
| Item 2 | -1.60 | .109 | NS |
| Item 3 | -1.83 | .068 | NS |
| Item 4 | -1.34 | .180 | NS |
| Item 5 | -1.60 | .109 | NS |
| Item 6 | -1.10 | .273 | NS |
| Item 7 | -0.36 | .715 | NS |
| Item 8 | -0.94 | .345 | NS |
| Item 9 | -0.24 | .813 | NS |
| Item 10 | -0.63 | .529 | NS |
| Item 11 | -0.51 | .612 | NS |
| Item 12 | -0.91 | .361 | NS |
| Item 13 | -1.48 | .138 | NS |
| Item 14 | -1.34 | .180 | NS |
| Item 15 | -1.60 | .109 | NS |
| Item 16 | -0.73 | .465 | NS |
| Item 17 | -2.01 | .028 | * |
| Item 18 | -2.01 | .028 | * |

TABLE (xiii) WILCOXON tests on pre and post-treatment scores for each item of the Sleep Behaviour Rating Scale for subjects in the stimulus control group (n = 11; 3 cases with missing data) * $p \leq .05$

| Variable | df | F Ratio | F Prob | Pair | Scheffe .05 | Range .10 | Planned t .01 |
|---|---|---|---|---|---|---|---|
| ZAS | (4,57) | 0.970 | 0.432 | - | | | |
| ZDS | (4,57) | 3.994 | 0.006 | 1 v 5 | | * | * |
| | | | | 2 v 5 | | | * |
| SBRS | (4,57) | 3.158 | 0.021 | 2 v 4 | | * | * |
| | | | | 2 v 3 | | | * |
| | | | | 2 v 5 | | | * |
| | | | | 2 v 1 | | | * |
| ARS | (4,59) | 2.020 | 0.104 | - | | | |
| ARS 1 | (4,59) | 1.129 | 0.352 | - | | | |
| ARS 2 | (4,58) | 0.556 | 0.670 | - | | | |
| ARS 3 | (4,53) | 2.259 | 0.076 | - | | | |
| ARS 4 | (4,59) | 0.246 | 0.911 | - | | | |
| ARS 5 | (4,59) | 0.348 | 0.844 | - | | | |
| ARS 6 | (4,59) | 0.331 | 0.856 | - | | | |
| ARS 7 | (4,59) | 1.794 | 0.143 | - | | | |
| ARS 8 | (4,58) | 1.440 | 0.233 | - | | | |
| ARS 9 | (4,59) | 4.320 | 0.004 | 1 V 5 | * | * | * |
| | | | | 3 V 5 | * | * | * |
| | | | | 4 V 5 | * | * | * |
| | | | | 2 V 5 | | * | * |
| ARS 10 | (4,59) | 1.534 | 0.205 | - | | | |

TABLE (xiv) ONEWAY ANOVAs on pre-post change scores for each of the rating scale assessments across the experimental conditions (Relaxation=1, stimulus control=2, paradox=3, placebo=4, no treatment=5). Results of range tests are also presented

| | AGE | DURATION | MEDPAST | ZASPRE | ZDSPRE |
|---|---|---|---|---|---|
| SOLB | .1162 | .1739 | .1629 | -.0420 | -.0069 |
| SOLSDB | .1239 | .0652 | -.0303 | .1014 | .1026 |
| TOTB | -.1928 | -.1105 | .0059 | .0875 | .1502 |
| TOTSDB | -.2417 | -.1773 | -.2955* | .0852 | .2299 |
| WAKEB | .1426 | .1195 | .0107 | -.1853 | -.2164 |
| WAKESDB | -.0961 | -.0659 | -.3040* | .0336 | .0215 |
| REPTHB | -.0877 | -.0111 | -.0358 | .4444** | .3108* |
| RESTEDB | .2103 | .1989 | .2856* | -.2036 | -.3228* |
| ENJOYB | .0732 | .1037 | .2169 | -.1098 | -.1987 |
| IRRITB | -.2817* | -.2470 | -.3590* | .6502** | .6413** |
| NAPDAYB | -.0605 | -.0071 | -.0274 | .0377 | -.0502 |
| SOLCH | .1150 | .0450 | .0156 | -.1208 | .0889 |
| SOLSDCH | -.0532 | -.0618 | -.1432 | .1411 | .1637 |
| TOTCH | -.1063 | -.0588 | .0691 | .1976 | .0802 |
| TOTSDCH | -.1345 | -.0988 | -.0883 | .0575 | .1506 |
| WAKECH | .0622 | -.0629 | -.1466 | -.0007 | -.0082 |
| WAKESDCH | -.0700 | -.2052 | -.3377* | .2139 | .1859 |
| REPTHCH | .0924 | -.3021* | -.3571* | .1976 | .2991* |
| RESTCH | -.1964 | -.0146 | .1210 | .0361 | -.0363 |
| ENJCH | -.2120 | .0675 | .1851 | .0225 | -.0545 |
| IRRITCH | .0632 | .0274 | -.1264 | .2228 | .1704 |
| NAPCH | -.2071 | -.2605 | -.1226 | -.0523 | -.0973 |
| ZASCH | .2091 | .0356 | -.0599 | .3497* | .2735 |
| ZDSCH | .0348 | -.1414 | -.2205 | .1862 | .3491* |

-* - SIGNIF. LE .01 ** - SIGNIF. LE .001

TABLE (xv) Predictor variables and their relationship with baseline and change score (outcome) measures. PEARSON co-efficients and probability value are presented

T-TEST

GROUP 1 - MEDS EQ 1.
GROUP 2 - MEDS EQ 2.

| VARIABLE | NUMBER OF CASES | MEAN | STANDARD DEVIATION | STANDARD ERROR | F VALUE | 2-TAIL PROB. | POOLED VARIANCE ESTIMATE T VALUE | DEGREES OF FREEDOM | 2-TAIL PROB. | SEPARATE VARIANCE ESTIMATE T VALUE | DEGREES OF FREEDOM | 2-TAIL PROB. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| AGE | | | | | | | | | | | | |
| GROUP 1 | 15 | 50.2000 | 13.115 | 3.903 | 1.11 | 0.869 | 1.26 | 82 | 0.206 | 1.32 | 21.35 | 0.204 |
| GROUP 2 | 69 | 44.4493 | 15.959 | 1.921 | | | | | | | | |
| DURATION DURATION OF INSOMNIA(YRS) | | | | | | | | | | | | |
| GROUP 1 | 15 | 13.8667 | 11.407 | 2.945 | 1.04 | 1.000 | 0.75 | 82 | 0.455 | 0.76 | 20.82 | 0.465 |
| GROUP 2 | 69 | 11.3913 | 11.624 | 1.399 | | | | | | | | |
| SOLB | | | | | | | | | | | | |
| GROUP 1 | 15 | 93.7333 | 52.366 | 13.784 | 1.27 | 0.503 | 1.09 | 82 | 0.279 | 1.01 | 19.11 | 0.327 |
| GROUP 2 | 69 | 80.6739 | 47.451 | 5.712 | | | | | | | | |
| SOLSDB | | | | | | | | | | | | |
| GROUP 1 | 15 | 60.1000 | 40.839 | 10.544 | 2.64 | 0.047 | 0.33 | 82 | 0.739 | 0.45 | 32.45 | 0.654 |
| GROUP 2 | 69 | 54.1232 | 44.305 | 7.982 | | | | | | | | |
| TOTB | | | | | | | | | | | | |
| GROUP 1 | 15 | 5.5837 | 1.247 | 0.322 | 1.15 | 0.819 | 0.03 | 82 | 0.972 | 0.04 | 21.57 | 0.972 |
| GROUP 2 | 69 | 5.5707 | 1.335 | 0.161 | | | | | | | | |
| TOTSDB | | | | | | | | | | | | |
| GROUP 1 | 15 | 1.3823 | 0.685 | 0.177 | 1.92 | 0.078 | 0.77 | 82 | 0.441 | 0.63 | 17.30 | 0.542 |
| GROUP 2 | 69 | 1.2451 | 0.494 | 0.059 | | | | | | | | |
| WAKEB | | | | | | | | | | | | |
| GROUP 1 | 15 | 1.2330 | 0.968 | 0.250 | 1.24 | 0.650 | -0.37 | 82 | 0.709 | -0.40 | 22.40 | 0.693 |
| GROUP 2 | 69 | 1.3470 | 1.088 | 0.131 | | | | | | | | |
| WAKESDB | | | | | | | | | | | | |
| GROUP 1 | 15 | 0.7353 | 0.584 | 0.151 | 1.15 | 0.672 | -1.02 | 82 | 0.309 | -0.98 | 19.67 | 0.348 |
| GROUP 2 | 69 | 0.8943 | 0.545 | 0.066 | | | | | | | | |
| REPTHB | | | | | | | | | | | | |
| GROUP 1 | 15 | 1.4560 | 1.022 | 0.264 | 1.21 | 0.584 | 0.45 | 82 | 0.652 | 0.43 | 19.36 | 0.676 |
| GROUP 2 | 69 | 1.5359 | 0.931 | 0.112 | | | | | | | | |
| RESTLDB | | | | | | | | | | | | |
| GROUP 1 | 15 | 1.9150 | 0.530 | 0.137 | 1.19 | 0.746 | 1.12 | 82 | 0.267 | 1.18 | 21.91 | 0.260 |
| GROUP 2 | 69 | 1.7334 | 0.579 | 0.070 | | | | | | | | |
| ENJOYB | | | | | | | | | | | | |
| GROUP 1 | 15 | 2.0020 | 0.545 | 0.141 | 1.43 | 0.469 | 0.89 | 82 | 0.374 | 1.00 | 23.56 | 0.333 |
| GROUP 2 | 69 | 1.8406 | 0.650 | 0.078 | | | | | | | | |
| IRRITB | | | | | | | | | | | | |
| GROUP 1 | 15 | 1.7053 | 0.814 | 0.210 | 1.28 | 0.631 | 1.45 | 82 | 0.151 | 1.57 | 22.51 | 0.135 |
| GROUP 2 | 69 | 1.3322 | 0.920 | 0.111 | | | | | | | | |
| NAPDAYB | | | | | | | | | | | | |
| GROUP 1 | 15 | 5.9000 | 7.935 | 2.049 | 4.55 | 0.003 | -0.78 | 82 | 0.436 | -1.22 | 44.09 | 0.230 |
| GROUP 2 | 69 | 9.4130 | 16.923 | 2.037 | | | | | | | | |
| SOLCH | | | | | | | | | | | | |
| GROUP 1 | 14 | 30.3214 | 39.829 | 10.645 | 1.28 | 0.643 | 0.29 | 67 | 0.698 | 0.42 | 22.32 | 0.681 |
| GROUP 2 | 55 | 25.1727 | 45.146 | 6.087 | | | | | | | | |
| SOLSDCH | | | | | | | | | | | | |
| GROUP 1 | 14 | 20.1071 | 41.142 | 10.996 | 3.67 | 0.013 | -0.20 | 67 | 0.839 | -0.29 | 40.18 | 0.772 |
| GROUP 2 | 55 | 24.5636 | 78.800 | 10.625 | | | | | | | | |
| TOTCH | | | | | | | | | | | | |
| GROUP 1 | 14 | -0.3575 | 0.519 | 0.139 | 3.56 | 0.015 | 0.13 | 67 | 0.899 | 0.18 | 39.45 | 0.858 |
| GROUP 2 | 55 | -0.3721 | 0.960 | 0.132 | | | | | | | | |
| TOTSDCH | | | | | | | | | | | | |
| GROUP 1 | 14 | 0.2479 | 0.776 | 0.207 | 1.64 | 0.204 | 0.16 | 67 | 0.875 | 0.14 | 17.24 | 0.894 |
| GROUP 2 | 55 | 0.2175 | 0.606 | 0.082 | | | | | | | | |
| WAKECH | | | | | | | | | | | | |
| GROUP 1 | 14 | 0.1236 | 0.617 | 0.165 | 1.47 | 0.449 | -1.78 | 68 | 0.080 | -2.00 | 23.59 | 0.041 |
| GROUP 2 | 56 | 0.5093 | 0.749 | 0.100 | | | | | | | | |
| WAKESDCH | | | | | | | | | | | | |
| GROUP 1 | 14 | 0.1196 | 0.505 | 0.135 | 1.29 | 0.641 | -0.78 | 67 | 0.440 | -0.84 | 22.33 | 0.414 |
| GROUP 2 | 55 | 0.2501 | 0.573 | 0.077 | | | | | | | | |
| REPTHCH | | | | | | | | | | | | |
| GROUP 1 | 14 | 0.2429 | 0.611 | 0.163 | 1.74 | 0.275 | 0.04 | 68 | 0.970 | 0.05 | 23.59 | 0.965 |
| GROUP 2 | 56 | 0.2340 | 0.804 | 0.107 | | | | | | | | |
| RESTCH | | | | | | | | | | | | |
| GROUP 1 | 14 | -0.1857 | 0.418 | 0.112 | 2.52 | 0.070 | 0.48 | 68 | 0.630 | 0.63 | 31.57 | 0.532 |
| GROUP 2 | 56 | -0.2758 | 0.663 | 0.089 | | | | | | | | |
| ENJCH | | | | | | | | | | | | |
| GROUP 1 | 14 | -0.2214 | 0.470 | 0.126 | 2.18 | 0.122 | 0.60 | 67 | 0.549 | 0.76 | 29.29 | 0.456 |
| GROUP 2 | 55 | -0.3399 | 0.695 | 0.094 | | | | | | | | |
| IRRITCH | | | | | | | | | | | | |
| GROUP 1 | 14 | 0.1329 | 0.454 | 0.121 | 3.18 | 0.026 | -0.77 | 66 | 0.442 | -1.07 | 37.08 | 0.293 |
| GROUP 2 | 54 | 0.3075 | 0.809 | 0.110 | | | | | | | | |
| NAPCH | | | | | | | | | | | | |
| GROUP 1 | 14 | 4.1786 | 8.083 | 2.160 | 1.30 | 0.627 | 0.60 | 67 | 0.549 | 0.65 | 22.42 | 0.525 |
| GROUP 2 | 55 | 2.3545 | 9.208 | 1.242 | | | | | | | | |

TABLE (xvi) Independent sample T-TESTs on drug status for demographic variables and baseline and change score (outcome) DSQ measures

# APPENDIX J

## Sleep Disturbance Questionnaire

SLEEP DISTURBANCE QUESTIONNAIRE

Please put a X in the appropriate box, depending upon how true you feel each of the following statements is for your typical sleep pattern.

On the nights when I don't sleep well the problem seems to be that:-

| | Never True | Seldom True | Sometimes True | Often True | Very Often True |
|---|---|---|---|---|---|
| 1. I can't get into a comfortable position in bed. | | | | | |
| 2. My mind keeps turning things over. | | | | | |
| 3. I can't get my sleep pattern into a proper routine. | | | | | |
| 4. I get too "worked up" at not sleeping. | | | | | |
| 5. I find it hard to physically "let go" and relax my body. | | | | | |
| 6. My thinking takes a long time to "unwind". | | | | | |
| 7. I don't feel tired enough at bedtime. | | | | | |
| 8. I try too hard to get to sleep. | | | | | |
| 9. My body is full of tension. | | | | | |
| 10. I am unable to empty my mind. | | | | | |
| 11. I spend time reading/watching TV in bed when I should be sleeping. | | | | | |
| 12. I worry that I won't cope tomorrow if I don't sleep well. | | | | | |

Which one of the above statements is most relevant to you. | No. |

Are there any other factors associated with your poor sleep pattern? If so, please write a short note in this space.

# APPENDIX K

## Additional Tables for Chapter 10

| Factor | Eigenvalue | % Variance | Cumulative % |
|---|---|---|---|
| 1 | 4.780 | 39.7 | 39.7 |
| 2 | 2.162 | 18.0 | 57.7 |
| 3 | 1.278 | 10.7 | 68.3 |
| 4 | .995 | 8.3 | 76.6 |
| 5 | .685 | 5.7 | 82.3 |
| 6 | .551 | 4.6 | 86.9 |
| 7 | .540 | 4.5 | 91.4 |
| 8 | .444 | 3.7 | 95.1 |
| 9 | .207 | 1.7 | 96.8 |
| 10 | .173 | 1.4 | 98.3 |
| 11 | .124 | 1.0 | 99.3 |
| 12 | .081 | 0.7 | 100.0 |

TABLE (xvii) Complete factor extraction table for the Sleep Disturbance Questionnaire under Principal Components Analysis

| Variable | df | F Ratio | F Prob | Pair | Scheffe Range .05 | Scheffe Range .10 |
|---|---|---|---|---|---|---|
| SOL mean | (3,80) | 0.036 | 0.990 | - | | |
| SOL SD | (3,80) | 2.334 | 0.080 | - | | |
| TOT mean | (3,80) | 5.080 | 0.003 | 1 v 2 | * | * |
| | | | | 4 v 2 | | * |
| TOT SD | (3,80) | 0.016 | 0.956 | - | | |
| WAKE mean | (3,80) | 1.494 | 0.223 | - | | |
| WAKE SD | (3,80) | 1.791 | 0.156 | - | | |
| REPTH | (3,80) | 1.077 | 0.364 | - | | |
| RESTED | (3,80) | 0.592 | 0.622 | - | | |
| ENJOY | (3,80) | 0.903 | 0.443 | - | | |
| IRRIT | (3,80) | 0.106 | 0.956 | - | | |
| NAPDAY | (3,80) | 2.717 | 0.051 | 4 v 1 | | * |

TABLE (xviii) ONEWAY ANOVAs on baseline mean scores for each of the DSQ variables across random therapy (1), tailored therapy (2), placebo (3) and no treatment (4) conditions. The results of range testing are also presented

| Measure | Baseline Mean | Treatment Week 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 |
|---|---|---|---|---|---|---|---|---|---|
| SOL mean | 85.3(50.9) | 63.2(50.2) | 56.7(46.1) | 55.1(37.0) | 57.9(46.9) | 58.9(65.8) | 56.6(61.7) | 52.5(53.8) | 55.8(46.4) |
| SOL SD | 57.0(35.9) | 39.1(47.9) | 28.0(47.6) | 32.6(29.6) | 33.3(37.0) | 31.1(46.1) | 31.1(51.3) | 36.5(50.1) | 40.7(56.4) |
| TOT mean | 4.50(1.51) | 5.00(1.39) | 5.05(1.11) | 5.09(1.55) | 5.14(1.69) | 5.00(1.65) | 5.05(1.64) | 5.27(1.79) | 5.17(1.80) |
| TOT SD | 1.32(0.63) | 0.99(0.70) | 0.94(0.71) | 0.96(0.64) | 0.86(0.43) | 0.92(0.61) | 0.93(0.78) | 0.88(0.82) | 0.98(0.53) |
| WAKE mean | 1.53(0.80) | 1.38(0.95) | 1.22(0.84) | 1.03(0.56) | 0.94(0.64) | 0.92(0.62) | 0.96(0.76) | 0.70(0.53) | 0.77(0.69) |
| WAKE SD | 1.12(0.64) | 0.81(0.41) | 0.79(0.29) | 0.73(0.31) | 0.62(0.25) | 0.63(0.40) | 0.62(0.25) | 0.58(0.33) | 0.61(0.48) |
| REPTH | 1.56(0.95) | 1.12(0.65) | 1.02(0.63) | 0.89(0.65) | 1.07(0.78) | 0.84(0.72) | 0.82(0.80) | 0.91(0.86) | 1.00(0.89) |
| RESTED | 1.66(0.55) | 1.73(0.68) | 1.84(0.66) | 2.01(0.59) | 1.84(0.48) | 1.92(0.56) | 1.92(0.55) | 2.15(0.55) | 1.67(0.84) |
| ENJOY | 1.76(0.42) | 1.89(0.64) | 1.90(0.78) | 2.05(0.71) | 1.99(0.48) | 2.06(0.62) | 2.00(0.58) | 2.35(0.51) | 1.97(0.65) |
| IRRIT | 1.28(0.84) | 1.04(0.85) | 1.01(0.81) | 0.81(0.72) | 0.91(0.73) | 1.02(0.75) | 1.16(0.89) | 1.01(0.82) | 1.04(0.92) |
| NAPDAY | 6.8(10.3) | 0.9( 3.4) | 1.5( 4.6) | 2.2( 4.9) | 0.3( 1.2) | 3.2( 7.7) | 0.2( 0.8) | 0.2( 0.8) | 0.0 (0.0) |

TABLE (xix) Mean scores with SD scores (bracketted) for the tailored therapy group on each of the DSQ measures at baseline and each week of the experimental period.

| Variable | pre | post | SE diff | T | df | Prob | Sig Level |
|---|---|---|---|---|---|---|---|
| ARS 1 | 3.79 | 4.50 | 0.48 | -1.47 | 10 | 0.179 | NS |
| ARS 2 | 5.26 | 4.96 | 0.39 | 0.77 | 10 | 0.462 | NS |
| ARS 3 | 4.77 | 5.43 | 0.89 | -0.51 | 10 | 0.621 | NS |
| ARS 4 | 6.72 | 6.68 | 0.87 | 0.05 | 10 | 0.960 | NS |
| ARS 5 | 4.34 | 4.65 | 0.59 | -0.53 | 10 | 0.613 | NS |
| ARS 6 | 4.56 | 3.55 | 0.92 | 1.11 | 9 | 0.305 | NS |
| ARS 7 | 3.69 | 3.79 | 0.72 | -0.14 | 10 | 0.892 | NS |
| ARS 8 | 7.33 | 5.73 | 1.01 | 1.49 | 8 | 0.188 | NS |
| ARS 9 | 1.70 | 5.34 | 1.06 | -3.45 | 10 | 0.009 | ** |
| ARS 10 | 4.13 | 4.84 | 0.72 | -0.98 | 10 | 0.354 | NS |

TABLE (xx) Mean scores for the pre and post-treatment ARS items and results of paired T-TEST comparisons for the tailored therapy group (* $p \leq .01$)

# APPENDIX L

## Additional Tables for Chapter 11

| Treatment/ Subject | Baseline mean SOL | Treatment Week 1 | Treatment Week 2 |
|---|---|---|---|
| RELAXATION | | | |
| 1 | 37 | 43 | 56 |
| 2 | 76 | 86 | 90 |
| 3 | 166 | 239 * | 101 |
| 4 | 136 | 34 | 122 |
| 5 | 89 | 59 | 64 |
| 6 | 143 | 75 | 93 |
| 7 | 226 | 247 | 154 |
| 8 | 76 | 89 | 43 |
| 9 | 70 | 131 * | 88 |
| 10 | 26 | 31 | 29 |
| 11 | 57 | 66 | 39 |
| 12 | 75 | 43 | 30 |
| 13 | 24 | 19 | 20 |
| 14 | 65 | 56 | 54 |
| STIMULUS CONTROL | | | |
| 1 | 128 | 41 | 43 |
| 2 | 80 | 44 | 34 |
| 3 | 67 | 45 | 32 |
| 4 | 35 | 39 | 16 |
| 5 | 32 | 9 | 11 |
| 6 | 129 | 66 | 86 |
| 7 | 73 | 33 | 61 |
| 8 | 41 | 31 | 51 |
| 9 | 51 | 30 | 28 |
| 10 | 88 | 56 | 47 |
| 11 | 66 | 23 | 14 |
| 12 | 208 | 99 | 73 |
| 13 | 112 | 86 | 69 |
| 14 | 48 | 16 | 19 |
| PARADOX | | | |
| 1 | 48 | 36 | 26 |
| 2 | 77 | 47 | 51 |
| 3 | 94 | 90 | 63 |
| 4 | 226 | 214 | 326 * |
| 5 | 32 | 69 * | 49 |
| 6 | 104 | 154 * | 146 * |
| 7 | 25 | 26 | 27 |
| 8 | 42 | 49 | 11 |
| 9 | 31 | 5 | 14 |
| 10 | 71 | 26 | 13 |
| 11 | 87 | 73 | 90 |
| 12 | 26 | 59 * | 28 |
| 13 | 75 | 103 * | 50 |
| 14 | 76 | 109 * | 89 |
| 15 | 71 | 61 | 47 |

TABLE (xxi) SOL mean scores at baseline and during the first two weeks of treatment for each of the active treatments in the main study. Items marked * indicate increments of 33% or greater over baseline value

| Treatment/ Subject | Baseline SOL | Final SOL | Absolute Reduction | Reduction >= 30' | Reduction >= 50% |
|---|---|---|---|---|---|
| RELAXATION | | | | | |
| 1 | 37 | 42 | | | |
| 2 | 76 | 68 | * | | |
| 3 | 166 | 59 | * | | * |
| 4 | 136 | 76 | * | | |
| 5 | 89 | 68 | * | | |
| 6 | 143 | 47 | * | | * |
| 7 | 226 | 120 | * | | |
| 8 | 76 | 48 | * | | |
| 9 | 70 | 79 | | | |
| 10 | 26 | 35 | | | |
| 11 | 57 | 47 | * | | |
| 12 | 75 | 26 | * | * | * |
| 13 | 24 | 41 | | | |
| 14 | 65 | 45 | * | | |
| STIMULUS CONTROL | | | | | |
| 1 | 128 | 28 | * | * | * |
| 2 | 80 | 12 | * | * | * |
| 3 | 67 | 34 | * | | |
| 4 | 35 | 20 | * | * | |
| 5 | 32 | 7 | * | * | * |
| 6 | 129 | 103 | * | | |
| 7 | 73 | 15 | * | * | * |
| 8 | 41 | 19 | * | * | * |
| 9 | 51 | 27 | * | * | |
| 10 | 88 | 39 | * | | * |
| 11 | 66 | 14 | * | * | * |
| 12 | 208 | 47 | * | | * |
| 13 | 112 | 39 | * | | * |
| 14 | 48 | 14 | * | * | * |
| PARADOX | | | | | |
| 1 | 48 | 6 | * | * | * |
| 2 | 77 | 28 | * | * | * |
| 3 | 94 | 43 | * | | * |
| 4 | 226 | 121 | * | | |
| 5 | 32 | 12 | * | * | * |
| 6 | 104 | 118 | | | |
| 7 | 25 | 29 | | | |
| 8 | 42 | 8 | * | * | * |
| 9 | 31 | 13 | * | * | * |
| 10 | 71 | 8 | * | * | * |
| 11 | 87 | 51 | * | | |
| 12 | 26 | 15 | * | | |
| 13 | 75 | 75 | | | |
| 14 | 76 | 60 | * | | |
| 15 | 71 | 43 | * | | |
| TAILORED THERAPY | | | | | |
| 1 | 132 | 79 | * | | |
| 2 | 104 | 144 | | | |
| 3 | 31 | 77 | | | |
| 4 | 64 | 128 | | | |
| 5 | 86 | 14 | * | * | * |
| 6 | 21 | 21 | | | |
| 7 | 143 | 69 | * | | * |
| 8 | 139 | 60 | * | | * |
| 9 | 34 | 5 | * | * | * |
| 10 | 31 | 19 | * | * | |
| 11 | 45 | 19 | * | * | * |
| 12 | 178 | 29 | * | * | * |
| 13 | 98 | 30 | * | * | * |
| 14 | 94 | 84 | * | | |

TABLE (xxii) SOL mean scores at baseline and the final week of treatment for each of the active treatment conditions. The clinical significance of therapy-induced change is indicated in terms of the three criteria; absolute SOL reduction (ie. greater than 1 min), SOL reduction greater than 30 min, and 50% SOL reduction

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