UNIVERSITY
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SLEEP RESEARCH
GROUP

The Quarter of an Hour Rule:

a simplified cognitive-behavioural intervention for insomnia improves sleep

Author: Marina Malaffo

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Department of Psychological Medicine,
University of Glasgow

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My gratitude goes also to the sleep research group for providing a space to discuss ideas constructively, to my parents for their constant encouragement and to Gregor for kindly proof-reading my writing.

Thanks also to the GPs who referred their patients to the trial and, especially, to the participants who kindly devoted considerable amount of their time to be tested and provide daily data.

Declaration

I, Marina Malaffo, declare that this is my own work, carried out under the normal term of supervision.

Abstract

Stimulus control (SC) is a core component of cognitive behavioural therapy (CBT) for insomnia and is the single intervention for which there is most empirical evidence. Nonetheless, little is known about whether all of the elements within SC are critical to sleep improvement. This study, therefore, investigated the impact on sleep of the Quarter of an Hour Rule (QHR) a single, situational element considered central to SC for insomnia. The mechanisms of effect of SC intervention remain also unclear. An associated aim of the present study, therefore, was to contrast two forms of administration of the QHR to test aspects of the learning theory presumed to underlie the SC model. In addition adherence to the behavioural intervention was investigated and the possibility of using actigraphy to measure adherence objectively was explored.

Prior to the randomised controlled trial (primary study), two preliminary studies were conducted. The first preliminary study aimed at determining the optimal cut-off to represent normalcy in sleep onset latency (SOL). The results indicated it to be fifteen minutes and, therefore, participants in studies two and three were asked to apply the QHR if they were not asleep within a quarter of an hour. Study two comprised three single cases and tested the feasibility of the QHR as a standalone therapy for insomnia. Visual inspection of the data and interrupted time series analyses evidenced SOL, wake after sleep onset (WASO) and sleep efficiency (S.E.) improvements in two out of three participants. Their Pittsburgh Sleep Quality Index (PSQI) score at the end of the intervention was reduced by 50% compared to baseline. The participant, whose sleep was not improved, following the intervention, had not applied the QHR. The results of this exploratory, single case, study warranted further investigation of the QHR.

In study three forty-one GP and self referred volunteers, aged 18-72 years, with SOL and/or WASO complaints, formed 3 randomised groups: QHRin bed, QHRout of bed and control. Both QHR conditions required to 'read if not asleep within a quarter of an hour', with groups differing only with the location (in bed versus out of bed) where to apply the QHR. Sleep diary pre-treatment (two weeks) and post-treatment (three weeks), home polysomnography (PSG) (two nights pre-, two post-

treatment) and sleep related questionnaire (pre and post) data were collected. Adherence with the QHR was measured objectively (actigraphy + light monitoring) and subjectively (adherence diary).

Following QHR treatments, statistically significant reductions in SOL (QHRout) and WASO (QHRin and QHRout), an increase in S.E. (QHRin and QHRout) and a decrease in PSQI score (QHRin and QHRout) were found. Trends also indicated increased total sleep time (TST). Clinically significant improvements (SOL and WASO ≤ 31 minutes or reduced by 50%, PSQI ≤5 or reduced by 50%) were obtained in 33-57% of active groups participants. Furthermore, 43% of QHRin and 66% of QHRout participants achieved S.E.≥85%. Magnitude of effect was greater for the QHRout than the QHRin and changes were achieved within the first week of treatment. No differences were found in PSG defined sleep variables. Nevertheless, subjective-objective discrepancies (i.e. differences between sleep diary and PSG defined sleep) were reduced following intervention. Sleep related questionnaires indicated fewer sleep pattern problems, less cognitive and somatic arousal and less cognitive activity (sleep effort, problem solving and sensory engagement) following QHR interventions. Participants tended to adhere in categorical terms (either adhering most times (majority) or not adhering at all). Adherence was adequate indicating that the QHR is well tolerated and accepted. Importantly, the combination of actigraphy and light provided a tool to measure adherence objectively.

The QHR, a single instruction administered in the form of brief therapy, was found to improve subjectively reported sleep both statistically and clinically. The finding that both QHRin and QHRout were efficacious for WASO sheds doubt on the argument that for difficulties maintaining sleep, the QHR component works solely via conditioning principles. However it is possible that re-conditioning effects apply not only to external (environmental) factors as posited by SC theory but, also, to internal (cognitive) factors. The finding that sub-ob discrepancies decreased following QHR intervention is discussed in terms of levels of arousal and stimuli engagement at the time of falling asleep and during PSG defined sleep. Further research is warranted.

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CHAPTER 1

Introduction

1.1 Overview

This chapter presents an overview of insomnia, its classifications, characteristics and epidemiology. Insomnia assessment and diagnostic issues are also considered with a view to what questions each of these methods can help to answer. Thereafter the three main perspectives of insomnia and some theoretical models within which it is possible to understand the emergence, maintenance and exacerbation of insomnia are outlined and evidence supporting and / or disconfirming these perspectives and models are presented. By taking into account the psychological and behavioural factors presumed to be involved in insomnia, a rationale for the use of psychological treatments is proposed. Psychological interventions for insomnia are briefly outlined and treatment outcome studies are critically reviewed so as to highlight what is still to be done. Furthermore, variables moderating treatment efficacy are discussed and a call for investigation of adherence to treatment is made. Drawing on the models and current limitations in insomnia treatment research, and the recent guidelines on insomnia, it is proposed that the investigation of one single component of the behavioural treatment for insomnia stimulus control, namely the quarter of an hour rule (QHR), can be employed not only to mitigate sleep difficulties but, importantly, to shed light on mechanisms of effects and the minimum dose needed to achieve therapeutic gains. In addition, employing only one single element permits a closer

look at adherence and to the testing of an objective methodology to measure and monitor adherence and its effects on therapy outcomes.

1.2 Insomnia

Lay-person definitions of insomnia include "*not being able to sleep well*", "*taking ages to fall asleep*", "*spending hours awake during the night*" and, as a result, "*feeling very tired during the day with difficulties concentrating*", "*feeling lethargic and with snappy moods*". The self-labelled 'insomniacs' will probably go on to say that they would love to have a good night's sleep, that they have tried everything (but nothing worked), that they don't want drugs or that drugs are not working anymore. Some will remember how easy it was to sleep once, and how good it feels when they have the occasional good night.

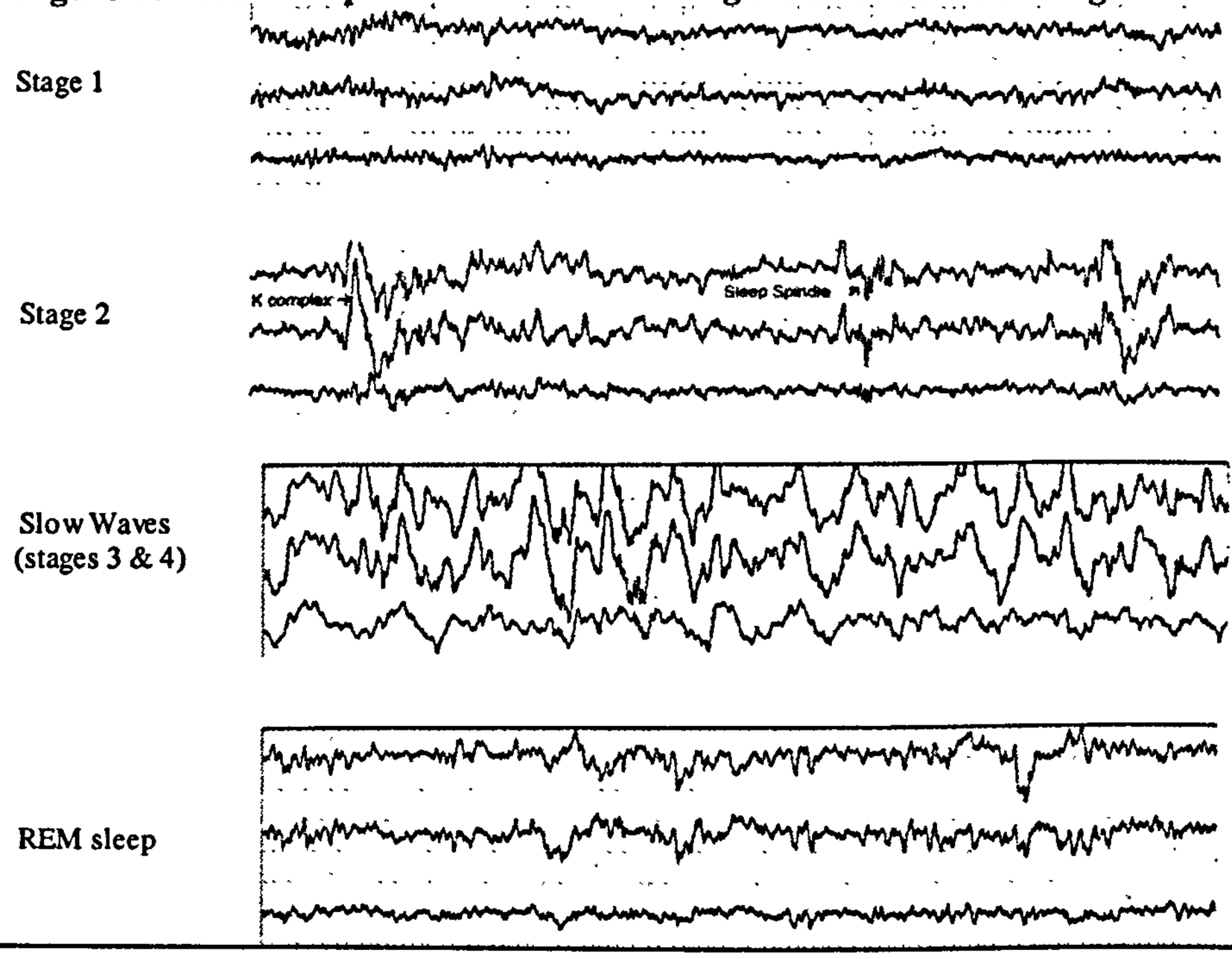
Insomnia is so widespread that most people have an idea of what insomnia is, although the 'insomnia ingredients' vary according to personal (one's own or someone else's) experiences. But what is insomnia really? Perhaps, as proposed by Espie (1991, 2002) the best way to start answering this question is to understand what normal sleep is and what regulates it.

1.2.1 Basic Facts about Sleep

Sleep is not a unitary state: while the observer can only see the two opposite states, that is wakefulness and sleep, the individual experiences different types of sleep such as deep and light sleep and dreaming. Research has corroborated the subjective experience of sleep as a non-unitary state by the measuring of the electrical currents of the brain.

The first division of sleep is that of non-rapid eye movement (NREM) sleep and REM sleep. As illustrated in figure 1, NREM sleep is further subdivided into four distinct stages.

Figure 1: An example of EEG traces during NREM and REM stages



Stage 1 is a very light sleep characterised by a relatively low-voltage and mixed frequency activity where theta rhythm (3 to 7 cycles per second - cps) and vertex sharps are common. Stage 2 represents light sleep as well and the EEG shows a pattern of relatively low voltage with mixed frequency activity. Two specific EEG patterns enable the distinction of stage 2 from stage 1: the sleep spindles (12-14 cps, with a duration of 0.5 to 1.5 seconds) and k-complex (a well delineated sharp wave of duration exceeding 0.5 seconds which is immediately followed by a positive component and stands out from the ongoing background activity). Each epoch (30 seconds) in stage 2 comprises up to 20% of delta waves (delta waves have high

voltage, i.e. amplitudes greater than 75 millivolts, and there are maximum 2 for cps). Deep sleep is seen in stage 3 and 4. Stage 3 is defined by the presence of delta waves in a percentage between 20 and 50 per each epoch of EEG recording. Stage 4, finally, is characterised by delta waves in more than 50% of the epoch. REM sleep is characterized by EEG activation: relatively low voltage, mixed frequency (typical of relaxed wakefulness) at the scalp level coupled to rapid eye movement and motor atonia (i.e. loss of muscle tone). Detailed guidelines and criteria for staging sleep are provided by the standard sleep staging manual (Rechtschaffen and Kales, 1968).

Sleep is, then, a highly organised activity and it follows a cyclic pattern. A normal adult enters sleep via NREM stages (from 1 to 4); this sequence is then reversed from stage 4 to 2 leading to the first REM episode. This first cycle of sleep takes about 70 to 120 minutes and on average, during one night, four to five of these cycles happen.

In Table 1 generalisations about sleep in normal and primary insomnia adults are provided. These PSG values for sleep continuity and sleep architecture measures were estimated by Smith, Nowakowski, Soeffing, Orff and Perlis (2003) from literature.

Table 1 – Average PSG Sleep variables for Healthy Normal Sleepers and Primary Insomnia

Mean Measures		Normal	Primary Insomnia
Sleep Continuity			
	SOL	10 (minutes)	56 (minutes)
	WASO	10 (minutes)	45 (minutes)
	TST	420 (minutes)	352 (minutes)
	SE	>90%	79%
Sleep Architecture			
	Wake	5%	11%
	Stage 1	5%	10%
	Stage 2	60%	56%
	Stage 3 & 4	15%	12%
	Stage REM	15%	22%

The question is: what regulates sleep?

In the 1970s Borbely and his colleagues in Zurich, began the exploration of a two-factor theory of the regulation of sleep based on experimental work with both animals and humans, which was then formulated in 1984 (Daan, Beersma and Borbely, 1984). In brief, they proposed that two 'opponent processes' regulate sleep: on one hand a homeostatic sleep drive, which is constantly active, promotes and maintains sleep (the longer one is without sleep the greater the sleep debt and as a consequence the drive to sleep); on the other hand wakefulness is fostered by a biological clock (regulated by a circadian timer), active during the day and inactive at night which acts as a kind of arousal system (Carskadon and Dement, 1981; Dement and Vaughan, 1999). In addition environmental and social factors influence sleep (e.g. shift work) (Webb, 1988).

An important point was made by Espie (2002): normal sleep is a 'passive' state that does not require effort, the 'good' sleeper falls asleep automatically without forcefully trying, in fact without trying at all.

Having discussed normal sleep, its regulation and 'passivity' it is easier to define and understand insomnia.

1.2.2 Definition and Classification of Insomnia

Broadly speaking insomnia refers to difficulty initiating and/or maintaining sleep. A more specific set of sleep disorders, all of which have in common a complaint of insomnia, has been provided by classification systems such as the Diagnostic and Statistical Manual of Mental Disorders (DSM) and the International Classification of Sleep Disorders (ICSD). Insomnia can be broadly subdivided into two categories: primary insomnia (i.e. independent, no comorbid symptoms) and secondary insomnia

(i.e. insomnia is instigated by other sleep, psychiatric or medical conditions). It is noteworthy that determining causality is often difficult (Smith, Smith, Nowakosky and Perlis, 2003) and that a diagnosis of primary insomnia is often made by exclusion (Morin, 1993).

Insomnia is also characterized by its duration: in the DSM insomnia is defined as 'acute' if it lasts for less than a month and it is accompanied by specific events precipitating the sleep problems (e.g. pain, bereavement). Insomnia is, instead, considered 'chronic' when the symptoms last longer than one month despite the stressor having been resolved or the person having adjusted to it. According to the ICSD-R, instead, insomnia is considered chronic if it lasts for at least six months.

Typically if the individual presents with secondary insomnia the treatment will focus on the disorder (for example depression) deemed to give rise to the insomnia symptoms. In instances of acute insomnia the treatment choice, if treatment is provided, is pharmacotherapy. When the individual presents with primary insomnia symptoms, instead, behavioural interventions for insomnia are provided, if available, as either standalone treatment or in addition to pharmacotherapy.

The present research investigates psychological interventions for insomnia, hence attention will focus on insomnia when it is chronic and a primary disorder.

In short, primary insomnia is defined by DSM-IV as a complaint of initiating and/or maintaining sleep or non-restorative sleep lasting for at least one month associated to decreased functioning during the day (e.g. lack of concentration, tiredness, decreased productivity) (American Psychiatric Association, 2000). Table 2, here below, provides the DSM-IV diagnostic criteria for primary insomnia.

Table 2 - DSM-IV - Diagnostic Criteria for 307.42 Primary Insomnia

A. The predominant complaint is difficulty initiating or maintaining sleep or non-restorative sleep, for at least one month.

B. The sleep disturbance (or associated daytime fatigue) causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

C. The sleep disturbance does not occur exclusively during the course of narcolepsy, breathing related sleep disorder, circadian rhythm sleep disorder, or a parasomnia.

D. The disturbance does not occur exclusively during the course of another mental disorder (e.g. major depressive disorder, generalised anxiety disorder, a delirium).

E. The disturbance is not due to the direct physiological effects of a substance (e.g. a drug of abuse, a medication) or a general medical condition.

Primary insomnia as defined by the DSM-IV subsumes to a number of insomnia categories in the ICSD. The category most closely resembling the DSM-IV definition of primary insomnia is that of psychophysiologic insomnia. According to the ICSD-R (American Sleep Disorder Association, 1997) psychophysiologic insomnia is a disorder of somatized tension and learned sleep-preventing associations resulting in a complaint of insomnia and associated decreased functioning during wakefulness (table 3 reports diagnostic criteria).

It should be noted that this definition, as compared to that of primary insomnia, is more tied up with the underpinning of the disorder (i.e. how insomnia started and it is maintained). Somatized tension refers to somatic hyperarousal, either perceived or objectively measured. Learned sleep-preventing associations refer to either internal cognitions (mainly worry about the inability to sleep) or external stimuli (conditioned

association of sleeplessness with situations and behaviours related to sleep such as lying in bed) both provoking pre-sleep arousal. The patient's focused attention on sleep is of paramount importance in psychophysiologic insomnia.

Table 3 - ICSD-R - Diagnostic Criteria: Psychophysiologic Insomnia 307.42-0

a. A complaint of insomnia is present and is combined with a complaint of decreased functioning during wakefulness.
b. Indication of learned sleep-preventing associations are found and include the following:
    1. Trying too hard to sleep, suggested by an inability to fall asleep when desired, but ease to fall asleep during other relatively monotonous pursuits, such as watching television or reading.
    2. Conditioned arousal to bedroom or sleep-related activities, indicated by sleeping poorly at home but sleeping better away from the home or when not carrying out bedtime routines.
c. There is evidence that the patient has increased somatized tension (e.g. agitation, muscle tension, or increased vasoconstriction)
d. Polysomnographic monitoring demonstrates all of the following:
    1. An increased sleep latency
    2. Reduced sleep efficiency
    3. An increased number and duration of awakenings
e. No other medical or mental disorders accounts for the sleep disturbance.
f. Other sleep disorders can coexist with the insomnia, e.g. inadequate sleep hygiene, obstructive sleep apnoea syndrome, etc.

Minimal criteria: A+B

The ICSD-R has recently been revised and a new version has been published (ICSD-2, American Academy of Sleep Medicine, 2005). This new version, informed by current research and guidelines, incorporates changes with regard to sleep disorders. In particular psychophysiologic insomnia, is renamed psychophysiological

insomnia in the ICSD-2 and described as a complaint of primary insomnia with the essential features of heightened arousal and sleep preventing associations (appendix 1 presents the revised criteria). It is noteworthy that although the characteristics pertaining to psychophysiological insomnia have hardly changed from one version to the next, in the latest version cognitive activity (in the form of intrusive, ruminative presleep cognitions, often related to the inability to sleep and its consequences) are explicitly mentioned.

Having clearly defined primary insomnia (as per DSM-IV) and psychophysiological insomnia (as per ICSD-R, ICSD-2), attention can be turned to the epidemiology of insomnia worldwide.

1.2.3 Epidemiology of Insomnia

The prevalence of primary insomnia in the adult population, as evidenced by epidemiologic surveys, is around 10–15 % increasing to around 20 % in older adults with a 'women to men' ratio of roughly 3:2 (Ancoli-Israel and Roth, 1999; Foley, Monjan, Brown, Simonsick, Wallace et al, 1995; Ford and Kamerow, 1989; Gallup Organisation, 1995; Morgan, 2003; Ohayon, 2002). Although there are variations in percentage of insomnia incidence, it is a worldwide complaint (Cirignotta, Mondini, Zucconi, Lenzi and Lugaresi, 1985; Gislason and Almqvist, 1987; Kim, Uchiyama, Okawa, Liu and Ogihara, 2000; Leger, Guilleminault, Dreyfus, Delahaye and Paillard, 2000; McGhie and Russell, 1962; Ohahion and Partinen, 2002, Ohahion and Hong, 2002). For example, an epidemiological survey of the French population found that 29% of the respondents had insomnia symptoms at least three times per week during the previous month (Leger et al, 2000). Cirignotta and colleagues

(1985) found it in around 15% of the population of San Marino. Similarly a survey of the South Korean general population found insomnia symptoms at least three times per week in 17% of those surveyed (Ohahion and Hong, 2002) and the overall prevalence of insomnia in the Japanese general population has been reported as 21% (Kim, Uchiyama, Okawa, Lin and Oshihara, 2000). A survey carried out in the UK evidenced that sleep problems are by far the most common mental health complaint (Singleton, Bumpstead, O'Brien, Lee, Meltzer, 2001) and primary insomnia is the most common of all sleep complaints (Bixler, Kales, Soldatos, Kales, and Healy, 1979). It is noteworthy that around 2% of the general population meet criteria for psychophysiological insomnia (American Academy of Sleep Medicine, 2005).

Insomnia is often dismissed as less of a serious problem (Simon and vonKorff, 1997) and often ignored at the clinical level despite being a considerable public health problem which is reported by around 20% of patients seen in general practice (Shocat, Umphress, Israel and Ancoli-Israel, 1999) and being associated with increased economic costs both in health care and industry (Simon et al, 1997). Indeed, insomnia is a contributing factor to reduced productivity, absenteeism, and accidents and, once established, it is difficult to extinguish (Brassington, King and Bliwise, 2000; Katz and McHorney, 1998; Mendelson, 1995; Roth and Ancoli-Israel, 1999).

In sum, primary insomnia is a persistent, common and difficult to treat condition associated with reduced daytime functioning, distress and sizeable economic costs. In addition insomnia has been found in two meta-analyses to be a key risk factor in the first episode and recurrence of depression (Cole and Dendukuri, 2003; Riemann and Voderholzer, 2003). It seems, therefore, important to understand what

mechanisms underlie the development and maintenance of insomnia and effective ways of treating it. First of all, however, ways of assessing insomnia and treatment outcomes need to be discussed.

1.3 Assessment of Insomnia and Monitoring Treatment Efficacy

The main methods for measuring sleep are subjective measures, in the form of retrospective and prospective self-reports and objective measures in the form of actigraphy and polysomnography (PSG). Each of these will be briefly described and evaluated so as to outline their strengths and weaknesses.

Typically, the sleep data collected by means of prospective self-reports and objective measures are i) sleep onset latency (SOL) that is the time taken to fall asleep at the beginning of the night; ii) wake after sleep onset (WASO) that is the total time spent awake during the night after sleep onset; iii) total time in bed (TIB) that is the amount of time spent in bed; iv) total sleep time (TST, TST=TIB – [SOL+WASO]) that is the amount of time actually spent asleep while in bed, v) sleep efficiency (S.E., S.E.=[TST/TIB]*100) that is the percentage of time spent asleep while in bed, and vi) number and length of arousals (only objective data). It should be noted that WASO can comprise all awakenings including early morning awakenings or it can be subdivided into WASO during the night and 'early wakening' that is the amount of time from the last wakening in the morning until rising.

1.3.1 Subjective Measures

The two most commonly used self-report measures of sleep are sleep questionnaires (retrospective self reports) and sleep diaries (prospective self-reports).

1.3.1.1 Sleep Questionnaires

Sleep questionnaires have sufficient diagnostic sensitivity in discriminating between normal and poor sleepers and they provide an easy, quick and inexpensive way to measure sleep patterns. They are, therefore, particularly in use where a large number of people are to be screened or surveyed, although they are also used as pre- and post- treatment measures of the individual's perception of their sleep quality and quantity. For example The Pittsburgh Sleep Quality Index (PSQI: Buysse, Reynolds, Monk, Berman, and Kupfer; 1989) was expressly designed for this latter purpose (Smith, Nowakowski, et al, 2003).

One important limitation of sleep questionnaires is their validity: they usually require the individual to provide estimates in terms of average which are most likely influenced by the particular mood of the individual at the time of completion and the most salient or recent experience of sleep (e.g. the particularly bad night). In addition, an average figure raises validity issues given that in insomnia night-to-night variability is high and, importantly, a feature of insomnia: most researchers and clinicians will have come across questionnaires with replies such as "it varies" or "5 minutes to 4 hours", to the questionnaire item: 'average time to fall asleep in the last month'.

In sum, sleep questionnaires provide a quick, convenient and inexpensive tool to measure sleep but have the disadvantage of being based on estimates drawn from memory of a number of past nights and of giving one global figure for each sleep variable under scrutiny. Sleep diaries, critically outlined below, partly avoid these pitfalls.

1.3.1.2 Sleep Diary

This measurement is the standard of practice for behavioural sleep medicine. Unlike sleep questionnaires, the sleep diary is completed each morning upon awakening for a given number of days so that sleep can be sampled across time. Therefore, although sleep measures are taken retrospectively (i.e. the previous night), they are applied prospectively in that they are gathered for a number of (future) consecutive nights. Similarly to retrospective sleep measures the individual is required to estimate variables such as 'time to fall a sleep' and 'duration of wakenings during the night'. However, sleep diaries require such estimates to only one time point (the previous night) hence the response biases (e.g. using averages) discussed above are avoided. Of course sleep diaries collect subjective judgements of sleep and, therefore, are not as precise as objective measures (section 1.3.2 here below). Nonetheless a very important feature of subjective measures is that, unlike PSG or actigraphy, they enable the gathering of information about the perception both of sleep disturbance (i.e. the complaint) and of recovery following treatment.

An issue with the diary is adherence to the instruction 'fill it in every morning': people might fill the diary in later on during the day or even worse a few days later. Another issue with sleep diaries, worth addressing especially for research purposes,

relates to standardisation: although there is a general agreement across sleep laboratories regarding the sleep parameters to be gathered, a standardised sleep diary is not in use and this can result in variations in the characterisation of sleep continuity across studies. These problems could be resolved by employing simple ways to store the daily sleep diary entries (e.g. I-pod type) and by agreeing on a minimal standard form, both in terms of sleep parameters to be collected and wording of questions, to be used across all sleep laboratories.

As it has been shown in the preceding discussion the sleep diary is an inexpensive tool for measuring sleep continuity and sleep quality variables night after night. The fact that it relies on the individual's report of the previous night is a weakness but also a major strength of sleep diaries. On one hand the reported values are not free of the memory biases typical of retrospective measures but on the other hand sleep diaries capture the subjective perception of the sleep difficulties. Given that the essential feature of insomnia is the individual's complaint of poor sleep (minimum three days per week) and its consequences during the day, it can be argued that the sleep diary is the most valid tool to measure this feature of insomnia and it is, therefore, not surprising that it is standard practice in behavioural sleep medicine.

Sleep diaries measure sleep continuity and collect the individual's perception of being either asleep or awake. As already mentioned, the subjective report of sleep argues against a unitary sleep state and the different 'layers' of sleep can be monitored via PSG, an objective measurement of sleep and wakefulness.

1.3.2 Objective Measures of Sleep

In this section polysomnography and actigraphy will be described and critically evaluated.

1.3.2.1 Polysomnography

Polysomnography (PSG) permits researchers and clinicians to measure sleep objectively, to obtain an exact temporal (second-by-second) resolution of sleep, to have direct and quantitative measures of brain and muscle activity and to differentiate sleep into substates (stages 1, 2, 3, 4 and REM) which are not detectable with other sleep measurements (Smith, Nowakowski et al, 2003).

Three measures are sufficient for monitoring and scoring sleep: electroencephalography (EEG) in particular the recording of brain wave activity from central and occipital areas of the scalp, electrooculography (EOG) which measures a differential of electrical potential, generated by each eye movement, between the cornea and the retina; and electromyography (EMG), which monitors muscle tone and is generally recorded at the chin site. These electrical currents can be represented continuously and plotted over time and their combination permits the detection and categorization of sleep stages and wakefulness. In order to obtain reliable PSG data the electrodes need to be placed accurately: the 10-20 International System (Jasper, 1958) is the most commonly used system for electrode attachment.

The PSG data are divided into sleep stages employing criteria for staging normal human sleep provided in the Rechtschaffen and Kales' (1968) manual (section 1.2.1

and figure 1 give explanation and example of sleep stages, and figure 15a shows a PSG recording device).

An important issue is that PSG recording usually takes place in sleep laboratories and several studies have noted that PSG sleep differences between poor and normal sleepers are at best modest (Means, Edinger, Glenn and Fins, 2003; Sugarman, Stern and Walsh, 1985). It could be argued that in laboratory settings the conditioned cues for wakefulness typical of the poor sleeper's bedroom are absent. Additionally the laboratory setting may provide a pre-bed routine that obviates to sleep-disruptive habits and the novelty might distract the individual from the usual pattern of worrying in bed. The fact that poor sleep is expected can paradoxically promote a good night's sleep by removing sleep anxiety performance. The opposite is true for the normal sleeper: habitual cues for sleep are absent, the usual pre-bed routine is disrupted and they might question their ability to sleep in a novel environment.

In other words, it can be argued that a short series of sleep recordings conducted in laboratory settings might not be representative of individuals' typical sleep. For example, Edinger and colleagues conducted a series of PSG studies where PSG recordings were conducted both at participants' homes and in laboratory settings. One study showed that individuals, regardless of their sleep status, acclimatise more readily to PSG recording when in their home as compared to the laboratory setting and that PSG recording at home enabled capture of the relatively high night-to-night variability typical of people with insomnia (Edinger, Fins, Sullivan, Marsh, Dailey, Hope, Young, Shaw, Carlson and Vasilas, 1997). In a subsequent study Edinger and colleagues found that people with insomnia who had a bed partner showed sleep differences with regard to normal sleepers only when PSGs were recorded at home,

once again suggesting that home PSG is a suitable option if capturing 'typical' sleep patterns is important (Edinger, Glenn, Bastian, Marsh, Daile, Hope, Young, Shaw and Meeks, 2001).

It seems, therefore, worthwhile to measure PSG sleep at home as this increases ecological validity in comparison with laboratory based trials. Simple to use, non-intrusive, relatively cheap, portable EEG systems have been recently devised which make it feasible to conduct home PSG so as to monitor objective sleep changes following insomnia interventions whilst the individuals sleep in the 'reality' of their own usual sleep environment.

It is noteworthy that subjective measures of sleep and PSG sleep are very often mismatched in both young adults and the elderly (Carskadon, Dement, Mitler, Guilleminault, Zarcone and Spiegel, 1976; Mercer, Bootzin and Lacks, 2002; Schramm, Hohagen, Backhaus, Lis and Berger, 1995; Webb and Schneider-Helmert, 1988). In order to explain such discrepancies, Borkovec (1979) suggested that when an overly active reticular system (which governs arousal) is present or a neurotransmitter-dependant sleep system fails to switch on then PSG confirms insomnia. In contrast, when reported insomnia is not confirmed by PSG defined sleep the individual has an overly active cognitive processing of anxiety-laden thoughts. It could be argued that, in the latter case, sleep staging does not capture features of insomnia such as alertness. Alertness in the form of EEG fast activity can be detected by performing power spectral analysis to the PSG recorded night (Perlis, Smith, Orff, Andrews and Gill, 2001).

Interestingly, several studies highlighted that despite positive treatment effects being evidenced by sleep diary data, there were only very small treatment effects or

none at all if PSG data were considered (e.g. Morin, Kowatch, Barry and Walton, 1993; Engle-Friedman, Bootzin, Hazlewood and Tsao, 1992; Morin, Stone, Trinkle, Mercer and Remsberg, 1993; Schramm et al, 1995).

Another important point, related to the difference between subjective and objective measures of sleep, could be made by taking into account Espie's (1991) observation that the subjective complaint normally incorporates an element of dissatisfaction with sleep quality variables such as 'not feeling refreshed in the morning', or 'sleep was of very poor quality'. Such complaints are not invalidated by an absence of EEG evidence of sleep pattern disruption: indeed not being sensitive to sleep quality variables weakens the role of PSG as a measurement criterion.

1.3.2.2 Actigraphy

Actigraphy measures movement and its absence i.e. this measurement, rather than measuring sleep directly, infers sleep by the absence of movement (Sadeh and Acebo, 2002). The movement detector, a small piezoelectric accelerometer that generates voltages when there is movement, is housed in a wristwatch-like device. The detected movement is translated into digital counts accumulated across epoch intervals, whose length can range from 2 seconds to 15 minutes, and stored in the internal memory of the watch. The epoch data collected are transferred to a computer that has the set up and the reader interface and each epoch is evaluated for whether it represents sleep or wakefulness.

Given that actigraphy is a proxy measure of sleep its validity as a measurement of sleep variables is inferior when compared to that of sleep diary and PSG. Indeed a problem with actigraphy is validity: good agreement (r=0.94) between actigraphy

and PSG for epoch by epoch sleep has been found in normal sleepers (Jean-Louis, Kripke, Cole, Assmus and Langer, 2001), but epoch by epoch wake have lower validity most probably because people, especially poor sleepers, tend to lie still in an attempt to fall asleep. In addition, the discrepancy is much higher when the sleep of people with insomnia is assessed (Chambers, 1994; Hauri and Wisbey, 1992). Furthermore, it has been shown that there are gender and age differences in actigraph estimates of sleep (for a review see Sadeh and Acebo, 2002). It could be argued that such variability decreases the usefulness of actigraphy when the focus of research is sleep. Kushida, Chang, Gadkary, Guilleminault, Carrillo, and Dement, (2001) compared PSG, actigraphy and sleep diary measures for one night in one hundred people with sleep difficulties. They found that the accuracy of actigraphy declines as sleep diminishes: actigraphy was excellent in detecting sleep (sensitivity = 0.92) but its ability to detect wakefulness (specificity= 0.48) was rather poor and the ability to detect both sleep and wakefulness (accuracy = 0.78) was low. Pollack, Tryon, Nagaraja and Dzwonczyk (2001) investigated the ability of actigraphy to predict PSG defined sleep and wakefulness and argued that actigraphy is not an accurate sleep-wake detector, although it may be useful as a measure of rest/activity. In particular, Sadeh and Acebo (2002, p120) cautioned that "*actigraphy is not the best method if interest is in the precise duration of sleep onset*" because the largest discrepancies between actigraphy and PSG measures are typically around sleep-wake and wake-sleep transitions.

It is important to note that actigraphs can collect other measures in addition to movement: this can be very useful if actigraphy is employed for collecting data other than sleep. For example one model (Actiwatch-L by Cambridge Neurotechnology,

figure 22 in section 6.3.3.5a for an example of actiwatch) incorporates an integral light sensor that monitors light intensity range measured from 0 to 50,000 lux. In this way, both movement and light are detected and stored. A secondary aim of the present research is the exploration of the impact of adherence on insomnia treatment outcomes: this extra feature of actigraphy can be exploited to objectively monitor adherence to a behavioural instruction asking participants to read (study 3, ch. 5).

In sum, there is not an absolute 'gold standard measure' of sleep. While sleep questionnaires are an important tool to quickly gather information regarding the perception of sleep problems, sleep diaries provide nightly information regarding the perception of sleep continuity and, very importantly, the subjective complaint. PSG measures objective sleep continuity and sleep architecture and enables power spectral analysis. However, given that PSG recordings are costly and time consuming they are not really feasible as a measurement of many consecutive nights. For this reason actigraphy is employed when the measurement of sleep over time is to be collected objectively; even though it presents the important problem of validity discussed above.

It can be concluded that subjective and objective measures of sleep might be complementary and, most importantly, the questions one is trying to answer and the issues and hypotheses under investigation (e.g. endorsed beliefs about sleep, EEG activity during sleep) should inform which are the best to use.

The next section introduces insomnia aetiological systems and related research.

1.4 Theoretical Perspectives on Insomnia

The aim of this section is to critically outline the major perspectives on insomnia (i.e. physiological, behavioural and cognitive) and related research. In addition 'purist' models within a given perspective and models integrating different perspectives are discussed.

Whilst there is limited information regarding mechanisms underlying pathophysiology and genetic or heritability factors in insomnia (Richardson and Roth, 2001; Yves, Morin, Cervena, Carlander, Beset and Billiard, 2003), most insomnia models agree on the hypothesis that insomnia is a disorder of arousal: the person complaining of insomnia has a level of arousal inhibiting sleep onset at bedtime and/or during the night. The debate regarding the origin (physiological, psychological and/or environmental) of such hyperarousal is, however, ongoing.

1.4.1 Physiological Perspective of Insomnia

This perspective proposes that physiological arousal and sleep are incompatible and that individuals with chronic insomnia experience sleep difficulties because of a high level of physiological arousal either in general or around bedtime and during the night.

In the nervous system arousal can be maintained by a failure of central nervous system (CNS) processes to promote sleep (homeostatic sleep drive) and/or to inhibit wakefulness (biological clock) (Daan et al, 1984). There is evidence of heightened cortical arousal during the sleep of people with insomnia. For example, Merica, Blois and Gaillard's (1998) PSG study found increased beta power (beta waves are

characteristic of alertness) in their insomnia group as compared to the control group. Similar results were obtained by Perlis, Kehr, Smith, Andrew, Orff and Giles (2001): high frequency EEG activity during the light sleep stages (stages 1 and 2) was increased in individuals with primary insomnia as compared both to controls and individuals with insomnia secondary to major depression.

High levels of autonomic activity associated with tension and problems in physiological relaxation (e.g. muscle tension, respiration rate) can be another form of arousal that, less directly, inhibits sleep. For example Monroe (1967) conducted an important study with 16 normal and 16 poor sleepers and found that poor sleepers exhibited heightened autonomic arousal both during and prior to sleep (evidenced by higher body temperature, body movements per hour, vasoconstriction per minute, perspiration rate and skin conductance). Other studies, however, did not find differences between normal and poor sleepers in autonomic variables (for a review see Bootzin and Nicassio, 1978; Borkovec, 1982). More recently, Bonnet and colleagues assessed physiological arousal employing a measure of oxygen consumption (VO_2) and found that the insomnia group, as compared to controls, displayed higher metabolic rate (autonomic arousal) both during the day and at night (Bonnet and Arand, 1995, 1997; Bonnet, McNulty and Arand, 1993). A limitation of these studies is that physical fitness and caloric intake greatly influence body oxygen consumption, hence the observed differences could have been due, for example, to the insomnia group being less physically fit than the control group.

In sum, although there are findings that physiological hyperarousal (autonomic and/or cortical) correlates with insomnia, research has not unequivocally supported the physiological perspective. It is noteworthy that this perspective informs on the

interaction between insomnia and arousal but lacks explanatory power regarding the development and exacerbation of insomnia. Although research evidence of physiological arousal is often used to provide support for aspects of some theoretical models of insomnia, a formal model of insomnia within the physiological perspective has not been proposed.

The behavioural perspective, discussed in the next section, attempts to provide an analysis of the maintenance and exacerbation of insomnia.

1.4.2 Environmental/Behavioural Perspective of Insomnia

According to the behavioural perspective, acute insomnia might be precipitated by a number of psychosocial events but chronic insomnia has its source in the environment. The source of the individual's recurrent difficulties with sleep is not considered to be physiological or psychological in origin, rather the individual's arousal is caused by situation and temporal factors or the interaction between the individual and their environment.

This perspective posits that the environment may elicit arousal responses by either failing to predict sleep at the right time for sleeping or predicting wakefulness at the wrong times. It is suggested that sleep disruptive habits (e.g. extending the time spent in bed, napping) and conditioned responses (e.g. lying in bed elicits wakefulness rather than sleep) interfere with sleep drive and serve as environmental inhibitors of sleep (Bootzin and Epstein, 2000; Edinger and Wohlgemuth, 1999; Morin, Savard and Blais, 2000; Spielman, Saskin and Thorpy, 1987).

However, results from the couple of studies investigating, directly or indirectly, conditioning in insomnia call into question the conceptual basis of the behavioural

perspective of insomnia. Kazarian, Howe and Csapo (1979) found support for the hypothesis that poor sleepers engage in sleep incompatible behaviour (both in the form of overt behaviour such as reading and covert such as thinking). However, two other studies found that normal sleepers, similarly to poor sleepers, engaged in sleep incompatible activity in the bedroom and that the two groups did not differ in pre-sleep activities (Harvey, 2000a; Haynes, Adams, West, Kamens and Safranek, 1982). The findings that poor and normal sleepers endorse similar behaviour at bedtime do not automatically rule out the role of conditioning in insomnia: it could be, for example, that the concept of 'preparedness' applies to poor sleepers but not to normal sleepers with regard to sleep incompatible behaviours and sleep. In learning theory, the concept of 'preparedness' refers to the observation that depending on the organism's make-up and social modelling, the organism is prepared (predisposed) to learn some associations more readily than others (Seligman, 1971; Hygge and Ohman, 1978). Learned taste aversion studies, for example, suggest that there is a biological preparedness to learn certain relations more readily than others and similar effects occur in operant learning with some responses more readily strengthened by some reinforcers than others (Bolles, 1970; Shettleworth, 1972; Bernstein, 1978). As discussed in section 1.4.2.2, predisposition is one of the three main factors theorised to play a major role in insomnia (Spielman et al, 1987). Additional research, perhaps employing prospective rather than retrospective measures, is much warranted to validate this hypothesis.

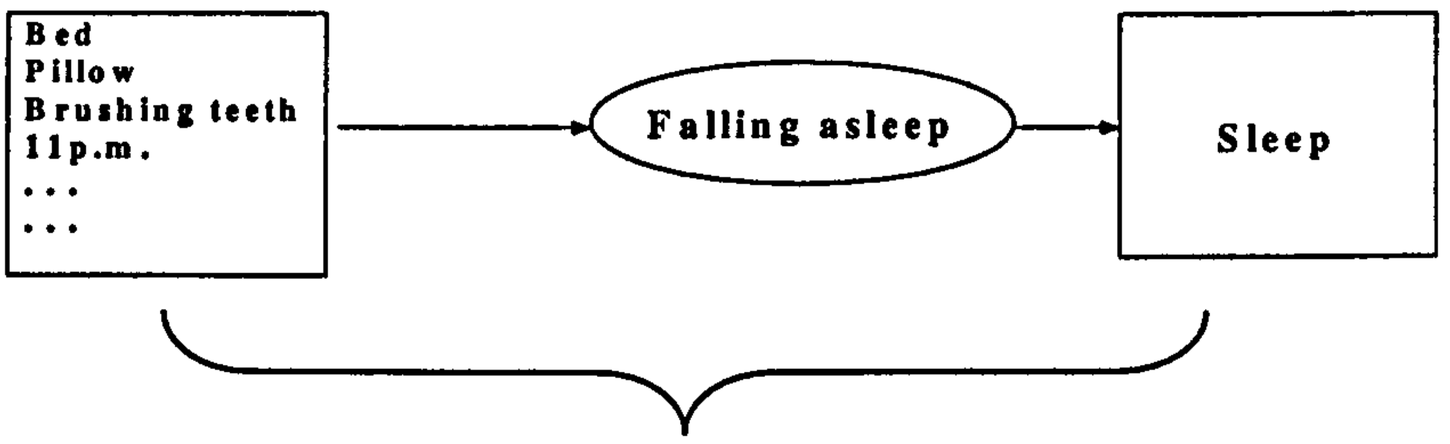
Two behavioural models of insomnia (stimulus control and the 3 factor model of insomnia) have gained extreme popularity. Perhaps, especially with regard to

stimulus control, this is mainly due to the positive outcomes repeatedly obtained with stimulus control therapy for insomnia and sleep restriction.

1.4.2.1 Stimulus Control (Bootzin, 1972, Bootzin and Nicassio, 1978)

Bootzin (1972) conceptualised sleep and insomnia in terms of learning theory and developed stimulus control therapy for insomnia. Stimulus control is based on operant conditioning concepts: stimuli can elicit a number of responses depending on the conditioning history. Stimulus control can be defined as the degree to which an antecedent stimulus (or class of stimuli) determines the probability that the organism gives the conditioned response i.e. the antecedent stimulus has some control over that particular response. Briefly, applying stimulus control concepts to sleep, falling asleep (operant) is emitted so as to produce sleep (reinforcement) and stimuli associated with sleep become discriminative stimuli for the occurrence of sleep (Bootzin and Nicassio, 1978). Stimuli associated with sleep can be temporal (i.e. bedtime) and/ or environmental (i.e. bedroom).

Fig 2 – Operant Analysis of Sleep



In normal sleep stimuli such as one's bed are associated to sleep

As shown in figure 2, this model proposes that in normal sleep the bedroom environment provides stimuli signalling sleep and such stimuli elicit falling asleep.

Bootzin and colleagues proposed that in the case of insomnia the discriminative stimuli for sleep are not established (e.g. lack of association sleep–bedroom) or stimuli for activities that are not compatible with sleep (e.g. television) are present. Situational triggers (sleep-preventing activities such as watching TV, reading, problem solving and bed aversion) may elicit arousal responses by becoming a cue for wakefulness or by failing to predict sleep. Espie (2002) suggested that poor stimulus control can inhibit bedtime de-arousal responses that in turn disrupt both the homeostatic sleep drive and the wakefulness-sleep circadian cycle.

Evidence for this model is provided by the success of the therapy intervention (section 1.7.2) derived by the stimulus control analysis of insomnia rather than by testing directly the conditioning aspects theorised to be responsible for insomnia. As critically presented in sections 1.6.2 and 1.7.2, many studies found that stimulus control therapy for insomnia markedly decreased SOL and WASO by 50 to 80% as compared to pre-treatment values and this findings have been taken as evidence for the plausibility of this model. Notwithstanding its efficacy, the mechanisms by which stimulus control achieves its effects are unclear and it cannot be ruled out that the therapy works for reasons other than re-establishing stimulus control. This point has been emphasised by Salkovskis (2002) who argued that treatment evidence does not shed light on the underlying mechanisms of effect. Indeed, as already discussed in the previous section, only a few studies have investigated pre-bed activities in normal and poor sleepers and no differences have been observed. Contrary to conventional wisdom (ICSD-R), it has, recently, been reported that people with insomnia sleep worse when they are away from home and it has been argued that this finding runs contrary to the hypothesis of conditioning to the bedroom environment (Vallieres,

Belanger, Bastien, Espie, Morin, 2005). However, it could also be argued that, in the case of insomnia, falling asleep is not under the stimulus control of any bedroom environment (either one's own or a hotel room), which coupled to other factors (e.g. drinking more, uncomfortable mattress) increases the sleep problem. It could also be that any bedroom environment sustains wakefulness, rather than falling asleep, in people with insomnia.

This model explains how insomnia is maintained and exacerbated but it lacks explanatory power with regards to the emergence of insomnia. The Spielman model, to which attention is now turned, while emphasising conditioning as the main factor maintaining insomnia, attempts to explain the emergence of insomnia.

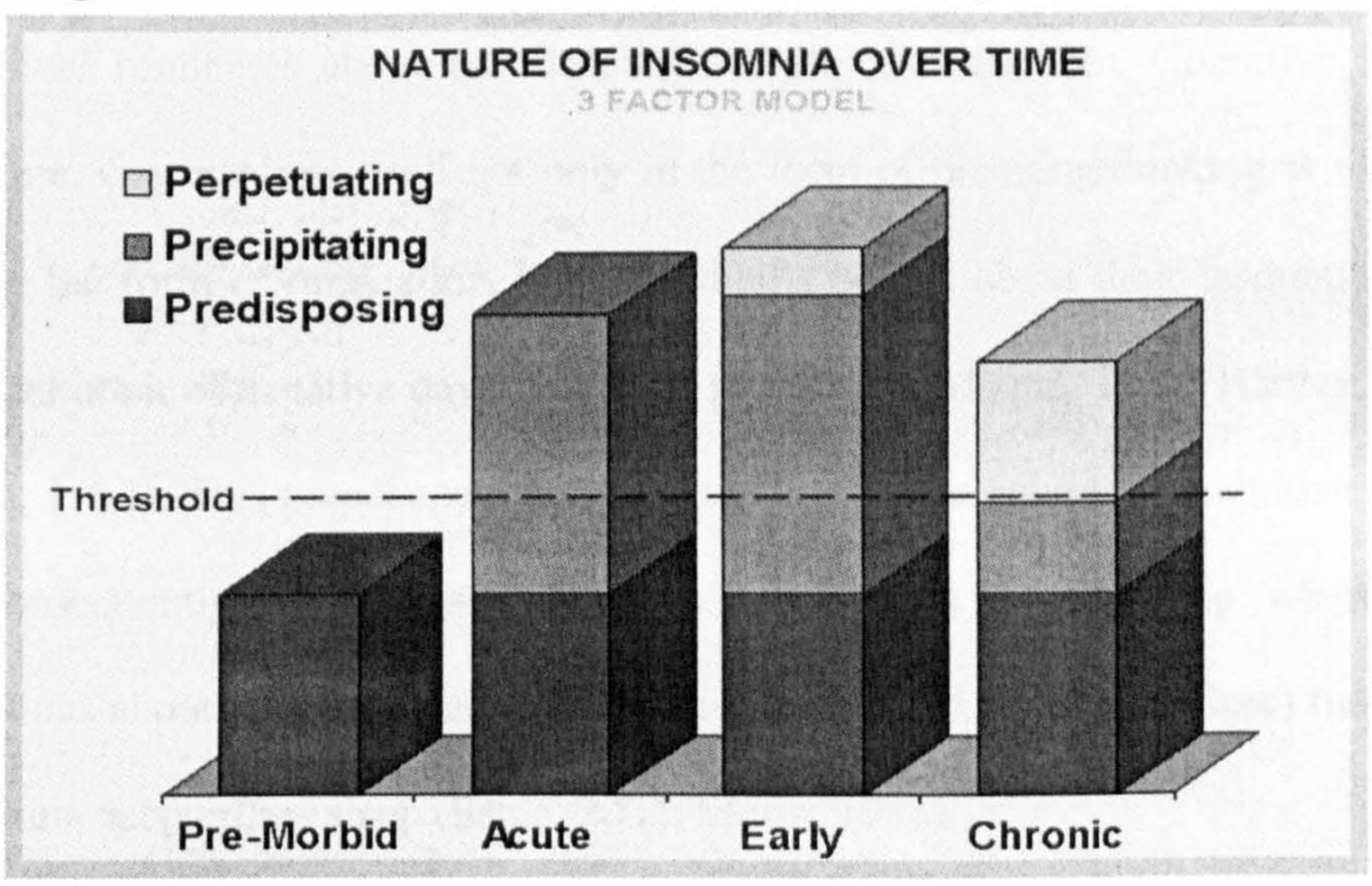
1.4.2.2 The Three Factor Model of Insomnia (Spielman, Caruso, and Glovinsky 1987)

Departing from an exclusively environmental/behavioural framework Spielman, Caruso and Glovinsky (1987) proposed a stress diathesis model of insomnia with an additional behavioural component. In brief, this model hypothesises that insomnia occurs acutely in relation to both trait and state (predisposing and precipitating) factors. In its early stages maintaining factors are developed and if not extinguished such maintaining factors will give rise to chronic insomnia.

Figure 3 shows the factors responsible for the emergence, exacerbation and maintenance of insomnia over time. The individual may be predisposed to develop insomnia due to trait characteristics such as personality characteristics, physiological arousal (Perlis, Giles, Mendelson and Bootzin, 1997; Kales and Kales, 1984; Stepanski, 2000) and genetic predisposition (Bastien and Morin, 2000). However,

precipitating events (e.g. situational stress, bereavement, pain) need to occur for the individual to experience acute insomnia, and factors adopted in an attempt to cope with insomnia symptoms (e.g. napping, extending the sleep opportunity window) are proposed to play a major role in chronic insomnia.

Fig. 3 - The Three Factor Model of Insomnia (Spielman et al, 1987)



Similarly to the stimulus control model, the three factor model is based on operant conditioning and does not take into account the possibility that classical conditioning plays a role in the maintenance of insomnia.

As already mentioned, both the stimulus control and the three factor models derive evidence from the efficacy of behavioural treatment of insomnia. However, their central tenets have never been tested empirically.

Neither of these models of insomnia nor the behavioural perspective in general, take cognition into consideration, yet a common complaint of people with insomnia is that of 'an overactive mind' during the night. Attention is, therefore, now turned to a perspective addressing this reported feature of insomnia.

1.4.3 Cognitive Perspective on Insomnia

According to the cognitive perspective of insomnia cognitive and/or emotional hyperarousal predisposes, precipitates and maintains insomnia. In brief, mental alertness (e.g. thinking, planning) may interfere with sleep and, sleeping difficulties can become a source of cognition with consequent effects upon self-perception: the individual ruminates about not sleeping and its consequences. Cognitive arousal, therefore, can manifest itself not only in the form of planning/thinking at night but also in the form of rumination, the individual's beliefs about their insomnia and/or the attribution of negative daytime events to insomnia (Espie, 1991; Harvey, 2002a, Morin, 1993). This preoccupation with sleep becomes a source of cognitive arousal and consequently exacerbates insomnia in a continuous negative loop where faulty cognitions about sleep (e.g. amount of sleep needed, effects of sleep loss) fuel worry and worry jeopardises sleep (Espie, 2002; Morin, 1993).

Research findings lend support to this perspective: people with insomnia were found to complain of cognitive arousal (e.g. inability to 'switch off their mind') more than of physiological arousal (Nicassio, Mendlowitz, Fussel and Petras, 1985) and of higher levels of presleep rumination as compared to normal sleepers (Espie, Brooks and Lindsay, 1989; Harvey, 2000c; Lichstein and Rosenthal, 1980). For example Lichstein and Rosenthal (1980) found that 55% of their 296 people with insomnia sample, regarded their sleep problems as arising from cognitive arousal and only 5% as arising from somatic arousal. Espie and collaborators found that two thirds (42 people) of their sample regarded cognitive intrusion as an important source of their sleep problems (Espie, Lindsay, Brooks, Hood and Turvey, 1989). These findings have been replicated by Harvey (2000b). Nicassio et al (1985) found, in their sample

comprising both college students and adults with sleep problems, that the correlation between pre-sleep cognitive arousal and sleep onset latencies was higher than that with pre-sleep somatic arousal. However, Sanavio (1988) found hardly any correlation (0.09) between measures of pre-sleep cognitions and self-reported SOL.

It is noteworthy that poor sleepers' cognitive activity at bedtime is generally emotionally-laden: poor sleepers typically report more negative thoughts regarding their sleep and its impact on their lives than controls (e.g. Espie, 1991; Edinger, Fins et al, 2000; Kuisk, Bertelson and Walsh, 1989). Bedtime is a period during which competing external stimulation is minimal and it is not, therefore, surprising that it becomes the time for focusing upon problems. It has been proposed (Kales and Kales, 1984) that people with insomnia internalise stress during the day which then re-emerges at sleep time. Indeed, Nelson and Harvey (2003) explored the amount (employing a press-holder counter device) and content of pre-sleep imagery and found that poor sleepers, as compared to normal sleepers, reported fewer images. However, unlike those of normal sleepers, such images were unpleasant (and related to topics such as intimate relationships and sleep); furthermore, SOL and amount of negative images were positively correlated.

In addition, research findings, employing self-report questionnaires (e.g. Edinger et al, 2000; Harvey, 2000b; Morin, 1993) or audiotape recordings of pre-sleep thoughts (Wicklow and Espie, 2000) suggest that insomniacs hold greater dysfunctional beliefs and attitudes about sleep than normal sleepers (e.g. 'I need 8 hours of sleep to function well the next day'). Indeed people with insomnia experience anxiety about being able to sleep (sleep performance anxiety): they try to

control the 'deviant' performance via coping efforts which might actually have the opposite effect of increasing SOL and WASO.

Another line of evidence for a cognitive model has been provided by studies where emotional and cognitive arousals have been manipulated (DeValck, Cluydts and Pirrera, 2004; Gross and Borkovec, 1982). For example, Gross and Borkovec (1982) found that individuals who were told they had to give a speech (manipulation of sleep cognition) had nap onset latencies almost twice as long than those who were not told so. In contrast, however, Haynes, Adams and Franzen (1981) found that a pre-sleep arithmetic mental task shortened SOL in people with insomnia. Although this finding suggests that mentation does not impact on sleep, it can also be argued that the fairly simple task employed by Haynes and colleagues inhibited self-generated and emotionally-laden mental activity, hence enabling cognitive de-arousal.

Cognition and affect, therefore, seem to interact in insomnia and it is possible that failure to find associations between sleep and cognitive arousal in some studies may have depended on the type of cognitions (and whether they were affect-laden) under examination.

It is important to consider that reinforcement contingencies are not always negative: thinking and planning can be enjoyable and valued by the individual and if this is the case avoiding mentation in bed might be difficult. In addition a distinction between difficulty sleeping because of thinking and thinking because of wakefulness must be made: taking into account that research has not always found an association between sleep latency and cognitive alertness it has been suggested that cognitive

arousal may be epiphenomenal to wakefulness (Espie, 1991; Morin, 1993, Perlis, Giles, Mendelson, Bootzin and Wyatt, 1997).

Nevertheless a sizeable amount of evidence for cognitive arousal in insomnia has been gathered and it should be noted that this perspective, as compared to the physiological and behavioural ones, has provoked a lot of research, most probably fuelled by patients' reports of 'a racing mind' and the popularity of cognitive theories and therapies for psychopathologies and psychological distress in general.

In the next section a cognitive model of insomnia that acknowledges and gives equal weight to (faulty) cognition both at night and during the day is presented.

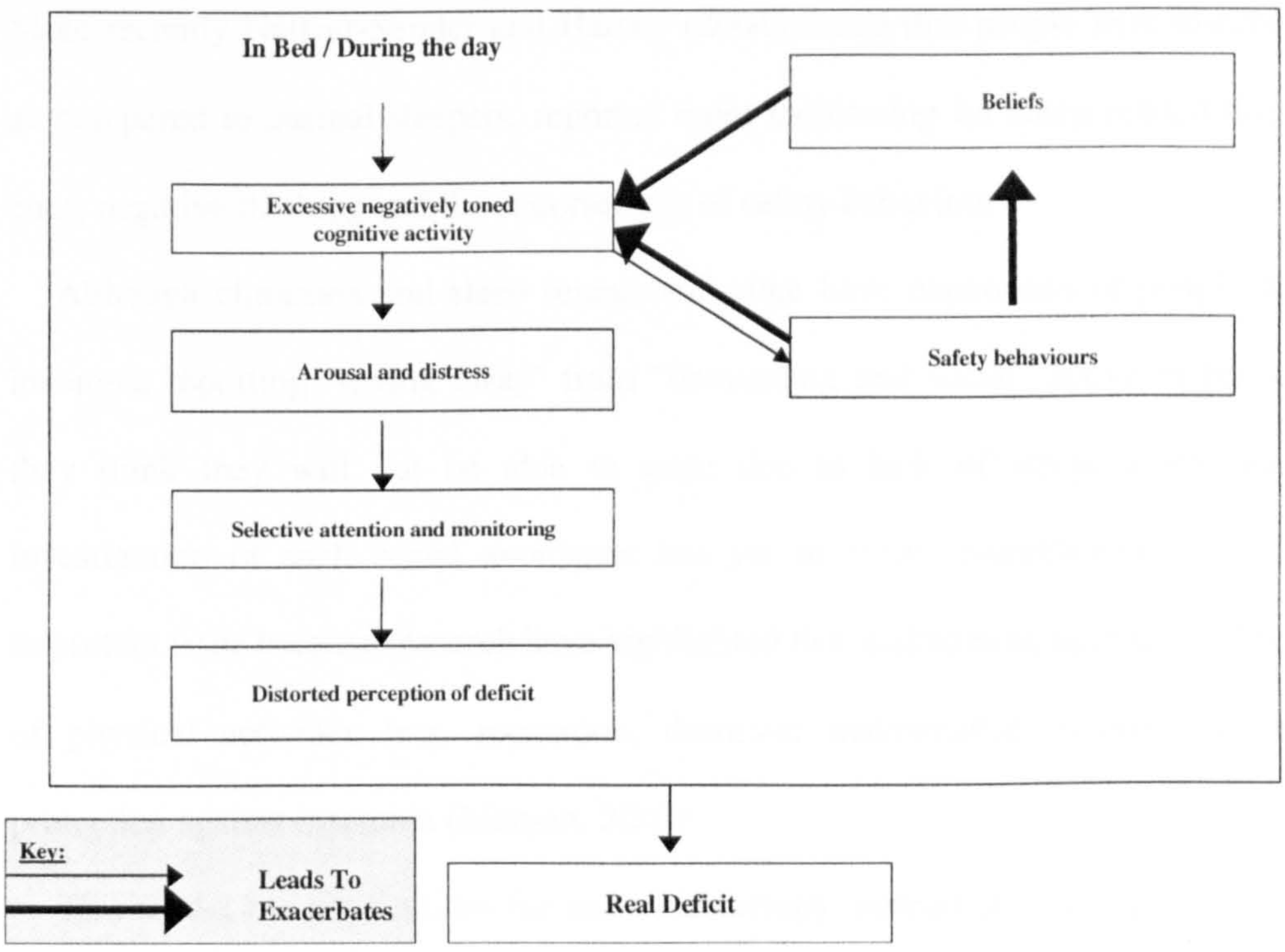
1.4.3.1 Cognitive Model of the Maintenance of Insomnia (Harvey, 2002a)

Harvey's (2002a) cognitive model of insomnia draws on contemporary experimental cognitive approaches to psychopathologies in general and anxiety disorder in particular. This model argues that selective attention and the processes hindering the automatic corrections of distortion of perception typically seen in anxiety disorders maintain insomnia.

According to this model (depicted in figure 4) insomnia is fuelled by worry and rumination about sleep and the daytime consequences of little sleep. This negatively valenced cognitive activity triggers autonomic arousal. The 'fight or flight' state produces selective attention towards threat-cues related to the initiation of sleep and the individual monitors body sensations and environmental stimuli (Neitzer-Semler and Harvey, 2004). In turn, monitoring internal and external events that could disrupt sleep increases both cognitive and physiologic arousal so that the individual is more likely to find evidence of arousal, which reinforces monitoring behaviour. In

addition, selective attention and monitoring the consequences of poor sleep can lead to detecting daytime deficits and to distorted perception of the causes of such deficits. This process ultimately leads to the endorsement of counterproductive safety behaviour (e.g. avoiding demanding work and social activities), which, in turn, increases worry regarding lack of sleep (Harvey, 2002a).

Fig. 4 - Cognitive Model of the Maintenance of Insomnia (Harvey, 2002a)



A key point of this model is that insomnia is maintained by cognitive processes and not by deficits in the sleep/wake cycle. This model makes a novel contribution to the cognitive perspective in that it introduces the concepts of selective attention, distorted perception and safety behaviours and, very importantly it places equal weight upon cognitive activity at night and during the day.

Research findings supporting the concept of negatively toned cognitive activity at bedtime has been reviewed in the preceding section and lends support to this model.

With regard to selective attention to the environment, Harvey and Schmidt (2000) found that clock monitoring increased SOL in both normal and poor sleepers. While this finding is evidence that looking at the clock impacts negatively on SOL, it does not 'prove' that clock monitoring is a typical behaviour engaged by people with insomnia. From a conditioning perspective, on the other hand, it could be argued that clock monitoring is a sleep-incompatible behaviour hence not eliciting falling asleep. More recently Neitzer-Semler and Harvey (2004) found that people with insomnia, as compared to normal sleepers, reported more monitoring for sleep related threat-cues, negative thinking and the endorsement of safety behaviours.

Although clinicians and sleep researchers alike have experience of people with insomnia reporting 'shying away' from "demanding and social" activities because they think they will not be able to cope due to lack of sleep, a systematic investigation of such social avoidance has yet to come. Nonetheless this is an important issue because research have highlighted that maintaining appropriate levels of physical activities (e.g. recreation, domestic maintenance, walking) affords protection against insomnia (Morgan, 2003).

This model has implications for insomnia therapy: instead of focusing directly on increasing amount of sleep and decreasing SOL and WASO, therapy should aim at i) decreasing selective attention and monitoring for sleep related threat cues; ii) correcting distorted perceptions about lack of sleep and its consequences and iii) eliminating the endorsement of safety behaviours.

None of the models discussed in the previous sections stand alone as a comprehensive theoretical model of the emergence, exacerbation and maintenance of

insomnia: each explains part of the empirical findings regarding physiological, behavioural and cognitive relation with insomnia. These frameworks are obviously not mutually exclusive and, as the next section highlights, some sleep scholars have proposed models of insomnia integrating the physiological, environmental and cognitive perspectives.

1.4.4 Integrative Models of Insomnia

Several integrative models of insomnia have been put forward over the past decades and furthered insight into factors likely to be implicated in the maintenance and exacerbation of insomnia. In this section, however, only four integrative models will be critically appraised. Kales and Kales' (1984) model has been selected because it incorporates psychodynamic concepts and explicitly points out secondary gains which may hinder the person's desire to overcome insomnia. Morin's (1993) model takes into account individual and environmental factors and argues that the consequences of insomnia can become the cause in a bi-directional loop. Perlis et al's (1997) model introduces the concept of classical conditioning in insomnia and provides a rationale of how this can activate cortical arousal producing enhanced sensory and information processing in the quiet hours of sleep. Espie's (2002) model stands out because, differently from the general consensus that insomnia is linked to hyperarousal, it proposes that mechanisms inhibiting de-arousal are responsible for the inability to fall asleep.

1.4.4.1 Insomnia as a Psychobehavioural Disorder (Kales and Kales, 1984)

Kales and Kales (1984), building on their experience of evaluating and treating individuals with insomnia, proposed that insomnia is a psychobehavioural disorder (characterised by excessive somatic symptomatology despite being functional in nature) and as such it is best understood by using the biopsychosocial model of illness. Similarly to other psychobehavioural disorders, insomnia affects and is affected by a number of facets of the person's life: personality factors, the illness itself, the family, the physician-patient interaction and society.

Supported by research evidence and strongly rooted in psychodynamic theories, Kales and Kales proposed that a tendency to internalise emotions and to have certain vulnerabilities rooted in childhood (i.e. emotional deprivation in childhood leading them to a feeling of inadequacy, insecurity and dependency in adulthood) can cause the emergence and maintenance of insomnia. It is therefore understandable that the insomniac may develop feelings of entitlement and expect to be nurtured and cared for by others (Healy, Kales, Monroe et al, 1981 as cited in Kales and Kales, 1984; Coursey, Buchsbaum and Frankel, 1975; Kales, Kales and Bixler, 1976).

In other words the internalisation of psychological conflicts is a source of chronic emotional arousal, which produces physiological activation, which in turn causes insomnia. In support of their model, Kales and Kales (1984) cited two studies involving more than 500 participants employing the Minnesota Multiphasic Personality Inventory. It was found that the predominant personality characteristic of people with insomnia, as compared to controls, was a strong tendency to internalise psychological conflicts rather then to express them openly. It can be argued that internalisation of conflicts gives rise to constant worry and frustration and,

interestingly, people with insomnia scored higher than normal sleepers in personality factors related to trait worry (Dorsey and Bootzin, 1997). In addition, Kales and Kales (1984) proposed that the initial sleep difficulty can give rise to a fear of sleeplessness as well as an expectation of poor sleep, both contributing to further emotional and psychological arousal and consequently maintenance and exacerbation of insomnia.

An important point often overlooked is that insomnia symptoms can generate secondary gains such as sympathy and attention (Kales et al, 1976). The social environment may contribute to the persistence of insomnia by providing benefits that hinder willingness to rehabilitation. In this way, insomnia itself may have a reinforcing effect by offering the individual a way to manipulate their environment and avoid unwanted aspects of daily life.

1.4.4.2 An Integrative Model of Insomnia (Morin, 1993)

In 1993 Morin, putting together research findings and clinical experience, proposed an integrative model of insomnia based on social learning theory, in particular the stimulus-organism-response-consequence model (see Haynes and O'Brien, 1990). Morin (1993) reasoned that the main mediating feature of insomnia must be hyperarousal because arousal regulates the balance between wakefulness and sleep and excessive arousal is incompatible with sleep. Depending on the individual, hyperarousal can manifest itself in the motoric, cognitive-affective and /or physiological (both central and/or autonomic systems) domains.

In addition sleeplessness can exacerbate hyperarousal in a vicious loop. A variety of stimulus conditions (e.g. associations about bedtime routines and worry about not

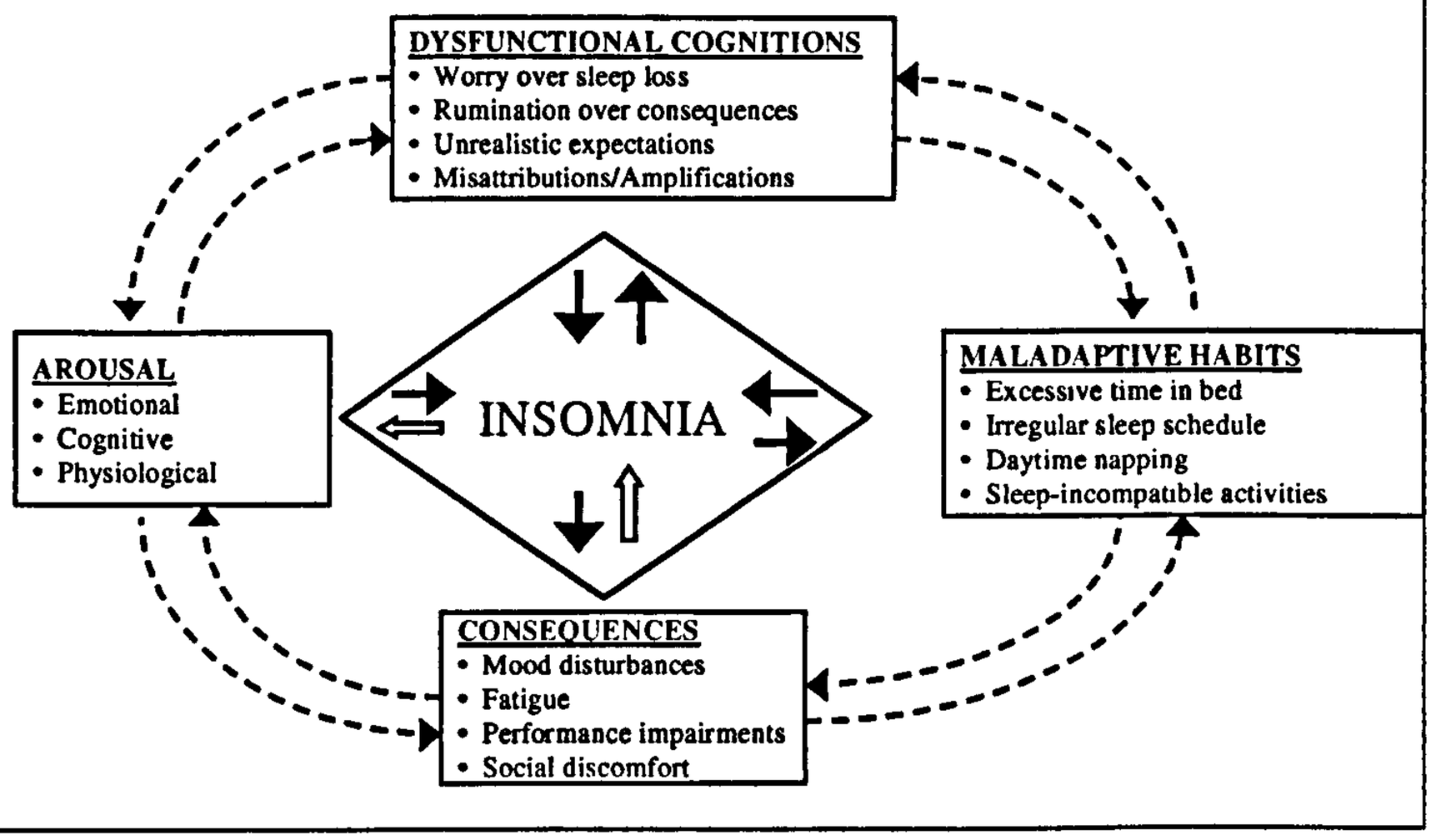
being able of falling asleep) can elevate arousal above a critical threshold and interrupt the natural sequence of relaxation, drowsiness and falling asleep. Morin suggested that people prone to insomnia might be more prone to conditioning. As proposed in section 1.4.2, people with insomnia might display a biological preparedness to learn certain associations but this hypothesis has not been empirically tested. Furthermore events during the day may influence the inability to sleep: internalisation of psychological conflicts, as proposed by Kales and Kales in 1984, may fuel arousal at bedtime and strengthen the underlying conditioning process.

Typical responses to sleeplessness include worry over sleep loss, rumination about daytime events, muscle tension and restlessness. In addition the person tries harder to fall asleep which increases performance anxiety. Next day consequences (perceived or real) involve fatigue, mood disturbances and performance impairments and these, in turns, fuels the memory of the bad night and can, over time, produce a sense of helplessness. In order to cope with insomnia people develop maladaptive behaviours such as napping which, while temporarily minimising sleep loss, overtime interfere with the synchronising effect of regular sleep-wake rhythm. Similarly to the three factor model (Spielman et al, 1987), Morin's (1993) model proposes that chronic insomnia is generally preceded by situational insomnia, brought on by stressful life events, that failed to improve once stressors disappeared.

This model highlights that the consequences of insomnia can become the causes and vice-versa: there is a bi-directional influence among these controlling variables (figure 5). This is a strength of Morin's model in that it has explanatory power of the

persistence of insomnia complaints and of the fact that insomnia is unlikely to remit spontaneously.

Fig. 5 - An Integrative Model of Insomnia (Morin, 1993)



There is evidence (albeit not unequivocal) for the components of this model. In previous sections evidence for cognitive, emotional and physiological arousal in insomnia have been discussed and causality has been questioned. The hypothesis that people with insomnia, as compared to controls, endorse dysfunctional cognitions regarding sleep and consequences of lack of sleep has been evaluated and many examples of research findings supporting this hypothesis have been provided. It has also been pointed out that there is scarce direct testing of the hypothesis that people with insomnia endorse more maladaptive behaviour than normal sleepers. Finally, although most insomnia patients complain of daily deficits (Morin and Espie, 2003) objective evaluation of daily functioning has found only mild impairments (Riedel and Lichstein, 2000).

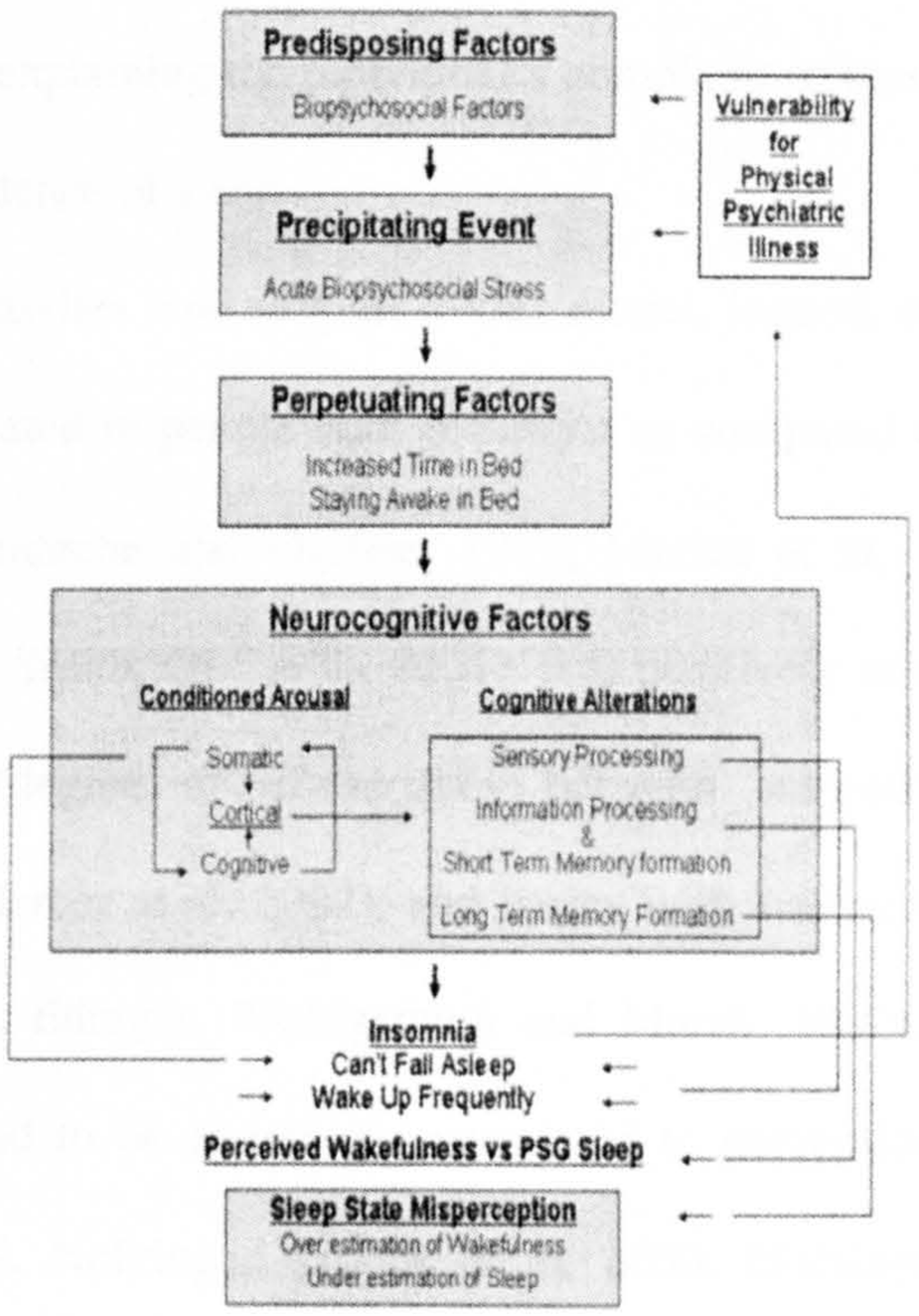
Morin points out that this theoretical framework has several implications for the management of chronic insomnia. First, learned behavioural and cognitive responses play a major role in maintaining insomnia, thus, treatment should focus on maladaptive sleep habits and dysfunctional sleep cognitions. Given that teaching to control sleep might exacerbate performance anxiety and, consequently, ability to sleep, this model posits training people with insomnia to control temporal, contextual, behavioural and cognitive factors that facilitate sleep rather than to control sleep itself.

1.4.4.3 The Neurocognitive Model of Insomnia (Perlis et al, 1997)

The neurocognitive model (Perlis et al, 1997) can be viewed as an extension of the behavioural model proposed by Spielman et al (1987). As illustrated in figure 6, this model acknowledges that cognitive factors might be mediating the occurrence and severity of insomnia in its acute phase and that cognitive activity might extend wakefulness. However, it argues that chronic insomnia is a 'reversible' central nervous system disorder due to operant and classical conditioning (Perlis, Smith and Pegeon, 2005) and that dysfunctional beliefs and worry are epiphenomena to (that is a by-product of) insomnia rather than a cause of insomnia. As discussed previously this possibility has been raised by other researchers (e.g. Morin, 1993, Espie, 2002).

Perlis and colleagues reasoned that wakefulness in bed might elicit arousal and that the continuing pairing of bed environment and wakefulness might be classically conditioning the individual to be aroused by the bedroom itself. Such conditioned arousal might perpetuate insomnia even when sleep incompatible or maladaptive behaviours (e.g. watching TV in bed, staying in bed for long periods) are removed.

Fig. 6 : The Neurocognitive Model of Insomnia (Perlis et al, 1997)



The neurocognitive model took its lead from previous work (e.g. Mendelson et al, 1986) on the perception of being awake while actually being asleep according to EEG sleep staging. It argues that cortical arousal is the result of classical conditioning and it is observable in people with primary insomnia as a high-frequency EEG activity (such as Beta activity, 14-45 Hertz) prior to falling asleep and during NREM sleep. Beta activity allows for higher levels of sensory and information processing making the individual more sensitive to environmental

stimuli. Being more sensitive to stimuli interferes with the ability to 'let go of the surrounding' and initiate sleep and can, hence, explain longer SOL.

Likewise increased information processing during PSG-defined sleep is proposed to interfere with the person's ability to distinguish sleep from wakefulness and, therefore, can help in explaining the individual's complaint of hardly having slept all night despite PSG evidence of sleep.

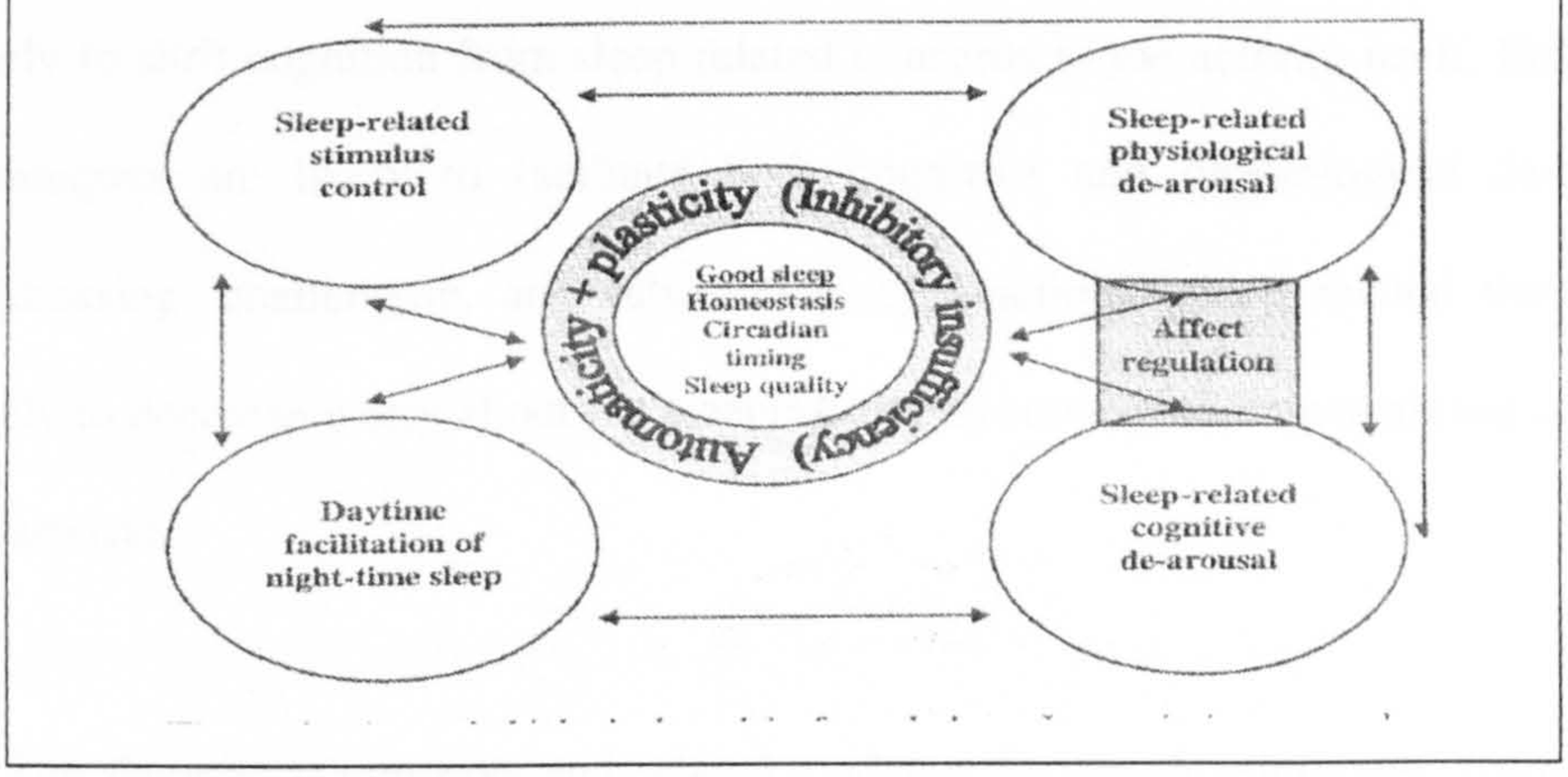
Fast EEG activity studies lend support to this model. Indeed, studies have shown that Beta EEG is elevated in people with insomnia as compared to normal sleepers (Freedman, 1986; Lamarche and Ogilvie, 1997; Merica et al, 1998; Merica and Gaillard, 1992; Perlis, Smith, Orff et al, 2001), it is positively associated with sleep state misperception (degree of discrepancy between subjective and objective measures of sleep) (Mercer et al, 2002), and varies with successful CBT treatment for insomnia (Krystal, Edinger, Wohlgemuth and Marsh, 2002). In addition Beta activity has been found to be negatively associated to perception of sleep quality (Hall, Buysse, Nowell, Nofzinger, Houck et al, 2000; Nofzinger, Price, Metzer, Buysse, Villemagne et al, 2000; Perlis, Merica et al, 2001). It is important to note that the evidence that increased high frequency activity appeared to be in the Beta and Gamma bands of the EEG suggests that the primary source potential for the signal is EEG (i.e. cortical arousal), rather than EMG (i.e. somatic arousal) which supports the suggestion that cortical and somatic arousals are distinct phenomena. Although the findings from these studies reveal cortical arousal the possibility that it is a consequence of cognitive arousal (e.g. being more alert because of scanning the environment for events threatening sleep) rather than being classically conditioned cannot be ruled out.

1.4.4.4 Psychobiological Inhibition Model of Insomnia (Espie, 2002)

The models of insomnia discussed in the previous sections focus on hyperarousal. Espie (2002), by contrast, proposed that insomnia is associated to the individual's inability to de-arouse. His psychobiological inhibition model examined insomnia from a different angle than the one traditionally taken. Rather than considering insomnia as the result of hyperarousal and its sleep inhibiting effects (inhibition of the 'falling asleep' system), Espie conceptualised insomnia as the malfunction of mechanisms and processes inhibiting wakefulness (inhibition of the 'wake-off' system). According to this model, and typical of neurobiological systems, good sleep is the natural state for the individual with both functional plasticity (accommodating night-to-night variations) and automaticity. Espie (2002) proposed automaticity of 'sleep homeostasis' and 'circadian timing' (for details see Dijk and Czeisler, 1995) to be the controlling feature of good sleep. Importantly, the concept of automaticity encapsulates the idea that good sleep is a 'passive' state that does not require effort.

As depicted in figure 7, good sleep is maintained by four interacting components: sleep-stimulus control, physiological de-arousal, cognitive de-arousal and daytime facilitation. In normal sleep both physiological and mental cues of sleep readiness are accurately interpreted and cues in the home environment reinforce cognitive and physiological de-arousal so that the individual enters sleep effortlessly. In insomnia, instead, cognitive phenomena (preoccupation with sleep, intention to sleep and effort to sleep) inhibit such de-arousal processes and make sleep difficult both at the beginning and during the night (for a formal conceptualisation of the attention-intention-effort pathway to insomnia see Espie, Broomfield, MacMahon, Macphee and Taylor; 2006).

Fig. 7 - Psychobiological Model of Good Sleep (Espie, 2002)



This model helps explain the transition from acute to chronic insomnia: attempts to re-gain normal sleep (e.g. focussing on ways to sleep well, going to bed earlier if the night before was bad) are likely to give rise to homeostatic dysregulation, circadian timing problems and conditioned arousal.

Perlis et al (2005) pointed out that considering hyper-arousal and de-arousal as two separate concepts enables us to define insomnia more clearly and to search for its neurobiological basis. In addition such differentiation permits to better understand mechanisms of effect in insomnia treatment programmes. Empirical research on the distinction of these two concepts is, therefore, warranted.

This model suggests that the efficacy of multicomponent cognitive-behavioural therapies (CBT) (outlined and discussed in the next session) may rely on the de-activation of inhibitory mechanisms of de-arousal. Sleep hygiene might remove some potential inhibitors of physiological de-arousal. Sleep scheduling (temporal instruction of stimulus control therapy and sleep restriction), re-adjusts the balance between the homeostatic sleep drive and the biological clock, and might also de-activate inhibition processes of cognitive de-arousal. In other words, when falling

asleep is difficult, giving up trying to sleep enables to decrease mental effort and consequently to fall asleep. Performing an activity rather than lying in bed awake is likely to shift cognition from sleep related concerns to the activity itself. Relaxation techniques are likely to facilitate both cognitive and physiological de-arousal. Addressing, challenging, and substituting dysfunctional sleep related thoughts is likely to decrease worry about not sleeping and its consequences permitting cognitive de-arousal.

The theoretical concepts and related evidence for the development, maintenance and exacerbation of insomnia inform insomnia therapy to which attention is now turned.

1.5 Therapeutic Approaches to the Treatment of Insomnia

In this section the principal therapies for insomnia (table 4), informed by one or the integration of the perspectives discussed in the preceding section, are briefly outlined.

Table 4 - Insomnia Therapy according to Underlying Perspective

Therapy Perspectives:	Physiological	Behavioural	Cognitive
Pharmacotherapy	v		
Sleep Hygiene	v	(v)	
Progressive Muscle Relaxation	v		
Diaphragmatic Breathing	v		
Biofeedback Assisted Relaxation	v		
Stimulus Control		v	
Sleep Restriction		v	
Paradoxical Intention			v
Imagery			v
Suppression			v
Cognitive Restructuring			v
CBT	v	v	v

Given that the experiments devised and carried out in the present research relate to stimulus control therapy, in addition to being presented here, stimulus control is explained in details and critically appraised in subsequent sections.

1.5.1 Treatments Related to the Physiological Perspective

1.5.1.1 Pharmacotherapy

Pharmacotherapy derives from the physiological perspective and it is the most popular approach for treating insomnia patients in primary care (Dement, 1994 as cited in Edinger and Wohlgemuth, 1999). It is noteworthy that Greater Glasgow remains amongst the highest prescribing areas in Scotland per head of weighted population during 1999 according to ISD Primary Care Unit statistics. Although pharmacotherapy has been shown to produce short-term sleep improvements, there is little evidence of its long-term efficacy (Kramer, 2000) and prolonged use of hypnotics is not recommended due to risks of dependence, sedative side effects, misuse and associated morbidity (Russell and Lader, 1992).

Perlis, Pigeon and Riemann (2006, submitted) summarised the findings of four meta-analyses on the use of pharmacotherapy (hypnotics and off label treatment with sedating antidepressant) and concluded that there is no firm evidence of the efficacy of pharmacotherapy for the treatment of persistent insomnia. Furthermore, they warned that, in the elderly, treatment benefits are outweighed by the risks. Their summary is in agreement with the NIH Statement produced in 2005 regarding treatment of insomnia (National Institute of Health, 2005).

Kripke (2000) argued that prescription should be limited to short courses and substituted by cognitive behavioural treatment (CBT). Indeed, more recently NICE (2004) recommended GPs to consider non-medicine as the first line of insomnia treatment and to prescribe hypnotic drugs only in the most severe cases of insomnia and only for short periods. Importantly, a pragmatic randomised controlled trial evidenced the efficacy and cost effectiveness of CBT for insomnia (CBT-I) in primary care patients presenting with a history of long term hypnotic drug use (Morgan, Dixon, Mathers, Thompson and Tomeny, 2003). Similarly to Morin, Colecchi, Ling, and Sood's (1995) results, Morgan et al's (2003) study showed that CBT-I facilitated hypnotic drug use decrease. Another important finding of Morgan et al's (2003) study was that following CBT-I, participants' sleep and quality of life were improved as compared to participants who continued with their usual treatment (i.e. hypnotic drugs).

Given that research findings and guidelines suggest that pharmacotherapy for persistent insomnia is not the best line of treatment (Kramer, 2000), it is not surprising that a variety of behavioural and cognitive therapies for insomnia have been devised.

1.5.1.2 Sleep Hygiene

Sleep hygiene is typically regarded as a behavioural strategy which, being educational in nature, sits well within a cognitive framework. Nonetheless, its aim is to ensure that agents heightening physiological arousal are avoided in the hours before bedtime and during sleep. In fact, sleep hygiene most often involves providing the patient with a list of behaviour (e.g. ingesting too much caffeine, smoking,

alcohol consumption) that may influence sleep quantity and quality. Although sleep hygiene is very popular, research findings evidenced that it is not efficacious as a standalone insomnia treatment (Morin, Culbert and Schwartz, 1994).

1.5.1.3 Relaxation

A number of relaxation techniques have been devised, each targeting a particular physiological system. The rationale being that a person with physiological arousal will find it difficult to fall asleep, hence if relaxation is induced sleep will follow. For example 'progressive muscle relaxation' aims at decreasing muscle tension (Haynes, Woodward, Moran and Alexander, 1974). Diaphragmatic breathing aims at moving the patient from thorax to abdomen breathing (i.e. deeper and slower). Biofeedback assisted relaxation aims at inducing muscle relaxation by frontalis EMG biofeedback (see Bootzin and Nicassio, 1978 for a review). It should be noted that the efficacy of relaxation in improving sleep does not lend support, per se, to the physiological framework. Indeed research found that sleep improvements following relaxation were not coupled to post treatment changes on physiological measures (e.g. heart rate, respiration) (Hauri, 1981; Borkovec, Grayson, O'Brien, Weerts, 1979).

1.5.2 Treatments related to the behavioural perspective

1.5.2.1 Sleep restriction

As discussed previously, poor sleepers tend to extend the sleep window opportunity (going to bed earlier and staying in bed longer, especially if the night before was a bad night) in an effort to provide more opportunity to sleep: this

strategy, however, is likely to result in fragmented and poor quality sleep. Spielman and colleagues (1987), drawing on the idea that homeostatic sleep drive and accurate circadian timing regulate sleep and that the individual's coping strategies disrupt these process, devised a therapy named 'sleep restriction'.

Sleep restriction aims at increasing S.E. by restricting the amount of time spent in bed (so as to approximate it to the actual time spent asleep) and at regularising sleep patterns by going to bed and getting up at the same time each day. Once S.E. is improved (i.e. S.E. ≥85%) the time in bed is extended by 15 minutes per night per week until the patient's S.E. starts to decline again. Morin (2005) proposed that sleep restriction improves sleep continuity due to a mild sleep deprivation and a reduction of sleep performance anxiety. Morin et al's (1994) meta-analysis found sleep restriction to improve both SOL and WASO.

1.5.2.2 Stimulus Control Therapy for Insomnia

By applying the conditioning conceptualisation of insomnia already discussed (section 1.4.2.1) a stimulus control therapy for insomnia was devised (Bootzin, 1972; Bootzin and Nicassio, 1978). Given that stimulus control is the most efficacious single therapy for insomnia, and the present research focus on one of its elements, the stimulus control treatment and associated efficacy studies are discussed in details in section 1.7. In brief the stimulus control package aims at bringing the act of falling asleep quickly under the stimulus control of the bedroom environment by training the person with insomnia to re-associate temporal (bedtime) and environmental (bedroom) cues for sleep with rapid sleep onset.

Before turning attention to cognitive therapies for insomnia it should be noted that sleep restriction and stimulus control are most often used together: for example, Espie and colleagues call this combination 'sleep scheduling' (e.g. Espie, Inglis, Tessier and Harvey, 2001) in their CBT intervention.

1.5.3 Cognitive Treatments for Insomnia

As discussed in previous sections there is ample evidence that individuals with insomnia report heightened cognitive arousal at bedtime. The basic premise of cognitive therapy is that appraisal of sleeplessness can trigger negative cognitions, emotions and beliefs about lack of sleep and its consequences that are incompatible with sleep. A number of cognitive treatments for insomnia aiming to produce sleep by altering faulty beliefs and attitudes about sleep have been devised. For example 'paradoxical intention' aims at increasing the chance of falling asleep quickly by obviating the performance anxiety that develops because of trying to control sleep. It directs the individual to try at any cost not to fall asleep: it is a "let it be attitude" and it is the only cognitive technique that does not directly try to decrease cognitive activity. 'Imagery', in contrast, attempts to distract the individual from thinking about sleep related topics. 'Suppression' aims at blocking out the thought content itself. Cognitive restructuring teaches the individual to rationally re-appraise their thought contents and to de-catastrophise the consequences of a bad night's sleep. These techniques might all be cognitive but they focus upon different solutions to the mental problem and represent variants of a cognitive model of insomnia. Interestingly, given the amount of research and related theoretical debate regarding

the importance of cognition in insomnia, the majority of therapeutic intervention and outcome studies are not based solely on cognitive therapy.

1.5.4 Multifaceted Cognitive Behavioural Therapy

The different interventions described so far are obviously not incompatible with each other. Indeed they can be combined and in recent years treatment packages, each including several single therapies for insomnia, have become the standard practice (Morin, 2005). Cognitive Behaviour Therapy (CBT) for insomnia typically includes stimulus control, sleep restriction, cognitive restructuring and sleep hygiene. The CBT package might also include other single therapies such as relaxation, paradoxical intention and imagery.

In sum, a number of psychological therapies for insomnia have been devised and in recent years insomnia therapy has typically been delivered in a multicomponent package (CBT). Having briefly outlined the principal insomnia therapies it is important to turn attention to evidence of treatment efficacy. Meta-analytic reviews pertaining to behavioural and psychological therapies for insomnia permit evaluation of treatments across studies and to identify therapies standing out as most efficacious.

1.6 Treatment Outcome Evidence

This section presents a summary of the evidence currently available in support of non-pharmacological interventions for insomnia. Findings from meta-analyses are summarised and discussed and a brief overview of recent clinical trials is provided

taking into account issues of clinical significance and long-term impact. Firstly, meta-analytic procedures are briefly explained.

1.6.1 Meta-Analytic Procedures

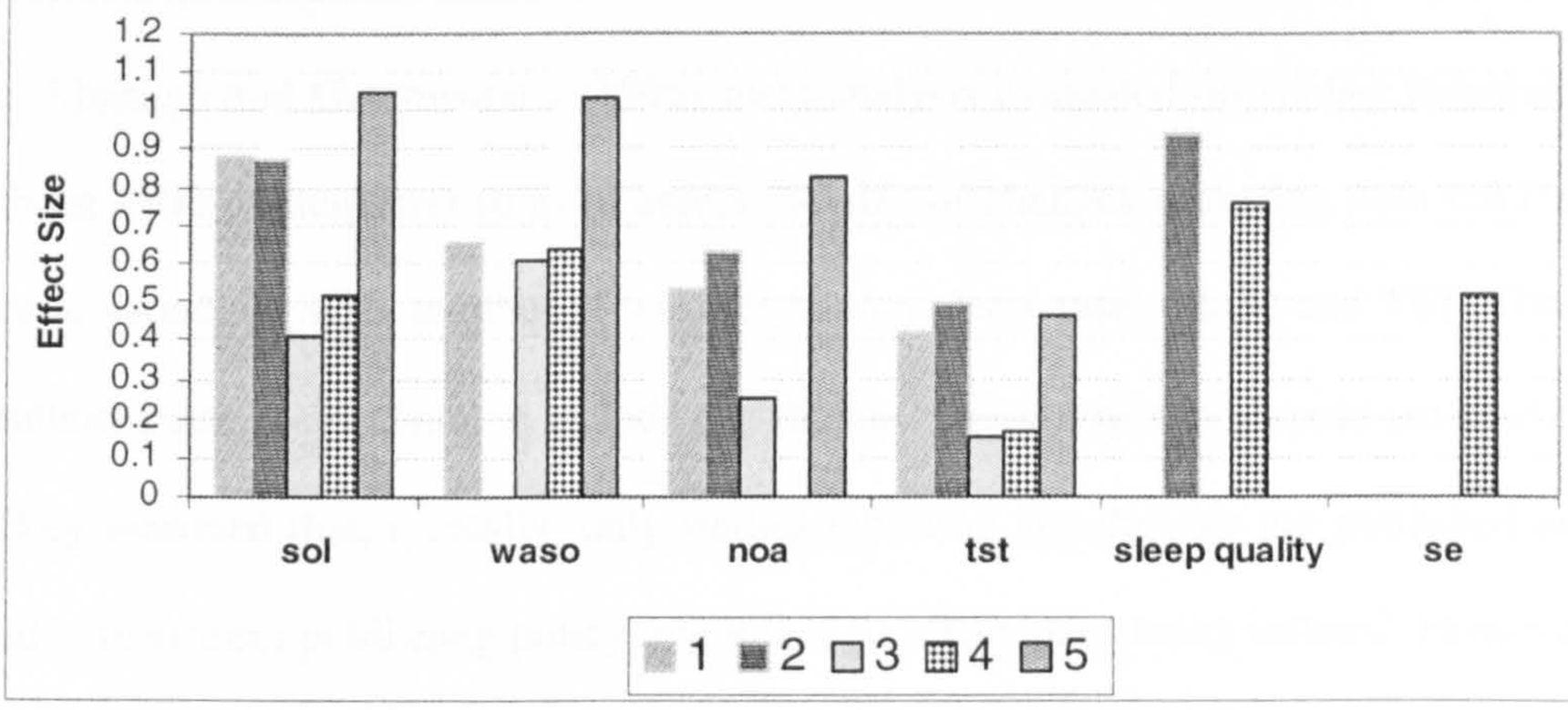
Meta-analytic procedures are an important tool as they permit the systematic evaluation of treatment outcome across multiple studies. Statistics such as t tests or F tests are not appropriate for such comparisons as the values obtained with these tests are partially a function of the sample size. In other words, studies with equivalent differences between treatment and control conditions can have very different t and F values depending on the sample sizes. In contrast, estimates of effect size provide a standard metric for assessing treatment effectiveness across multiple trials because they are not influenced by sample sizes. The most common estimate of effect size is Cohen's d (Cohen, 1988). Cohen's d is a measure of effect that may be thought of as determining how many standard deviations separate the means of two groups. Cohen (1988) hesitantly defined an effect size of 0.20 as a small effect, 0.50 as a medium effect and 0.80 as a large effect and acknowledged the danger of using terms such as 'small', 'medium' and 'large' out of context ("there is a certain risk in offering conventional operational definitions for those terms for use in power analysis in as diverse a field of inquiry as behavioural science", p.25).

1.6.2 Meta-Analytic Findings

Outcomes of studies evaluating non-pharmacological interventions for insomnia have been summarised in six meta-analyses (Irwin, Cole and Nicassio, 2006; Morin et al, 1994; Montgomery and Dennis, 2004; Murtagh and Greenwood 1995; Pallesen,

Nordhus and Kvale, 1998; Smith, Perlis, Park, Smith, Pennington, et al, 2002). In figure 8 the effect sizes reported by each of these meta-analyses are reported.

Fig: 8 - Effect Sizes for Sleep Parameters Obtained with Psychological Interventions as reported in 5 Meta-Analyses



1=Morin et al, 1994; 2=Murtagh et al, 1995; 3=Pallesen et al, 1998; 4=Irwin et al, 2006; 5=Smith et al, 2002 (Montgomery et al, 2004 did not provide ES)

In 1994, Morin et al reviewed 59 studies (involving more than 2000 participants) on the psychological treatment of insomnia so as to assess the size of changes occurring with CBT. With regard to SOL the effect size was 0.88 (i.e. on average the treated person was 0.88 standard deviation better than the untreated people) and the average treated individual was falling asleep faster than 81% of those who did not receive treatment. With regard to WASO the effect size was 0.65 and the treated person had WASO shorter than 74% of those who did not receive treatment. TST effect size was found to be 0.42. The absolute values of improvement rate were 27.7 minutes for SOL (43% improvement), 32.7 minutes for WASO (46% improvement) and 28.5 minutes for TST (8% improvement). Only the effects for SOL and WASO were statistically significant. The modest improvement in TST might be due to the initial impact of stimulus control and sleep restriction. Finally comparison of various treatment modalities showed that **stimulus control** and sleep restriction (although

the authors advised to be caution regarding the latter therapy as only a few studies were available) were the **most effective** single therapies for both SOL (improvement of 32 minutes) and WASO (improvement of 40 minutes) while sleep hygiene was not effective as standalone therapy.

Murtagh and Greenwood's (1995) meta-analysis evaluated 66 studies (involving about 1500 participants) so as to assess the size of changes occurring with CBT on three variables: SOL, number of wakenings after sleep onset (NoA) and TST. These authors made the decision to include unpublished manuscripts they could get hold of. They reasoned that, typically, only studies reporting significance are published and this (unwritten) publishing policy might lead to effect sizes being inflated. However, they found effect sizes to be similar to those found by Morin et al (1994): 0.87 for SOL, 0.63 for NoA and 0.49 for TST. The absolute values of improvement rate were 24 minutes for SOL (39% improvement), –1.2 for NoA (73% improvement) and 32 minutes for TST (9% improvement). **Stimulus control** global effect size was higher than that of any other therapy and SOL improvement was 32 minutes. Similarly to Morin et al's (1994) meta-analysis findings, stimulus control appeared to **provide the greatest improvement in general.**

These two meta-analyses also examined follow-up data: the improvements obtained were maintained and for certain variables (SOL and TST) changes from baseline values actually increased.

In 1998 Pallesen et al evaluated the outcome of thirteen studies examining the effects of CBT-I in the older population. Significant effects were found for SOL (effect size was 0.41), NoA (effect size 0.25), WASO (effect size 0.61) and TST

(effect size 0.15). The effect sizes were higher at follow-ups. It was, therefore, concluded that CBT-I is also indicated in the older population.

By contrast, Montgomery and Dennis' (2004) summary of outcomes for CBT-I in the older population provided by six studies yielded positive results only for WASO and TST. They found that CBT had a modest impact on WASO (decrease of 22 minutes) and on TST (increase of 14.6 minutes) and practically no effect on SOL. They concluded that CBT-I is indicated for WASO in the older population.

In 2006 Irwin and colleagues carried out a further meta-analysis evaluating the impact of behavioural interventions for insomnia in older adults. Evaluation of 23 randomised controlled trials showed significant effects on sleep quality (effect size of 0.76), SOL (effect size of 0.52) and WASO (effect size of 0.64). Behavioural therapies (**stimulus control** and sleep restriction) effect sizes were found to be in the region of 0.91 for sleep quality, 0.59 for SOL and 0.82 for WASO.

Finally, Smith and colleagues in 2002 published a comparative meta-analysis of pharmacotherapy and behavioural therapies for chronic insomnia. With regard to the impact of behavioural therapies on sleep the following effect sizes and absolute changes were found. The effect size for both SOL and WASO was 1.0, for TST was 0.46 and for sleep quality rating was 1.44. Behavioural therapies fared better than pharmacotherapy for all values but TST. However, this outcome might be due to sleep restriction curtailing amount of sleep. Absolute values of improvement rate were 23.3 minutes (43% improvement) for SOL, 38.4 minutes (55.9% improvement) for WASO, 19.6 minutes (5.9% improvement) for TST and 0.96 (28.4% improvement) for sleep quality ratings.

In sum, the data from these meta-analyses show that cognitive/behavioural treatments for insomnia produce reliable and robust changes in a number of sleep continuity parameters and that these improvement hold at follow-ups. It is important to note that stimulus control was found to produce equivalent (or better) outcomes than multicomponent CBT and other single therapies for insomnia.

1.6.3 Clinical studies

Clinical trials have evidenced that CBT is clinically effective for persistent insomnia presenting in general practice (Espie, Inglis and Harvey, 2001; Morgan, Thompson, Dixon, Tomeny and Mathers, 2003), therapeutic gains are maintained at follow up and CBT can provide a cost effective alternative to pharmacotherapy in the treatment of insomnia (Morgan, Dixon, Mathers, Thompson and Tomeny, 2004). Taking into account these findings it is not surprising that Espie (1999) argued that cognitive-behavioural treatment therapies might be the treatment of choice for chronic primary insomnia rather than pharmacotherapy.

It is noteworthy that, although the majority of people treated improve their sleep, only a small percentage of them (around 25 %) return within the normal sleep parameters. The majority still experience some (albeit less severe) sleep problems (Espie, Inglis, Tessier and Harvey, 2001; Morin, Colecchi, Stone, Sood and Brink, 1999).

1.6.4 Is CBT the Treatment of Choice?

The meta-analyses and clinical studies discussed above provide convergent evidence regarding the efficacy and effectiveness of CBT-I in reducing sleep

continuity parameters. A review conducted by the Standards of Practice Committee of the AASM (Chesson, Ferber, Fry, Grigg-Damberger, Hartse et al, 1999; Morin, Hauri, Espie, Spielman, Buysse and Bootzin, 1999) reached similar conclusions, albeit a note of caution was stated because none of the published studies was awarded 'A level'. 'A level' methodology requires a placebo control while non-pharmacological studies of insomnia typically have a waiting-list control group. Indeed, only a few studies have used attention-placebo conditions (e.g. Lichstein, Riedel, Wilson et al, 2001; Backhaus, Hohagen, Voderholzer, Riemann, 2001) while majority of clinical trials have used waiting list control groups. Without a placebo control it is difficult to attribute, unequivocally, treatment effects to any specific elements of the therapy itself (e.g. relaxation, re-conditioning) rather than to factors such as the measurement process (e.g. self-monitoring) or factors such as social desirability (Crowne and Marlow, 1960), therapist attention or patient's expectation.

It is noteworthy that stimulus control was the only therapy being warranted a full Grade B recommendation. In addition it should be noted that effect sizes for SOL and WASO reported for stimulus control have ranged between 0.70 and 1.16 while for CBT from 0.92 to 1.05 (Morin et al, 1994; Murtagh et al, 1995) and that best outcomes from CBT have been found when stimulus control (and sleep restriction) were part of the package.

1.6.5 Considerations Regarding Treatment Outcome Findings

To conclude this section the following main points are to be considered.

First, although CBT has become in recent years the standard non pharmacological therapy for insomnia, stimulus control has yielded strong effects as evidenced by the

meta-analytic reviews just discussed. Significantly, the Standards of Practice Committee of the AASM (1999) recommended stimulus control as the gold standard treatment for insomnia.

Second, despite ample evidence supporting the efficacy of psychological and behavioural treatments for insomnia, there is still scarce information regarding the specificity of these treatment modalities and the active mechanisms responsible for sleep improvements (Morin, Bastien and Savard, 2003). Not surprisingly, given that stimulus control for insomnia fared better than other single therapies, one such investigation found that the most critical ingredients associated with long-term sleep improvements were stimulus control and sleep restriction (Harvey, Inglis and Espie, 2002).

It seems, therefore, timely to investigate what the critical components of CBT are and, furthermore, what elements of each single therapy are essential. One way to carry out such investigation is by a dismantling strategy to isolate individual components (Currie, Wilson, Pontefract and deLaplante, 2000).

An important pitfall in insomnia research is the dose-response curve. While it has been amply investigated in pharmacological research this has not been the case with regard to psychological treatment. That is, behavioural therapists have generally failed to explore what "dose" of CBT is needed to achieve minimally acceptable sleep improvement (Edinger and Means, 2005). Lichstein and Riedel (1994), after performing a review of treatments of insomnia, pointed out that there appears to be wide potential for brief interventions and limited therapist participation. For example, Edinger and Sampson (2003) have found that 2 sessions of CBT produced significant improvements in insomnia symptoms. More recently, Edinger and

colleagues have explored the dose-response effects of behaviour therapy for insomnia. They assessed the optimal dose of CBT for treating WASO by administering it in 1, 2, 4 and 8 bi-weekly sessions and found that the 1 and 4 session protocols had greater efficacy than the other two conditions. Specifically a 50% reduction WASO in 58% of participants was achieved by the 4 session condition, in 44% of participants by the 1 session condition and only 23% in the 8 session condition (Edinger, Wohlgemuth, Radtke and Marsh, 2004). This is a much needed and welcomed line of research: it helps in establishing the optimum dose of therapy required to achieve a given level of improvement and providing intervention guidelines accordingly.

Whilst significant progress has been made, the field is not, as yet, at a point where the patients can be offered a maximally effective psychological treatment. This is indicated by the significant percentage (20-30%) of patients who do not improve following CBT and by the average overall improvement (see the six meta-analyses just reviewed) among those who do respond being only around 40 to 60%. Although this degree of change is statistically significant, it is not enough to convincingly move the average patient into a state where they could be classified as, and they would call themselves, normal sleepers.

Given these premises it seems very important to examine systematically from first principles (almost at a microscopic level) the single components of individual therapies that are delivered as part of the multicomponent package CBT-I. This has theoretical and more pragmatic values. Testing one element at a time helps to shed light on the theoretical underpinning of the therapy (why it works). Likewise examining single elements of insomnia packages would enable the extraction of the

essential and most therapeutic ingredients and ultimately enable the provision of optimum service while containing health costs.

Stimulus control appears to be a good candidate for a detailed examination of single elements. Not only has it been favoured by outcome findings and been recommended as the gold standard therapy for insomnia; it is also easy to administer and positive outcomes are typically obtained in a few weeks.

1.7 Stimulus Control: a Detailed Description and Critical Appraisal

This section is in three parts. First, the six elements comprising stimulus control are analysed so as to assess what the theoretical bases underpinning these elements are. Second, previous research on stimulus control therapy for insomnia is outlined and evaluated. Third, by taking into consideration both theory and research evidence, the element of stimulus control therapy deemed to epitomise stimulus control is identified.

1.7.1 Stimulus Control Therapy: its Elements

As already discussed stimulus control therapy is based on learning theory, in particular operant conditioning and stimulus control (section 1.4.2.1). Briefly, stimulus control theory holds that in chronic insomnia, sleep incompatible behaviour diminishes or extinguishes the discriminative properties for sleep of the bedroom environment.

The aim of therapy, therefore, is for the patient to be in their bed only when asleep so that the bedroom environment acquires discriminative properties eliciting the physiological response and attitude consistent with a discriminative stimulus. In

other words, the bedroom environment (discriminative stimulus) would signal the imminence of sleep (reinforcer) and the person should follow asleep quickly (operant). Subsequent sleep experiences will reinforce this process.

It is important to note that stimulus control therapy for insomnia comprises both elements of strict stimulus control and elements of temporal harmonisation of the sleep-wake schedule. In particular, by inspecting table 5, it becomes apparent that instructions 2, 3, and 4 are situational in that they aim at ensuring that the bed is used only for sleep and that the patient does not spend time in bed awake. The other instructions (1, 5 and 6) are temporal in nature: they ensure a stable sleep-wake rhythm.

Table 5 – Components of the Stimulus Control Therapy for Insomnia

1. Lie down intending to sleep ONLY when you are sleepy.
2. Do not use your bed for anything except sleep: that is do not read, watch TV, eat or worry in bed. Sexual activity is the only exception to this rule. On such occasion the instructions are to be followed afterward, when you intend to sleep.
3. If you find yourself unable to fall asleep, get up and go to another room. Stay up as long as you wish and then return to the bedroom to sleep. Although we do not want you to watch the clock, we want you to get out of bed if you do not fall asleep immediately. Remember that the goal is to associate your bed with falling asleep quickly. If you are in bed more than about 10-15 minutes without falling asleep and have not gotten up, you are not following this instruction.
4. If you still cannot fall asleep, repeat step 3. Do this as often as it is necessary throughout the night.
5. Set your alarm and get up at the same time every morning irrespective of how much sleep you got during the night. This will help your body acquire a consistent sleep rhythm.
6. Do not nap during the day.

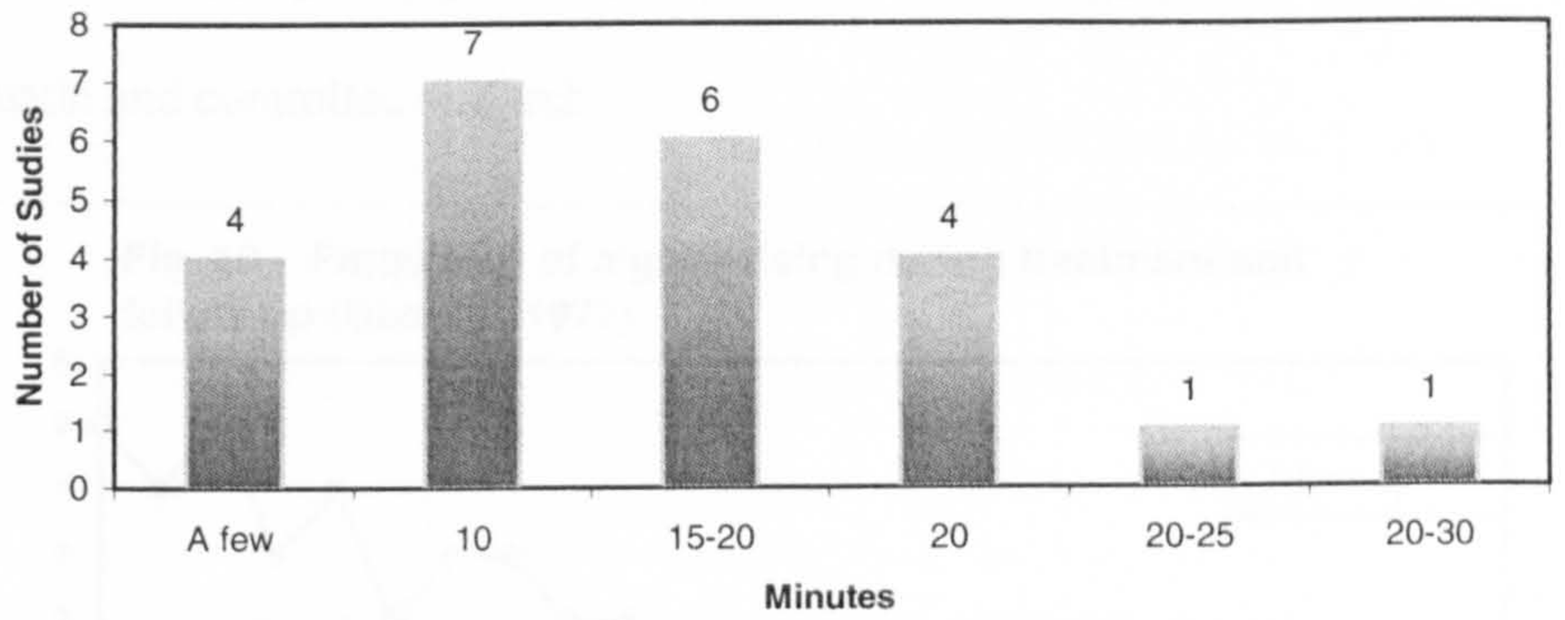
It could be argued that while situational elements sit well within a stimulus control framework, temporal elements do not. This suggests that the mechanism of effects of at least half of the stimulus control package for insomnia is likely to be due to factors other than those of operant learning theory. This reasoning restricts the choice of the best candidate to synthesise stimulus control to only two elements: 'using the bed only for sleeping' (instruction 2) and 'getting out of bed if not asleep quickly' (instruction 3 and 4). Both instructions aim at the same target: 'be in bed only when asleep'. In this way the association between bedroom and sleep is created and strengthened while associations between bedroom and wakefulness avoided. However, it is felt that instruction 2 does not explicitly tell the patient what to do if, once intending to sleep, sleep does not come. Instruction 3, by contrast, prescribes what to do (i.e. go to another room) if not asleep: in so doing it automatically prevents sleep-incompatible activities being performed in bed and ensures that the bed is used only for sleeping. The instruction "If you find yourself unable to fall asleep, get up and go to another room" can be proposed, therefore, as the element of stimulus control that better encapsulates the stimulus control essence.

It should be noted that instruction 3 requires the person to get out of bed if not asleep within 10-15 minutes. However, as pointed out by Espie (1991) this 10-15 minutes limit is arbitrary. Indeed, in his first application of stimulus control to insomnia Bootzin (1972) left this rule vague: 'if unable to sleep get up' so as to avoid the patient initiating a habit of clock watching. Espie and colleagues (Espie et al, 1989) used a 20 minutes period for initial sleep latency because in their clinical experience only few successfully treated people achieved post-treatment sleep onset latency of ten minutes. They argued that ten minutes might be too strict a threshold

when compared to the sleep pattern of normal sleepers for whom taking ten minutes to fall asleep might not be considered problematic.

As shown in figure 9, the amount of time lying awake in bed prior to "getting up and going to another room" varies from study to study (a few minutes to 25 minutes range).

Figure: 9 - Time frame for the Stimulus Control instruction 'get up and go to another room'

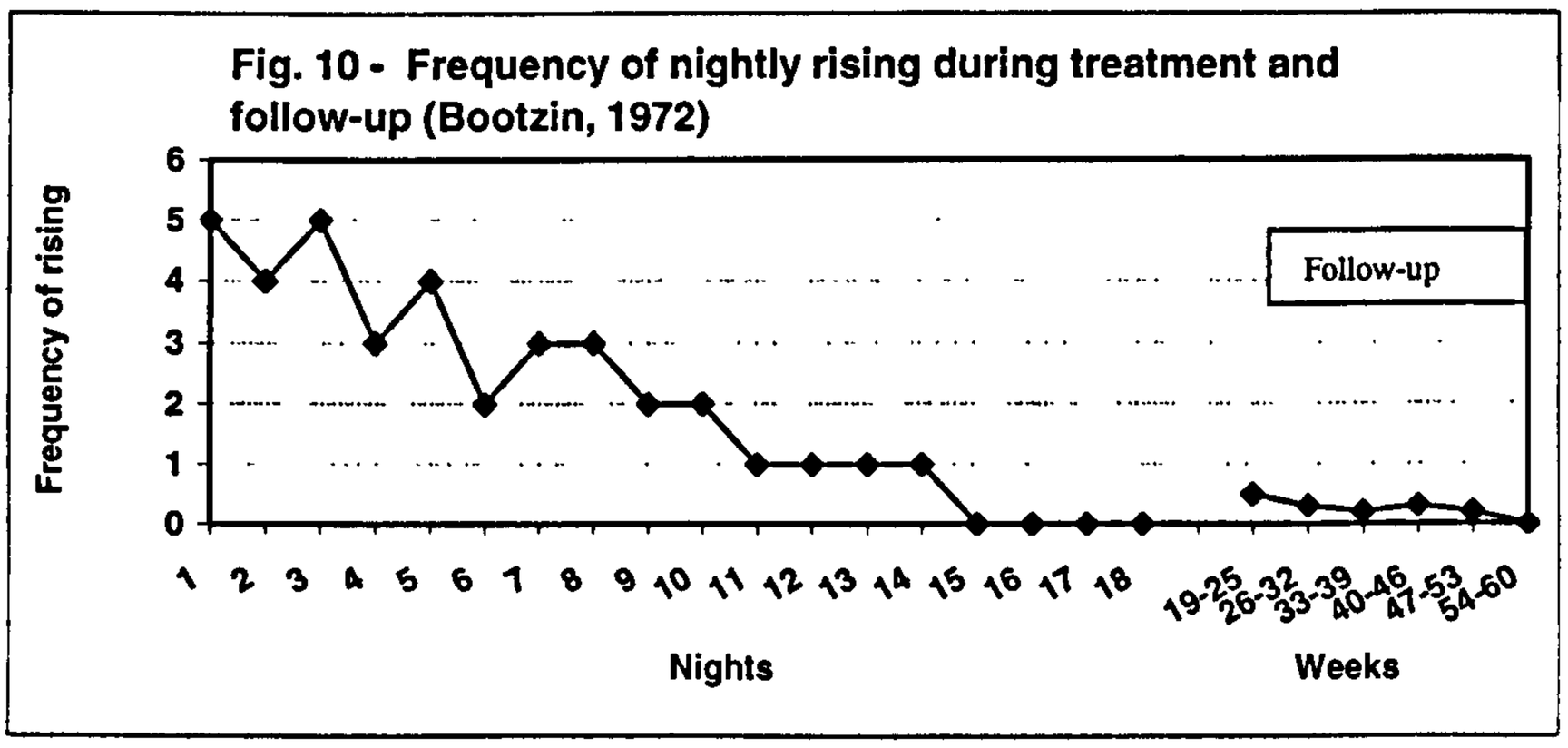


A few minutes/reasonable time: Bootzin, 1972; Toler, 1978; Norton et al, 1979; Haynes et al, 1975
10 minutes: Bootzin et al, 1991; Bootzin et al, 2000; Lacks, et al, 1983a; Lacks et al, 1983b; Puder et al, 1983; Turner et al, 1979a,b; Zwart et al, 1979;
15-20 minutes: Bootzin et al, 2000; Morin et al, 1988; Morin et al, 1990; Morin et al, 1994; Morin, Colecchi et al, 1999; Morin, Hauri et al, 1999.
20 minutes: Morin et al, 1995; Sanavio et al, 1990; Espie et al, 1989; Espie et al, 1999
20-25 minutes: Jacob et al, 1993
20-30 minutes: Hoelscher et al, 1988

Ultimately the aim of therapy is to move the patient to normalcy: it is, therefore, important to explore the length of normal sleepers' SOL so as to determine the optimum timeframe for this stimulus control instruction. The average PSG measured SOL for normal young adults is around 10 minutes (see table 1 p. 4), but it seems important to examine SOL also in terms of sensitivity and specificity so as to establish what duration best represents SOL in normal sleep. This issue is addressed in the first study of the present research (chapter 3).

1.7.2 Stimulus Control Therapy: Outcome Studies

Bootzin (1972) first used stimulus control in a single case study and within three weeks of stimulus control treatment the number of times the patient had to get out of his bed at sleep onset were dramatically reduced. Importantly, as depicted in figure 10, improvement was maintained at six weeks follow up and confirmed by the individual's wife. The improvement was very encouraging and warranted more systematic and controlled research.



Bootzin (1975) (as reported in Bootzin and Nicassio's review, 1978) allocated a sample of 66 participants, with severe and persistent insomnia, to: stimulus control, relaxation, self-relaxation placebo or waiting list control groups. Participants self-reported severe insomnia although the length of insomnia was not stated. Stimulus control reduced SOL by 67 minutes, while relaxation reduced it by 30 minutes. In addition 57% of those receiving stimulus control averaged less than 25 minutes to fall asleep as compared to 29% of those in the relaxation group and 22% of those in the two untreated groups.

Following these first two insomnia treatment studies employing stimulus control many investigations have shown that stimulus control is effective in reducing SOL and WASO and increasing TST .

For example, Haynes et al's (1975) found stimulus control therapy to be associated with shorter SOL (average reduction of 40 minutes) in all four single cases by the end of treatment. There were also improvements for all four participants in terms of TST and feeling rested after sleep, and three of them reported fewer wakenings. In addition, in three out of four cases, improvements were maintained at a nine-month follow up (telephone contact). In view of previous findings that poor and normal sleepers engage in similar sleep-incompatible behaviours in the pre-bed period (Haynes, Follingstad and McGowan, 1974), they proposed that stimulus control increases sleep continuity by conditioning low levels of physiological arousal to bedroom stimuli. Reading, talking working in bed may increase arousal and thus make sleep more difficult. It is important to note that their design ABAB (baseline, stimulus control, back to baseline, stimulus control) did not show a reversal effect after the first treatment phase; it could, however, be argued that rather than a return to baseline it represented a maintenance phase. Therefore the re-introduction of stimulus control treatment in the following phase was of little experimental value (Espie, 1991).

Lawrence and Tokarz (1976, as reported in Bootzin and Nicassio, 1978) recruited a student population with moderate SOL and assigned them to individual stimulus control, small group stimulus control, progressive relaxation training or desensitisation placebo. Both stimulus control groups reduced mean SOL to less than 20 minutes and improvement was considerably better than relaxation (75% vs 30%

improvement SOL improvement at post-treatment). Unfortunately no follow-up data is available.

Hughes and Hughes (1978) assigned 36 media recruited people with insomnia for at least 4 months to one of four groups: stimulus control, relaxation, EMG feedback or pseudo-feedback. Two weeks baseline and two weeks post-treatment sleep diary were collected. A drawback of this study is that treatment length varied considerably from eight weeks for EMG feedback, to four weeks for relaxation and to only two weeks for stimulus control. All groups improved and no differences were found in efficacy among treatments. Interestingly stimulus control produced SOL improvements very rapidly (two weeks). Similarly to other researchers they proposed that the equivalence in treatment outcome might be due to a cognitively mediated process common to all treatments. Follow-up data suggested that results were maintained but, unfortunately, given that data was available for only 33% of participants (12 out of 36), it was impossible to differentiate between treatments.

Turner and Asher's (1979b) treatment study employed a multiple baseline design with six severe insomniacs. After one-week baseline, three participants received eight weeks of stimulus control and the other three participants four weeks of quasi-desensitisation placebo treatment followed by four weeks of stimulus control. The placebo treatment had little impact on SOL, while stimulus control reduced SOL in both when treatment lasted eight and four weeks. Interestingly the authors reported that five out of the six participants achieved SOL shorter than thirty minutes within four weeks of stimulus control therapy. The sleep diary data were corroborated by reports of spouses or roommates. Turner and Asher also collected participants' suggestions regarding stimulus control efficacy. All six individuals suggested that

stimulus control worked by "breaking up lying in bed and thinking"; in other words it provided a way of controlling bedtime cognitions by simply getting out of bed. These comments suggest that stimulus control decreases anxiety regarding sleeplessness.

Turner and Ascher (1979a) devised a well controlled comparative study and showed that stimulus control, similarly to progressive muscle relaxation and paradoxical intention, improved the sleep of severe insomniacs while control conditions (desensitisation placebo and waiting-list) did not. Each group comprised ten media recruited participants who received four weekly therapy sessions following a baseline week. All three active groups reduced SOL (from more than an hour to less than thirty minutes) and number of awakenings after four weeks of therapy. Stimulus control was not found to be superior to relaxation or paradoxical intention but it produced the greatest absolute SOL reduction. Three shortcomings to this study should be noted. First there was no follow-up and it could be that gains might have been kept differentially across treatments. Second, there was no indication of what point in treatment therapeutic effects started to emerge. Third uncontrolled use of sleep medication was allowed: both increment and reduction of sleep medication might have contributed to the post-treatment results.

Lacks, Bertelson, Gans and Kunkel (1983a) replicated Turner and Asher's (1979a) study and they also investigated the interaction between insomnia severity and treatment. Sixty-four media and GPs recruited participants divided, upon completion of one week sleep diary, into three groups: mild (SOL 15-44 minutes), moderate (SOL 45-75 minutes) and severe (SOL 76-152 minutes) insomnia and randomly allocated within groups to each of the treatments. Participants received

therapy for four weekly sessions in small groups. Differently from Turner and Ascher's (1979a) study, SOL measures showed that stimulus control outcomes were considerably superior not only to no-treatment and placebo, but also to paradoxical intention and relaxation at each assessment point after week one. They also found a modest positive correlation between insomnia severity and degree of improvement and they concluded that stimulus control is the most effective treatment choice regardless of insomnia severity.

Lacks, Bertelson, Sugerman and Kunkel (1983b) compared the effect of stimulus control with quasi desensitisation placebo on WASO. Fifteen participants, all presenting with sleep maintenance insomnia (average 75 minutes), were recruited via media advertisements and letters to GPs. After one week baseline they were given treatment in small groups during (four weekly sessions of 60-90 minutes). WASO was reduced from 60 to 25 minutes in the stimulus control group and from 88 to 51 minutes in the placebo group (note that both groups reduced WASO by thirty-five minutes). The two treatments did not differ from one another and both treatments maintained these gains at three months follow-up. Two important points can be made from this study: first that stimulus control is efficacious in reducing WASO as well as SOL and number of awakenings. Second the quasi-desensitisation placebo treatment may actually have an active component: specifically the mental rehearsal of pre-bedtime routine and imagery is likely to preclude intrusive cognitions and in this way facilitating returning to sleep.

The studies reviewed so far assessed stimulus control efficacy in students and adults. Anderson and colleagues tested stimulus control efficacy in improving sleep

in older adults. Stimulus control improved WASO and S.E. but not SOL in their group of older adults (Anderson, Zendell, Rosa, Rubinstein, Herrera et al, 1988).

Sanavio et al (1990) allocated 40 insomniacs to one of four experimental (cognitive, biofeedback, stimulus control coupled to relaxation and no treatment) groups. Each group received six intervention sessions. The three active groups all reduced both SOL and WASO as compared to no improvements in the control group. However, only cognitive therapy resulted in an increase in TST. These authors provided three years follow up data (23 participants out of 30 provided data) showing that further reductions in both SOL and WASO were achieved for all three active treatments. In particular WASO was reduced from roughly an hour at baseline to about 15 minutes three years after the six weeks treatment.

Puder, Lacks, Bertelson and Storandt (1983) found that stimulus control was efficacious in improving SOL in older adults when a clinical psychologist administered therapy in small groups in four weekly sessions. A cohort of sixteen older adults was assigned to either immediate or delayed (following ten weeks baseline) stimulus control. Both groups, once stimulus control was administered, reduced their sleep latency by around fifty per cent and such reduction was maintained at two months follow-up.

Stimulus control efficacy in reducing sleep difficulties in 'older' people (average age 57) was also investigated by Morin and Azrin (1987). Twenty-seven media recruited people meeting criteria for insomnia were randomly allocated to stimulus control, imagery training and no treatment control. Following one-week baseline participants attended four weekly one-hour therapy sessions conducted in small groups. The stimulus control group achieved a statistically significant reduction of

WASO (by 65%) as compared to the other two groups. At 12-months follow up, however, stimulus control and imagery displayed almost identical WASO (around 25 minutes). They argued that psychological therapy for insomnia is indicated to decrease WASO in older adults too.

Morin and Azrin (1988) conducted another study, similar to that of the previous year with regard to number of participant and treatments, collecting data both via sleep diary and a switch-activated clock to record, objectively, WASO and SOL. Both active treatments, but not the waiting-list control group, reduced WASO and stimulus control produced an increase in TST of 65 minutes on average. Therapeutic gains were maintained at 12 months follow-up. SOL data were also reported and showed improvement both at the end of treatment and at follow-up for stimulus control (56, 40 and 23 minutes respectively), at a lesser extent for imagery training (38, 26, 25) but, interestingly, also for the waiting list control (52, 31).

Espie, Lindsay et al (1989) recruited eighty-four GPs referred outpatient adults, seventy participants were allocated to either stimulus control, relaxation, paradoxical intention, desensitisation placebo or no treatment. The remaining fourteen were allocated to a tailored intervention. Sleep diaries, incorporating both measures of sleep quantity and quality, were completed for two weeks baseline and eight weeks intervention. It is noteworthy that the stimulus control group almost halved SOL during the first week of treatment and by the eighth week SOL was 31 minutes (pre to post change of 62%). In line with Lacks et al's (1983a) findings stimulus control produced significant improvements in short time. By the end of the eighth week paradoxical intention reduced SOL to 36 minutes (51% pre to post change). In contrast, relaxation effects were similar to placebo. With regard to sleep quality,

while relaxation produced improvements both in terms of perceived restedness upon wakening in the morning and sleep enjoyment, stimulus control produced no measurable impact upon these variables. To summarize, stimulus control produced quick and effective improvements in SOL, and the treatment achieved clinical significance: indeed every patient improved, two-thirds reduced SOL by greater than half and a higher percentage of participants had sleep onset latencies of 30 minutes or less at follow-up. As pointed out by the authors the instruction to go to bed only when sleepy/tired might have guaranteed, per se, shorter SOL.

It is noteworthy that the efficacy of stimulus control in improving sleep has been typically tested by employing only subjective measures (sleep diary). To the author's knowledge only two studies looked at subjective and objective sleep improvements following stimulus control. In Anderson et al's (1988) study stimulus control improved subjective estimates of WASO and S.E but S.E. was the only PSG sleep parameter improved. Similarly, Engle-Friedman et al (1992) found that stimulus control improved subjectively measured sleep parameters and was the most effective condition in improving sleep when compared to relaxation and control. However such subjective changes were not corroborated by polysomnographic data. It should be noted that lack of PSG changes following CBT-I treatments are not uncommon (Krystal, Edinger, Wohlgemuth, and Michaels, 2002).

The studies reviewed so far highlight that stimulus control is efficacious in reducing estimates of SOL, WASO and NoA per night and, in general, it has yielded greater therapeutic effects when compared to other treatments. Furthermore therapeutic effects have generally been achieved very rapidly (Espie, Lindsay et al, 1989; Hughes and Hughes, 1978). In addition it has been found to be efficacious

with both young and older populations and to maintain therapeutic effects at follow-ups.

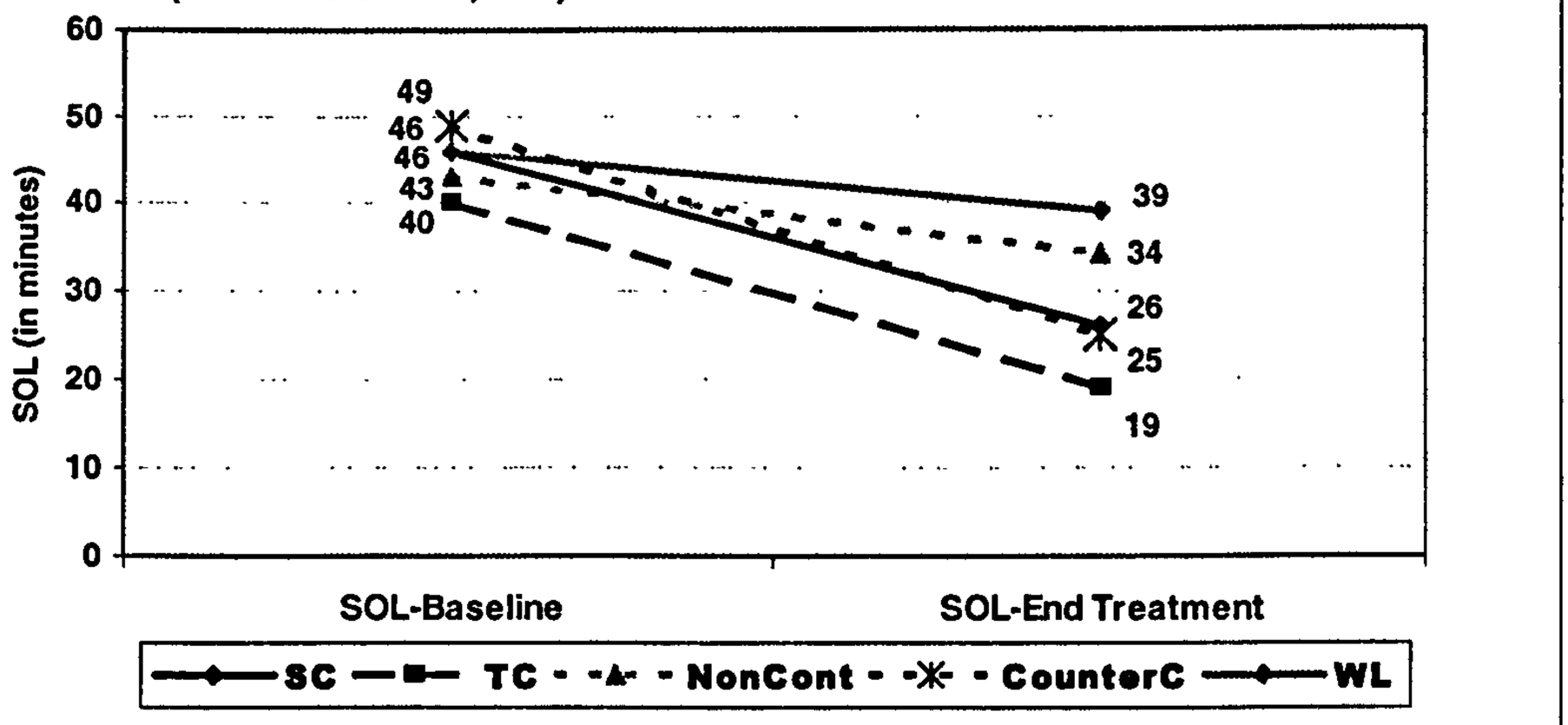
The mechanisms of effects of stimulus control are, however, less clear and participants' comments on the possible ways in which this therapy works are suggestive of 'breaking a pattern of lying in bed thinking', 'being able of stop thinking by getting out of bed'. In addition the findings of four studies cast doubts on the stimulus paradigm by either using only some instructions or by using a paradigm that seems to run counter to the whole conditioning aspect.

Tokarz and Lawrence (1974) (as reviewed in Bootzin and Nicassio, 1978) separated temporal components aimed at regularising the sleeping pattern according to the constraints of time (sleep scheduling) and stimulus components aimed at making the bedroom environment function as a discriminative stimulus for falling asleep (e.g. not watching TV in bed) of stimulus control. Results showed these two factors to yield equivalent efficacy in reducing SOL (from 50 minutes at baseline to around 10 at post-treatment) in their student sample when therapy was delivered in small groups once a week over four weeks. This is an important finding as it suggests that only half of the stimulus control instructions (either temporal or situational instructions) are sufficient to produce remarkable improvements. It is, therefore, surprising, that only a couple of studies have been carried out to replicate and explain these findings especially given that stimulus control is the core of CBT, the most delivered insomnia therapy in the last 25 years.

Another important study with regard to the mechanisms of effect of stimulus control was carried out by Zwart and Lisman in 1979. They assigned 47 undergraduate students, all reporting SOL greater than 30 minutes, to five different

conditions: the 'whole stimulus control' package, 'only temporal control' instructions, 'non-contingent control' (that is getting out of bed a fixed number of times at fixed intervals), 'countercontrol' (that is sit up in bed and do something if unable to sleep within 10 minutes) and 'waiting list'. Each group, other than the waiting list one, met 4 times on a weekly basis for 30 minutes. Figure 11 shows SOL at baseline and at the end of treatment (4 weeks) for the five conditions: interestingly the whole stimulus control package, temporal control instructions and countercontrol instructions form three parallel lines suggesting equivalence of improvement (confirmed by statistical analyses).

Fig. 11 - Mean SOL for Groups at Baseline and End of Treatment (Zwart and Lisman, 1979)



SC= whole stimulus control, TC= temporal control, NonCont=non-contingent control, CounterC=counter control, WL=waiting list

In line with Tokarz and Laurence's (1974) findings, 'temporal control' instructions were effective and sufficient to decrease insomnia symptoms. This finding made the authors suggest that stimulus control may function both for reconditioning of responses to the environment but also via improved harmonisation of the circadian cycle. Of particular importance was the finding that 'counter control instructions' were as effective as the stimulus control package. This finding shed

doubts on an environmental conditioning paradigm. Zwart and Lisman suggested that counter control ensured disruption of bedtime as cues for mental arousal. In other words 'doing something' acts as a distractor: the person who cannot sleep is occupied and does not focus on lying there not sleeping.

Alperson and Biglan (1979) investigated the efficacy of self-administered (in the form of manual with minimal therapist contact) treatment of insomnia. After two weeks baseline, twenty two media-recruited insomniacs aged less than fifty-five years old, were assigned to one of three groups: relaxation and stimulus control, relaxation and 'counter-control' or self-monitored. A further group, comprising seven insomniacs aged over fifty-five received relaxation plus stimulus control. It was found that both the stimulus control and the counter-control groups decreased sleep latency significantly better than the self-monitor group, and, similarly to Zwart and Lisman's (1979) findings there were no differences between the stimulus control and the counter control groups. However it is not possible to rule out the possibility that the procedure most impacting on SOL reduction was relaxation (which was taught in both conditions). It is worth noticing that the older group responded significantly less well than the younger one. This latter finding could be due to stimulus control being less efficacious with older adults. However, given that other studies have shown stimulus control to improve sleep in this population (e.g. Morin and Azrin, 1987, 1988; Puder et al, 1983), this explanation seems unlikely. Different outcomes between the studies could be due to delivery modality: self-administered therapy might work less well than therapist led administration with older participants. It could also be that older participants found it difficult getting out of

bed (e.g. mobility issues, cold) and gave up adhering to some components of stimulus control.

Indeed, when counter-stimulus control was administered to older adults, a statistically significant reduction in WASO was obtained (Davies, Lacks, Storandt and Bertelson, 1986).

In sum these four studies, in line with the stimulus control studies discussed previously, evidenced the efficacy of stimulus control. In addition, and very importantly, Tokarz and Lawrence's (1974) and Zwart and Lisman's (1979) studies also highlighted that stimulus control may function via at least two separate mechanisms: a stimulus control paradigm (situational components of stimulus control) and an 'improved harmonisation of the individual's circadian cycle' (temporal instructions) rather than solely via reconditioning of responses to the environment (Espie, 1991, p. 55). Furthermore, the findings that counter-stimulus control instructions improved sleep call into question the conceptual basis of stimulus control. Should the mechanism of effects be the strengthening of an association between bedroom environment and sleep, then asking people to stay in bed awake (and perform activities in bed) should weaken even further such association. Taking this point into consideration it could be argued that counter-stimulus control produced sleep improvements because of its temporal instructions.

1.7.3 'Get up and go to another room if not asleep': a feasible therapy?

The stimulus control studies discussed in the previous section evidenced the efficacy of stimulus control therapy for insomnia: importantly, stimulus control has been consistently found to be as efficacious as other therapies or to produce stronger

effects. Just as importantly, such effects emerged after only one or two weeks of therapy. However, participants' comments suggested that positive outcomes might not be due only to bringing back falling asleep under the stimulus control of the bedroom. Three studies (Alperson and Biglan, 1979; Davies et al, 1986; Zwart and Lisman, 1979) showed that both the original stimulus control and counter-stimulus control improved sleep. These findings would suggest caution about considering conditioning as the only mechanism underpinning stimulus control efficacy.

In section 1.7.1 it has also been argued that stimulus control therapy for insomnia comprises two different sets of instructions: one set aiming at re-establishing temporal harmonisation of the sleep-wake schedule and the other one aiming at bringing falling asleep under the stimulus control of the bedroom (situational elements). Indeed, two studies evidenced that therapeutic effects can be obtained when only situational or only temporal instructions are delivered (Tokarz and Lawrence, 1974; Zwart and Lisman, 1979).

The results of these four studies, coupled to the findings that both poor and normal sleepers endorsed similar sleep incompatible behaviours at bedtime (e.g. Harvey, 2000a; Haynes et al, 1982), highlight the importance of carrying out further research on the situational aspect of stimulus control. As already mentioned, it is surprising that this line of investigation has not been pursued and it seems timely to address this.

Given that situational elements of stimulus control have been shown to produce sleep improvements and that the instruction 'If you find yourself unable to fall asleep, get up and go to another room' seemed to epitomise the essence of stimulus control theory (section 1.7.1), it seems reasonable to propose a simplified stimulus

control therapy comprising only this instruction and to test its efficacy in improving sleep.

Before concluding this chapter it is important to highlight moderating variables, that is variables that can influence treatment outcomes.

1.8 Treatment Response and Moderating Variables

Meta-analyses have evidenced that behavioural therapies for insomnia improve sleep in around 70-80% of treated individuals - that is around 20-30% of people undergo insomnia treatment without benefiting from it. This finding raises an important question: what factors inhibit or moderate negatively sleep improvements in some of the treated individuals?

Chambers (1992) suggested that non-adherence might be the single greatest drawback to the success of behavioural treatment for insomnia, and attention is now turned to this moderating variable. Thereafter therapy credibility and expectancy of improvement are discussed. Finally the importance of the individual's readiness to change is highlighted.

1.8.1 Treatment Adherence

In the provision of health, the term adherence refers to the implementation of medical and treatment advice (Spilker, 1991) and it has been highlighted that non-adherence with treatment instructions can mask treatment effects (Frangakis and Baker, 2001).

Sleep researchers are becoming increasingly aware that adherence is an extremely important construct in therapeutic interventions (Bouchard, Bastien and Morin,

2003; Buysse, 2003; Harvey et al, 2002; Morgan Thompson et al, 2003; Riedel and Lichstein, 2001; Vincent and Hameed, 2003), yet very little systematic research on its impact on therapeutic gains have been carried out and no standardized methodology for measuring adherence exists.

The few times adherence has been measured, measurement has relied on subjective (mainly examination of sleep diaries) or important others' reports (Morin, Colecchi et al 1999; Bouchard et al, 2003). For example, Bouchard and colleagues (2003) used sleep diaries to measure adherence during 8 weeks CBT treatment. Seven daily criteria reflecting stimulus control and sleep restriction were extracted from the sleep diaries and rated. It was found that participants complied with at least 6 criteria each day. Harvey et al (2002) asked patients (both responders and non-responders to treatment) to rate their use of the behavioural treatment components of CBT-I 1 year after treatment was completed. Although the component sleep scheduling (a combination of stimulus control and sleep restriction) was used by less than 50% of patients at one year follow-up, it was found to be the strongest and most consistent predictor of SOL and WASO reduction.

The possibility that stimulus control could present a challenge in terms of adherence has also been evidenced by Schramm and colleagues. Their insomnia sample was asked, at the end of therapy, about their use of CBT modules and responses showed that stimulus control was rarely practised (Schramm et al, 1995). Similarly, Chambers (1992) found that only about 30%, of a sample of 103 insomnia patients, strictly followed treatment recommendations. In contrast, Riedel and Lichstein (2001) found, in their sample of older adults, that adherence to sleep restriction instructions was 'reasonably good'; interestingly, consistency in sleep

schedule predicted sleep improvements at post-treatment while degree of bedtime reduction did not.

These studies investigated adherence to CBT and, therefore, do not permit assessment of whether participants applied all components of stimulus control. In this regard it has been suggested that the instruction 'get out of bed if unable to sleep', particularly when elderly people are involved, can be difficult to comply with (Bootzin and Epstein, 2000). Indeed, Lichstein and Morin (2000) argued that the component of stimulus control 'get out of your bed if not asleep in 20 minutes' is particularly difficult to be followed by older adults because of mobility issues and the reluctance to leave the bed during winter months: non adherence with this instruction might help explain findings that stimulus control for insomnia is less efficacious with older adults.

As already mentioned these studies relied either on adherence being inferred by sleep diary entries or on retrospective (end of studies and follow-ups) estimates of adherence. This can be misleading as reports of adherence can be affected by the researcher's expectation or the participant's desire to conform or please the therapist raising the possibility that conclusions on the effects and active therapeutic ingredients of an intervention could be compromised (Buysse, 2003).

One instrument, namely actigraphy, already used in sleep studies, could lend itself to monitoring adherence objectively. Carney, Lajos and Waters (2003) proposed that using actigraphy to verify adherence to treatment instructions could be very important. In their elegant study they found that participants who were told that actiwatches would provide information on application of sleep instructions (bed and wake time) adhered more than those who were not informed that the 'watch' would

verify their adherence. A pitfall of this study is that sleep schedule was to be followed only for 2 nights; it is therefore possible that the undergraduates complied with instructions only because of the short time they were required to do so. In addition the group was composed of normal sleepers. It might be that poor sleepers would find it more difficult to follow instructions. Nonetheless, this study highlighted an objective method to evaluate adherence which is worth pursuing. In study two and three of the present research the possibility to use actigraphy to measure adherence objectively is explored.

It is important to pay attention to two other factors, namely credibility of treatment and expectancy of improvement following treatment, because they can impact on treatment outcomes (Kazdin, 1979). For example, if the individual does not believe that therapy will help improve sleep, they might be less likely to adhere to the treatment modality.

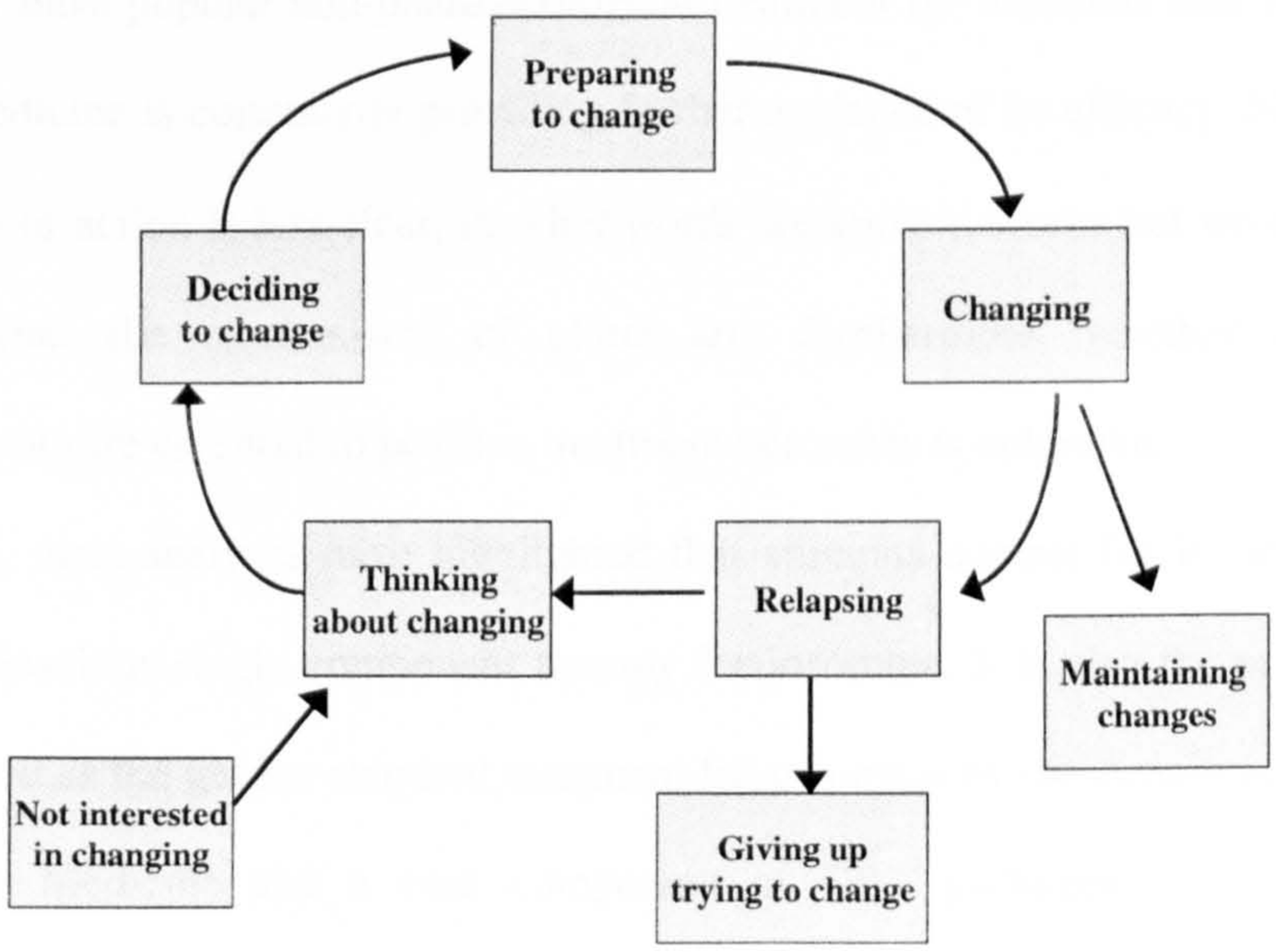
1.8.2 Credibility and Expectancy

Assessing credibility of treatment and expectancy of positive treatment outcomes enables the investigators to check that these two variables are not responsible for any differences in observed outcome between the compared conditions. Expectancy and credibility have been found to be influenced by the amount of information given and the kind of language used (Kazdin and Krouse, 1983), and to correlate with therapy outcome (Chambless, Tran and Glass, 1997; Borkovec and Costello, 1993). Riedel and Lichstein (2001) found that some of their patients did not adhere to spending less time in bed (sleep restriction) because this seemed counterintuitive to what one ought to do when having insomnia.

1.8.3 Other Moderating Variables

It can be argued that independently of the efficacy of a given treatment, the individual must be ready (that is motivated and willing) to learn and apply therapy instructions (Edinger and Means, 2005). In other words, if the individual is not "ready to change", even the most efficacious treatments will not produce positive therapeutic outcomes. As suggested by Morin and Espie (2003) the 'stages of change' model put forward by Prochaska, DiClemente and Norcross (1992) is a useful tool to assess readiness and motivation to change (figure 12). It is noteworthy that Morgan et al's (2003) study highlighted that higher expectations of change predicted better treatment outcomes.

Fig. 12 – Assessment of Readiness to Implement Insomnia Therapy using the Process of Change Model



For example, secondary gains (e.g. sympathy, relief from demanding tasks) might influence readiness to change (Kales and Kales, 1984).

1.9 Summary of the Introduction

The preceding sections critically reviewed the literature pertaining to insomnia, its non-pharmacological treatment and variables that can hinder treatment efficacy. Several distinct findings can be extracted from the literature.

First, although there is empirical evidence supporting the physiological, behavioural and cognitive perspectives of insomnia none seems to be exhaustive. Single factor theories are attractive for their simplicity but recent research suggests that insomnia is multi-dimensional in that physiological, psychological, behavioural and social domains interacts in the emergence, maintenance and exacerbation of insomnia.

Second, in the last 20 years CBT-I, which comprises several single therapies, has been the most popular non-pharmacological treatment for insomnia and behavioural sleep medicine is continually providing further evidence of its efficacy. Nonetheless its mode of action is less clear, in other words we know it works but we don't quite know what the mechanisms of effect are. Furthermore, whether all CBT-I components are essential to positive treatment outcomes is unknown.

Third, meta-analyses have highlighted that stimulus control for insomnia is the most efficacious single component therapy for insomnia. It is also the only therapy recognised as the golden standard treatment for insomnia by the American Academy of Sleep Medicine and a core component of CBT packages. Nonetheless, its mechanisms of effect are unclear. Despite stimulus control being based on the notion that in insomnia falling asleep is not under the stimulus control of the bedroom (because sleep incompatible behaviours are in the bedroom and/or sleep inducing stimuli are not established), research findings raised doubts about this hypothesis.

Two studies on pre-sleep activities failed to find differences between normal and poor sleepers, hence suggesting that behaviour incompatible with sleep might not play, per se, an essential role. Furthermore, a counter-stimulus control paradigm, requiring participants to spend time awake sitting up in bed, was found to improve sleep. This finding argues against situational conditioning. In addition, participants' comments suggested that stimulus control breaks a pattern of lying awake in bed thinking. Finally, a detailed examination of the elements of stimulus control highlighted that it comprises both situational (aiming at re-establishing good stimulus control) and temporal instructions (aiming at re-harmonising sleep and wakefulness).

Fourth, although research has consistently found stimulus control to improve sleep, most studies relied only on subjective estimates of sleep. Indeed, the two studies employing objective measurements (PSG) of sleep failed to find changes in the sleep parameters under investigation.

Fifth, it has been shown that, although insomnia treatments produce sleep improvements in around 70-80% of the population, not everyone responds to treatment. The possibility that this is in part due to non-adherence has been raised but systematic research on adherence and its impact on treatment outcomes is lacking. Furthermore, in the few instances in which adherence has been investigated, its measurement was provided by retrospective and subjective tools (e.g. sleep diary, participants' comments). This finding highlighted the importance of measuring adherence systematically and objectively.

The next chapter details the present research, which comprises three studies designed to investigate the issues just highlighted above.

Chapter 2

Overview of the Present Research

2.1 Rationale

The literature reviewed in the previous chapter highlighted that CBT is an efficacious treatment for insomnia comprising several discrete therapies each of which, in turn, encompasses several elements. The fact that it is multicomponent makes it difficult to assess if all its components are critical to therapeutic gains and to identify mechanisms of treatment effect. The assessment of what elements are critical to positive outcomes is important because of the need to provide cost-effective care. Understanding the mechanisms of effect underpinning insomnia treatment is also important because it may help to shed light on factors that maintain and/or exacerbate insomnia. This, in turn, could lead to possible ways of preventing acute insomnia from becoming chronic, and so maximise the effectiveness of insomnia treatment packages.

However, most research on insomnia therapy has been 'macroscopic' in nature focussing on the efficacy and effectiveness of complex multicomponent packages, with a tendency, if anything, to add modules rather than striving to obtain maximum effect with minimum intervention. It seems important, therefore, to address this issue by looking not just at single therapies within CBT but also 'microscopically' at single elements of these therapies. A good analogy would be: having built a fine house it is as if we have neglected to pay sufficient attention to what its foundations and cornerstones are. The present research was devised to look into this matter.

Having evaluated the literature (chapter one), stimulus control for insomnia emerged as a core component of CBT, the most efficacious single therapy and the only therapy recognised as the gold standard treatment for insomnia by the American Academy of Sleep Medicine. Notwithstanding its efficacy, previous research findings cast doubts on whether all its elements are critical to positive outcomes and on the mechanisms of effect of stimulus control. For this reason stimulus control was examined in more detail. Analysis of its six component parts suggested that the instruction 'if you are not asleep within a reasonable time get up and go to another room and return to bed only when sleepy again' captures the essence of learning theory posited to underlie stimulus control therapy and, in this sense, may be a cornerstone element worth of close examination.

Interestingly, the duration of waking time before applying this instruction was found to be somewhat arbitrary in that it varied from study to study. If this instruction is to return the individual into the normal range then what constitutes normalcy with regard to SOL needs to be clear and study one was devised to address this issue.

Furthermore, findings that poor and normal sleepers endorse similar sleep incompatible behaviours at bedtime questioned conditioning as the sole mechanism underpinning stimulus control. This possibility was strengthened by findings that a counter-stimulus control paradigm produced positive outcomes.

The literature review also highlighted that the impact of stimulus control on PSG defined sleep seeming largely unknown. It should be appreciated that it is only in the past 10-15 years that PSG measures have started to be employed in efficacy studies and that results are equivocal: some studies have evidenced PSG defined sleep

improvements while other studies have not (for a review see Stepanski, 2000). The majority of outcome studies employing PSG are CBT trials perhaps because most single therapy studies were carried out in the 1970s and early 1980s when, due to technology constraints, PSG recording was more cumbersome than nowadays. One stimulus control study employing PSG measures found only S.E. improvements (Anderson et al, 1988). It seems, therefore, important to attempt to address the need for objective data on sleep changes following therapy. In particular, given that stimulus control for insomnia attempts to condition falling asleep to the bedroom environment, home PSG seems a more logical choice than PSG recorded in laboratory settings.

In addition, examination of previous research revealed that not everyone responds to insomnia treatment. Several researchers have argued that poor treatment response is likely to be related to adherence, yet adherence has been rarely investigated. In addition the few studies that examined adherence have done so subjectively or have inferred it from sleep diary entries. It was reasoned that in the case of a single element therapy, whose implementation requires overt behaviour (i.e. get up and read), actigraphy could be employed to measure adherence objectively.

The present research, therefore, represents an investigation into the impact of a single element therapy, named the Quarter of an Hour Rule (QHR), on subjective and objective (PSG) sleep in people presenting with insomnia in primary care or self-referred as having insomnia. Additionally, whether conditioning to the bedroom environment is the sole mechanism of effect of stimulus control therapy for insomnia was explored. Finally, the relationship between treatment outcome and adherence

and, importantly, the feasibility of employing actigraphy to measure adherence objectively were examined.

2.2 Aims

The principal aim of the present research was to examine the impact of the QHR on sleep parameters of individuals with psychophysiological insomnia. Both subjective (sleep diaries) and objective measures (home PSGs) of sleep were gathered. The second aim was to investigate its mechanisms of effect. The third objective was to explore the relationship between therapy outcomes and adherence and the feasibility of employing actigraphy to measure adherence objectively.

This research comprises three studies. Two preliminary studies were completed to inform the randomised controlled experimental trial (study three). The first aim of study one (chapter three) was to determine what constitutes normalcy with regard to SOL so as to have a 'reasoned' duration of time to wait before applying the QHR. The results of study one informed the second and the third studies.

Prior to conducting a randomised controlled experimental study, it was deemed important to carry out a few single case studies to test if the QHR would impact on sleep. The first aim of study two (chapter four) was, therefore, to assess the effect of the QHR on subjective and objective sleep of three individuals presenting with insomnia. In particular, the validity of home portable EEG equipment, whose set up could be performed by the participants themselves, was tested. Another aim of study two was to investigate adherence to the QHR both subjectively via adherence diaries and objectively using actigraphy. The results of this explorative study informed on the feasibility of the QHR as a standalone therapy. Data gathered from study two also

enabled optimal calibration of study three methodology with regards to objective measurements of sleep and adherence.

The first aim of the randomised controlled experimental trial (study three, chapter five) was to investigate the impact of the QHR on subjective and objective sleep of GP referred and self-referred people with insomnia. The second aim was to explore possible mechanisms of effect of the QHR by requiring to apply the QHR in two different locations out of bed (QHRout) and in bed (QHRin). In addition, a battery of sleep related questionnaires measuring sleep related behaviours, cognitions and arousability were administered. As in study two, another aim of this research was to investigate the impact of adherence on treatment effects. Adherence with the QHR treatments was measured daily subjectively, via an adherence diary, and objectively by means of actigraphs incorporating a light detector.

Each of these studies are described and discussed in the following chapters.

2.3 Ethical Approval and Ethical Issues

Ethical approval for the present research and two amendments to the original approved protocol were granted by the Greater Glasgow NHS Primary Care Trust. Copies of the original and the amendments were forwarded to the University Ethics Committee as is standard practice (appendix 2).

It should be noted that based on prior work in this area (e.g. Bootzin, 1972; Zwart and Lisman, 1979; Davies at al, 1986) it was assumed that the QHR intervention would be either beneficial or would carry no adverse effects.

With regard to home PSG, the procedure was well tolerated and minimal discomfort was reported. Participants were given a mobile telephone contact number

so as to have access to the researcher during the PSG nights. Participants were encouraged to contact the researcher if they had questions or problems related to the PSG recording equipment or if emergencies requiring the removal of PSG equipment arose. Only one participant phoned, soon after the researcher left her home, as she thought she might have switched the EEG recording equipment off.

CHAPTER 3 – Study One

A Cross-Sectional Comparison of Sleep Behaviour in Normal and Poor Sleepers

This study was carried out so as to assess normal and poor sleepers' SOL with the aim of determining the duration of SOL in normal sleep. In addition, the portion of the sleep opportunity window that should be considered in calculating TIB was also explored with the aim of wording the sleep diary so as to best capture sleep continuity variables. The findings of this study informed methodology for study two and study three (chapters four and five respectively) with regard to component therapy (called the QHR).

3.1 Rationale

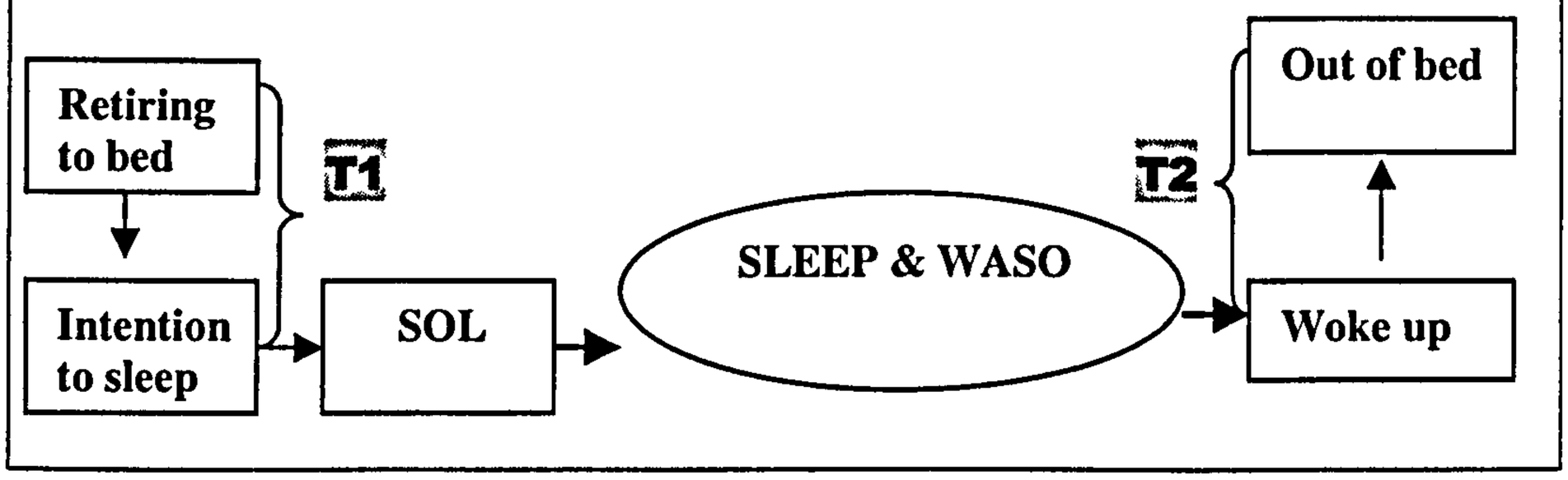
Research typically focuses on the 'abnormal' and on therapies to decrease 'disease'. Ideally, however, the ultimate goal of therapy is not just to achieve symptom control but also to return the individual to normalcy. It seems very important, therefore, also to study normal sleep behaviour to inform both theory and treatment.

In the present context, decisions concerning the amount of time to wait before applying the instruction 'get up and go to another room' (table 5, instruction 3 section 1.7.1) would be somewhat arbitrary without data on what latency constitutes SOL in normal sleep. Whereas to the author's knowledge what constitutes normal subjective SOL has never been formally assessed in its own right, such data can be inferred from the literature comparing normal and poor sleepers. For example, Means

et al (2003) reported normal sleepers' SOL of 12 minutes and Wohlgemuth, Edinger, Fins and Sullivan (1999) of 23 minutes.

Likewise, it is also important to standardise research and clinical practice by employing a 'reasoned' definition of sleep efficiency. Sleep diaries routinely used in research include sleep continuity variables (e.g. SOL, WASO), however, sleep behaviour at bedtime and in the morning is not always measured in a uniform manner. For example, it is possible to measure TIB in several ways: TIB can be defined as the time elapsed from 'intention to sleep' to 'woke up' (i.e. the narrowest sleep window opportunity), or it can be calculated as the time between 'retiring to bed' to 'out of bed' (i.e. the widest sleep opportunity). Providing TIB is measured consistently (i.e. it is measured identically across conditions and at the considered time points) this does not represent an issue within each single study. However when studies carried out by different researchers are compared, the way in which TIB is measured might be in part responsible for differences in treatment outcomes. Importantly, if the sequences of behaviour, at sleep time (T1) and in the morning upon awakening (T2) (fig. 13), differ between normal and poor sleepers then when calculating TIB T1 and/or T2 would acquire importance for theory (potentially helping explaining some aspects of insomnia) and, consequently, for therapy.

Fig. 13: Sequence of Behaviour at Sleep Time and in the Morning



3.2 Aims and Hypotheses

The first aim of this study was to investigate the duration of subjective SOL that is typical of normal sleep. In particular what duration would be the best cut-off to include normal sleepers and to exclude poor sleepers (sensitivity/specificity) was examined.

The second aim was to investigate sleep behaviour at bedtime and in the morning in normal and poor sleepers. Based on the stimulus control argument that in insomnia sleep incompatible behaviours are present, it was hypothesised that poor and normal sleepers would differ with regard to the periods of time spent awake in bed, both at bedtime pre-sleep and in the morning after wakening. The first prediction was that T1 would be greater for poor sleepers than normal sleepers. The second prediction was that T2 would be greater for poor sleepers than normal sleepers.

In addition, information regarding reasons for waking up in the morning and behaviour endorsed when sleep did not come easily was collected.

3.3 Methods

3.3.1 Design

This was a cross sectional comparison to examine sleep behaviour in normal and poor sleepers. The areas of interest regarded the sleep pattern at SOL, T1 and T2 (measured in minutes). There was one between subjects factor of 'Group' with two levels (normal sleeper and poor sleeper).

3.3.2 Participants

One hundred potential participants were approached. These were acquaintances and/ or colleagues of the researcher, with English as their first language, aged 20 to 60 years. Fourteen individuals decided not to participate and six participants did not return the forms. A total of eighty volunteers took part in the survey (45 females and 35 males). The majority was employed either full or part time (77%), while a small percentage was retired (9%) or in full time university education (14%). They all self-reported as free from psychological or neurological disorders. To analyse the data in relation to sleep problems, participants were divided into two groups according to their total score in the PSQI (Buysse et al, 1989). Demographic and PSQI characteristics are reported in table 6 (section 3.4.1).

3.3.3 Materials and Apparatus

3.3.3.1 Diary

Sleep parameters were gathered by means of sleep diaries (adapted from Morin and Espie's (2003) sleep diary), which participants completed each morning upon rising for one week (appendix 3). Via the sleep diary, daily information on 'sleep continuity' such as SOL, WASO, which indicated and / or confirmed the disorder (i.e. insomnia) were gathered. The sleep diary is routinely used to gather sleep-continuity measures in sleep research (Espie, 1991; Morin, 1993).

The sleep diary, however, can also be used to gather sleep behaviour information such as pre-bed routine. This investigation concentrated on sleep behaviour at night (when retiring to bed) and in the morning by explicitly differentiating the 'retiring to

bed' from 'intention to sleep' times and 'wake up' from 'out of bed' times (fig. 14 in section 3.1). Additionally, the diary included three questions so as to gather information about: i) endorsed behaviour when 'sleep does not come easily' at sleep onset, ii) endorsed behaviour when 'sleep does not come easily' during the night and iii) reasons for waking up in the morning.

Similar to Monk, Buysse, Rose, Hall and Kupfer's (2000) study, information regarding restedness and alertness on awakening was collected. Participants judged their quality of sleep, restedness and alertness in the morning on a rating scale (0= not at all, .., 4=very much).

3.3.3.2 Sleep Questionnaire

The Pittsburgh Sleep Quality Index (PSQI: Buysse et al, 1989) is a retrospective self-rated questionnaire comprising 19 items assessing the individual's sleeping habits and sleep quality during the past month (appendix 4). The PSQI produces seven component scores (each producing a score between 0 and 3), the sum of which provide a global index of sleep quality. A score above 5 on the global index (score ranging from 0-21) identifies significant sleep disturbance with high sensitivity and specificity (Buysse et al, 1989; Backhaus, Junghanns, Broocks, Riemann and Hohagen, 2002). Test-retest reliability for the PSQI global score, over a one-month period, was found to be 0.85 in the original study (Buysse et al, 1989) and Carpenter and Andrykowski (1998) reported good internal consistency in a mixed sample of medical patients with normal and poor sleep (Cronbach's $\alpha = 0.80$).

3.3.4 Data Processing

The primary sleep diary variables were 'Duration of SOL', T1 and T2 as previously defined. In order to validate the cross sectional investigation, however, the sleep parameters indicating a disorder of insomnia were first studied.

Data were analysed using a statistical package (SPSS for Windows version 11.5).

3.3.5 Procedure

Potential participants were informed that the study investigated whether people who sleep well act differently from those who do not sleep well both during typical nights and when sleep does not come so easily. People, who expressed an interest in participating, were told that they were required to fill in a sleep questionnaire (PSQI) and, thereafter, to complete sleep diary every morning for one week. Both the PSQI and the sleep diary were to be forwarded to the researcher in the provided envelope at the end of the week. As the diary and the PSQI did not have an ID number, they were stapled together.

The PSQI takes around 10 minutes to complete and the sleep diary around 5 minutes per day, thus requiring around 45 minutes of participants' time to complete the investigation.

3.4 Results

3.4.1 Participant Characteristics

Table 6 presents information regarding number of participants in each group, their gender, age, and the mean and standard deviation (SD) for PSQI total scores.

Table 6 – Summary Data for the normal sleeper and the poor sleeper Groups

	Normal sleeper	Poor sleeper	Total
Participants	50	30	80
Female/Male	29/21	16/14	45/35
Age band:			
(20-30 years)	22	10	32
(31-40 years)	13	9	22
(41-50 years)	8	6	14
(51-60 years)	7	5	12
PSQI total score (SD)	2.5 (1.0)	10.0 (1.9)	

As expected, given that the normal and poor sleeper groups were formed by splitting the participants into two groups depending on their PSQI score (Buysse et al, 1989), an independent t-test confirmed that the 2 groups differed significantly in terms of their quality of sleep level [$t(78)= -23.53$, $p=0.0001$]. There were no significant differences in age group [$\chi^2(3)=0.90$, $p=0.82$] or gender [$\chi^2(1)=0.16$, $p=0.68$].

3.4.2 Sleep Continuity and Sleep Quality Data

Sleep continuity variables are generally used to confirm sleep status initially.

As shown in table 7, diary sleep data demonstrated differences between normal and poor sleepers. In order to assess if these differences were statistically significant separate Mann-Whitney U tests were conducted on SOL, WASO and TST with group (normal sleeper vs. poor sleeper) as the between subjects factor. The choice of a non-parametric test was made because visual inspection of the data suggested that the scores were not normally distributed. This was confirmed by examination of kurtosis and skewness (values higher than 1.9).

Table 7 – Mean and SD for Sleep Continuity (in minutes) and Sleep Quality (0-4) for the Normal Sleeper and Poor Sleeper Groups

	Normal Sleeper (N=50)	Poor Sleeper (N=30)
SOL	9.38(7.69)	47.22(31)
WASO	8.72(8.87)	53.73(58)
TST	442(43)	342(85)
TIB ('Intention to sleep' to 'woken up')	460(43)	444(66)
T1 ('retiring to bed' to 'intention to sleep)	14(14)	17(14)
T2 ('waken up' to 'out of bed')	29(20)	57(42)
Quality of Sleep	3.04(0.48)	1.60(0.72)
Restedness	2.91(0.50)	1.58(0.63)
Alertness	2.91(0.59)	1.79(0.80)

Mann Whitney analyses revealed that there were highly significant differences with regard to SOL (U=101.000, N_1=50, N_2=30, P= 0.0001 two tailed), WASO (U= 219.50, N_1=50, N_2=30, P= 0.0001 two tailed) and TST (U=182.000, N_1=50, N_2=30, P= 0.0001 two tailed). Specifically, participants in the poor sleeper group took longer to fall asleep, were awake for longer during the night and slept less than normal sleepers. These results, therefore, confirmed the initial differentiation of the two groups on the basis of their PSQI scores.

Each morning participants reported the quality of the previous night's sleep, and their level of alertness and restedness upon awakening. Examination of table 7 indicates that ratings of quality of sleep, restedness and alertness on final waking of normal sleepers differed from those of the poor sleeper group. The data met criteria for parametric tests and independent t-tests confirmed that normal sleepers reported better quality of sleep, felt more rested and alert upon awakening than poor sleepers [t(78)= 10.70, $p<0.001$; t(78)=10.30, $p<0.001$; t(78)=7.12, $p<0.001$ respectively].

Interestingly, on occasions when normal sleepers could not fall asleep easily they tended to 'just lie there waiting for sleep' (84% of times). Although poor sleepers

reported 'lying there' as their most endorsed behaviour when they could not sleep (66%), they often reported 'watching TV' and/or 'reading a book' and/or 'listening to the radio' and/or 'thinking about how to sleep'.

3.4.3 Upper Limit of Sleep Onset for Normal Sleepers

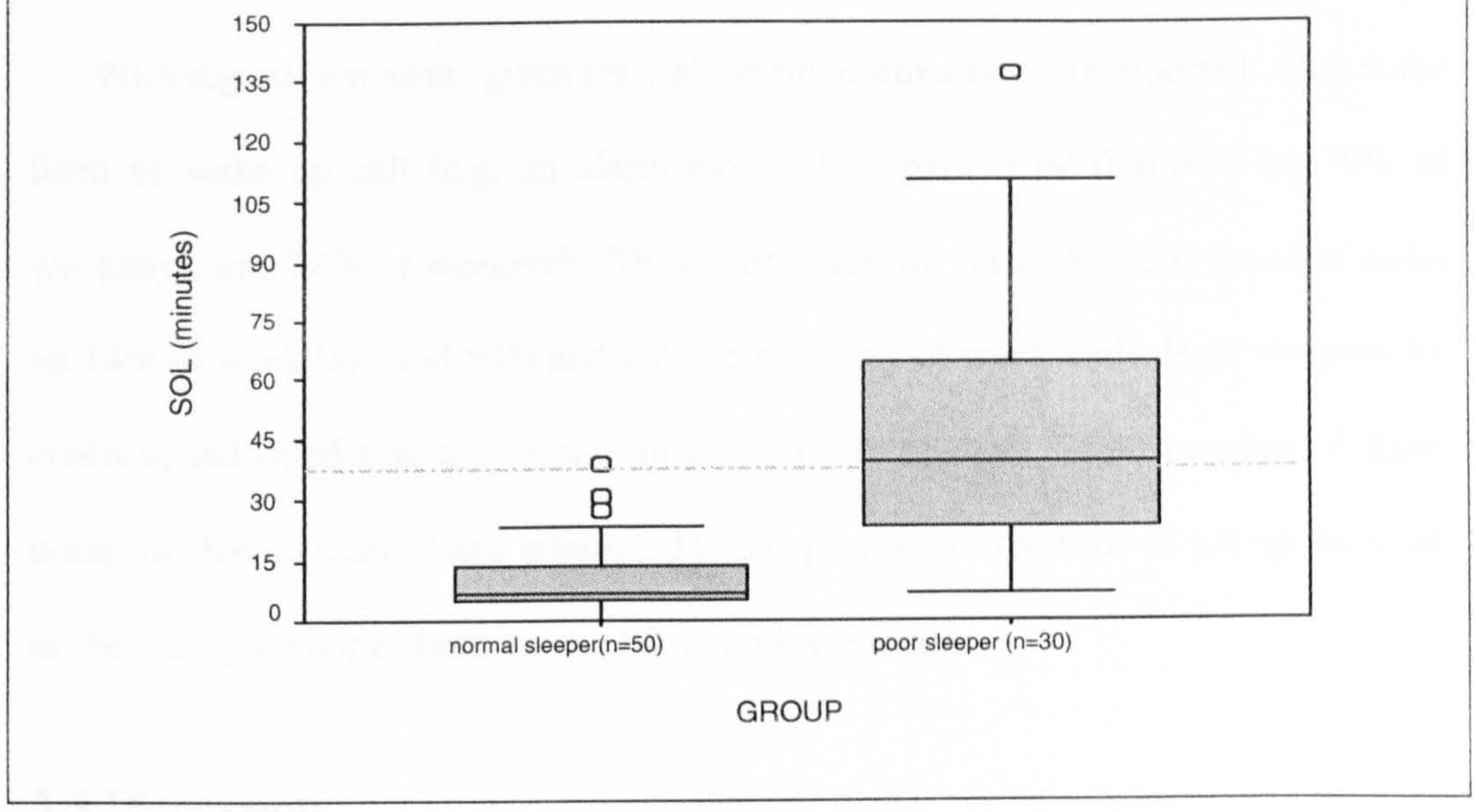
To establish the upper limit of normal sleep behaviour at sleep onset, data from the normal sleeper group were examined in terms of sensitivity and specificity. Sensitivity refers to the probability that a person having a given characteristic will be correctly identified as having such characteristic. Specificity is the probability that a person without that characteristic will be properly classified as not having it.

Analysis of sensitivity and specificity data indicated that SOL of 5 minutes correctly identified only 38% (19 individuals) of normal sleepers and correctly excluded 100% (30 individuals) of poor sleepers, SOL of 10 minutes correctly identified 72% (36 individuals) of normal sleepers and correctly excluded 90% (27 individuals) of poor sleepers, SOL of 15 minutes correctly identified 92% (46 individuals) of normal sleepers and correctly excluded 86.7% (26 individuals) of poor sleeper and SOL of 20 minutes correctly identified 92% (46 individuals) of normal sleepers and correctly excluded 76.7% (23 individuals) of poor sleepers.

As depicted in figure 14, these results indicate that a SOL of 15 minutes is the best cut-off to include normal sleepers and exclude poor sleepers.

In light of these findings, it seems plausible to consider a quarter of an hour as the upper limit of normal sleep behaviour at sleep onset and, to specify such time when instruction 3 of stimulus control is delivered (table 3, section 1.7.1).

Fig. 14: Box and Whiskers Plot indicating that a quarter of an hour is the best cut-off for normal sleep onset behaviour



3.4.4 Sleep Behaviour at Bedtime and in the Morning

Data for T1 and T2 were also not normally distributed hence non-parametric statistical analyses were carried out. Separate Mann-Whitney U tests were conducted on these variables. There was no significant difference between the two groups for T1 ($U=656.500$, $N_1=50$, $N_2=30$, $P=0.35$ n.s.) indicating that the amount of time spent in bed prior to switching off the light with the intention to sleep, did not differ between normal and poor sleepers.

In contrast, there was a significant difference between the two groups with regard to T2 ($U=443.500$, $N_1=50$, $N_2=30$, $P=0.002$ two tailed) suggesting that the amount of time spent lying in bed once awake in the morning was greater for poor sleepers. This finding highlighted the importance of considering this portion of sleep opportunity window when calculating TIB.

3.4.5 Reasons for Waking Up in the Morning

With regard to reasons given for waking up, normal sleepers reported using some form of wake up call (e.g. an alarm clock, their partner or flatmate) on 70% of weekdays, and 38% of weekends. They 'just woke up' on 16% and because of noise on 14% of weekdays and 50% and 12% respectively of week-ends. Poor sleepers, by contrast, indicated that they woke up on majority of days (76%) because of light, noise, or 'for no reason' and attempted to sleep again till the time to get up for work or their daily activities both on weekdays and weekends.

3.5 Discussion

In this section results obtained in the survey will be discussed with particular attention to the design of study two (exploratory single case study, chapter 4) and study three (experimental study, chapter 5).

3.5.1 Summary of the Results

The first aim of this cross sectional study was to assess the duration of SOL typical of normal sleep behaviour. Results indicated that 15 minutes was the best cut-off to discriminate normal SOL. The second aim was to investigate whether or not sleep behaviour at bedtime and in the morning differed between normal and poor sleepers. The first prediction, that the amount of time spent in bed before switching off the light with the intention to sleep would be greater for poor sleepers compared to normal sleepers, was not supported. The second prediction, that poor sleepers would spend a longer time in bed in the mornings than normal sleepers, was supported.

Finally, descriptive information regarding reasons for waking up in the morning and behaviour endorsed when sleep did not come easily was collected. This information suggests that normal sleepers during weekdays tended to use a form of 'wake up' call in order to get up in the morning while poor sleepers tended to wake up for no apparent reasons or because of noise. Additionally, in the few occasions when normal sleepers could not fall asleep easily they tended to lie in bed waiting for sleep to come while poor sleepers tended to endorse a variety of behaviour (e.g. lie in bed, watching TV, read, get a juice).

3.5.2 - Interpretation of the Results

Importantly for insomnia therapy instructions, this study highlighted that 15 minutes provides the best sensitivity-specificity cut-off to discriminate normal SOL from SOL of poor sleepers. For example, as already pointed out in the introduction, the amount of time to wait before applying the stimulus control instruction 'get out of your bed and go to another room' is rather arbitrary varying from a few minutes (Haynes et al, 1975) to 20-30 minutes (Hoelscher et al, 1988). The present finding suggests that 15 minutes may be the most appropriate amount of time to return the poor sleeper to normalcy.

The first prediction, that the amount of time spent in bed before switching off the light with the intention to sleep would be greater for poor sleepers as compared to normal sleepers, was not supported. However, on reflection, this finding may not be surprising, given that some previous research has shown that poor and normal sleepers endorse similar pre-sleep activities (Haynes et al, 1982). In poor sleepers, considering TIB from the intention to sleep could result in heightened arousal due to

the pairing of being in bed with being awake and frustrated/worried. Accordingly from a learning theory perspective, any time spent awake should be avoided so as to re-establish appropriate stimulus control.

The present results seem to suggest that both normal and poor sleepers do spend a similar amount of time in bed prior to attempting to sleep. However, it could be speculated that the reason for spending time awake in bed prior to attempting to sleep differed depending on the group. In particular it might be that normal sleepers decide to switch off the light so as to sleep when they feel sleepy, while poor sleepers might make such decision on the basis of external cues (e.g. clock, partner) rather than to internal cues for sleep. This possibility has been raised in a recent paper on the attention-intention-effort pathway to insomnia (Espie et al, 2006). Espie et al (2006) proposed that normal sleepers read in bed and attempt to stay awake (but they cannot), while poor sleepers read because they cannot fall asleep/cannot stay asleep. In other words, normal sleepers intend to read in bed but sleep takes over while poor sleepers unsuccessfully intend to fall asleep and because of wakefulness they start to read. Although this is a possibility, it was not tested in this study.

The second prediction, that time spent in bed from waking up to getting out of bed would be greater in poor sleepers than in normal sleepers, was supported. This result highlights the importance of considering the period between waking up and getting up when calculating TIB. Whereas normal sleepers once they woke up tended to get out of their bed, poor sleepers seemed to lie there, presumably, in the hope of getting some more sleep. This finding is potentially important with regard to stimulus control theory at WASO. The poor sleeper, by staying in bed when awake in the morning, might weaken the association between the bed and sleep.

The finding that 70% of normal sleepers woke up during week-days because of an alarm clock replicate Monk et al's (2000) findings. They suggested that this finding coupled to the fact that normal sleepers tended to sleep longer at weekends (when they could wake up at leisure), was an indication of sleep debt. It could be argued that normal sleepers maintain a healthy tension between sleep drive and sleep behaviour, that is having a modicum of sleep debt helping to maintain sleep homeostasis. Poor sleepers, in contrast, reported waking up because of environmental noises or for no apparent reasons. However, this finding cannot be taken as an indication that they do not have a sleep debt. Indeed, they felt less rested and less alert in the morning than normal sleepers and they tried to satisfy their need for more sleep by staying in bed and 'trying to fall back to sleep' once they woke up.

3.5.3 Considerations for Study Two

These findings inform the methodology for study two and three.

First, the results indicated that a window of 15 minutes gives ample opportunity to fall asleep and may be the optimal cut-off when normalcy in sleep onset is considered desirable. It seems reasonable, therefore, to ask people complaining of insomnia to apply the stimulus control instruction 'get out of your bed and go into another room if not asleep within a quarter of an hour'. Bearing in mind that the ultimate goal of therapy is reinstating the person with insomnia as a normal sleeper, by setting the limit of being in bed awake to quarter of an hour, the goal of sleep normalcy in the treatment programme is reinforced. It was decided to focus on a quarter of an hour rather than 15 minutes, so as to move away from the concept of 'being absolutely precise'.

Second, the study highlighted the importance of including for analysis T2 (i.e. period between wake and rise) so as to capture early wakening. Importantly, with regard to stimulus control therapy, considering this period is important in order to make sure that the stimulus control instruction is applied every time the person is in bed but not asleep. Therefore, the sleep diary employed in study two and three explicitly asked 'What time did you rise from bed this morning'.

Chapter Four: Study Two

Could a Single Element of the Stimulus Control Package Improve Sleep? An Exploratory Study

This chapter reports three single case studies, conducted to examine the feasibility of a single element therapy to improve sleep of people with insomnia, and data on adherence gathered subjectively and objectively.

4.1 Rationale

In the introduction it was reasoned that, despite CBT-I being consistently found to improve sleep of people with insomnia, its mechanisms of effect and whether all components are critical to positive outcomes are unclear. Furthermore, although stimulus control is central to diagnostic conceptualisation of psychophysiological insomnia (ICSD-R), a core component of CBT-I and, also, the best evidenced non-pharmacological intervention, whether re-conditioning is the mechanism responsible for sleep improvements is uncertain. In addition research findings highlighted that administering only some of the components of the stimulus control package produced outcomes equivalent to those obtained by the whole package.

These findings highlighted the importance of de-constructing stimulus control. Following a detailed analysis of its components, one element (spending night-time wakeful periods out of the bedroom) was deemed to best epitomise the stimulus control essence and, therefore, merit of analysis in its own right. In study one a quarter of an hour was identified as the best amount of time to represent SOL of

normal sleepers, hence this single element therapy was named the quarter of an hour rule (QHR). The present exploratory study comprises three single cases and was conducted to assess the impact of the QHR on sleep of people with insomnia.

The literature reviewed in chapter one also highlighted the need to measure stimulus control efficacy objectively because its efficacy in reducing insomnia has typically been measured only subjectively. Given that home PSG has the advantage of increasing ecological validity and that stimulus control hypotheses conditioning to the bedroom environment, PSGs were recorded in participants' homes. In particular, this study explored the feasibility of a novel program, (Biosleep), requiring recording EEG only at Fz and either A1 or A2. If this program proved to be valid, then it could be readily used for home PSG studies because participants could set up EEG recording by themselves once trained to do so.

Finally, given that adherence has been suggested to be an important factor in treatment outcomes but it has been rarely studied systematically, this study examined adherence by measuring it both subjectively and objectively.

4.2 Aims and Hypotheses

The first aim of the present study was to investigate the impact of the QHR on sleep parameters. In line with most research regarding the efficacy of cognitive and behavioural treatments for insomnia, this study employed subjective sleep measures in the form of sleep diary and the diagnostic tool PSQI (Buysse et al, 1989).

Based on previous research showing that stimulus control improved sleep parameters (e.g. Bootzin, 1972, 1975; Haynes et al, 1975; Puder at al, 1983) and produced clinically significant changes (e.g. Sanavio, 1990), it was predicted that:

i) Participants' subjective SOL and WASO would be reduced and S.E. would be increased following intervention; ii) The QHR would produce clinically significant improvements.

In addition to diary data objective sleep continuity parameters were also recorded so as to explore if meaningful PSG data could be obtained by 'Biosleep' a software package requiring recordings only at one site.

Another aim of the present study was to assess daily adherence with the QHR and to gather comments related to its implementation. The feasibility of employing actigraphy to measure adherence objectively was explored.

4.3 Methods

4.3.1 Design

The study comprised 3 single cases: P1, P2 and P3. Single case studies have been widely used by cognitive-behavioural researchers and this methodology is of great importance in the preliminary assessment of clinical techniques and innovations (Morley, 1987). Each single case was an A-B design (A=baseline of 21-22 days, B= treatment of 23 days). Treatment phase length was decided on the basis of previous findings that sleep improvements were observed within two to three weeks after stimulus control implementation (e.g. Bootzin, 1972; Espie, Brooks et al, 1989; Sloan, Hauri, Bootzin, Morin, Stevenson and Shapiro, 1993; Zwart and Lisman, 1979). The data collected during baseline, via participants' responses to a nightly sleep diary, served as a point of comparison to evaluate i) changes in sleep patterns and ii) therapeutic gains following treatment.

4.3.2 Participants

A campus e-mail recruitment procedure was introduced to identify individuals with sleep difficulties for an experimental study on insomnia. The researcher evaluated interested individuals' eligibility via a screening interview according to DSM-IV (APA, 1994) and ICSD-R (ASDA, 1997) diagnostic criteria.

Inclusion criteria were: difficulty in initiating and/or maintaining sleep (SOL and/or WASO greater than 30 minutes) occurring at least three nights per week for at least six months (consistent with Lichstein, Durrence, Taylor, Bush and Riedel, 2003) and perceived as causing distress or deterioration in daytime functioning, and a PSQI (Buysse et al, 1989) score greater than 5. Exclusion criteria were a score greater than 14 on either of the two subscales of the HADS (Zigmond and Snaith, 1983), major medical and/or psychiatric disorder (self reported), psychotropic drug use over the last year, sleep medication use during the last month, alcohol and/or drug abuse, report of symptoms suggestive of sleep apnoea, periodic leg movement (PLMS) or restless legs syndrome (RLS).

Of an initial eighteen potential participants who were screened, 4 met all diagnostic criteria. However, one participant was excluded because diary baseline SOL, WASO, TST and S.E. values were in the normative range for good sleepers (flow chart as per Consort guidelines in fig. 17). The present study, therefore, includes 3 participants.

4.3.3 Materials and Apparatus

4.3.3.1 Sleep Diary

Subjective sleep continuity parameters were gathered by means of sleep diaries (adapted from Morin and Espie's (2003) sleep diary), which participants completed each morning upon arising during baseline and intervention phases. Via the sleep diary, daily information on SOL, WASO, bed-time, rising time, alcohol consumption, sleep medication and daytime napping were obtained (appendix 5). In addition information regarding quality of sleep, restfulness and alertness upon rising (5-point Likert scale) were collected. The variables retained from the sleep diaries were S.E., SOL and WASO. WASO was defined as the summation of the time spent awake after sleep onset and, therefore, comprised duration of all awakenings including early morning awakenings.

4.3.3.2 Sleep Assessment and Anxiety and Depression Questionnaire

4.3.3.2a The Pittsburgh Sleep Quality Index

The Pittsburgh Sleep Quality Index (PSQI: Buysse, et al, 1989) was used as a global measure of sleep quality (section 3.3.3.2, appendix 4). In this study the global index was used in the analysis of treatment outcome as well as for individual assessment of clinical significance.

4.3.3.2b The Screening Interview

The screening interview was a standard protocol (Espie, 2000; Morin and Espie, 2003) based on the ICSD-R and DSM-IV criteria for chronic insomnia. This was

completed during a face-to face interview and considered sleep complaints (chronicity, number of nights with sleep difficulties, SOL and WASO), daytime functioning, use of sleeping tablets, mood, sleep-disruptive medical conditions and other sleep disorders were assessed.

4.3.3.2c The Hospital Anxiety and Depression Scale

The Hospital Anxiety and Depression Scale *(*HADS: Zigmond and Snaith, 1983*)* is a self-report questionnaire measured on a 4 point Likert scale (0-3): 7 items measure depression and 7 items measure anxiety (appendix 6). In particular a score between 0 and 7 is considered as normal, 8 to 10 as mild, 11 to 14 as moderate and 15 to 21 as severe. Internal consistency and test-retest reliability were found to be good (Cronbach alpha= 0.89 for both sub-scales, test-retest correlation = 0.72, $p<0.001$) (Dunbar, Ford, Hunt and Der, 2000;).

4.3.3.3 Objective Measurement of Sleep

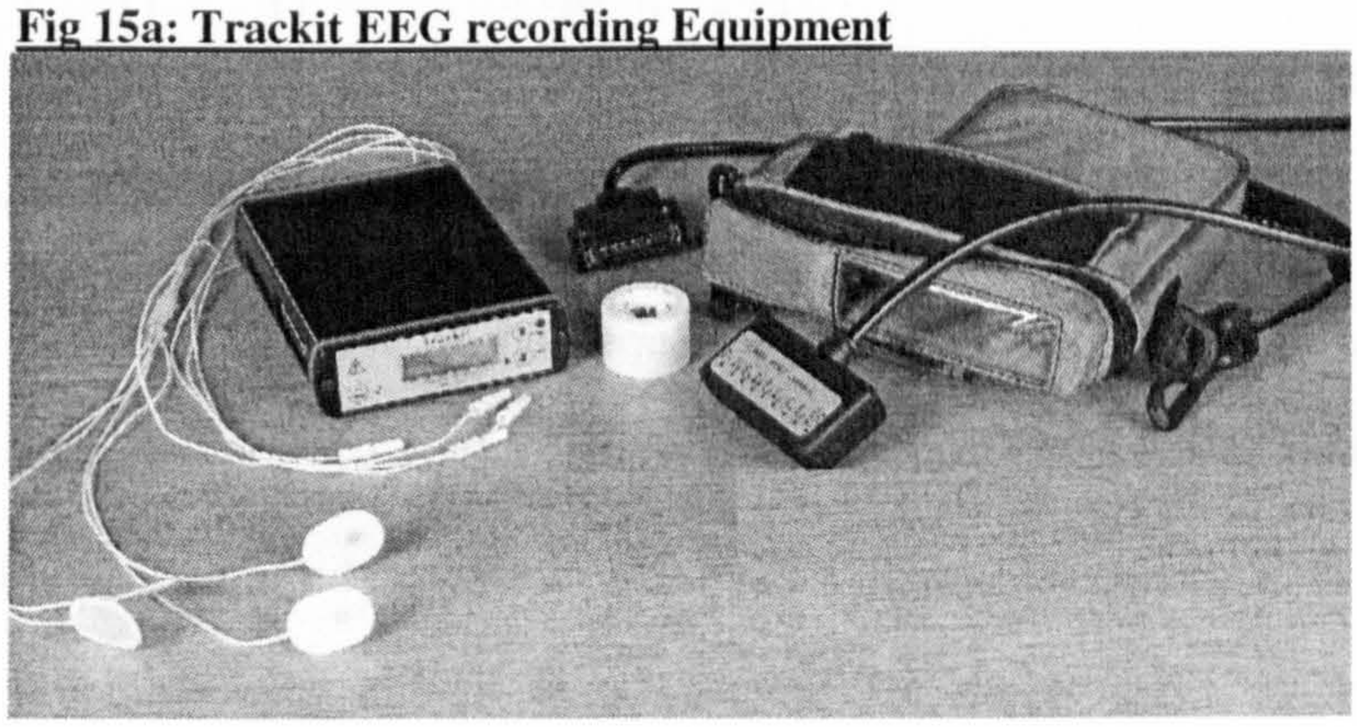
4.3.3.3a Ambulatory Electroencephalogram/Polygraph Recording Device

The Ambulatory EEG/PSG recording device employed in this study was Trackit (Lifelines Ltd, UK). It is a multi channel ambulatory electroencephalograph, comprising of 24-channel EEG amplifier and an 8-polychannel acquisition board. The device is powered by 3 batteries, it is the size of a small hand-bag and weighs approximately 750 grams (fig. 15a). The Patient Connection Unit is a block of moulded plastic with touch-proof sockets and a shielded cable, which connects the EEG recording electrodes attached to the individual to the Trackit unit. The Trackit

set up software is used to program the Trackit recorder for an ambulatory recording session.

The EEG data are stored on an internal ATA flash card in the form of an EDF (European Data Format) file, which enables subsequent review by any EDF-compatible EEG software package.

Fig 15a: Trackit EEG recording Equipment

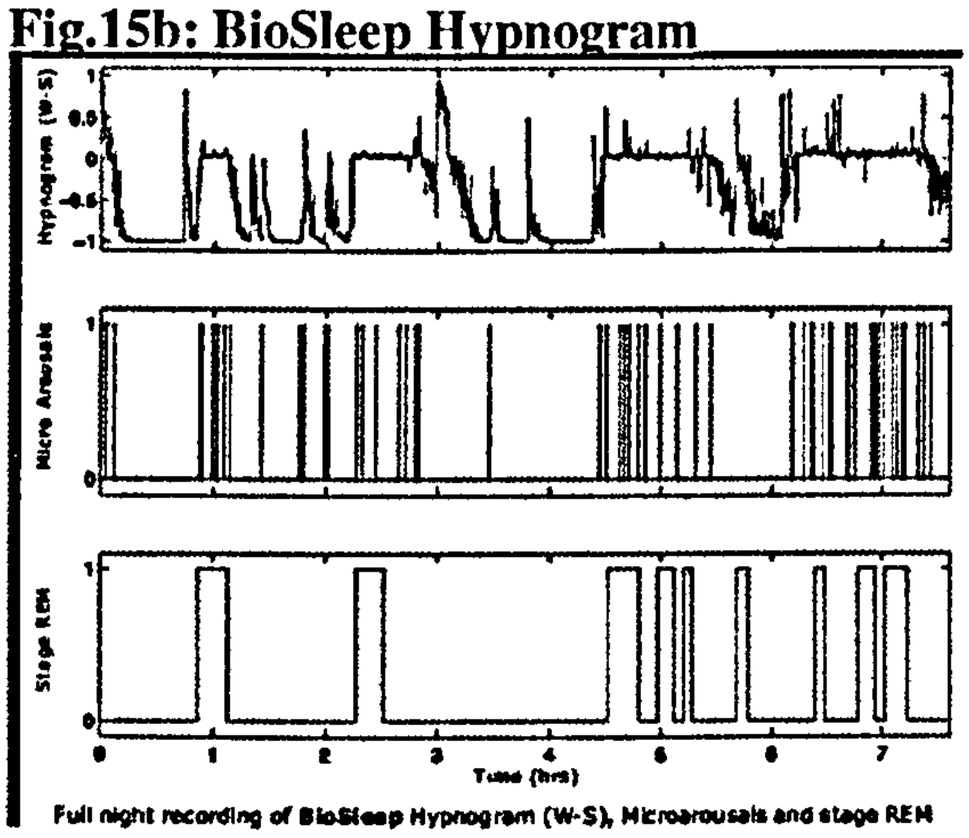


4.3.3.3b Analysis of the Electroencephalographic Recording

The EEG data in the form of EDF files were analysed by employing BioSleep software (Oxford Bio Signal Ltd). Biosleep is a computer program which uses a neural network (Roberts and Tarassenko, 1992; Pardey, Roberts, Tarassenko and Stradling, 1996) to quantify sleep and wakefulness on a second-by second basis, using a single channel of recorded EEG. In particular the EOG signal is proposed to contain both eye movement and EEG signal.

Biosleep first indirectly performs frequency analysis of the EEG, then it presents this analysis as input to a neural network which has been trained to recognise combinations of three different types of EEG activity: wakefulness, REM or light sleep and deep sleep. Figure 15b shows an example of BioSleep Hypnogram.

Fig.15b: BioSleep Hypnogram



For each one-second epoch, Biosleep assigns a probability value to each of these three processes so as to extract the probability that one particular process was dominant in the brain during a particular one-second epoch.

Biosleep's analysis of the frequency content of the EEG uses a technique known as autoregressive modelling. It constructs its model using the kind of least-square technique employed in regression analysis.

It is important to note that Biosleep is a simple and user friendly package, however it provides pseudo R&K. A study by McGrogan, Braithwaite and Tarassenko (2001) showed that over the nine nights recordings of sleep EEG the correlation between the pseudo- R&K obtained with Biosleep and R&K stages from a consensus of three human experts was 72.2%.

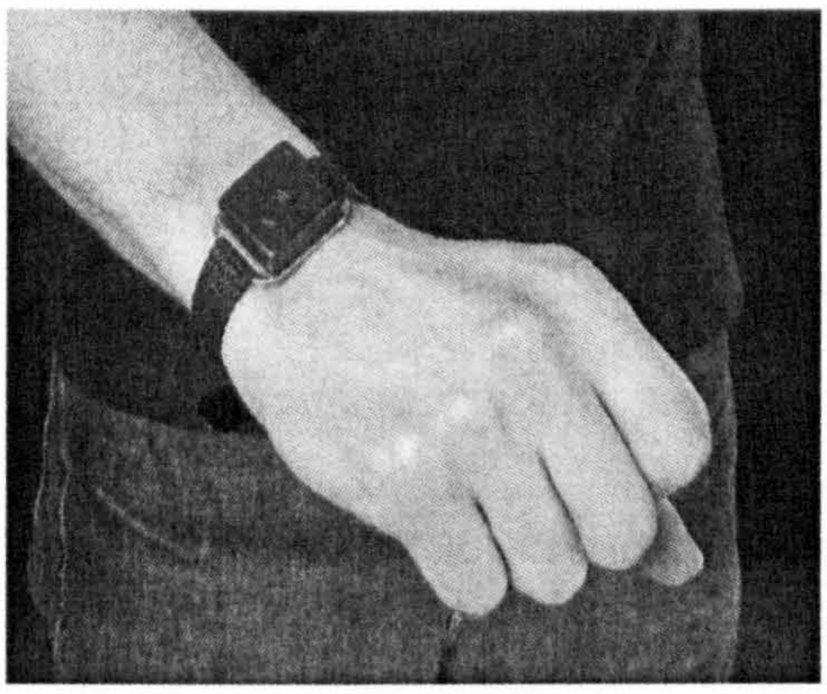
4.3.3.4 Measures of Adherence

Adherence was measured both subjectively via adherence diary and objectively via actigraphy. In particular, with regard to objective adherence, it was reasoned that implementation of the QHR required movement (e.g. going to another room, reading or having a juice) and therefore, actigraphy could be a useful tool.

4.3.3.4a Actigraphy

The Actiwatch (Cambridge Neurotechnology Ltd) is a lightweight wristwatch-like electronic device that measures and records physical movements by the means of a piezo-electric accelerometer that is set up to record the intensity, amount and duration of movement (figure 16). The corresponding voltage produced is converted and stored as an activity count in the Actiwatch memory unit. The data collected are then analysed via a software package produced by Cambridge Neurotechnology Ltd.

Fig. 16: Actiwatch



Given that the QHR required participants to 'get out of your bed if not asleep within a quarter of an hour' one minute epochs recorded by the ACTW were considered. If there was continued movement for at least 16 minutes, the QHR should have been applied.

4.3.3.6b Adherence Diary

Adherence with the QHR was measured daily, from the first night of implementation, via a small 'intervention diary' devised by the researcher by adapting Lichstein's CBT adherence checklist (personal communication, 2003). This served to collect data regarding whether, for each night of intervention, the QHR had to: a) be applied to fall asleep and/or during the night if awake, b) how many times, c) after how long it was applied (if at all) and d) how difficult it was to apply it (measured on a Likert type scale: 1-10 with 1=not difficult at all and 10= extremely difficult) (appendix 7). In order to minimise social desirability issues (e.g. pleasing

the researcher) participants were told that their responses would help the researcher to understand the extent to which people found it difficult to implement the QHR.

4.3.3.6c Adherence Score

An adherence score was calculated using the following formula:

$$\text{Adherence Score} = \frac{\text{Number of Times QHR was adhered to}}{\text{Number of Times Implementation was Required}}$$

4.3.3.5 The Quarter of an Hour Rule Intervention

The intervention was named 'The quarter of an hour rule'(QHR) and was extracted from the original protocol proposed by Bootzin in 1972. The QHR stated:

> **If you are NOT asleep within a quarter of an hour,**
> **Then get up and do the chosen activity in the chosen room.**
> **Go back to your bed when you feel ready to sleep again.**

The activity to perform and the room to go to were decided by the participant during the delivery of the QHR intervention. The words 'when you feel sleepy again' of the original protocol (Bootzin, 1972) were changed into 'when you feel ready to sleep again' to reduce the impact of the implicit instruction of monitoring an internal state for sleep.

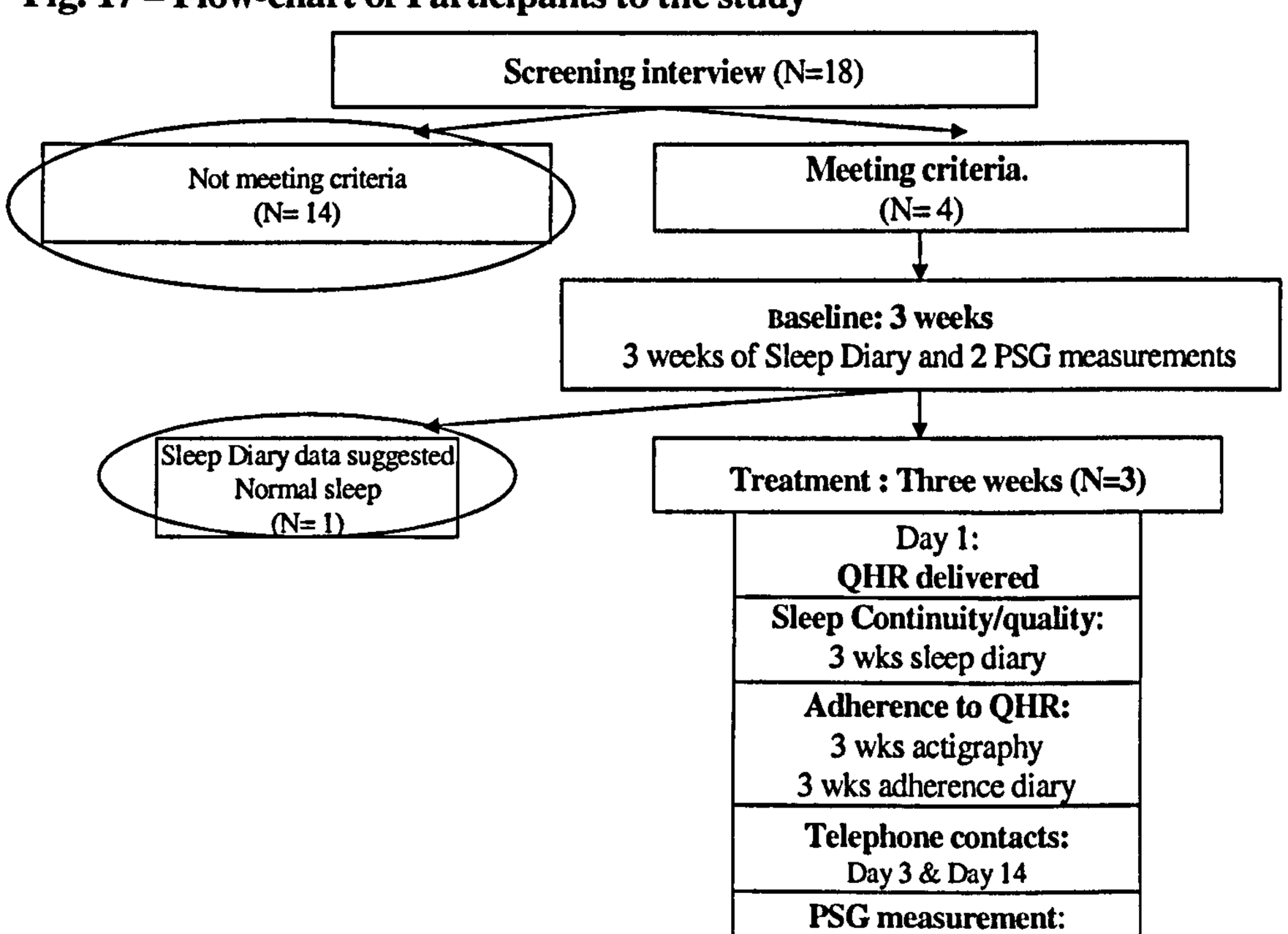
Participants were requested to follow the instruction again if, once returned to their bed they were not asleep within a quarter of an hour or if they woke up during the night and could not fall to back to sleep within a quarter of an hour.

It was made clear that clock watching, so as to decide when a quarter of an hour had elapsed, was to be avoided as this might keep them awake and to get up if they felt that they had been lying in bed for about a quarter of an hour. The intervention was delivered individually, by the researcher, following a written protocol (appendix 12) and it took only 30 minutes. A flow-chart instruction sheet was given to participants to help them follow the QHR when at home (appendix 8).

4.3.4 Procedure

People who replied to the recruitment email showing an interest in the study were sent an information pack (appendix 9) and they were invited to contact the researcher if they wanted to participate.

Fig. 17 – Flow-chart of Participants to the study



Once potential participants replied, they were invited to meet with the researcher (on an individual basis) and sign a consent form. Eligibility was then assessed via structured interview (chapter 3 in Morin and Espie, 2003), the PSQI (Buysse et al, 1989) and the HADS (Zigmond and Snaith, 1983). Assessment lasted around forty-five minutes. Participants not meeting criteria were debriefed and thanked for their interest and time but did not continue with the study. Participants meeting criteria were trained in the correct use of the EEG/PSG recording equipment (Trackit, section 4.3.3.3a), had a practice session and were handed in a written memo (appendix 11). The practice session ensured that participants had understood how to set up Trackit properly and that they felt comfortable using it. Participants were then provided with the sleep diary to be filled in each morning upon rising for three weeks and the EEG/PSG recording equipment. The training/practice session lasted around forty-five minutes. The researcher collected the EEG/PSG recording equipment three days after this meeting.

Three weeks after baseline, participants met, on an individual basis, with the researcher who delivered the QHR intervention following a written version so as to avoid confounding in therapy outcome due to different wordings (appendix 12). Participants were given an easy to follow memo about the QHR (appendix 8), were instructed to keep completing the sleep diaries and, in addition, the 'adherence with the QHR diary' (appendix 7) for a further three weeks. Finally, it was explained that the actiwatch detected movement and would provide the research with information regarding when they were active or resting. They were asked to wear the actiwatch all the time (unless they were getting in contact with water) during the three week

intervention and were given a memo about actiwatch use (appendix 13). The intervention session lasted around thirty minutes.

During the three weeks intervention there were two informal contacts (the third and the fourteenth day of the intervention) over the phone, lasting no more than 10 minutes each, during which the QHR was reinforced. Two days prior to the end of the intervention period the researcher gave participants the EEG recording equipment and reminded them how to use it. The morning after the second EEG night, participants met with the researcher and filled in the PSQI. The researcher collected comments (verbatim) about their sleep and how they felt about the intervention. Participants returned the EEG recording equipment, the sleep and adherence diary and the Actiwatch. They were fully debriefed and their sleep results were sent to them within a month. This session lasted around forty-five minutes.

4.3.5 Data Analysis

Efficacy of the QHR was explored via subjective (sleep diary) and objective (PSG recording) measures of SOL and WASO. Daily sleep diary data were first analysed by visual inspection of graphic representations, a standard method used in single case studies (Franklin, Gorman, Beasley and Allison, 1996). Thereafter Interrupted Time Series Analyses (ITSACORR, Crosbie, 1993) were conducted to determine whether visually observed changes between baseline and post intervention were statistically significant. Time series analysis permits identification, across adjacent (baseline and treatment) phases in an experimental design, of level change (abrupt change in the mean level of the data) and slope change (gradual change) once the treatment is introduced.

In accordance with Lichstein and Fischer's (1985) and Kazdin's (1980) proposal, the following indices of clinical significance of results (therapeutic gains obtained at outcome) were employed: i) SOL and WASO shorter than 31 minutes and SE greater than 85%, ii) PSQI global score equal to or less than 5 (which measure provided collateral validation of measures obtained via sleep diaries) and iii) individual subject variable effect sizes. These were calculated by dividing the pre-post treatment change by the pooled standard deviation. A positive effect size indicated a reduction in insomnia symptomology.

No inferential statistics were calculated for objective sleep data or adherence. Objective sleep continuity data were compared to subjective data. As already mentioned, Biosleep provides pseudo R&K and sleep architecture values were reported. Adherence scores and participants' comments regarding applying the QHR were reported.

4.4 Results

4.4.1 Participants' Characteristics

Participant P1 was a 25 years old male complaining of both sleep onset and sleep maintenance difficulties since the age of sixteen. He could not relate the onset of sleep difficulties to any particular event. He had a higher degree, was working full time, and shared a flat with two friends. He did not have a regular bed partner.

Participant P2 was a female third year accountancy undergraduate student, 22 years of age, who complained of having had sleep onset and sleep maintenance

difficulties for eighteen months (grandmother died). She was sharing a flat with two people and did not have a regular bed partner.

Participant P3 was a 45 years old final year history undergraduate student, married and with no children. His sleeping difficulties started in his mid-twenties probably due to shift work. His sleep difficulties had not disappeared despite having left the army (and shift work) for over 15 years. He complained of difficulties falling asleep.

Participant P4 was a male support worker aged 32, who complained of both sleep onset and sleep maintenance difficulties and, particularly, of not feeling refreshed in the morning (non-restorative sleep). His sleep difficulties started when he was a teenager and his family moved from south of England to Scotland and he was teased at school for his accent. Criteria of psychophysiologic insomnia were met at intake, however, analysis of the three weeks baseline showed that all sleep parameters were within the norms for good sleepers (hence not confirming intake diagnosis). For this reason he was excluded from the study. He was fully debriefed, and the good sleeping guide prepared by Espie, together with a sleep relaxation tape, were given to the participant.

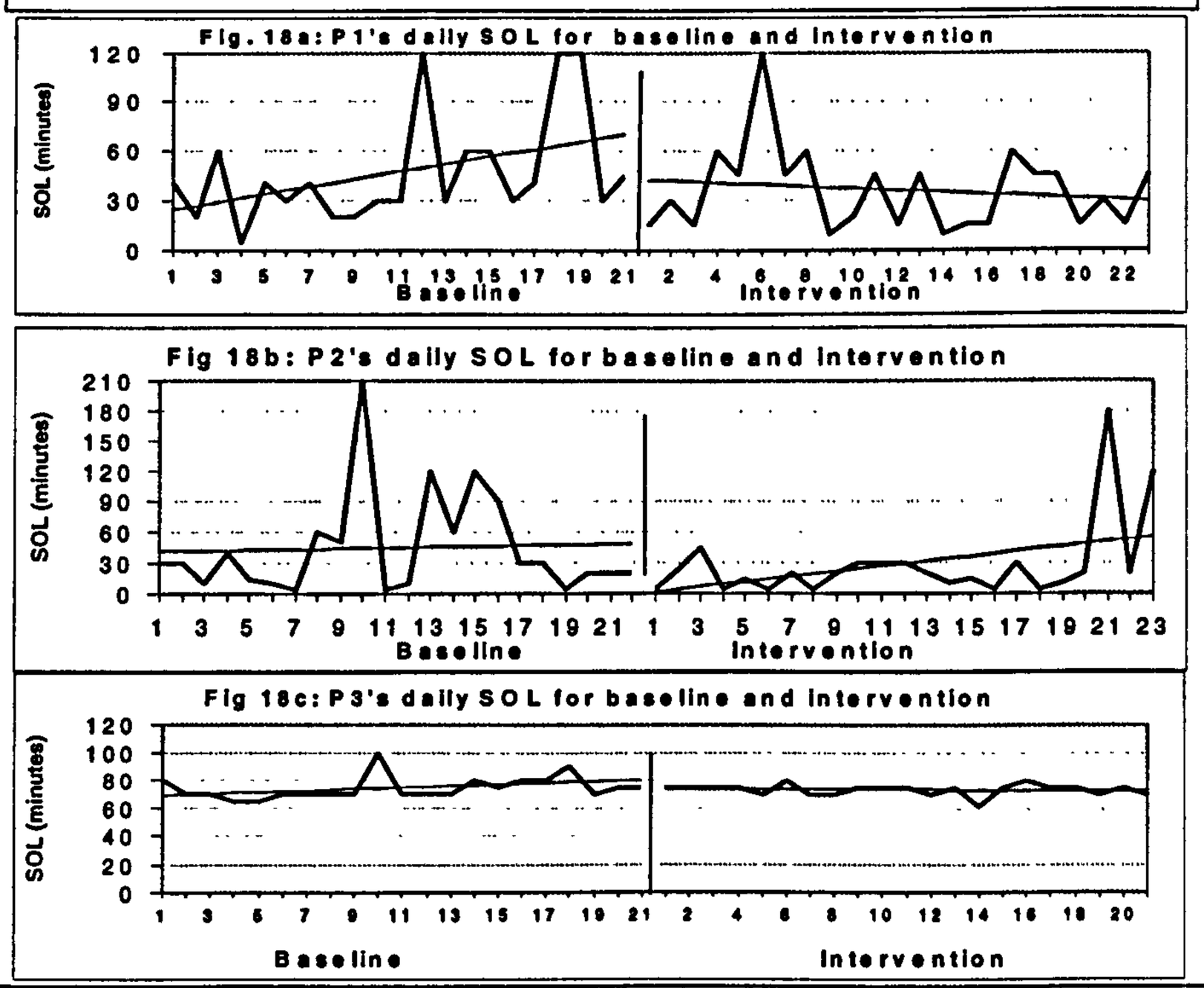
4.4.2 Sleep Pattern Changes: Visual Inspection of Sleep Diary Data and ITSA

4.4.2.1 *Complaint of difficulty of falling asleep (SOL)*

Figure 18 presents SOL data for P1, P2 and P3. Baseline values reflect typical sleep pattern variations inherent in insomnia with no indication of spontaneous improvement. Mean values for each phase are presented in table 8.

Following intervention with the QHR, P1's mean SOL reduced by 24% (table 8) although visual inspection of figure 18a does not reveal a clear pattern of change. By contrast P2's SOL reduced by 35% and in particular only 3 out of the 23 treatment nights were in the clinical range (greater than 31 minutes) compared with 8 in the 22 baseline nights. Visual inspection suggests reduced variability in sleep pattern. ITSA revealed a trend with regard to change in slope [t(39)= -1.75, p=0.08] from baseline to treatment for P1. With regard to P3, this participant never applied the QHR during the 21 intervention nights and it is not, therefore, surprising that no changes were observed between baseline and intervention periods.

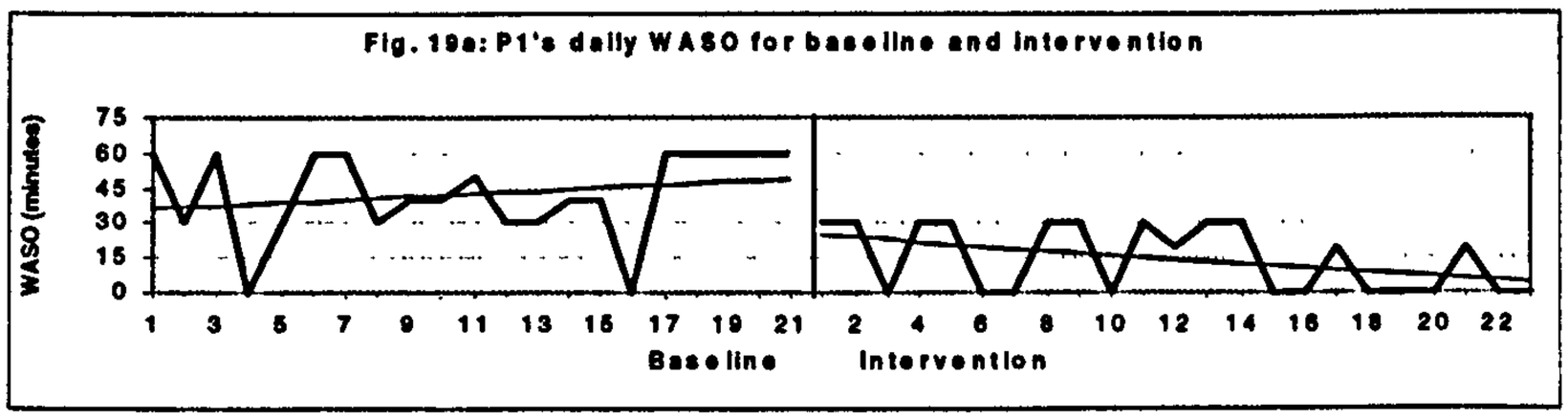
Fig. 18: Daily SOL at Baseline and Intervention

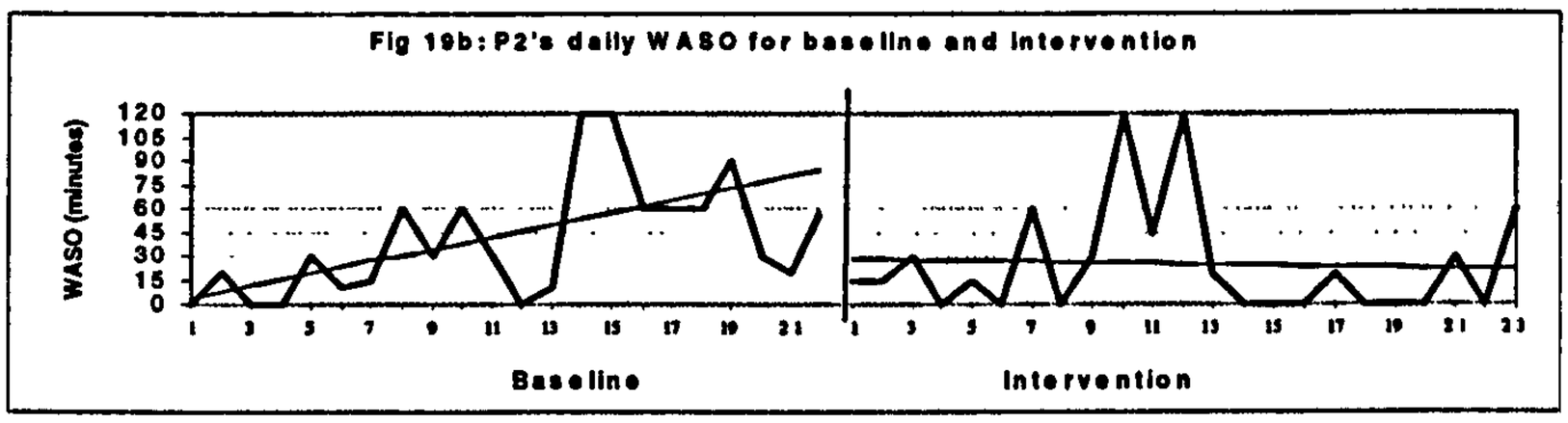


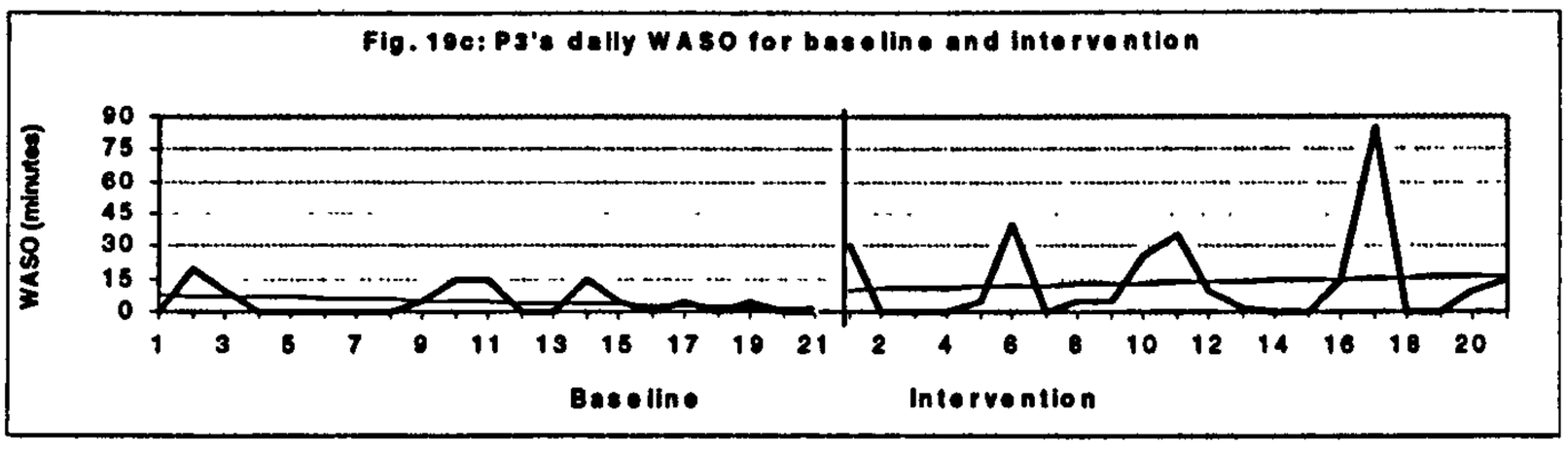
4.4.2.2 Complaint of difficulty of staying asleep (WASO)

Figure 19 presents WASO data for P1, P2 and P3. Baseline values, again, reflect typical sleep pattern variations inherent in insomnia with no indication of spontaneous improvement. Mean values for each phase are presented in table 8.

Fig. 19: Daily WASO at Baseline and Intervention







Visual inspection of fig. 19a suggests decrease in WASO for P1 confirmed by mean reduction of 28 minutes (67%, table 8), in particular 14 out of the 21 nights of baseline were in the clinical range (greater than 31 minutes), but none of the 23 post-intervention nights were. Reduction in WASO for P2 was less marked (37%) nonetheless 5 of the 23 post-intervention nights were in the clinical range compared with 9 out of the 22 baseline nights. It should be noted that she was camping at a

concert site during nights 10, 11 and 12 of the intervention phase and people were coming and going all night. ITSA confirmed a significant overall change [F(2,39)=4.61, p=0.02] and a significant change in slope [t(39)= -2.06, p=0.05] from baseline to treatment for participant P1.

4.4.3 Clinical Significance

Clinical significance was evaluated on three indices: end state scores for SOL, WASO, S.E. and PSQI global score change and effect sizes. In particular clinical significance was measured on two criteria: a post-treatment change over baseline greater than 50% (Lichstein and Fischer, 1985) and/or the individual has moved from the dysfunctional to the functional range (Kazdin, 1977).

As depicted in table 8, P1's WASO and S.E. and P2's SOL and WASO returned within the normative average range following intervention. In addition, outcome measure on the PSQI showed a significant improvement following QHR intervention for both participants. Although their PSQI scores did not quite fall below the recognised threshold for sleep disturbance (≤5) scores were reduced by 50%: from 13 to 6 (P1) and from 14 to 7 (P2) respectively. It is noteworthy that a significant improvement was found for the PSQI component score 'subjective sleep quality', which changed, following intervention, to 'fairly good' for both P1 and P2 (table 8). Finally, SOL effect sizes were in the small to medium range, and WASO and S.E. effect sizes in the medium to large range for both participants. With regard to P3, he did not show positive changes in the above mentioned values following treatment. His PSQI score changed from 9 to 8 and his PSQI component score 'subjective sleep quality' changed from 'fairly bad' to 'fairly good'.

Table 8: Sleep parameters and clinical indices of change for P1, P2 & P3

1a) Mean (SD) for SOL WASO and SE at baseline and post- intervention, post-treatment changes and effect sizes

	Baseline	Post-Interv	Pre to Post Change Value	%	Treatment Effect Size
SOL (minutes)					
P1	47.1(33.4)	35.6(25.3)	- 11.5	24.4	0.39
P2	45.0(50.3)	29.1(40.6)	- 15.9	35.3	0.35
P3	74.5(8.3)	73.4(4.0)	- 1.1	1.5	0.17
WASO (minutes)					
P1	42.9(19.0)	14.3(14.4)	- 28.6	66.6	1.69
P2	40.2(36.3)	25.2(35.4)	- 15.0	37.3	0.42
P3	4.5(6.5)	13.4(20.6)	+ 8.9*	66.4 *	- 0.58*
* WASO worsened in the intervention period					
S.E. (%)					
P1	75(11)	86(11)	11	14.6	0.99
P2	64(16)	74(14)	10	15.6	0.67
P3	84(03)	82(05)	- 2*	2.4*	- 0.75*
* this value shows a deterioration of sleep variables from baseline to post-treatment					

1b) PSQI at 1st day baseline and 23rd day post-intervention

	PSQI global score		PSQI 'subjective sleep quality' component	
	Baseline	Post-Interv	Baseline	Post-Interv
P1	13	6	Fairly bad	Fairly good
P2	14	7	Very bad	Fairly good
P3	9	8	Fairly bad	Fairly good

4.4.4 Objective Sleep Continuity Data

Table 9 presents the sleep continuity and sleep architecture data recorded during the second night of baseline and the last night of treatment.

Inspection of table 9 shows that Participant P1's objective SOL and WASO were shorter the last night of treatment than at baseline, participant P2's SOL decreased and participant P3's SOL and WASO increased. The objective and subjective data do not show any clear association. It is noteworthy that pseudo R&K values for REM

are extremely high as compared to those reported in the literature (p. 16) for both normal and poor sleepers.

Table 9 – Sleep Continuity Variables gathered by PSG at Baseline (night 2) and during the Last Night of Treatment Phase (and correspondent diary values)

	BASELINE			END OF TREATMENT		
	PSG	Diary	Pseudo R&K	PSG	Diary	Pseudo R&K
Participant P1						
S.E.	0.92	0.61		0.98	0.97	
WASO	29	60		7	0	
SOL	9	40		1	15	
% REM			54.9%			85.2%
% Stage 1			12.2%			12.6%
% Stage 2			20.3%			2.2%
% Stage 3&4			12.6%			0.0%
Participant P2						
S.E.	0.95	0.88		0.92	0.97	
WASO	9	0		30	0	
SOL	10	30		1	20	
% REM			39.5%			40.2%
% Stage 1			3.8%			20.7%
% Stage 2			11.1%			12.8%
% Stage 3&4			45.6%			26.2%
Participant P3						
S.E.	0.98	0.71		0.93	0.79	
WASO	4	35		25	15	
SOL	0	80		23	80	
% REM			54%			58%
% Stage 1			13%			15%
% Stage 2			20%			17%
% Stage 3&4			13%			10%

4.4.5 Adherence with the Quarter of an Hour Rule

Participant P1 reported (daily adherence diary) applying the QHR at SOL 93% of the times (14 of 15) he was not asleep within a quarter of an hour. Data gathered via actigraphy showed that there was movement on 13 occasions. With regard to WASO participant P1 stated that he never had to apply the rule. However, actigraphy recorded seven periods of movements greater than 16 minutes suggesting that he should have applied the rule at least 7 times during the intervention phase.

P2 applied the QHR 70% of the times (12 of 17) she was not asleep within a quarter of an hour at SOL and data gathered via actigraphy confirmed that there was movement at SOL in 12 occasions. With regard to WASO, participant P2 applied the rule only 29% of the times (2 out of 7) she did not return to fall asleep within 15 minutes and actigraphy detected movement on 7 occasions.

Participant P3 never applied the rule.

4.4.6 Adherence Comments

P1 commented that he wanted to ***"really give it a try"***, noticed the intervention helped his sleep and this, in turn, made him ***"more determined to follow the rule"***. he enjoyed ***"reading my book"*** instead of ***"turning in my bed wondering whether I will fall asleep and thinking about everything until eventually I fall asleep"***.

P2 felt that by staying in bed, even if awake, she could not be blamed for her poor sleep ***"It shows I am really trying to sleep it is not my fault if I can not"***. For the same reason it was hard for her to stay up till she felt ready to sleep again ***"I feel obliged to go back and try hard to sleep"***. Having the QHR to apply enabled her to ***"get rid of the blame"*** and, therefore, to ***"stop trying harder and harder and getting more frustrated"***.

P3 did not apply the rule. When the researcher met the participant to debrief him, he said that he had realised that **"family and friends know I don't sleep"** and ***"I can get on with my study and things at my own pace"***. He told the researcher that not sleeping much at night and then having a nap actually suited him ok.

4.5 Discussion

In this section results are discussed in relation of the feasibility of employing the QHR as a stand alone therapy and in relation to design improvements for the main study (study three). A more general discussion is provided when results of the main study are analysed and general issues raised.

4.5.1 Summary of the Results

The primary aim of this study was to explore if the QHR would reduce symptomatic sleep complaint at a meaningful clinical level. In addition, this study explored the possibility of acquiring meaningful PSG data by recording at one site only. Finally this study assessed daily adherence and participants' comments regarding the implementation of the QHR. The feasibility of actigraphy to measure adherence objectively was also explored.

Two specific predictions were made. The first prediction was that, following the QHR, participants' estimates of SOL and WASO would be reduced and S.E. increased. This prediction was partially supported. Inspection of SOL and WASO mean and raw score values suggested that the QHR was associated with positive improvements in the two participants (P1 and P2) who applied the QHR. Conservative time series analyses evidenced statistically significant changes in WASO, and a trend toward significance for SOL for participant P1. Objective measures of sleep did not show a clear trend: P1's SOL and WASO and P2's SOL were reduced.

The second prediction was that the QHR would produce clinically significant improvements. This prediction was also supported. WASO of participants P1 and P2, P2's SOL and P1's S.E. fell within the normative range at post-treatment and their PSQI global scores were reduced by 50%, approaching normal range.

Another aim of this study was to collect adherence information. Results suggested that better adherence (participant P1 reported following the QHR most of the time) was associated with more convincing data evidencing change (including ITSA). It is noteworthy that participant P2 obtained therapeutic gains despite partial adherence to the protocol. Importantly participant P3 never adhered to the QHR and, indeed, his sleep did not improve.

With regard to objective measures of sleep acquired via Trackit and analysed via a novel program requiring EEG recording at one site only, no clear patterns were found. Importantly, sleep architecture data differed greatly from those reported in EEG literature for both normal and poor sleepers.

4.5.2 Interpretation of the Results

As reported in the previous section, following the QHR sleep difficulties were decreased in two out of three participants. With regard to the participant whose sleep did not improve, it is noteworthy that he never adhered to the QHR. Interestingly, these results are comparable to those obtained in previous studies of the full stimulus control treatment and of multicomponent CBT (Morin et al, 1994).

Effect size calculations indicated that treatment gains were in the small to moderate range for SOL and in the moderate to large range for WASO. Previous studies have reported larger effect sizes for SOL (Morin et al, 1994; Morin et al,

1999; Murtagh and Greenwood, 1995) but initial insomnia severity in the present study was moderate thus limiting power. Indeed, a positive relationship between SOL and WASO reductions and initial insomnia severity in clinical samples has been found in previous studies (Espie et al, 2001; Lacks et al, 1983a).

Importantly, for the two participants who applied the intervention, improvements were also clinically significant with sleep parameters and PSQI scores reduced by 50% or returned to normalcy. Furthermore both participants P1 and P2 reported greater satisfaction with their sleep. This is an important finding as perception that one's quality of sleep has improved, rather than insomnia symptoms simply being less, might be critical to reduce the psychological distress often accompanying insomnia (Morin et al, 1994). Subjective distress may be the primary motivation prompting people with insomnia to seek treatment (Stepanski, Koshorek, Zorick, Glinn, Rohers, and Roth 1989).

In sum, three weeks after the delivery of the QHR (a single session intervention requiring 30 minutes to be delivered) positive effects, on both quality of sleep and sleep parameters, were obtained.

Adherence data are important both from a clinical (adherence-response relationship) and theoretical (mechanisms of effect) point of view. An interesting finding, which highlights the need to research the issue systematically, regards the adherence-response relationship. Chambers (1992) proposed that adherence might be the most important factor in treatment success: indeed, better adherence (P1) was associated with more convincing data evidencing change (including ITSA). However, despite partial adherence to the protocol, therapeutic gains were also obtained by participant P2. This was an unexpected result: in light of Bootzin and

Epstein's (2000) suggestion that, for stimulus control to work, participants must follow the instructions 'fastidiously' (p. 175), it was assumed that the QHR was to be followed consistently and rigorously to produce sleep changes. Perhaps factors other than adherence (e.g. feeling in control) played a role.

Participant P1's comments suggest that the QHR might help the individual to feel differently about the time spent awake: rather than a frustrating period it becomes acceptable, even enjoyable. Speculatively, it could be argued that the QHR, by preventing the individual from using the time awake worrying, enabled him to reach the 'right conditions' to fall asleep. On the other hand, it is possible that the QHR 'broke' the association bedroom-wakefulness (i.e. sleep improvements were due to good stimulus control) but that the participant interpreted sleep improvement in a cognitive way.

Participant P2 waited longer than fifteen minutes before applying the QHR most times (67%) and her comments, not only highlight one of the causes of non-adherence, but are also an example of 'effortful sleep' typical of insomnia yet so atypical of 'normal sleepers' (Espie, 2002). However, her sleep improved and it could be, tentatively, suggested that the QHR by removing her responsibility for not being able to sleep (she would have tried harder but the QHR did not allow her) enabled her to 'do nothing' and hence fall asleep. As proposed by Espie (2002) good sleepers do not try or do something to fall asleep: they just seem to do it.

Overall these comments are in line with Zwart and Lisman's (1979) proposal that stimulus control might work by disrupting cognitive activity at bedtime: be it thinking, planning, trying to sleep. Indeed, the six participants in Turner and Ascher's (1979a, b) studies pointed out that stimulus control provided them with a

means of avoiding thinking while lying in bed. Although this is an interesting proposal further, direct testing, of this hypothesis should be devised.

This study suggested that home PSG was well tolerated as participants commented on Trackit being easy to carry around (e.g. going to the bathroom, watching TV in the sitting room; making a cup of tea) and that the recording equipment minimally disrupted their usual sleep. However, Biosleep output displayed very high percentage of REM sleep and little percentage of Stg 2 sleep as compared to PSG scored according to R&K guidelines. This finding argues against recording EEG at only one site and analysing data employing Biosleep because output does not seem valid.

In sum, this exploratory study highlighted that the QHR, requiring only 30 minutes to be explained, might be a promising therapy for insomnia. In addition home PSG was well tolerated and participants reported that they were able to apply the QHR.

4.5.3 Considerations for the main study

As summarised and discussed in the previous section the results of this preliminary study are encouraging: one single session followed by a couple of telephone contact were sufficient to decrease sleep difficulties and outcomes were clinically significant. The two participants, who applied the QHR, responded quickly to treatment, reported good adherence and that home PSG was minimally intrusive.

Furthermore this study highlighted changes to the methodology so as to better calibrate the main study. In particular the following points were noted.

First, the single case methodology permits assessment of an intervention fairly quickly and at modest cost and it is, therefore, well suited to test novel interventions. Indeed, the present single case study suggested that the QHR, a novel intervention requiring minimal contact with the therapist, decreases sleep difficulties. However, without a contrast intervention it is hard to assess whether any therapeutic gains are attributable to the QHR itself or to other factors such as passage of time or contacts with a therapist or the measurement process itself (i.e. monitoring one's sleep). The randomised controlled trials, which compare two or more treatments (one of which can be no-treatment) can help to address this issue. It seems, therefore, advisable, to carry out the next study employing this methodology.

Second, although participants to the present study met criteria for psychophysiological insomnia, they were self-referred. In order to explore the generalisability of the QHR outcomes the main study involved GPs' and consultants' referred participants.

Third, in order to shed light on whether the mechanisms of effects of the QHR are purely due to environmental conditioning and the re-establishment of good stimulus control, a counter control intervention (Zwart and Lisman, 1979) could be employed. For this reason a counter QHR instructing participants to apply the QHR in bed was devised and its outcomes contrasted to those of the canonical QHR.

Fourth, although actigraphy may have a value in monitoring adherence, the present study highlighted that actigraphy can only indicate if the person is moving but does not evidence whether the QHR is actually adhered to. It was reasoned that if the QHR asked participants to read if not asleep, then actigraphy with the added

feature of a light detector would enable measurement of adherence to the rule. In the main study objective adherence was, therefore, measured with actigraphy plus light.

Fifth, the pilot study highlighted that home PSG is feasible and well tolerated but that Biosleep has limited validation: it produces pseudo R&K and data seems implausible (note the REM for example). In the main study home PSG was carried out according to insomnia PSG recording criteria so as to enable traditional R&K scoring and interpretation of the collected EEG data.

Chapter 5

Study Three

Impact of the quarter of an hour rule intervention, implemented in bed and out of bed, on sleep parameters

5.1 Rationale

The results obtained with the single case studies in the preliminary study (study two, chapter 4) suggested that the QHR might be a promising therapy for insomnia and, therefore, worth exploring in a more systematic way. In addition, taking into account the methodological shortcomings highlighted by the preliminary study, a number of changes were made with regard to sleep measurements, objective measurement of adherence and procedure. Furthermore, sleep related questionnaires were introduced in order to shed light on the mechanism of effects of the QHR. The QHR was administered in two different formats and a control group was added.

Briefly, this study comprised three groups (QHR in bed, QHR out of bed and Control). Similarly to the preliminary study sleep parameters were measured subjectively and objectively; however, unlike study two, home PSG measurements were performed employing traditional recording sites as per R&K (Rechtschaffen and Kales, 1968). As in study two objective and subjective measures of adherence were also collected; however the objective measure of adherence incorporated both detection of movement and light. Finally, a battery of sleep related questionnaires, tapping into sleep related cognitions, behaviours and arousability at bed time, was administered at baseline and at the end of the intervention.

5.2 Aims and Hypotheses

The first aim of study three was to assess whether the QHR, proposed to capture the essence of the stimulus control therapy for insomnia, could be employed to reduce sleep difficulties in a clinical population.

The second aim of the present research was to explore possible mechanisms of effect of the QHR. This exploration was carried out by devising two QHR interventions: one to be implemented out of bed (QHRout) and one to be implemented in bed (QHRin). Additionally, sleep related questionnaires measuring sleep behaviours and cognitive activity at bed time were employed.

The third aim was to investigate the relationship between adherence and treatment response and to look into the feasibility of an objective measurement of adherence.

Specifically the following predictions were made:

First, resting on the same bases discussed in the rationale of study two, it was predicted that participants' self reported SOL and WASO would be reduced and TST and S.E. increased following the QHRout intervention.

The second prediction rested on evidence presented in the introduction (Alperson and Biglan, 1979; Zwart and Lisman, 1979; Davies et al, 1986) that a procedure named 'countercontrol' improved sleep. Specifically, it was predicted that self-reported SOL and WASO would decrease and TST and S.E. would increase following the QHRin intervention.

Third, it was predicted that, differently from the QHRout and the QHRin interventions, the control group condition would not impact on SOL, WASO, S.E. or TST.

Fourth, it was predicted that the outcome measure PSQI (Buysse et al, 1989) at the end of the intervention would be reduced, as compared to baseline, in both QHR groups. No changes in PSQI were expected for the control group.

Fifth, improvements in reported qualitative measures of sleep (i.e. quality of sleep, restedness and alertness upon awakening in the morning) were expected following intervention only for the active treatment groups.

Sixth, in view of evidence that stimulus control produces clinically significant changes (e.g. Bootzin, 1972; Sanavio et al, 1990) and the results of the pilot study, it was predicted that participants in the active treatment groups would meet criteria for clinically significant changes as compared to the control group. However, no predictions were made with regard to the differential magnitude of improvements obtained with the two different administrations of the QHR.

No firm predictions were made with regard to sleep related cognitions (e.g. sleep effort, sleep beliefs) and arousability at bedtime measured via questionnaires. As discussed in the introduction a number of authors (e.g. Espie, 1991, Turner and Ascher, 1979a,b; Zwart and Lisman, 1979) have suggested that stimulus control reduced sleep difficulties not only by re-conditioning mechanisms but also by decreasing cognitive interference. If this was the case then a decrease in cognitive activity would be expected. In contrast, if the mechanisms of effect were solely related to re-conditioning then no differences in sleep related cognitions should be found.

Taking into account that previous research has produced mixed results with regard to objective changes following non-pharmacological insomnia interventions (Stepanski, 2000) no firm predictions were made with regard to changes in PSG defined sleep.

Seventh, it was predicted that sleep continuity parameters measured via PSG and via sleep diary would be positively correlated. This prediction rests on evidence (e.g. Carskadon et al, 1976; Schramm et al, 1995) that sleep diary and PSG are correlated although sleep continuity parameters seem to be inflated as compared to objective ones.

The eighth prediction rested on claims that adherence might be an important aspect of treatment response (section 1.81): specifically it was predicted that greater adherence to treatment would be associated with better outcomes.

The third study set out to test these eight predictions and to shed light on the two research questions.

5.3 Methods

5.3.1 Design

This was a randomised experimental controlled trial (Clinical Registration N. NCT00170391) investigating the efficacy of the QHR carried out in bed (QHRin) and out of bed (QHRout) as compared with a sleep self-monitoring group (control), using parallel measurements of objective and subjective sleep parameters, taken at baseline and post-treatment. The impact of the QHR interventions on a number of cognitive factors was investigated by employing six, well-validated questionnaires. They are commonly used in sleep research (section 5.3.3.2a provides a detailed

explanation of the questionnaires used). Finally, the impact of adherence was also investigated both subjectively (adherence diary) and objectively (actigraphy plus light). Randomisation was performed by the statistics department at Glasgow University: a random sequence, controlling for participants' gender, was generated by SPSS.

This quasi-experiment with a mixed design had one between subject factor 'Group' with three levels ('QHRout', 'QHRin' and 'control') and one within subject factor 'Time' with two levels ('baseline' and 'end of treatment').

Table: 10 - Dependent Variables for PSQI, Sleep Continuity, Sleep Quality, Questionnaires and Adherence with the QHR

	Subjective		Objective	
	Diary	Questionnaire	PSG	ACTW-L
PSQI				
PSQI Total Score	--	✓	--	--
Sleep Continuity				
SOL (minutes)	✓	--	✓	--
WASO (minutes)	✓	--	✓	--
S.E. (%)	✓	--	✓	--
TST (minutes)	✓	--	✓	--
Number of Arousals	--	--	✓	--
Sleep Quality				
Quality of Sleep (0-4)	✓	--	--	--
Restedness (0-4)	✓	--	--	--
Alertness (0-4)	✓	--	--	--
Stage 1 & 2 (%)	--	--	✓	--
Stage 3 & 4 (%)	--	--	✓	--
REM (%)	--	--	✓	--
Sleep related Cognitions				
Q1: Total Sleep Behaviour	--	✓	--	--
Q2: Glasgow Sleep Effort	--	✓	--	--
Q3a: Physical Tension	--	✓	--	--
Q3b: Sleep Pattern Problem	--	✓	--	--
Q3c: Cognitive Arousal	--	✓	--	--
Q3d: Sleep Effort	--	✓	--	--
Q4a: Problem Solving	--	✓	--	--
Q4b: Sleep & Wakefulness	--	✓	--	--
Q4c: Somatic & Sensory Engagement	--	✓	--	--
Q5a: Cognitive Arousal	--	✓	--	--
Q5b: Somatic Arousal	--	✓	--	--
Q6a: Immediate Consequence of Insomnia	--	✓	--	--
Q6b: Long-term Consequence of Insomnia	--	✓	--	--
Q6c: Need for Control over Insomnia	--	✓	--	--
Adherence with the QHR				
Adherence score at SOL (0-10)	✓	--	--	✓
Adherence score at WASO (0-10)	✓	--	--	✓

Separate analyses were conducted for sleep data gathered subjectively (on the basis of participants' responses to a nightly sleep diary) and objectively (on the basis of PSG recordings). The sleep continuity dependent variables (DVs), sleep quality DVs, battery of questionnaires DVs, adherence with the QHR DVs and whether they were objectively or subjectively collected are listed in table 10.

5.3.2 Participants

Eighty-three individuals were screened. They were recruited via GP practices, a sleep clinic at the Southern General Hospital and via a media advertisement. People who responded to the media advertisement were invited to speak to their GP and ask to be referred. Participants' insomnia was assessed according to DSM-IV (APA, 1994) and ICSD-R (ASDA, 1997) diagnostic guidelines first during a telephone interview and then during a face-to-face interview.

Inclusion and exclusion criteria were identical to those applied in the preliminary study (study two, section 4.3.2). Thirty-nine possible participants were excluded, after assessment, due to the following: aged over 75 (n=4), drug use (n=1), alcohol use (n=5), suspected sleep apnoea (n=9), suspected PLMS/RLS (n=4), pain (n=4), shift-work (n=2), suspected sleep terrors (n=1), PTSD (n=2), physical illnesses (n=5) and nightmares (n=2).

A cohort of 44 participants entered the study, however, one participant withdrew from the study after ten baseline days due to personal circumstances. The first PSG night confirmed that the 44 individuals included in the study did not present with sleep disorders other than insomnia.

Six participants completed the study for all phases but the last PSG recordings (5 participants informed the researcher that they could not undergo the two PSG nights

due to family circumstances, one was deemed to be distressed on the night of the recording and, therefore, the researcher decided not to perform PSG assessment). In addition, one participant did not complete the diary during the treatment phase and one participant's diary was lost in the post. Finally, four participants did not return the end of treatment battery of questionnaires.

In summary baseline and treatment diary data and PSQI scores were gathered from 41 participants, baseline and end of treatment PSG data from 37 participants, complete sleep continuity and sleep quality data (diary and PSG) were obtained from 35 participants. Baseline and end of treatment questionnaires were available for 39 participants (flow chart as per Consort guidelines in fig. 25).

Demographic and clinical characteristics of participants in each group are summarised in table 11. In brief, participants' mean age was 47.5 years, there were 29 females and 15 males, 25 were living with a partner and 19 were living alone, 28 were in full employment, 4 were in full time education and 12 were retired. They were all educated at college level or above.

5.3.3 Materials and Apparatus

5.3.3.1 Screening Interview

The screening interview was identical to that used in study two (section 4.3.3.2b). Although reliance on self-report for screening out sleep disorders other than insomnia is prone to error (Hoffestein and Szalai, 1993) it provides a cost-effective way to initially screen each potential participant reducing, therefore, the number of (costly) PSGs to be carried out.

5.3.3.2 Subjective Measurements of Sleep and Sleep Related Questionnaires

5.3.3.2a Sleep Diary

The sleep diary was the same as that used in study two (section 4.3.3.1 and appendix 5). As in study two the variables retained from the sleep diaries were SOL, WASO, S.E. and, in addition, TST.

5.3.3.2b Sleep Related Questionnaires

As in study two the **PSQI** (Buysse, et al, 1989) and the **HADS** (Zigmond and Snaith, 1983) were administered (sections 4.3.3.2a and 4.3.3.2c and appendixes 4 and 6 respectively).

In addition psychological measures associated with sleep were collected by means of a battery comprising six, well validated, sleep related questionnaires that focus on symptoms, interpretation, cognitions and behaviour that may accompany sleep complaints.

Sleep Behaviour Self Rating Scale - Revised (SBSRS-R Kohn and Espie, 2005) is an experimental measure of the behavioural pattern around bedtime and it is a revised form of the original one produced in 1979 (Kazarian, Howe and Csapo, 1979). The revised version comprises 14 statements (appendix 14) rated on a 5 point Likert scale ('Never', 'Rarely', 'Sometimes', 'Often', 'Very Often'). The total score ranges from 14 to 70. It was developed to assess sleep incompatible behaviour associated with a person's bed/bedroom and it is the only published scale to quantify the stimulus control paradigm. Test-retest correlation was r=0.88 indicating acceptable reliability; internal consistency and discriminant validity were also acceptable (Cronbach alpha=0.69, K-R 20 =0.70).

The Glasgow Sleep Effort Scale (GSES, Broomfield and Espie, 2005) is composed of 7 statements measured on a 3-point Likert scale ('Very Much', 'To Some Extent', 'Not at all') and the total score ranges from 0 to 14 (appendix 15). It measures elements of effortful preoccupation with sleep (e.g. worrying over sleep and failure to sleep, need to control one's sleep). It has acceptable internal consistency (Cronbach alpha=0.77).

Sleep Disturbance Questionnaire (SDQ, Espie et al, 1989, 2000) comprises 12 statements (appendix 16). Using a 5 point Likert scale ('Never True', 'Seldom True', 'Sometimes True', 'Often True', 'Very often True') participants had to indicate what factors they perceived to be the sources of their sleep problem (e.g. 'I get too "worked up" at not sleeping'). This questionnaire differentiates four sleep interfering factors. In particular items 1, 5 and 9 give a total score for 'physical tension', items 3, 7 and 11 for 'sleep pattern problems', items 2, 6 and 10 for 'cognitive arousal' and items 4, 8 and 12 for 'sleep effort'. Each of these 4 factors' total score ranges from 3 to 15. It has good internal consistency (Cronbach alpha=0.82).

Glasgow Content of Thoughts Inventory (GCTI, Harvey and Espie, 2004) comprises 25 items scored on a 4 point Likert scale ('Never', 'Sometimes,', 'Often' and 'Always') (appendix 17). It measures the types of thoughts people have while trying to get to sleep and gathers a score for three different constructs: 'cognitive intrusions relating to active problem solving' (items 1, 3, 8, 12, 14, 15, 19, 21 and 23 with a total score range from 9 to 36), 'cognitive intrusions relating to sleep and wakefulness' (items 5, 6, 7, 9, 11, 18, 22, 24 and 25 with a total score range from 9 to 36) and 'cognitive intrusions relating to somatic and sensory engagement' (items 2, 4, 10, 13, 17 and 20 with a total score range from 7 to 28). Internal consistency is

good (Cronbach alpha=0.87) and test-retest reliability using the intra-class correlation coefficient was found to be highly satisfactory (ICC=0.87, p<0.001)

Pre-Sleep Arousal Scale (PSAS, Nicassio et al, 1985). The PSAS is a sixteen item questionnaire using a 5-point rating scale (not at all, slightly, moderately, a lot, extremely) assessing the intensity of pre-sleep somatic and cognitive aspects of arousal (appendix 18). Higher scores indicate greater arousal. Eight items assess cognitive arousal (e.g. 'thoughts keep running through your head') and eight assess somatic arousal (e.g. ' a tight tense feeling in your muscles') that can be experienced in the pre-sleep period. Scores of the two sub-scales (each scale ranges from 8 to 40) are computed separately by summing across cognitive and somatic arousal items. This scale is a helpful screening measure to discriminate poor from good sleepers. Somatic and cognitive sub-scales were shown to be internally consistent and stable over time (Cronbach alphas ranged from 0.68 on the cognitive subscale to 0.88 on the somatic subscale; 3 week test-retest correlations ranged from 0.72 on the cognitive to 0.76 on the somatic subscales). Mean scores of people with insomnia were significantly higher than those of normal sleepers on scores for both subscales. On the cognitive subscales means for those with and without insomnia were M=15.2(5.3) and M=11.34(3.4), respectively.

Dysfunctional Beliefs and Attitudes about Sleep -10 (DBAS-10, Espie, Inglis, Harvey and Tessier, 2000) is a 10 item scale rated on a visual analogue scale concerning sleep-related beliefs (e.g. needing 8 hours of sleep per night) (appendix 19). It is based on the original 30 item scale (Morin et al, 1993) but limited to items shown to be sensitive to change after CBT. It has acceptable internal consistency (Cronbach alpha=0.69). It measures the types of beliefs people hold regarding

insomnia and yields a score for three different constructs: 'beliefs about the immediate negative consequences of insomnia' (items 1, 2, 6, 7 and 9 with a total score ranging from 0 to 50), 'beliefs about the long term negative consequences of insomnia' (items 3, 5 and 8 with a total score ranging from 0 to 30) and 'beliefs about need for control over insomnia (items 4 and 10 with a total score ranging from 0 to 20). Its validity has been recently supported (Edinger, Wohlgemuth et al, 2001).

Credibility/Expectancy Questionnaire (modified). The original questionnaire was developed by Devilly and Borkovec (2000) and comprised six items using a 9-point rating scale (not at all, .., very). It measures rationale credibility (items 1, 2, 3 and 5) and treatment expectancy (items 4 and 6). The credibility factor was found to possess high internal consistency (Cronbach alphas ranged from 0.81 to 0.86), inter-item correlations across studies ranged between 0.62 and 0.78 and test-retest reliability was 0.75. In the present study only the four items of the credibility subscale were used. Furthermore the words' 'trauma symptoms' were changed to 'sleep difficulties'. In the present study the four items were measured on a 4-point scale (not at all, somewhat, quite, very) (appendix 20).

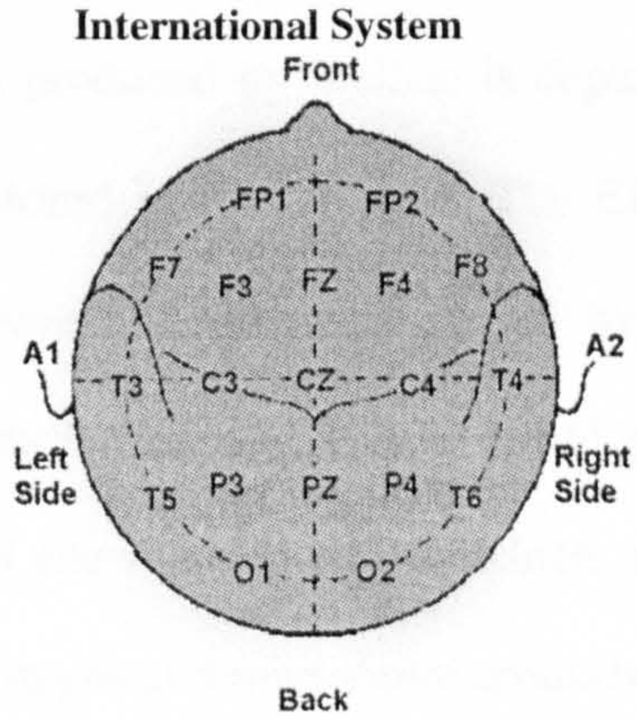
5.3.3.3 Objectively Measured Sleep

Data for objective sleep measures were obtained using PSG. These included data obtained from EEG, EOG and EMG (section 5.3.3.3a). The objective measures of sleep were acquired via an ambulatory recording device (section 5.3.3.3b) and downloaded to a proprietary software package (section 5.3.3.3c). They were then manually scored according to R&K manual (section 5.3.3.3c).

5.3.3.3a Objectively Measured Sleep

EEG was recorded at 4 scalp sites (C3, C4, O1, O2) according to the 10-20 international electrode system (Jasper, 1958) (fig. 20) using an ambulatory EEG/polygraphy recorder referenced to Cz.

Fig. 20 - The 10-20 Electrode Placement International System



EOG was recorded from 2 electrodes placed at the outer canthi of both eyes (LOC, ROC). System reference (the mean amplitude between Cz and the recording sites) was used for recording. Neutral was taken from Fz. The data were later re-referenced off line to the opposite mastoid bone (A1, A2).

The montage, therefore, included 2 EOG referenced to the left and right mastoid (LOC/A2 and ROC/A1), 4 EEG referenced to both mastoids (C3/A2, C4/A1, O1/A2, O2/A1) and a bipolar mentalis EMG. EOGs and EEGs were high pass filter at 0.3 Hz and low pass filtered at 70Hz. EMGs were high pass filtered at 2 Hz and low pass filtered at 120 HZ. Electrode impedance was kept below 5kΩ.

The researcher had been trained in correct electrode attachment and electrode removal by Mrs Popadopoulos the chief EEG technician of the department of Neurology at the Southern General Hospital in Glasgow.

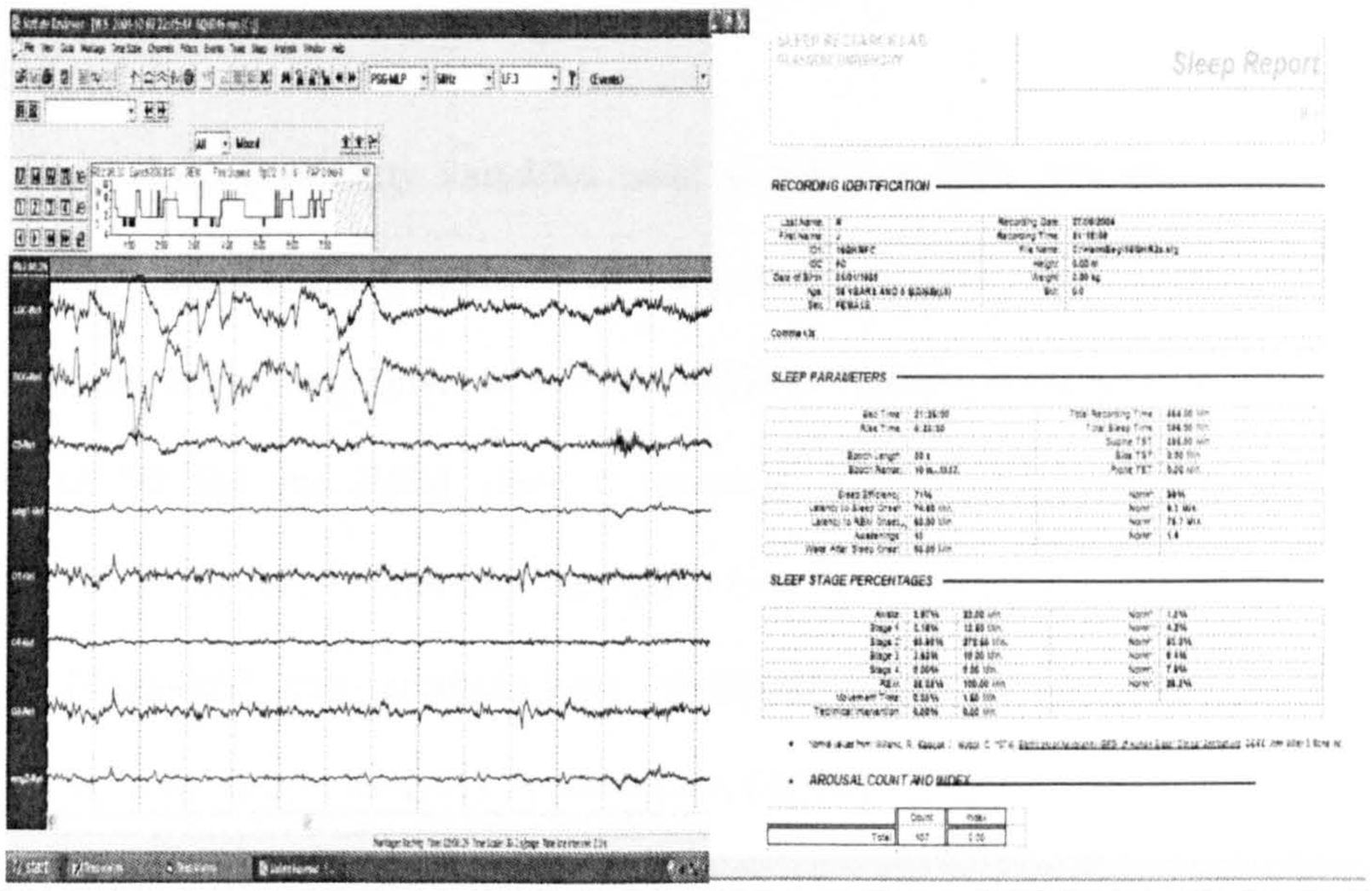
5.3.3.3b Ambulatory Electroencephalogram/Polygraph Recording Device

The ambulatory EEG/polygraphy recording device was, the Trackit (Lifelines, Ltd), the same as that employed in study two (4.3.3.3a).

5.3.3.3c Analysis of the Polygraphic Recording

Stellate Sleep System (Stellate Ltd, Canada) is a software package enabling the acquisition and storage of EEG data in a central database. It offers tools facilitating manual scoring of the recorded EEG (e.g. spindle detector tool, automatised division of the EDF file in 30 second epochs). An example of the sleep hypnogram and sleep report produced by Stellate is depicted in figure 21. The EEG acquired via Trackit was stored in EDF format. The EDF file was, subsequently, uploaded in Stellate Reviewer and manually scored by an expert PSG technician according to R&K (Rechtschaffen and Kales, 1968) guidelines. Thereafter, Stellate summarised the scored file in sleep reports which contain, among others, SOL, WASO, TST, S.E., sleep stages and micro/mini arousals data.

Fig. 21 - An example of a PSG scored record and relative sleep report. The PSG recording is manually scored and staging for each epoch is memorised in Stellate and thereafter a Sleep Report is generated by the Stellate Software



5.3.3.3d Sleep Scoring

The PSG recordings were blind scored in 30-second epochs from lights off to lights on according to R&K's (Rechtschaffen and Kales, 1968) standardised criteria by Ms Manon Lamy (Sleep Laboratory Coordinator, Universite' Laval, QC). Lights off and lights on were extracted by the 'eventit' feature of Trackit: basically participants pressed a button on Trackit when they switched off the light and in the morning when they got up. The time of button pressing was recorded by Trackit and retrieved by the experimenter at a later stage. The C3, EMG and EOG areas were used for scoring. However, for correct detection of the waking stage (EEG alpha activities), the O2 area was used as well. If C3 and/or O2 recordings were difficult to score due to artifact contaminations, C4 and/or O1 were used as alternatives. TIB was calculated from lights off to lights on. EEG defined sleep onset was scored as the occurrence of 3 consecutives epochs of stage 1, or 1 epoch of stage 2, 3, 4, or REM.

Thus sleep continuity variables were as follows: SOL was the amount of time elapsed in minutes from lights off to sleep onset, WASO was the sum of wake time in minutes from sleep onset to the last REM or NREM sleep episode, TST was the sum of NREM and REM sleep in minutes. The epochs contaminated by body movements for longer than half the epoch (i.e. 16-30 sec) were scored as movement time. Micro-and mini- arousals were computed visually according to the criteria of the American Sleep Disorders Associations (ASDA, 1992).

5.3.3.4 Subjective Measurement of Adherence

As in study two, adherence to the QHR was measured daily. In particular measurement of adherence to the QHRout diary was identical to that employed in the pilot study and gathered information such as the need to apply the QHR and whether it was applied or not (section 4.3.3.6 and appendix 7). The only difference between the adherence diary for the QHRout and QHRin (appendix 21) concerned the location where the rule was to be applied (e.g. 'Did you go to another room?' and 'Did you sit up in bed?' respectively).

5.3.3.5 Objective Measurement of Adherence

In the next two subsections actigraphy and the algorithm devised, by the researcher, to obtain an adherence score for objectively measured adherence are presented.

5.3.3.5a Actigraphy

The Actiwatch-Light (ACTW-L - Cambridge Neurotechnology Ltd) is similar to the Actiwatch used in study two (section 4.3.3.4 and figure 17). However, as shown on the left (fig. 22), the event button is replaced by a light detector. In this way in addition to collecting movement information, the ACTW-L measures light intensity via a lux sensor.

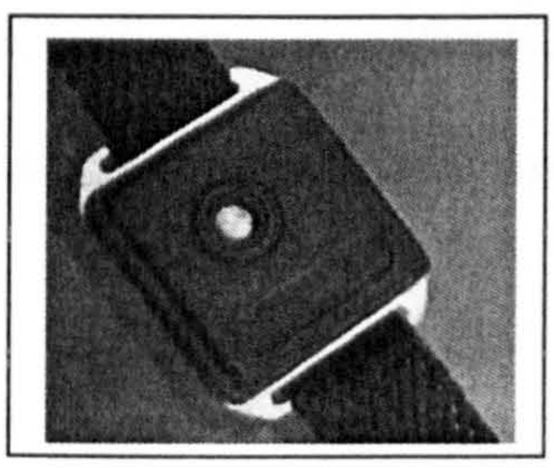


Fig. 22: ACTW-L

Data collected using the ACTW-L were analysed via a software package produced by Cambridge Neurotechnology Ltd (Actiwatch sleep 2002). As shown in

figure 23, this software produces an automated measure of sleep continuity and light during the recorded period. A string of numbers represents the amount of movement and the amount of lux (light) for each one-minute- epoch.

Fig. 23 – On the left side the Sleep analysis of the data gathered with ACTW-L is presented: the black lines represent movement and the yellow lines represent light. On the left side the epoch by epoch output with activity and light is presented.

1QHR			1QHR		
Thu 27-May-2004			Fri 28-May-2004		
Time	Movemen	Light	Time	Movemen	Light
00:00:00	0	0	00:00:00	13	0
00:01:00	0	0	00:01:00	50	1.09
00:02:00	0	0	00:02:00	11	0
00:03:00	0	0	00:03:00	80	0
00:04:00	0	0	00:04:00	92	0
00:05:00	0	0	00:05:00	122	0
00:06:00	0	0	00:06:00	43	0
00:07:00	0	0	00:07:00	3	0
00:08:00	0	0	00:08:00	75	0
00:09:00	0	0	00:09:00	122	0
00:10:00	0	0	00:10:00	3	19.31
00:11:00	0	0	00:11:00	0	8.9
00:12:00	0	0	00:12:00	0	4.56
00:13:00	0	0	00:13:00	88	8.9
00:14:00	0	0	00:14:00	550	4.56
00:15:00	0	0	00:15:00	806	8.9
00:16:00	0	0	00:16:00	181	25.19
00:17:00	0	0	00:17:00	453	31.47
00:18:00	0	0	00:18:00	516	4.56
00:19:00	0	0	00:19:00	206	4.56
00:20:00	0	0	00:20:00	200	75.99
00:21:00	0	0	00:21:00	103	111.38
00:22:00	0	0	00:22:00	221	75.99
00:23:00	0	0	00:23:00	483	8.9
00:24:00	0	0	00:24:00	55	8.9
00:25:00	0	0	00:25:00	162	31.47
00:26:00	0	0	00:26:00	162	84.43
00:27:00	0	0	00:27:00	412	25.19
00:28:00	0	0	00:28:00	550	8.9
00:29:00	0	0	00:29:00	516	1.09
00:30:00	0	0	00:30:00	168	8.9
00:31:00	0	0	00:31:00	0	38.1
00:32:00	0	0	00:32:00	0	45.07
00:33:00	0	0	00:33:00	361	52.35
00:34:00	0	0	00:34:00	328	31.47
00:35:00	0	0	00:35:00	252	8.9
00:36:00	0	0	00:36:00	14	4.56
00:37:00	0	0	00:37:00	26	4.56
00:38:00	0	0	00:38:00	43	4.56
00:39:00	0	0	00:39:00	0	4.56
00:40:00	0	0	00:40:00	31	1.09
00:41:00	0	0	00:41:00	55	1.09
00:42:00	0	0	00:42:00	23	1.09
00:43:00	0	0	00:43:00	28	1.09
00:44:00	0	0	00:44:00	228	4.56

5.3.3.5b - Adherence Algorithm and Adherence Score

Given the interest in adherence to the rule during the night, only the data gathered during the period from 'switching off the light with the intention to sleep' – to 'rising from bed' was considered. As depicted in fig. 23, Actiwatch Sleep produces 3 columns for each epoch: one with time, one with movement data and one with light data.

A rule-based algorithm was devised comprising binary digit notation to yield an index of movement and an index of light. These two indices were then linked to provide an overall adherence score. Specifically, each sixty-second epoch of the night was examined for movement and light sufficient to read (lux>6). In order to determine adherence with the QHR the following steps were applied:

1. Three columns (M=movement, L=light and A=adherence) were created adjacently to those reporting movement and light values.
2. In column M all epochs were given a score (0= no movement; 1= movement).
3. If one or two epochs of no movement (M=0) were observed between two epochs of movement (M=1), they were considered as movement and given a score of 1. In this way a column with a mix of zeros and ones was produced for movement.
4. In column L all light epochs were given a score (0= not enough light to read i.e. lux≤6; 1= enough light to read i.e. lux>6).
5. For each period of more than 15 consecutive movement epoch (M=1 for 15 consecutive epochs), the light scores (column L) were examined. If following movement only epochs (i.e. M=1, L=0) light was also detected (i.e. M=1 and L=1) then adherence with the QHR was successful; if no light was detected (i.e. M=1 and L=0) then the QHR was not implemented (i.e. no adherence).

This rule based algorithm permitted the researcher to count objectively the number of times the behavioural intervention 'QHR' should have been implemented, the number of times the QHR was actually adhered to and the length of time elapsed before the instruction was implemented.

As in study two the adherence score was calculated using the following formula:

$$\text{Adherence Score} = \frac{\text{Number of Times QHR was adhered to}}{\text{Number of Times Implementation was Required}}$$

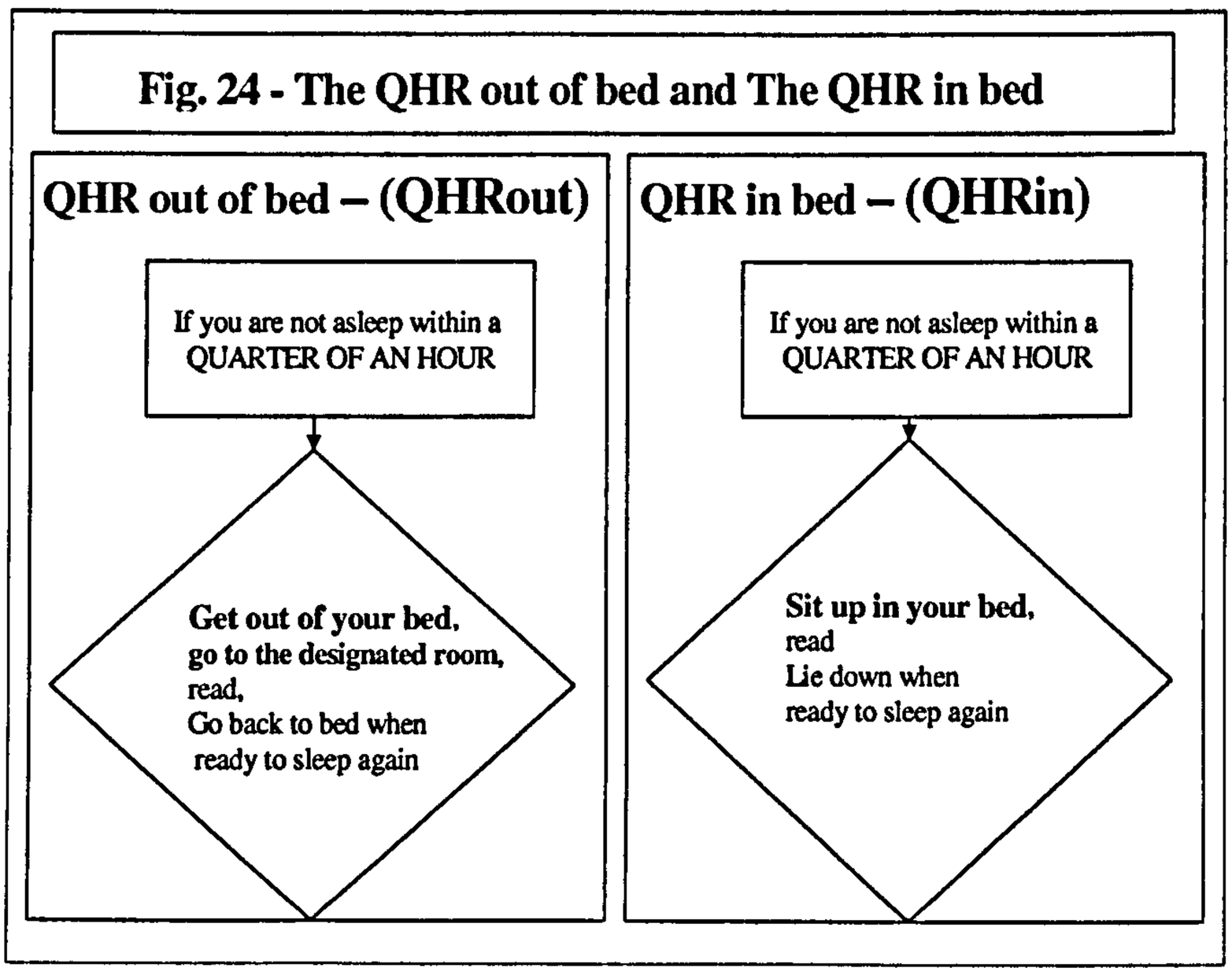
Similarly to Riedel, Lichstein, Peterson, Epperson, Means and Aguillard' (1998) study if a participant fell asleep within a quarter of an hour (i.e. no need to apply the QHR) a score of 100% was given.

5.3.3.6 Material for the intervention

5.3.3.6a The Single Component Interventions: QHR out of bed & QHR in bed

The Single Component Intervention, named 'the quarter of an hour rule' (QHR), was adapted from the situational component of stimulus control intervention (Bootzin, 1972) 'if you are not asleep within reasonable time get up and do something in another room until you feel sleepy. Repeat the instruction as many times as it is necessary.' In order to test the hypotheses, it varied regarding the location where it had to be applied (fig. 24).

Fig. 24 - The QHR out of bed and The QHR in bed



As shown in figure 24, if not asleep within a quarter of an hour then the QHRout asked participants to 'get out of bed, go to the designated room and read. Return to bed when you feel ready to sleep'. The QHRin, in contrast, asked participants to 'sit up in bed and read. Lie down when you are ready to sleep again'. The QHR

interventions were delivered individually by the researcher and, in order to enhance treatment fidelity, structured written protocols were followed. In this way it was ensured that delivery of the QHRout and QHRin interventions were consistent across participants (appendixes 22a and 22b respectively). A flow-chart type instruction sheet was handed to participants to help them follow the rule (appendixes 23a and 23b respectively).

5.3.3.6b Self-monitoring: the Control Group

The control group self monitored their sleep by completing the sleep diary for the whole duration of the study (five weeks). In order to ensure that outcomes were due to specific therapeutic ingredients rather than the measurement process (sleep self-monitoring) or other non- specific factors (e.g. patient's expectations, therapist's attention) the number and length of contacts between the control group and the researcher were identical to those with participants randomised to the active intervention groups. Importantly, the control group was not given sleep hygiene instructions or any other insomnia treatment: during contacts the researcher asked general questions such as "how was your sleep yesterday?" or "did you remember to fill in the sleep diary?".

5.3.4 - Data Processing

Preliminary calculations (e.g. TIB, S.E., global adherence score) were performed by inputting raw data in a spreadsheet (Microsoft Excel) and results were then used for statistical analyses. All data analyses (parametric and non-parametric tests of

differences and correlations) were performed employing the statistical software package SPSS 11.5 for Windows (Microsoft).

In the mixed model ANOVAs, 'Group' (QHRin vs. QHRout vs. Control) was the between subjects factor and time of measurement 'Time' (Baseline vs. End of Treatment) was the repeated measure. The alpha level was set at 0.05 (Sterling, Rosenbaum and Weinkam, 1995) and a p-value between 0.051 and 1.0 was considered as a non-significant trend. In instances where non-significant results were obtained, the test power to detect significant difference was inspected and reported. A statistically significant main effect for 'Group' was followed by pairwise Bonferroni corrected comparisons (p=0.05/3=0.017). Likewise a significant 'Group' by 'Time' interaction was followed by pairwise Bonferroni corrected comparisons (p= 0.05/6= 0.008) to examine the time effect within each group individually and the differences between groups at end of treatment.

Bonferroni corrections for each set of analyses were employed to control for type I error. It is appreciated that the Bonferroni correction is conservative and, therefore, while controlling for type I error it inflates type II error. A less conservative post-hoc test, such as the Tukey HSD, would be more appropriate to balance type I and type II errors. However, this test assumes that each group has an equal number of cases (Urdan, 2001). The present study employed unequal sample size groups and, therefore, the Tukey HSD was not deemed to be the best post-hoc test to use. Fisher LSD, a post-hoc test less conservative and indeed often criticised for not sufficiently controlling for Type I error, also requires equal size samples (Keppel and Wickens, 2004). It was decided, therefore, also to report uncorrected t-test values for those

pairwise comparisons that had obtained a $p \leq 0.05$ value but which were non-significant once Bonferroni correction was applied.

Cohen's effect sizes (Cohen, 1988) were calculated using the formula (for an explanation of effect sizes see section 1.6.1):

$$ES = \frac{M_1 - M_2}{\sqrt{(SD_1^2 + SD_2^2)/2}}$$

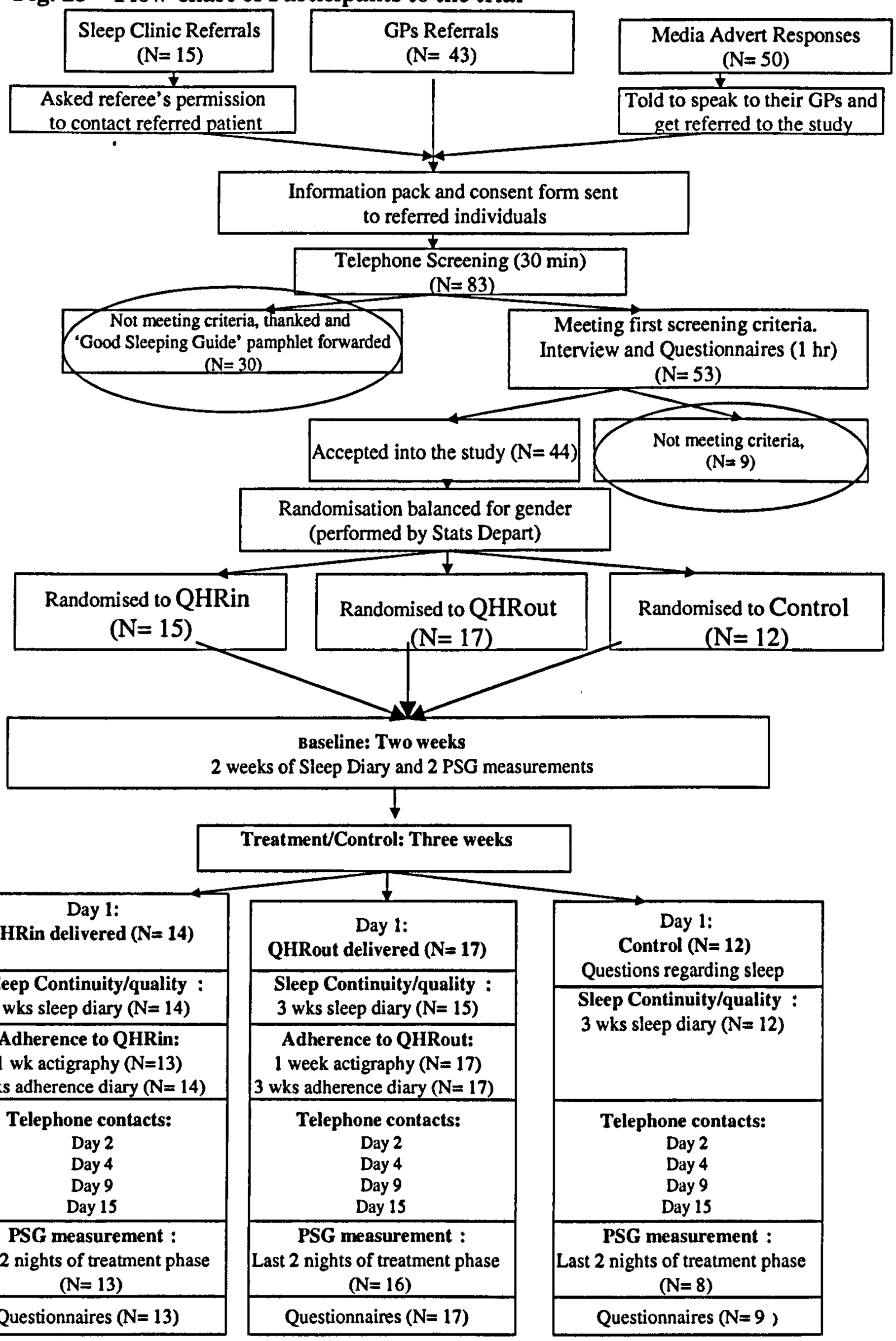
M_1 (SD_1)= Mean (SD) $Group_1$
M_2 (SD_2)= Mean (SD) $Group_2$

5.3.5 – Procedure

As depicted in figure 25 participant recruitment utilised three avenues: General Practioners' (GPs) referral, sleep clinic referred patients and media advertisement. The researcher contacted, by letter, 42 surgeries in Greater Glasgow explaining the study and inviting GPs to refer suitable patients (appendix 24). This first contact was followed by a telephone contact within one month and, if requested, a visit to the surgery. The surgeries to be contacted were decided on the basis of number of GPs per surgery (N>2). GPs referred possible participants on the basis of a quick 'fitting criteria for the study' checklist (appendix 25). With regard to recruitment via sleep clinic referrals, a letter asking permission to contact the referred patient was sent to the referrer (appendix 26). Individuals who responded to a media advertisement were asked to speak to their GP with a view to being referred to the study. There are no available data on number of people who responded to the advertisement and, when prompted to do so, asked their GPs to be referred to the study.

Referred individuals were sent an information pack (appendix 27) by post and were asked to reply if interested in the study. Eighty-three individuals agreed to be contacted.

Fig. 25 – Flow-chart of Participants to the trial



The telephone screening interview lasted around thirty minutes. Those presenting with suspected sleep disorders other than primary insomnia or not meeting other criteria (e.g. age, medication used) were thanked for their interest, the 'good sleep guide' (appendix 28) was forwarded to them but they did not participate further in the study.

Individuals who, as per preliminary assessment, seemed to fit criteria were asked to come to the sleep research laboratory for an in depth face to face interview who lasted around one hour. Those meeting DSM-IV/ICSD-R diagnostic criteria for primary/psychophysiologic insomnia were advised of the study procedure in detail. If they agreed to participate, they were, once again, informed of their right to withdraw at any time without having to explain their decision and were asked to read and sign a consent form (appendix 29). They were reminded that, as stated in the information leaflet, they might not be assigned to a treatment group and that they would be informed by phone of whether they had been randomised to a treatment or control group. Participants, then, completed the battery of questionnaires and the PSQI and the date to start the baseline phase, commencing with two home PSG recordings, was agreed. The first night was an adaptation night, as routinely conducted in PSG studies (e.g. Bonnet and Arand, 1997), it provided objective screening information but the data collected were not utilised for outcome analyses.

It is important to highlight that the researcher followed a 'safety procedure' during home visits (appendix 30). This was a revised form of the home visit procedure in place at that time at the Section of Psychological Medicine at the University of Glasgow.

On the first PSG night, the researcher went to the participant's house at the agreed time, explained how to fill in the sleep diaries, how the electrodes were going to be attached and removed and what to do with the ambulatory PSG recorder at bedtime and in the morning. Participants were provided with a mobile number to contact the researcher during the night should they have any question or problem. Thereafter the PSG was set up for the nightly measurement. This procedure lasted around 1½ hours. The following morning the researcher returned to the participant's house to remove electrodes. This contact lasted around half an hour. The second night and following morning procedures were identical to those of the first night and the associated morning.

After the baseline period (2 weeks) the researcher met again with the participant. If the participant had been assigned to either the QHRout or the QHRin then the appropriate intervention was delivered and the researcher checked that the participant had clearly understood how to implement the intervention. An easy to follow QHR memo was provided (appendices 8 and 23). Use of Actiwatch and adherence diary (appendices 7 and 21) was explained and the participant was reminded to keep filling the sleep diary in. In order to avoid inflating adherence by making the participant aware of the purpose of actigraphy, they were told that the actiwatch monitored movement and rest and could give an indication of rest-wake patterns during day and night. This meeting lasted around 45 minutes and the delivery of the intervention took no more than 30 minutes.

If the participant had been assigned to the control group the researcher met with them, collected the 2-week baseline diaries and asked a few general questions

regarding their sleep. They were then reminded to keep filling the sleep diary in for the next three weeks.

Four, 5-10 minute, telephone contacts were made during the treatment period so as to ensure that the QHR was implemented correctly each night. The participants in the control group were also contacted four times, and the telephone conversation regarded correct completion of the sleep diary. Participants were contacted on the 2^{nd}, 4^{th}, 9^{th} and 15^{th} day of the intervention period (fig. 25).

During the last two days of the treatment period PSG recordings were repeated. Participants were asked to fill in the PSQI and the battery of questionnaires on the morning after the last PSG recording. In instances where participants had to go to work early and did not have time to fill in the questionnaires, they completed them later in the day and posted them by the next morning.

As already discussed in study 2 length of treatment phase was decided based on previous findings that sleep improvements were observed within two to three weeks after stimulus control implementation (e.g. Bootzin, 1972; Espie, Brooks et al, 1989; Sloan et al, 1993; Zwart and Lisman, 1979).

5.4 Results

This section comprises descriptive and inferential statistics, for subjective and objective data. First, participants' clinical and demographic characteristics are reported. Second, the results of analyses carried out on the subjective sleep continuity, sleep quality, clinical significance and sleep related cognitions questionnaires are provided. Third, the results of statistical analyses on the objectively gathered sleep quantity and sleep architecture data are reported. Fourth,

results of the comparison of the subjective and objective data are given. Finally, the results of the subjective and objective measurements of adherence and their associations to sleep parameters are presented.

5.4.1 – Participants' Characteristics

Table 11 presents, for each of the 3 groups, information regarding the number of participants, their gender, if they were living with a partner or alone, their employment status and the type of insomnia they presented with [mixed, sleep onset (SOL) or sleep maintenance (WASO)]. All participants were educated at college level or above. In addition mean (SD) age of participants, PSQI total score, length of the insomnia complaint, HADS anxiety and depression scores are reported.

Table 11: Summary Data for participants randomised to the QHRin, QHRout and Control (SD in brackets)

	QHRin (n=15)	QHRout (n=17)	Control (n=12)
Sociodemographic			
Age (yrs) Mean (SD)	48.3 (14.7)	45.5 (14.1)	48.9 (14.9)
Age Range	21-70	18-64	24-72
Gender (number of f/m)	11/4	11/6	8/4
Living with Partner/Alone	6/9	11/6	8/4
Employment Status			
Full Time/Student/Retired	10/1/4	12/2/3	6/1/5
Sleep			
Mean (SD) PSQI total score	12.8 (1.4)	13.4 (1.9)	12.0 (2.6)
Mean (SD) Insomnia Duration (years)	11.5 (7.6)	10.2 (7.8)	10.9 (9.2)
Insomnia Type: Mixed/SOL/ WASO	11/1/5	10/2/5	6/0/6
Psychological			
Mean (SD) HADS Anxiety	8.1 (2.8)	9.5 (3.7)	7.5 (4.0)
Mean (SD) HADS Depression	4.6 (2.5)	5.5 (2.9)	5.7 (3.7)

One-way ANOVA revealed that there were no statistically significant between groups differences in age [$F(2,41)=0.24$, n.s.], duration of insomnia [$F(2,41)=0.11$, n.s.], PSQI total score [$F(2,41)=1.79$, n.s.], HADS anxiety [$F(2,41)=1.22$, n.s.] or HADS depression [$F(2,41)=0.50$, n.s.] (all $p_s>0.1$).

5.4.2 - Subjective Sleep Continuity Data and PSQI total Score

In this section sleep continuity parameters (SOL, WASO, TST and S.E.) gathered via sleep diary and the PSQI total score are reported descriptively and then formally analysed. Before analysing each of these variables some points common to all variables are addressed.

Diary entries for both baseline and intervention periods were completed by 41 participants. The baseline mean values correspond to the mean of the two-week baseline daily data for each participant. In order to assess that there were no differences between the mean values of week 1 and those of week 2, mixed ANOVAs were carried out for each of the sleep continuity variables and no statistically significant differences were found (all $p_s>0.1$). The end of treatment mean values correspond to the mean values of the daily data for the third week of treatment. Inspection of sleep diary data suggested that they were not normally distributed and, indeed, the Shapiro-Wilk test of normality confirmed this. Therefore, logarithmic transformations (base 10) were performed on SOL, WASO, TST and S.E. data. For clarity, however, the reported means in tables and figures correspond to non-transformed data.

One-way ANOVAs were performed on the baseline mean SOL, WASO, TST and S.E. values and the mean PSQI total score (table 12) to examine if there were

statistically significant differences in sleep continuity parameters among the groups prior to the treatment phase. No significant differences were found for SOL [F(2,38)=0.12, n.s.], WASO [F(2,38)=2.60, n.s.], TST [F(2,38)=0.58, n.s.], S.E. [F(2,38)=0.36, n.s.] or PSQI [F(2,38)= 2.56, n.s] (all p_s>.1).

As already discussed in section 5.3.4, if the mixed ANOVAs showed significant main effects or interactions then pairwise t-tests, with Bonferroni correction, were carried out to assess where the differences were located. In particular, as the one-way ANOVAs showed no significant differences between the 3 groups at baseline, independent t-tests were carried out only on the end of treatment mean values. Paired t-tests (baseline vs. end of treatment) were applied for within subjects differences.

Prior to reporting descriptive and inferential analyses it is important to provide data regarding the credibility of treatment.

5.4.2.1 Credibility of the QHR Treatment

Credibility questionnaires were completed by 13 participants in each of the 2 treatment groups (QHRin and QHRout) to rule out the possibility that outcomes were influenced by a treatment being more plausible than the other. The mean credibility score for the QHRin was 2.88 (0.55) and for the QHRout was 2.98 (0.39). An independent t-test showed that the two groups' credibility scores were not statistically different [t(26)= -0.55, n.s.).

5.4.2.2 Descriptive Sleep Continuity Data gathered via Sleep Diary

Table 12 presents mean (SD) data for sleep continuity variables (SOL, WASO, TST and S.E.) and PSQI at baseline and the third week of treatment for the QHRin,

QHRout and Control groups. In order to assess if the differences, suggested by inspection of table 12, between and within groups were statistically significant, inferential statistics were computed. The next sub-sections report the results for each of the sleep continuity variables examined (ANOVAs results in appendix 33).

Table 12: Mean and SD for Subjective Sleep Continuity Variables and PSQI at Baseline and End of Treatment for the QHRin, QHRout and Control Groups

	QHRin (N=14)	QHRout (N=15)	Control (N=12)
SOL Baseline	61.5 (50.4)	58.2 (57.1)*	49.1 (31.0)
SOL End Treatment	**51.9 (58.7)***	**30.1 (34.3)***	**64.1 (52.7)**
WASO Baseline	113.5 (89.5)	98.4 (59.2)*	111.54 (57.3)
WASO End Treatment	**63.5 (54.1)**	**45.7 (36.9)***	**113.0 (67.1)**
TST Baseline	314.6 (94.7)	342.6 (73.1)	321.3 (78.9)
TST End Treatment	**383.0 (76.7)**	**408.7 (36.7)**	**324.1 (72.7)***
S.E. Baseline	0.64 (0.18)	0.69 (0.14)	0.66 (0.15)
S.E. End Treatment	**0.77 (0.16)**	**0.83 (0.09)***	**0.65 (0.13)**
PSQI (SD) Baseline	12.6 (1.3)	13.7 (1.8)	12.0 (2.7)
PSQI (SD) End of Treatment	**8.8 (2.9)**	**8.1 (2.9)**	**12.0 (2.1)**

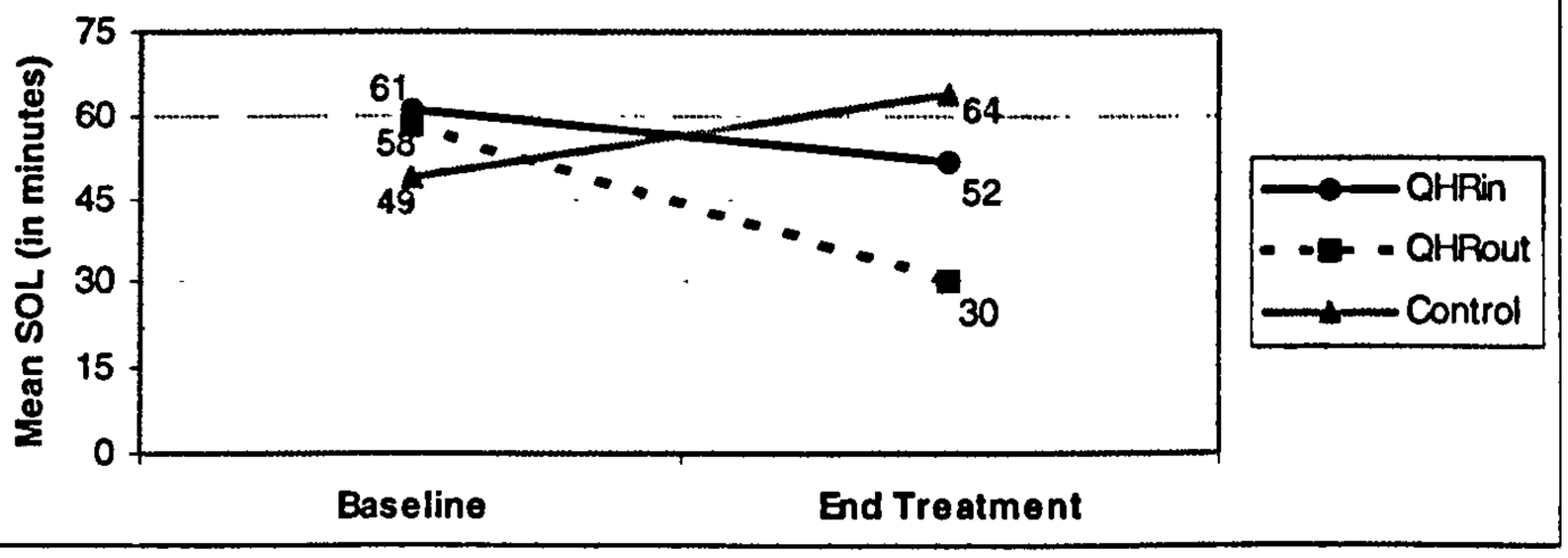
Note: * Shapiro-Wilk test of normality indicated that data are not normally distributed

5.4.2.3 Sleep Onset Latency

In figure 26 the mean SOL values for each group at baseline and end of treatment are presented graphically. A mixed model ANOVA was carried out on the log transformed mean SOL data. There was a significant main effect for time [F(1,38)=7.11, p=0.01] showing that the mean value at baseline [M= 56.16(47.5)] was statistically significantly higher than that at the end of treatment [M=47.56(50.0)]. No significant main effect for group [F(2,38)=0.81, n.s.] was found but a significant interaction effect was found between group and time [F(2,38)=4.36,

p=0.02]. Employing the Bonferroni post-hoc correction (p=0.006), only the difference between baseline and end of treatment mean times for the QHRout group was significant [t(14)=3.99, p=0.001].

Fig. 26 - Mean SOL at Baseline and End of Treatment for the QHRin, QHRout and Control Groups

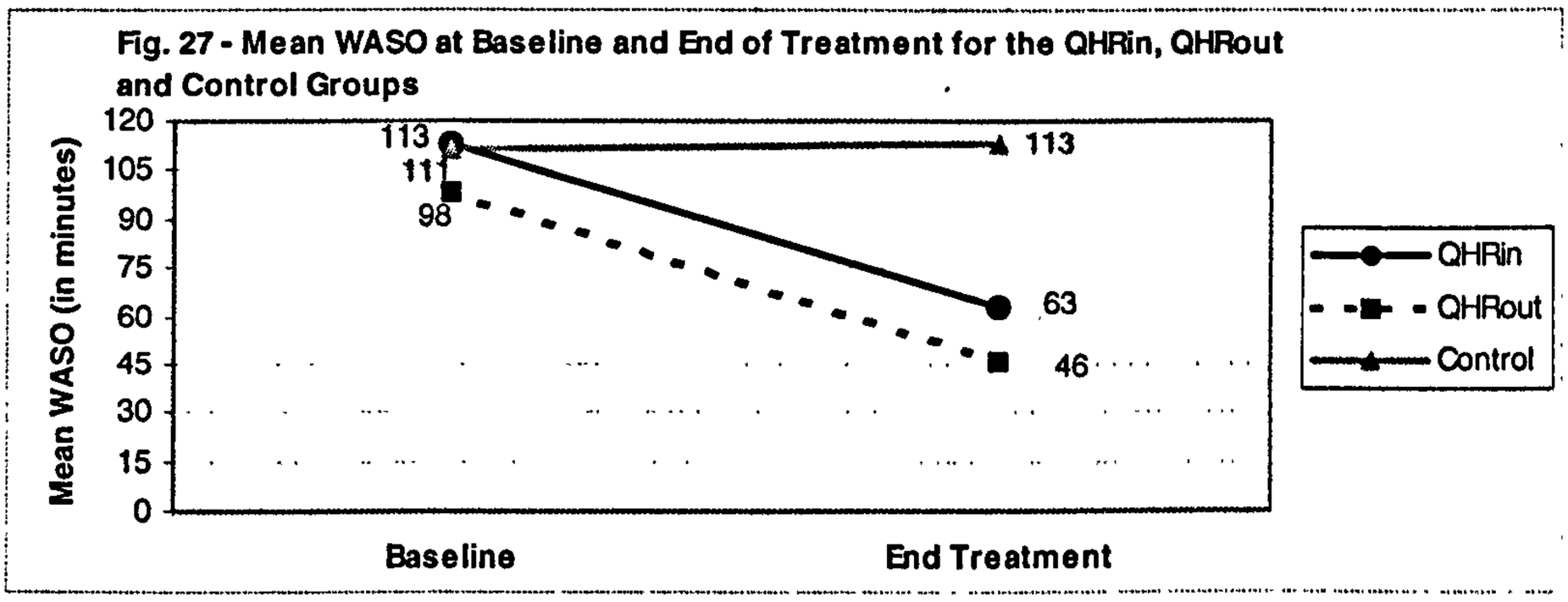


As depicted in figure 26 this result confirmed that following the QHR out of bed intervention the QHRout group's mean SOL was decreased. Regarding the uncorrected t-tests, the only significant result was that between the QHRout and the Control groups at end of treatment [t(25)=-2.14, p=0.04].

5.4.2.4 Wake after Sleep Onset

In figure 27 the mean WASO values for each group at baseline and end of treatment are presented graphically. A mixed ANOVA revealed no significant main effect for group but a significant main effect for time was found [F(1,38)=20.4, p<0.0005] showing that the mean value at baseline [M= 107.39(69.2)] was statistically significantly higher than that at the end of treatment [M=71.48(58.8)]. A significant interaction effect [F(2,38)=4.56, p=0.02] was explored with Bonferroni post-hoc correction. The difference between the baseline mean time and that at the end of treatment for the QHRout group was significant [t(14)=5.26, p<0.0005] as

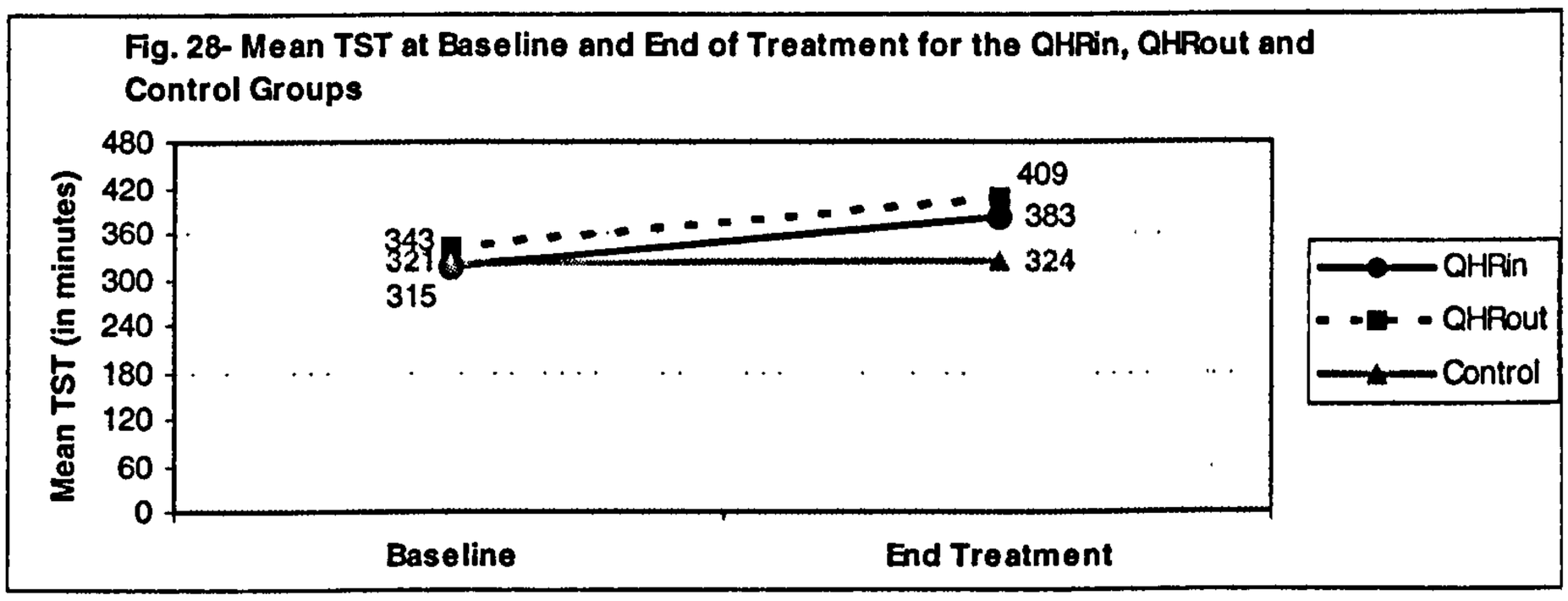
was the difference between the mean time between the QHRout and the Control groups at the end of treatment [t(25)=3.63, p=0.001].



These results indicate that following intervention WASO was decreased in the QHRout group and that it was significantly lower than that of the Control group at the end of the intervention. Regarding the uncorrected t-tests, statistically significant differences were found between baseline and end of treatment for the QHRin [t(13)=2.49, p=0.03] and between the QHRin and the Control groups at end of treatment [t(24)=-2.34, p=0.03].

5.4.2.5 Total Sleep Time

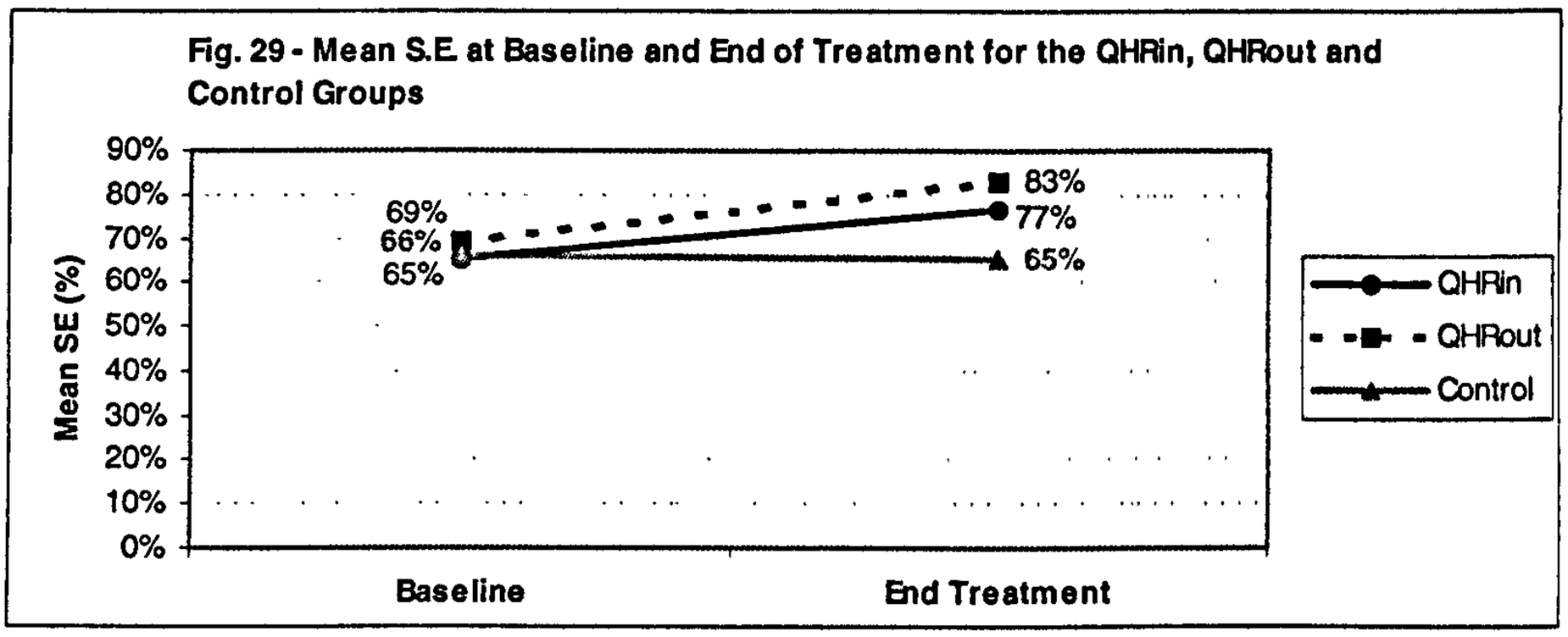
In figure 28 the mean TST values for each group at baseline and end of treatment are presented graphically. A mixed ANOVA revealed no significant main effect for group but a significant main effect for time was found [F(1,38)=13.89, p=0.001] showing that the mean TST value at baseline [M= 328(81.6)] was statistically significantly lower than that at end of treatment [M=374(71.1)]. Probably because the power to detect significant differences was low (power=0.53), the interaction between group and time showed only a non-significant trend [F(2,38)=2.89, p=0.07].



Inspection of figure 28 suggests that the QHRin and the QHRout groups slept longer following the intervention increased from baseline to treatment while the control group's TST did not change.

5.4.2.6 Sleep Efficiency

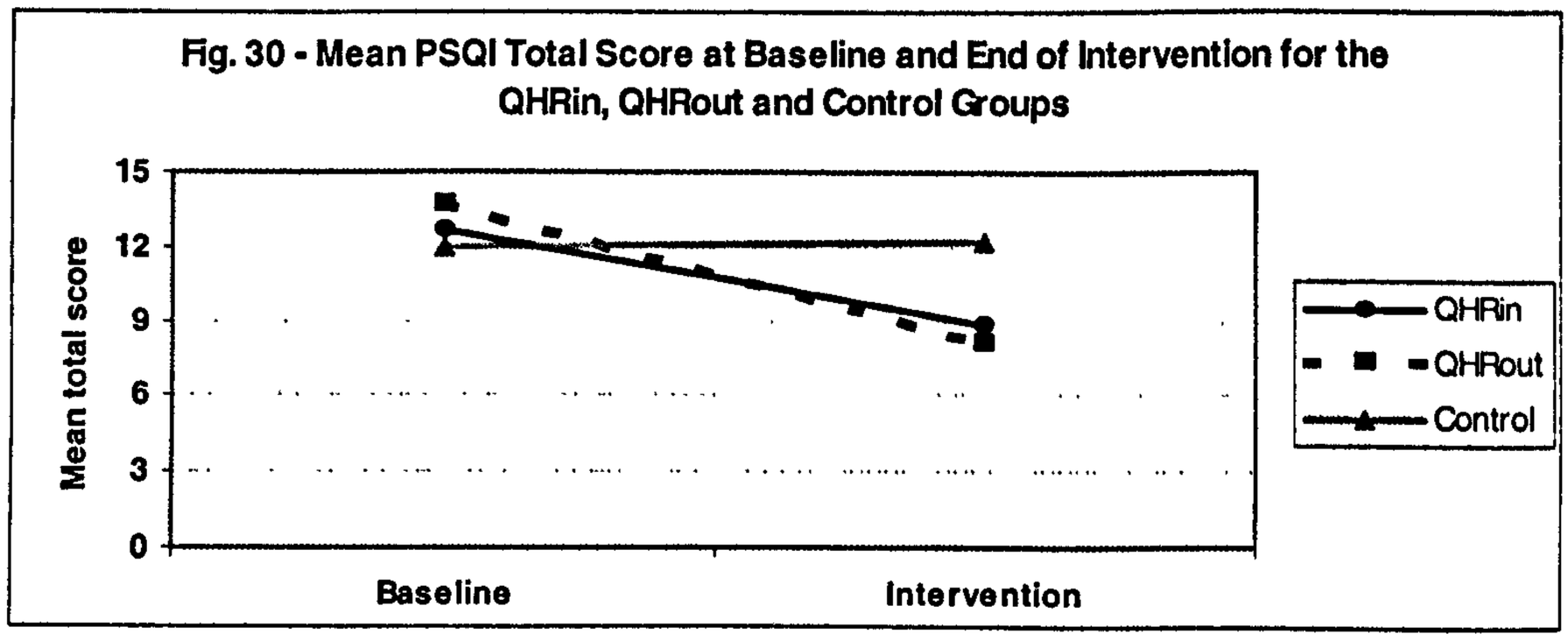
Figure 29 presents S.E. values for each group at baseline and end of treatment. No significant main effect of group was found but a main effect of time [F(1,38)=24.79, p<0.0005] was found. Mean S.E. value at baseline [M= 0.67(0.16)] was statistically significantly lower than that at the end of treatment [M=0.77(0.15)]. In addition, a significant interaction effect [F(2,38)=8.13, p=0.001] was explored employing the Bonferroni post-hoc test. This indicated differences between S.E. means at baseline and at end of treatment for the QHRout [t(14)=-5.52, p<0.0005] and for the QHRin [t(13)=-3.68, p=0.003] groups. Furthermore, the difference between the QHRout and the Control groups at the end of treatment [t(25)=3.27, p=0.003] was also significant. Regarding uncorrected t-tests, the only significant difference was between QHRin and Control groups at end of treatment [t(24)=2.51, p=0.02].



These results indicate that S.E. increased following both QHR interventions and that the S.E. at the end of treatment was higher in QHRout than the Control group.

5.4.2.7 Pittsburgh Sleep Quality Index Total Score

In figure 30 the mean PSQI total score values for each group at baseline and end of treatment are presented graphically.



There was no significant main effect for group [F(2,38)=2.00, n.s.] but a significant main effect for time [F(1,38)=46.04, p<0.0005] was found. This indicated that the PSQI at the end of treatment [M=9.54(3.1)] was lower than that at baseline [M=12.83(2.0)]. A significant interaction effect [F(2,38)=13.38, p<0.0005] was explored with Bonferroni correction. Pair-wise comparisons indicated that at end of

treatment the mean PSQI values of the QHRin and the QHRout groups were statistically significantly lower that that of the Control group [t(24)=-3.54, p=0.003 and t(25)=-3.95, p=0.001 respectively]. In addition, the mean PSQI values at end of treatment as compared to baseline were lower for both the QHRin [t(13)=4.19, p=0.001] and the QHRout [t(14)=6.68, p<0.0005].

5.4.2.8 Summary of subjectively measured sleep continuity parameters

The analyses reported in the sub-sections above suggest the QHR interventions produced statistically significant differences in some of the sleep parameters gathered via sleep diary while no changes were found between baseline and end of treatment for the Control group. In addition, while no statistically significant differences were found at baseline between the 3 groups, differences between the Control group and the QHRout and/or the QHRin were found at end of treatment. Furthermore, the QHR interventions produced highly statistically significant changes on the PSQI total scores within groups compared to the control group. However no differences were found between the two treatment groups QHRin and QHRout for any of the sleep continuity or PSQI mean values. These results will be critically examined in the discussion section.

The aims of insomnia treatments are not only to improve sleep continuity but also to improve the perception of quality of sleep and feelings upon awakening. The next section is concerned with subjective estimates of sleep quality.

5.4.3 Sleep Quality Data

In this section estimates of sleep quality factors are reported and analysed. Each morning participants reported the quality of the previous night's sleep, and their level of alertness and restedness upon awakening. First the descriptive data are presented and, thereafter, the results of separate mixed ANOVAs on each of these three dependent variables are reported.

One-way ANOVAs on the baseline means of quality of sleep, alertness and restedness (table 16) revealed no significant differences for quality of sleep [F(2,38)=.111, n.s.], restedness [F(2,38)=.79, n.s.] or alertness. [F(2,38)=.15, n.s.] (all p_s>0.1) between the groups.

5.4.3.1 Descriptive Sleep Quality Data

The Mean Score and SD for each group during baseline and during the last week of treatment are reported in table 13.

Table: 13- Mean (SD) scores for Quality of Sleep, Restedness and Alertness baseline and end of treatment for the QHRin, QHRout and

	Quality of Sleep	Alertness	Restedness
QHRin N=14			
Baseline	**2.01 (.48)**	**2.09 (.71)**	**1.93 (.71)**
End of Treatment	**2.48 (.73)**	**2.25 (.87)**	**2.26 (.83)**
QHRout N=15			
Baseline	**1.97 (.62)**	**1.99 (.79)**	**1.63 (.64)**
End of Treatment	**2.29 (.47)**	**2.21 (.55)**	**2.12 (.51)**
Control N=12			
Baseline	**1.70 (.57)**	**2.16 (.98)**	**1.69 (.67)**
End of Treatment	**1.70 (.68)**	**1.90 (1.0)**	**1.57 (.76)**

Inspection of table 13 suggests that there were differences between and within groups and inferential statistics were performed to assess if such differences were statistically significant.

5.4.3.2 Sleep Quality

In figure 31a the mean Sleep Quality values for each group at baseline and end of treatment are presented graphically.

A significant main effect of group was found [F(2,38)=3.80, p=0.03]. The sleep quality means were M=2.25(0.6) for the QHRin, M=2.13(0.9) for QHRout and M=1.70(0.6) for the Control. Employing Bonferroni post-hoc pairwise comparisons no significant differences between the groups' means were found. There was also a significant main effect for time [F(1,38)=9.82, p<0.0005] indicating that quality of sleep at the end of treatment [M=2.18(0.7)] was higher than that at baseline [M=1.90(0.6)]. The group by time interaction was not significant perhaps because power was low (power=0.49). However, a trend [F(2,38)=2.60, p=0.09] suggested that at the end of treatment the QHRin and the QHRout groups reported that the quality of their sleep was better compared to baseline while the control group's score remained virtually the same.

5.4.3.3 Restedness Upon Awakening

In figure 31b the mean restedness values for each group at baseline and end of treatment are presented graphically.

There was no significant main effect of group [F(2,38)=1.74, n.s] but a significant main effect of time [F(1,38)=6.95, p=0.01] was found indicating that the level of

restedness at the end of treatment [M=2.01(0.7)] was higher than that at baseline [M=1.75(0.7)]. In addition the group by time interaction was significant [F(2,38)=4.07, p=0.02]. However, employing the Bonferroni post-hoc test none of the pairwise comparisons was significant. Regarding uncorrected t-tests significant differences were detected for QHRin baseline vs. end of treatment [t(13)=-2.29, p=0.04], QHRout baseline vs. end of treatment [t(14)=-3.05, p=0.009], QHRin vs. Control at end of treatment [t(24)=2.16, p=0.04] and QHRout vs. Control at end of treatment [t(25)=2.16, p=0.04]. These differences indicate that both QHR interventions increased the level of restedness compared to baseline and that by the end of the intervention both QHR groups' estimate of restedness was higher than that of the Control group.

5.4.3.4 Alertness Upon Awakening

In figure 31c the mean alertness values for each group at baseline and end of treatment are presented graphically.

No significant time effect [F(1,38)=.24, n.s.] or group effect [F(2,38)=.10, n.s.] were found. Perhaps due to low power (power=0.55), only a non-significant group by time interaction trend [F(2,38)=2.89, p=0.07] was found. It suggested that the QHRin and QHRout groups estimated greater alertness at the end of treatment as compared to baseline while the control group's estimate of alertness was lower at the end of treatment as compared to baseline.

Interestingly, figure 31 shows almost parallel lines for the QHRin and QHRout suggesting that the intervention produced similar changes in direction and intensity

in the three sleep quality variables under examination. This raises the possibility that these questionnaires were measuring one single construct.

Fig. 31 - Mean scores for the variables 'Quality of Sleep', 'Restedness' and 'Alertness' at baseline and end of treatment for the three groups

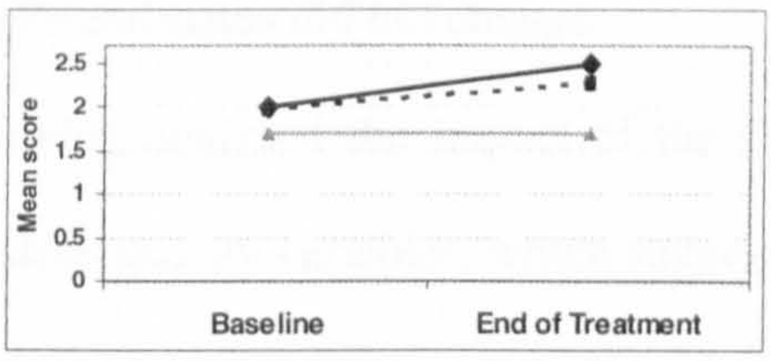


Fig. 31a 'Quality of Sleep'

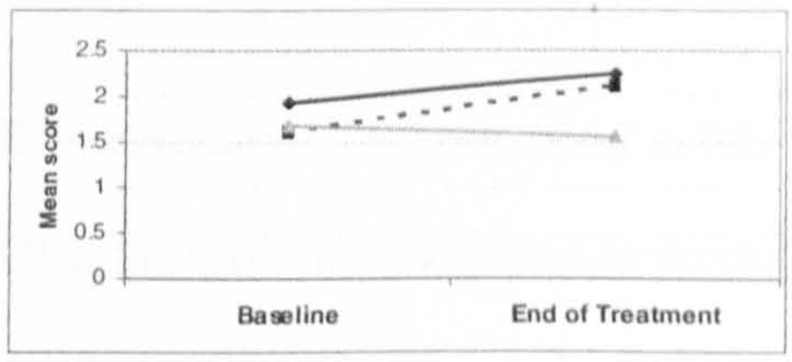


Fig. 31b 'Restedness'

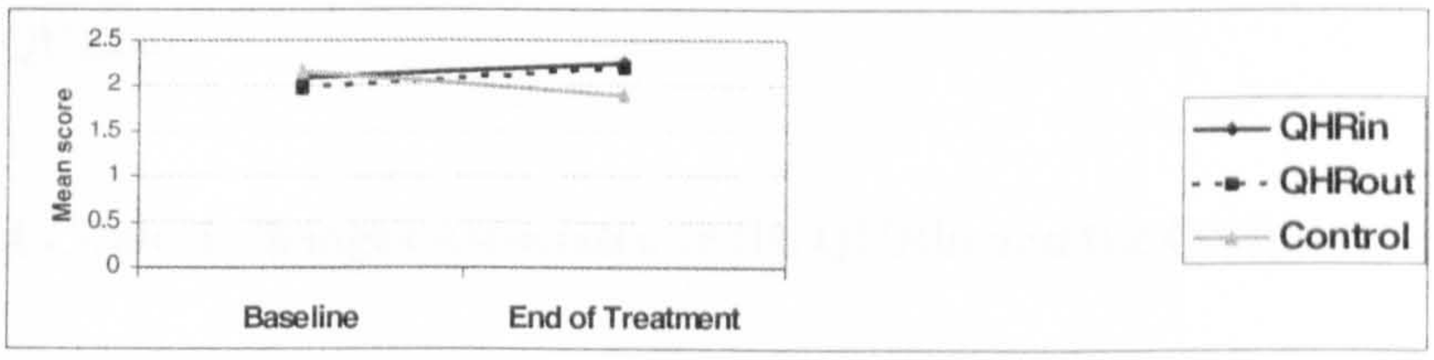


Fig. 31c 'Alertness'

In order to assess if this was the case a Pearson product correlation was carried out on the first baseline week of participants in study three and on the week of participants in study one (total N=121). Significant positive correlations between 'quality of sleep' and 'restedness' ($r=.875$, $p<.0005$), 'restedness' and 'alertness' ($r=.877$, $p<.0005$) and 'quality of sleep' and 'alertness' ($r=.809$, $p<.0005$) were found. Squaring these values resulted in $R^2=76\%$, $R^2=76\%$ and $R^2=65\%$ respectively indicating that there was substantial shared variance across this set of qualitative ratings.

5.4.3.5 Summary of the Sleep Quality Results

The results presented above indicate that both QHR groups reported improved sleep quality, restedness and alertness following the intervention while the control group's estimates did not change.

Having analysed the impact of the QHR interventions on both sleep continuity and sleep quality variables, which suggested that the QHR is efficacious in producing improvements, attention is now turned to the clinical significance of the QHRin and the QHRout.

5.4.4 Clinical Changes Associated to the QHRin and the QHRout

In this section the clinical significance of the QHR treatment is addressed. Kazdin (1977) regarded change in therapy as clinically significant when the individual moved from the dysfunctional to the functional range of normalcy. In addition, Lichstein and Fischer (1985) regarded a change over baseline greater than 50% as a useful clinical outcome measure. These two outcome measures were used to assess the clinical significance of the QHRin and QHRout interventions. They were applied to the sleep continuity variables SOL and WASO and to the PSQI total score.

5.4.4.1 Clinical Significance

Table 14 presents the percentage of participants in each group (QHRin, QHRout and Control) achieving a mean reduction of clinical significance (Kazdin, 1977, Lichstein et al, 1985) for SOL, WASO and PSQI and associated effect sizes.

Table: 14 - Percentage of Participants in Each Treatment Condition achieving Mean Reduction of Clinical Significance According to 2 Criteria(*) and Effect Sizes(**)

Treatment	N	Measures of Clinical Outcome		Effect Size
		50% Reduction	Returned to Normalcy	
SOL				
QHRin	14	21%	33%	0.2
QHRout	15	47%	33%	0.6
Control	12	17%	11%	0.3
WASO				
QHRin	14	57%	33%	0.7
QHRout	15	53%	33%	1.1
Control	12	0%	0%	0.0
PSQI				
QHRin	14	29%	7%	1.7
QHRout	15	33%	13%	2.3
Control	12	0%	0%	0.0

(*) Criteria proposed by Kazdin, 1977; Lichstein et al, 1985
(**) Effect size calculated baseline to end of intervention

As reported in table 14 the QHRin and the QHRout produced clinically significant changes in 30 to 50% of the participants. The effect sizes were in the small to medium range for SOL, in the medium to large range for WASO and in the large range for the PSQI. The effect sizes for both TST (0.8 for QHRin, 1.1 for QHRout and 0.0 for Control) and S.E. (0.8 for QHRin, 0.8 for QHRout and 0.0 for Control) were in the large range. Importantly 43% of participants in the QHRin, 66% of QHRout and only 8% of controls achieved a S.E.≥85%.

Interestingly, the magnitude of change achieved with the QHRout was superior to that achieved with the QHRin as evidenced by effect sizes.

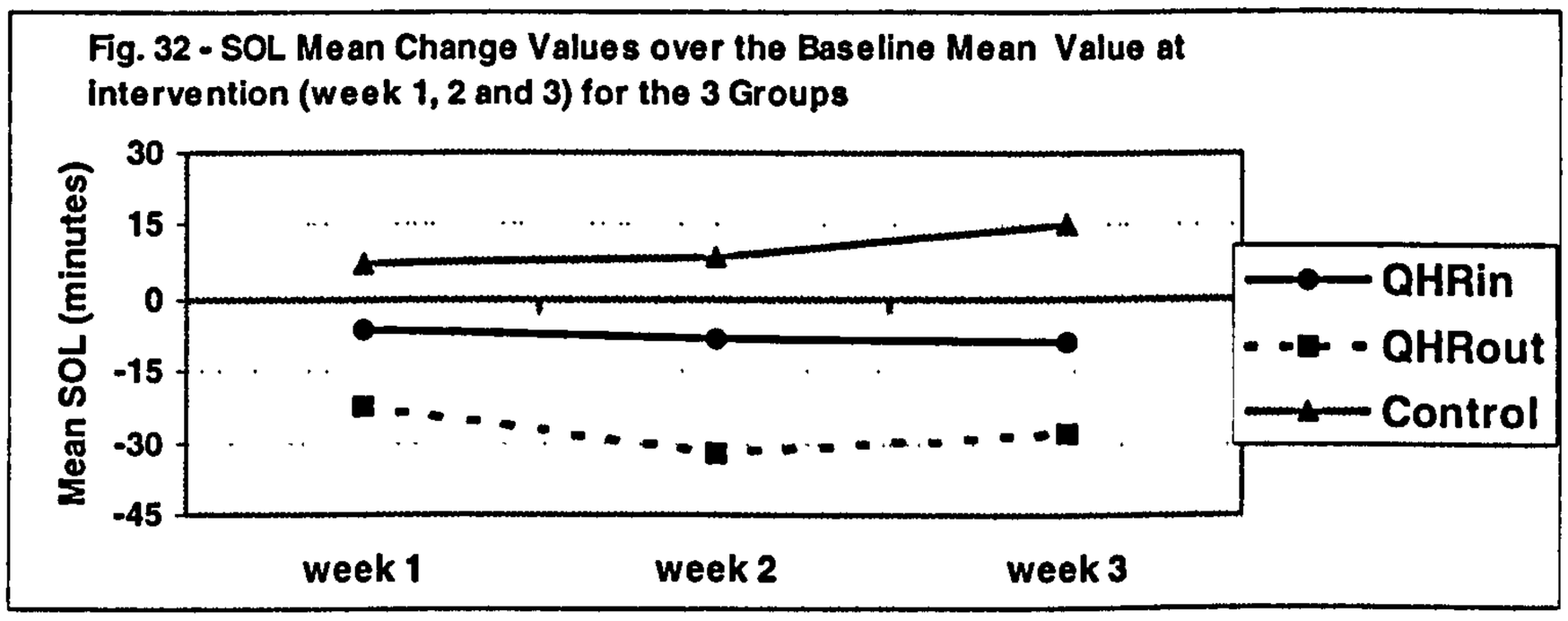
5.4.4.2 Therapeutic effects at week one, two and three

In order to explore when the QHR started to impact on sleep, the mean change scores over baseline at week 1, week 2 and week 3 were considered. Week 1, week 2 and week 3 represent the mean of the sleep diary data gathered during the first,

second and third week of intervention respectively. In the following sections non-parametric analyses (Kruskal-Wallis) were carried out on the change score values to assess after how many intervention days statistically significant differences between groups emerged.

5.4.4.2a Response Curve for Sleep Onset Latency

In figure 32 the mean SOL changes over the baseline mean values at week 1, 2, and 3 for each group are presented graphically.



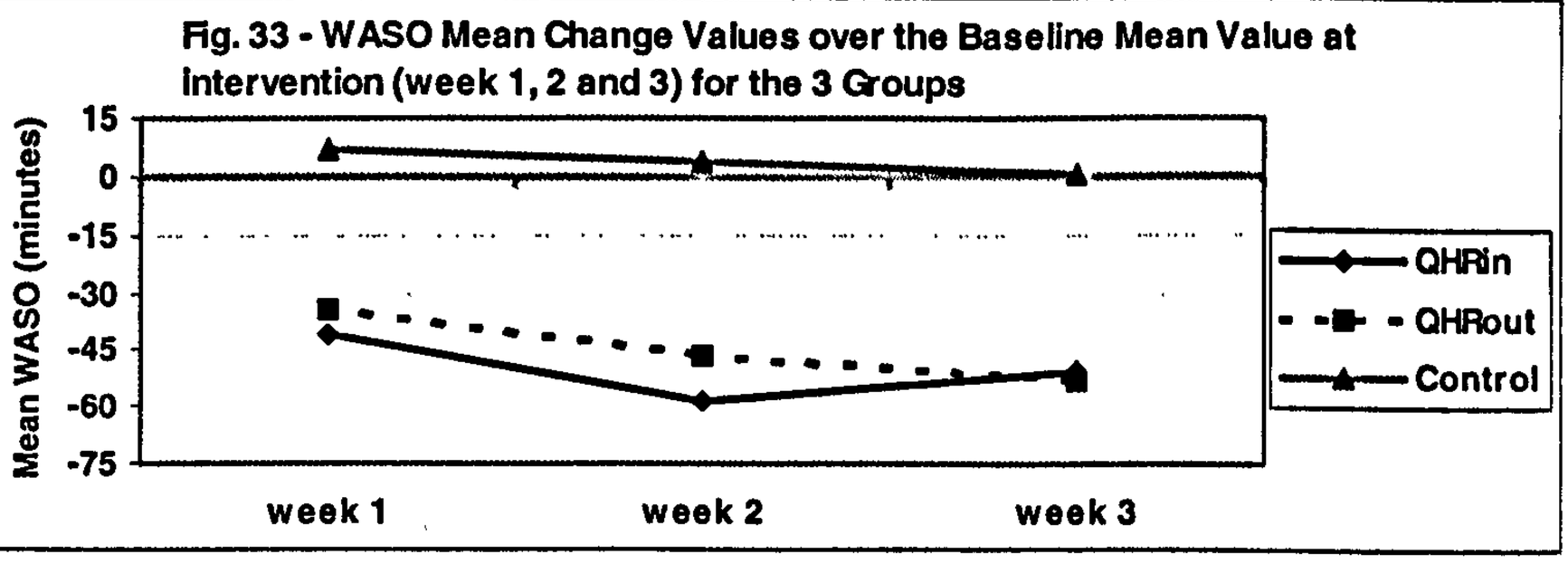
Statistically significant differences between the groups were found for SOL mean change values over baseline at week 1 [$\chi^2(2)= 9.700$, $p=0.008$], week 2 [$\chi^2(2)= 6.756$, $p=0.03$] and week 3 [$\chi^2(2)= 9.213$, $p=0.01$]. Bonferroni corrected Mann-Whitney comparisons between groups were performed for each week comparison. No statistically significant differences were found between the two treatment groups (QHRin and QHRout) at week 1, week 2 or week 3. With regard to week 1, statistically significant differences were found between the QHRin and the Control groups [$U=36.00$, $N_1=14$, $N_3=12$, $p=0.01$] and the QHRout and the control groups [$U=31.00$, $N_2=15$, $N_3=12$, $p=0.003$] indicating that already during the first week of intervention the two treatment groups took less time to fall asleep than the control

group. With regard to week 2 and 3, statistically significant differences were found between QHRout and the control [U=39.500, N_2=15, N_3=12, p=0.01 and U=26.000, N_2=15, N_3=12, p=0.001 respectively] showing that during the second and third weeks of intervention the QHRout took less time to fall asleep as compared to the control group. It is important to note that these results are due to the QHRout group reducing their SOL and the control group increasing theirs.

5.4.4.2b Response Curve for Wake After Sleep Onset

The graphic representation of the mean WASO changes over the baseline mean values at week 1, 2, and 3 for each group (fig. 33) suggests no change for the control group but positive changes from the first week of intervention for the QHRin and the QHRout.

Fig. 33 - WASO Mean Change Values over the Baseline Mean Value at Intervention (week 1, 2 and 3) for the 3 Groups

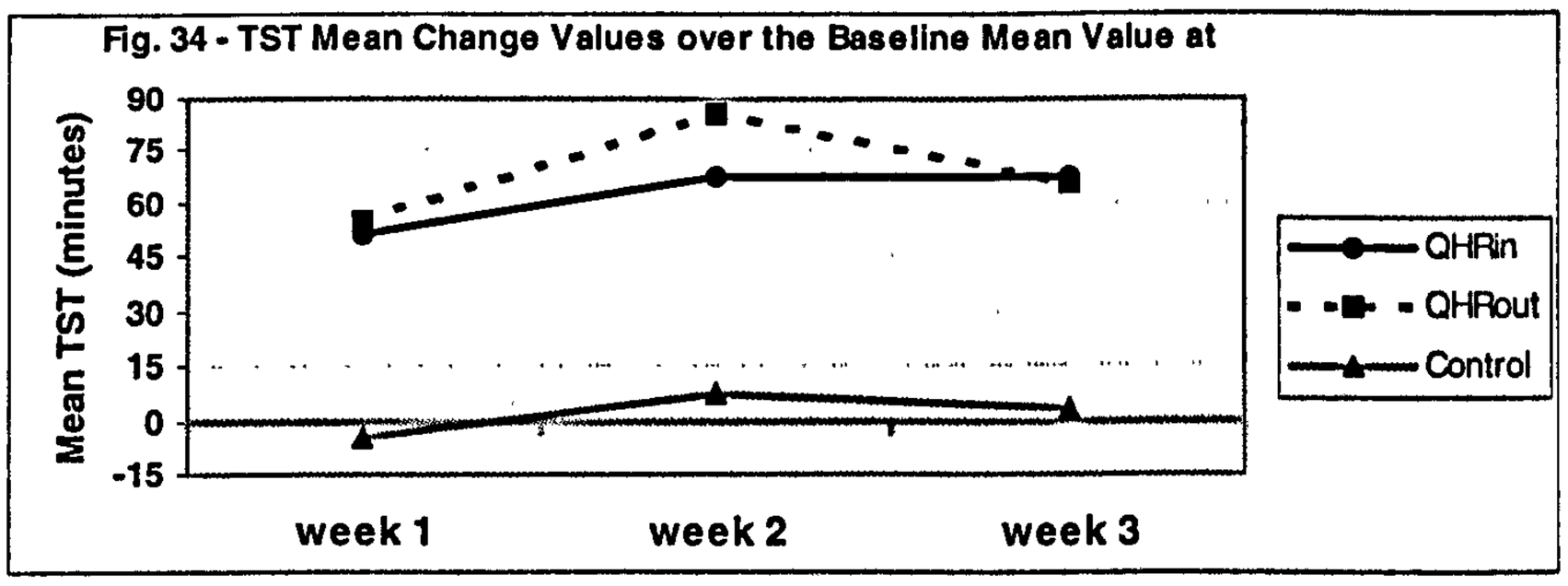


Statistically significant differences between the groups were found for WASO mean change scores over baseline at week 1 [$\chi^2(2)$= 11.050, p=0.004], week 2 [$\chi^2(2)$= 9.678, p=0.008] and week 3 [$\chi^2(2)$= 9.075, p=0.01]. Bonferroni corrected comparisons showed no statistically significant differences between the QHRin and the QHRout at week 1, week 2 or week 3. With regard to week 1, statistically significant differences were found between the QHRin and the Control groups [U=29.000, N_1=14, N_3=12, p=0.004] and the QHRout and the Control groups

[U=31.00, N_2=15, N_3=12, p=0.003] indicating that already during the first week of intervention the two treatment groups spent less time awake during the night as compared to the control group. These improvements were maintained during week 2 and 3 [QHRout and the control group U=44.000, N_2=15, N_3=12, p=0.02 and U=24.000, N_1=15, N_3=12, p=0.001 respectively and QHRin and the control group U=26.000, N_1=14, N_3=12, p=0.002 and U=46.000, N_2=14, N_3=12, p=0.05 respectively] (uncorrected t-tests for week 3).

5.4.4.2c Response Curve for Total Sleep Time

Inspection of figure 34 suggests that the mean TST over the baseline mean values at week 1, 2, and 3 differed only for the QHRin and the QHRout.

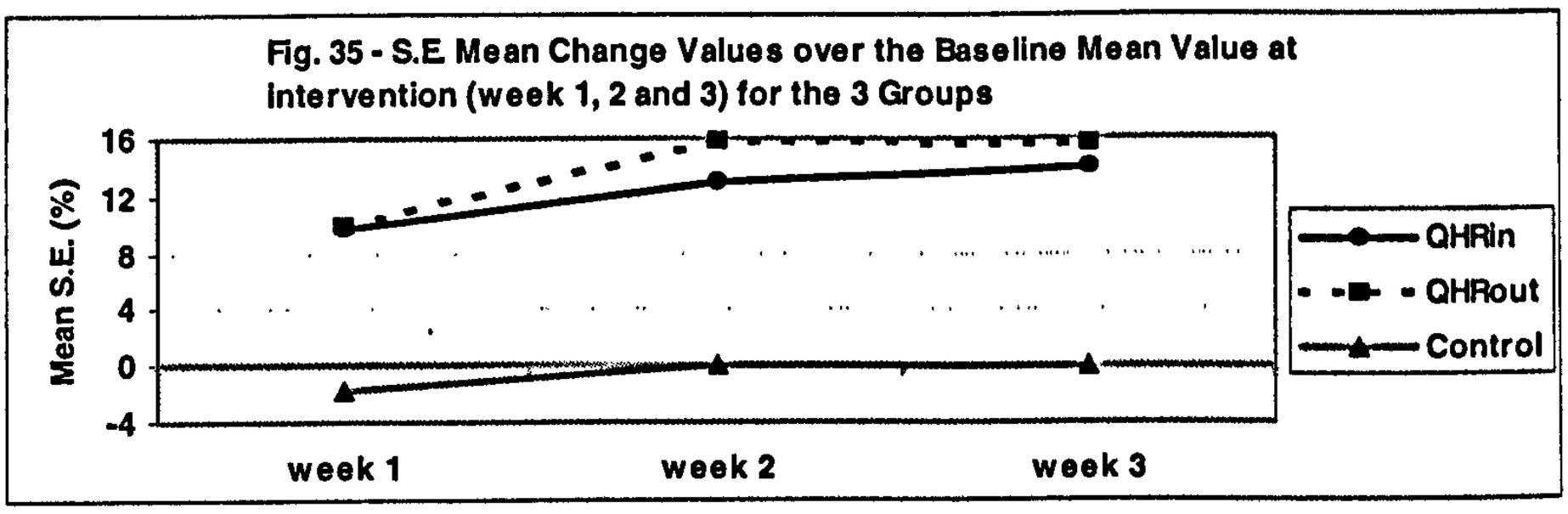


Statistically significant differences between the groups were found for the TST mean change values over baseline at week 1 [$\chi^2(2)$= 7.503, p=0.02], week 2 [$\chi^2(2)$= 10.000, p=0.07] and a non significant trend at week 3 [$\chi^2(2)$= 5.704, p=0.06]. Applying Bonferroni corrected comparisons no statistically significant differences were found between the QHRin and the QHRout at week 1, 2 or 3. With regard to week 1, a statistically significant difference was found between the QHRout and the control groups [U=37.00, N_2=15, N_3=12, p=0.009] indicating that already during the first week of intervention the QHRout slept longer as compared to the control group.

The difference between the QHRin and the control group approached significant difference [U=42.000, N_1=14, N_3=12, p=0.03]. With regard to week 2, a statistically significant difference was found between the QHRout and the control group [U=28.000, N_2=15, N_3=12, p=0.002]. In addition the difference between the QHRin and the control groups approached significance [U=38.000, N_1=14, N_3=12, p=0.018]. With regard to week 3 a non-significant trend was found and inspection of figure 34 suggests that during the third week of intervention the QHRout and QHRin slept longer than the control group.

5.4.4.2d Response Curve for Sleep Efficiency

In figure 35 the mean S.E. changes over the baseline mean values at week 1, 2, and 3 for each group are presented graphically.



Statistically significant differences between the groups were found for the S.E. mean change values over baseline at week 1 [$\chi^2(2)$= 11.40, p=0.003], week 2 [$\chi^2(2)$= 14.40, p=0.001] and week 3 [$\chi^2(2)$= 11.64, p=0.003]. Bonferroni corrected comparisons showed no statistically significant differences between the QHRin and the QHRout at week 1, 2 or 3. With regard to week 1, statistically significant differences were found between the QHRin and the control group [U=35.000, N_1=14, N_3=12, p=0.01] and the QHRout and the control group [U=21.500, N_2=15, N_3=12,

p<0.0005] indicating that already during the first week of intervention S.E. mean change values of the QHRin and the QHRout were greater that that of the control group. With regard to week 2, a statistically significant difference was found between the QHRin and the control group [U=26.500, N_1=14, N_3=12, p=0.002] and the QHRout and the control group [U=18.000, N_2=15, N_3=12, p<0.0005] showing that during the second week of intervention the QHRout and the QHRin had higher mean S.E. values as compared to the control group. With regard to week 3, a trend between the QHRin and the control group [U=45.000, N_1=14, N_3=12, p=0.05] and a statistically significant difference between the QHRout and the control groups [U=14.500, N_2=15, N_3=12, p<0.0005] were found.

5.4.4.2e Summary of the Response Curve Findings

The response curve results obtained by performing statistical analyses on the mean change scores suggested that the QHR interventions produced statistically significant differences in most of the sleep parameters already during the first week of treatment as compared to the control group. These results are critically examined in the discussion section.

Having shown that the QHR interventions improved subjectively estimated sleep continuity and quality, that it produced clinical significance changes and that these changes presented within the first week of treatment, attention is now turned to the possibility that the QHR intervention is associated to changes in sleep related cognitions and to arousability at bed time.

5.4.5 Sleep Related Questionnaires

In this section sleep related cognition and physical and mental arousal at bedtime, gathered via a battery of six, well validated questionnaires, are reported descriptively and then formally analysed. This analysis can help to shed light on whether the QHR had an impact on cognition and arousal as well as sleep parameters. Before analysing each of the questionnaires some common points may be made. One-way ANOVAs were performed on the baseline mean scores for each scale, or questionnaire sub-scales where appropriate, to examine if there were statistically significant differences among the groups prior to the treatment phase. The only significant differences indicated that at baseline the somatic and sensory engagement of the QHRin was lesser than those of the QHRout and of the control group. The results of one-way ANOVAs are reported in appendix 31.

5.4.5.1 Descriptive Data of sleep related cognitions and bed-time arousal

The Mean Score and SD for each group at baseline and end of treatment are reported in table 15. The next subsections report results for each of the questionnaires completed by participants.

5.4.5.2 Q1: Sleep Behaviour Self Rating Scale – R

With regard to the SBSRS-R scale no significant main effects for time [F(1,36)=0.18, n.s.] or group [F(2,36)=4.89, p=0.01] or significant interaction [F(2,36)=2.48, n.s] were found. It should be noted that power to detect differences was very low (power = 0.25)

Table 15 - Mean(SD) scores at baseline and end of treatment for the battery of questionnaires for the QHRin, QHRout and Control groups

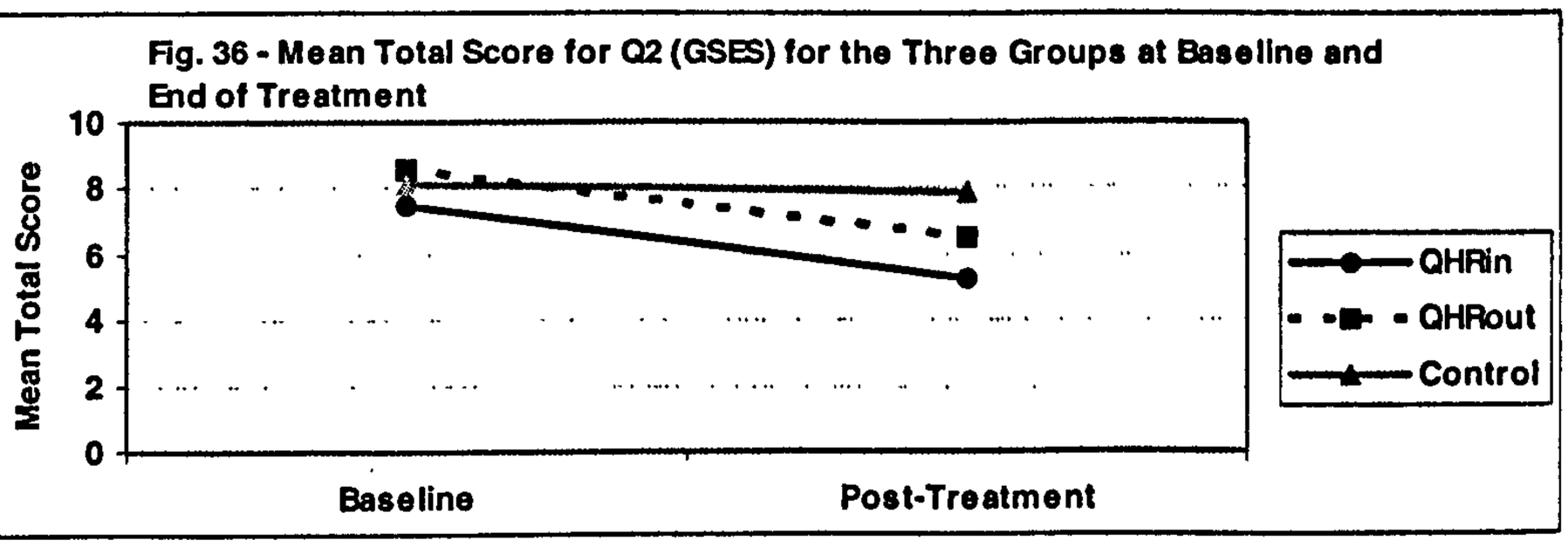
	QHRin(N=13)		QHRout (N=17)		Control (N=9)	
	Baseline	Intervention	Baseline	Intervention	Baseline	Intervention
Q1	29.8(6.8)	30.0(5.3)	29.8(5.5)	27.8(4.6)	31.3(5.9)	32.4(6.2)
Q2	**7.5(3.6)**	**5.3(2.8)**	**8.6(2.4)**	**6.5(2.4)**	**8.1((3.4)**	**7.9(2.8)**
Q3a	11.1(3.9)	9.9(4.1)	12.4(2.6)	11.7(2.6)	11.8(1.8)	12.1(1.3)
Q3b	**7.6(2.6)**	**6.1(2.2)**	**9.2(2.5)**	**8.5(4.1)**	**10.8(3.2)**	**9.7(2.8)**
Q3c	8.9(3.1)	5.1(1.8)	10.6(2.1)	7.9(2.2)	9.2(2.2)	7.1(2.2)
Q3d	**9.4(2.4)**	**6.2(1.7)**	**9.3(1.9)**	**6.3(1.8)**	**8.4(1.6)**	**8.2(2.2)**
Q4a	18.6(5.9)	17.9(6.5)	21.2(4.4)	18.6(5.3)	21.1(3.4)	22.1(2.9)
Q4b	**20.1(5.4)**	**18.7(6.2)**	**23.1(3.5)**	**19.5(5.0)**	**22.4(5.9)**	**24.5(4.2)**
Q4c	12.1(2.5)	12.0(3.1)	14.6(2.6)	11.6(2.3)	14.9(1.7)	15.7(3.3)
Q5a	**13.5(4.3)**	**11.6(3.8)**	**13.2(4.1)**	**11.5(3.7)**	**14.2(6.6)**	**14.1(5.8)**
Q5b	21.1(7.6)	19.1(7.9)	24.7(5.3)	21.8(6.7)	26.5(3.4)	25.2(4.7)
Q6a	**11.8(3.8)**	**11.1(3.9)**	**13.6(2.7)**	**12.9(2.4)**	**13.2(2.6)**	**11.6(3.7)**
Q6b	16.4(4.7)	16.8(4.2)	18.1(4.9)	18.7(3.2)	17.0(4.8)	16.4(4.5)
Q6c	**24.6(4.2)**	**23.6(6.9)**	**26.3(7.0)**	**24.6(5.9)**	**27.6(7.0)**	**26.7(8.0)**

Q1= SBSRS-R,
Q2=GSES,
Q3= SDQ. It comprises 4 subscales, measuring:
Q3a= cognitive arousal, Q3b= physical tension, Q3c= sleep effort, Q3d= sleep pattern problems
Q4= GCTI. It comprises 3 subscales measuring cognitive intrusions relating to
Q4a= active problem solving, Q4b= sleep and wakefulness Q4c=somatic and sensory engagement
Q5= PSAS. It comprises 2 subscales measuring Q5a= cognitive arousal, Q5b= somatic arousal
Q6= DBAS-10. It comprises 3 subscales measuring beliefs about insomnia. In particular
Q6a=need for control over insomnia Q6b= long term & Q6c= short term negative consequences

5.4.5.3 Q2: Glasgow Sleep Effort Scale

In figure 36 the mean total GSES scores for each group at baseline and end of treatment are presented graphically.

Fig. 36 - Mean Total Score for Q2 (GSES) for the Three Groups at Baseline and End of Treatment



No main effects of group [F(2,36)=0.32, n.s.] was found but a significant main effect of time [F(1,36)=12.93, p=0.001] was found showing that the total score was higher at baseline [M= 8.1(3.0)] than at end of treatment [M= 6.4(2.7)]. This result indicated a decrease in sleep effort at the end of treatment. No significant interaction [F(2,36)=2.00, n.s.] was found but power to detect differences was low (power=0.38).

5.4.5.4 Q3: Sleep Disturbance Questionnaire

The SDQ comprises four sub-scales which were analysed separately as they measure different constructs.

5.4.5.4a: Cognitive Arousal

With regard to the dimension 'cognitive arousal' of the SDQ no significant main effects of time [F(1,36)= 3.00, p=0.09] or group [F(2,36)=1.31, n.s.] or significant interactions [F(2,36)=1.90, n.s.] were found. The power to detect differences was low (power =0.39, 0.26 and 0.37 for Time, Group and interaction respectively).

5.4.5.4b: Physical Tension

With regard to the dimension 'physical tension' of the SDQ a significant main effect of group [F(2,36)=4.75, p=0.015] was found and post-hoc Bonferroni corrected pairwise comparisons indicated that physical tension of the QHRin [M=6.6(2.4)] was lower than that of the control group [M=10.2(2.9)] [t(20)=-3.01, p=0.007]. A significant time effect was also found [F(1,36)=4.50, p=0.04] showing that perception physical tension had decreased at the end of treatment [M= 8.0(3.4)]

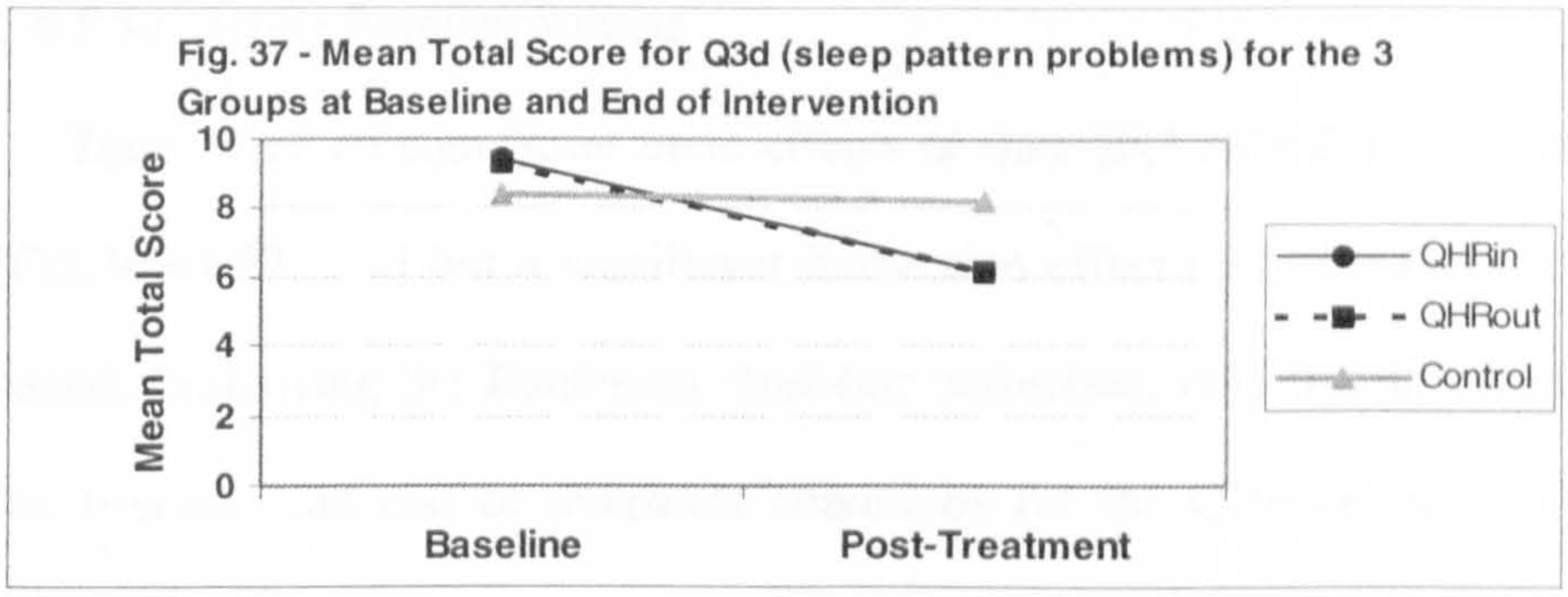
as compared to baseline [M= 9.0(2.9)]. No significant interaction was found [F(2,36)= 0.22, n.s.] however power to detect differences was low (power=0.08).

5.4.5.4c: Sleep Effort

With regard to the dimension 'sleep effort' of the SDQ a significant group effect [F(2,36)=6.67, p=0.003] was found and post-hoc Bonferroni corrected pairwise comparisons [t(280=-3.76, p=0.001] indicated that the QHRin sleep effort was lower than that of the QHRout [M=7.0(2.4) and M=9.2(2.1)]. A main effect of time [F(1,36)=28.73, p<0.0005] indicated that the total score was higher at baseline [M= 9.7(2.5)] than 3 weeks after intervention [M= 6.8(2.3)] (i.e. less sleep effort at the end of treatment). This result is similar to that obtained with the GSES (section 5.4.5.3). No significant interaction [F(2,36)=.805, n.s.] was found but power was low (power=0.18).

5.4.5.4d: Sleep Pattern Problems

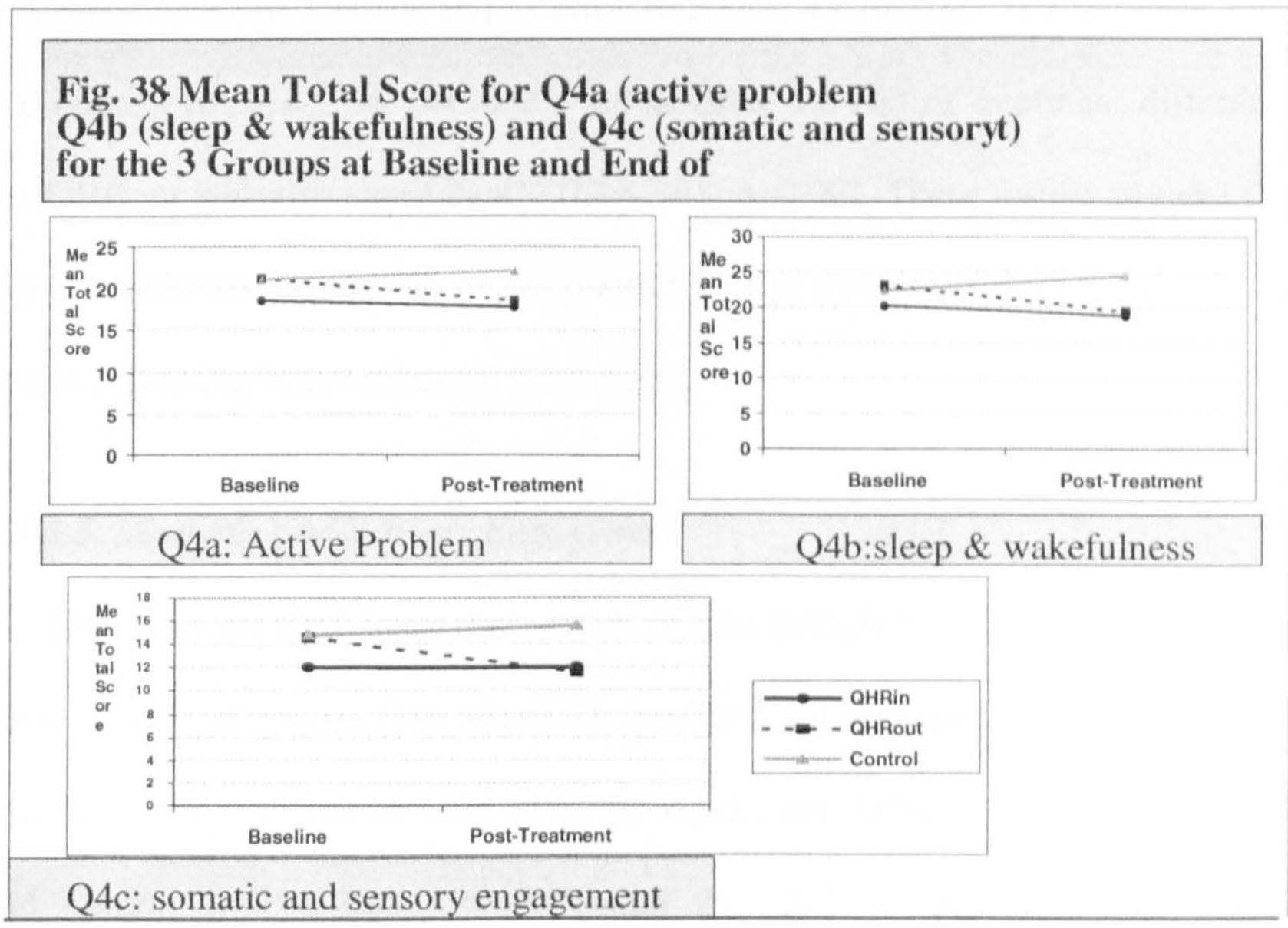
No significant main effect of group was found [F(2,36)=0.51, n.s.] but a significant main effect of time [F(1,36)=22.18, p<0.0005] showing that the total score was higher at baseline [M= 9.1(2.0)] than at end of treatment [M= 6.7(1.9)] was found. A significant interaction effect [F(2,36)=3.78, p=0.03] was explored with post-hoc Bonferroni corrected comparisons. Results showed that sleep patterns problems were less at the end of treatment as compared to baseline for both the QHRout [t(16)=4.47, p=0.0005] and the QHRin [t(12)=4.58, p=0.001]. The uncorrected pairwise comparisons were: end of treatment QHRin vs. control [t(20)=-2.4, p=0.025] and QHRout vs. control [t924)=-2.4, p=0.024].



As depicted in figure 37 these results indicated that both QHR interventions decreased sleep pattern problems while the control condition did not.

5.4.5.5 Q4: Glasgow Content of Thoughts Inventory

The GCTI comprises three sub-scales, which were analysed separately as they measure different constructs. In figure 38 the mean total scores of the GCTI for the three groups are presented graphically.



5.4.5.5a: Active Problem Solving

There were no significant main effects of time [F(1,36)= 2.47, n.s] or of group [F(2,36)=1.27, n.s.] but a significant interaction effect [F(2,36)=4.59, p=0.02] was found. Employing the Bonferroni post-hoc correction, only the difference between the baseline and end of treatment conditions for the QHRout group approached significant difference [t(16)=3.2, p=0.006] suggesting that the QHRout intervention decreased active problem solving (fig. 38-Q4a). All other comparisons did not differ (all p_s>0.05).

5.4.5.5b: Sleep and Wakefulness

There were no significant main effects of time [F(1,36)=1.59, n.s.] or of group [F92,36)=2.18, n.s.]. A significant interaction effect [F(2,36)=4.34, p=0.02] was explored employing the Bonferroni post-hoc tests which revealed no significant differences. The uncorrected t-tests indicated significant differences at the end of treatment between QHRin and control [t(20)=-2.43, p=0.02 and between QHRout and control [t(20)=-2.54, p=0.018]; the baseline vs. end of treatment difference for the QHRout was also significant[t(16)=-2.61, p=0.02]. These results suggest that the QHR conditions, compared to the control group, decreased sleep and wakefulness scores following intervention (fig. 38-4Qb).

5.4.5.5c: Somatic and Sensory Engagement

There was no significant main effect of time [F(1,36)=2.67, n.s.] but a significant main effect of group [F(2,36)=5.73, p<0.007] was found. Post-hoc Bonferroni corrected pairwise comparisons revealed significant differences between the QHRin and control [t(20)=-2.88, p=0.009] and the QHRout and control [t(24)=-2.74,

p=0.011] indicating that the QHRin and the QHRout somatic and sensory engagement was lower than the control [M=12.1(2.8), M=13.0(2.4) and M=15.2(2.6)] respectively). A significant interaction effect [F(2,36)=6.54, p=0.04] was explored with Bonferroni post-hoc tests. Significant differences between baseline and end of treatment for the QHRout group [t(16)=3.42, p=0.003] and between the QHRout and Control group [t(24)=-3.63, p=0.001] were found, indicating that the QHRout group decreased their somatic and sensory engagement following intervention and it was lower than that of the control group at end of treatment (fig. 38-Q4c). The uncorrected pairwise comparison between QHRin and Control [t(20)=-2.6, p=0.017] was significant, however, this was due to the QHRin score being statistically significantly lower than the control group already at baseline rather than to a difference due to the intervention.

5.4.5.6 Q5: Pre-Sleep Arousal Scale

The PSAS comprises two sub-scales, which were analysed separately as they measure different constructs.

5.4.5.6a: Cognitive Arousal

With regard to 'cognitive arousal' there was no significant main effect of group [F(2,36)=0.54, n.s.] or interaction effect [F(2,36)=0.92, n.s.] but a significant main effect of time [F(1,36)=5.18, p=0.03] was found indicating that cognitive arousal was higher at baseline [M=13.6(4.7)] than at end of treatment [M=12.1(4.3)].

5.4.5.6b: Somatic Arousal

With regard to 'somatic arousal' a non significant trend for the main effect group was found [F(2,36)=2.65, p=0.08]. A significant main effect of time [F(1,36)=7.03, p=0.01] indicated that the somatic arousal at baseline was higher [M=24.0(6.1)] than at end of treatment [M=21.7(7.0)]. No significant interaction effect was found [F(2,36)= 0.34, n.s.] but power was low (power=0.10).

5.4.5.7 Q6: Dysfunctional Beliefs and Attitudes about Sleep-10

The DBAS-10 comprises 3 sub-scales, which were analysed separately as they measure different constructs. However none of the main effects or interaction effects for any of the three subscales was significant. This is probably due to the fact that power to detect differences was low ranging from 0.9 to 0.43.

5.4.5.8 Summary of the Sleep Related Questionnaire Results

The results presented above suggest that the QHR interventions decreased sleep cognitive activity at bedtime and improved sleep patterns while changes were not found in the control group. Inspection of the figures reveals that baseline to treatment changes evidenced by this battery of questionnaires were similar raising the possibility that these measures are not independent from each other. A correlation matrix (table 16) was calculated which confirmed the overlap across these scales. Consequently the results obtained may suggest changes in an underlying common construct (e.g. decreasing anxiety about sleep) rather than changes in 14 different constructs.

Table 16 – SPSS Correlation Matrix Output for the Baseline Scores of 6 Sleep Related Questionnaires and their Sub-Scales

		Q1	Q2	Q3b	Q3d	Q3c	Q3a	Q4a	Q4b	Q4c	Q5a	Q5b	Q6a	Q6b
Q2	r	.076	-											
Q3b	r	.160	.263	-										
Q3d	r	-.186	.177	-.019	-									
Q3c	r	.149	.648 (**)	.344 (*)	.136	-								
Q3a	r	-.078	.078	.439 (**)	.092	.190	-							
Q4a	r	-.171	.067	.235	.147	.188	.702 (**)	-						
Q4b	r	.025	.470 (**)	.302	.332 (*)	.363 (*)	.440 (**)	.461 (**)	-					
Q4c	r	.139	.540 (**)	.338 (*)	.156	.607 (**)	.297	.400 (*)	.727 (**)	-				
Q5a	r	.382 (*)	.171	.134	-.165	.152	.096	.067	.087	.111	-			
Q5b	r	.022	.144	.402 (*)	.097	.317 (*)	.710 (**)	.809 (**)	.564 (**)	.472 (**)	.230	-		
Q6A	r	-.058	.303	.136	-.274	.053	.171	.319 (*)	.156	.267	.293	.180	-	
Q6b	r	.214	.464 (**)	.057	-.260	.307	-.038	.097	.147	.399 (*)	.155	.092	.458 (**)	-
Q6c	r	.105	.356 (*)	.460 (**)	-.125	.354 (*)	.559 (**)	.454 (**)	.491 (**)	.528 (**)	.205	.561 (**)	.303	.561 (**)

Q1= Sleep Behaviour Self Rating Scale
Q2= The Glasgow Sleep Effort Scale
Q3= Sleep Disturbance Questionnaire:
Q3a= cognitive arousal,
Q3b= physical tension,
Q3c= sleep effort,
Q3d= sleep pattern problems
Q4= Glasgow Content of Thoughts Inventory:
Q4a= active problem solving,
Q4b= sleep and wakefulness
Q4c=somatic and sensory engagement
Q5= Pre-Sleep Arousal Scale:
Q5a= cognitive arousal,
Q5b= somatic arousal
Q6= DBAS-10:
Q6a=need for control over insomnia
Q6b= long term negative consequences of Insomnia
Q6c= immediate negative Consequences of insomnia

* Correlation is significant at the 0.05 level (2-tailed).
** Correlation is significant at the 0.01 level (2-tailed)

5.4.6 Objective Sleep Continuity Data

In this section sleep continuity parameters gathered via PSG are reported and then analysed. Before analysing each of these variables some points common to all variables should be made. As explained in section 5.2.5 PSG data for both baseline and intervention periods were recorded in 37 of the 44 participants. One-way ANOVAs were performed on the baseline mean SOL, WASO TST and S.E. values and no significant differences were found for SOL [$F(2,34)=.067$, n.s.], WASO [$F(2,34)=.298$, n.s.], TST [$F(2,34)=3.23$, n.s.] or S.E. [$F(2,38)=.496$, n.s.] (all $p_s>0.05$).

5.4.6.1 Sleep Continuity Data gathered via Polysomnography

Table 17 presents the means and SD for sleep continuity variables recorded during the second night of baseline and the last night of treatment for the QHRin, QHRout and Control groups.

Table 17 - Mean and SD for Objectively Measured Sleep Continuity Variables at Baseline and End of Treatment for the 3 Groups

	QHRin (N=13)	QHRout (N=16)	Control (N=8)	Total (N=37)
SOL				
Baseline	27.73 (30.0)	24.03 (26.3)	26.31 (24.9)	25.82 (26.7)
End Treatment	33.92 (34.9)	17.35 (19.4)	18.87 (15.3)	23.51 (25.9)
WASO				
Baseline	54.11 (45.4)	49.12 (38.8)	62.50 (32.2)	53.77 (39.3)
End Treatment	70.6 (58.2)	61.62 (49.9)	62.18 (27.9)	64.90 (48.3)
TST				
Baseline	421.73 (45.3)	430.03 (60.3)	376.50 (27.2)	415.54 (52.8)
End Treatment	393.23 (75.9)	411.84 (55.9)	401.56 (70.1)	403.08 (65.2)
S.E.				
Baseline	.83 (.08)	.84 (.08)	.81 (.09)	.83 (.09)
End Treatment	.78 (.13)	.83 (.09)	.82 (.06)	.82 (.10)

Separate mixed model ANOVAs were carried out on each of the sleep continuity variables but no statistically significant differences were found. There were no time,

group or interaction effects for SOL [F(1,34)=.167, n.s.; F(2,34)=.513, n.s.; F(2.34)=1.12, n.s.], WASO [F(1,34)=1.130, n.s.; [F(2,34)=.264, n.s; F(2.34)=.165, n.s.], TST [F(1,34)=.373, n.s.; F(2,34)=1.595, n.s.; F(2.34)=1.121, n.s.] or S.E. [F(1,34)=.438, n.s.; F(2,34)=.626, n.s.; F(2.34)=.824, n.s.]. These results indicate that PSG defined sleep parameters were unchanged at end of treatment as compared to baseline. The positive subjective outcomes regarding sleep continuity parameters were, therefore, not confirmed by outcomes obtained via PSG. It is, therefore, important to consider whether or not sleep continuity variables measured subjectively were correlated with the same variables measured objectively (section 5.3.7.3). First, however, sleep architecture parameters will be analysed

5.4.6.2 Sleep Architecture Data

Table 18 presents the means and SD for sleep architecture variables (Stg 1, Stg 2, Stg 3 &4 and REM) recorded during the second night of baseline and the last night of treatment for the QHRin, QHRout and Control Groups.

Table 18- Mean and SD for Objectively Measured Sleep Architecture Variables at Baseline and End of Treatment for the 3 Groups

	QHRin (N=13)	QHRout (N=16)	Control (N=8)	Total (N=37)
Stg 1				
Baseline	22.69 (17.9)	18.37 (11.5)	17.56 (12.4)	19.71 (14.1)
End Treatment	21.65 (19.4)	20.78 (14.5)	16.06 (10.0)	20.06 (15.4)
Stg 2				
Baseline	255.03 (29.7)	247.18 (40.8)	242.50 (25.4)	248.93 (33.7)
End Treatment	226.26 (60.3)	241.35 (35.3)	251.00 (47.7)	238.14 (47.6)
Stg 3 & 4				
Baseline	31.96 (33.5)	39.15 (23.5)	18.50 (32.2)	32.16 (26.9)
End Treatment	34.07 (28.2)	33.68 (23.5)	17.06 (15.4)	30.22 (24.3)
REM				
Baseline	112.03 (25.7)	125.34 (33.8)	97.93 (19.4)	114.74 (29.7)
End Treatment	111.23 (42.7)	116.03 (30.0)	117.68 (30.6)	114.70 (34.3)

Separate mixed model ANOVAs found no statistically significant differences. Specifically, there were no time [F(1,34)=0.001, n.s.], group [F(2,34)=0.361, n.s.] or

interaction [F(2.34)=0.6.16, n.s.] effects for Stg 1., no time [F(1,34)=1.102, n.s.], group [F(2,34)=0.086, n.s.] or interaction [F(2.34)=1.632, n.s.] effects for Stg 2, no time [F(1,34)=0.367, n.s.], main group [F(2,34)=1.661, n.s.] or interaction [F(2.34)=0.876, n.s.] effects for Stg 3&4 and no time [F(1,34)=0.342, n.s.], no group [F(2,34)=0.699, n.s.] or interaction [F(2.34)=2.200, n.s.] effects for REM.

These results indicated that sleep architecture values gathered via PSG at baseline remained unchanged following the intervention phase.

These analyses and those reported in the previous section suggest that the QHR interventions had no impact on objective sleep continuity or sleep architecture variables. This is a net contrast with estimates of sleep continuity and sleep quality. It is, therefore, important to assess if the subjective and objective sleep parameters obtained in this study correlate.

5.4.7 Subjective and Objective Sleep Continuity: Association

In this section objective and subjective measures of sleep continuity are compared

5.4.7.1 Descriptive Subjective and Objective Sleep Continuity

To determine whether the subjective estimates of sleep on the recording nights were representative of the subjective sleep estimates of non-recording nights, the mean for self-report SOL, WASO, TST and S.E. on the recording nights were compared to the mean for self-report SOL, WASO, TST and S.E. for the baseline and end of intervention weeks for each group (Jacobs and Benson, 1993). As depicted in table 19 the means did not differ significantly, suggesting that the sleep recording night was representative of sleep on non-recording nights .

Table 19 - Wilcoxon Signed Rank Test values and significances comparing sleep diary data of recording night and mean week values at baseline and end of intervention

		Recording Night Values Vs Weekly Mean Values					
Group	**Sleep Variable**	**Baseline**			**Intervention**		
		z	N-ties	p	z	N-ties	p
QHRin							
	SOL	0.15	13	ns	0.45	13	ns
	WASO	1.15	13	ns	0.80	13	ns
	TST	0.31	13	ns	1.50	13	ns
	S.E.	0.52	13	ns	0.53	11	ns
QHRout							
	SOL	0.85	14	ns	2.66	12	ns
	WASO	1.47	14	ns	1.03	14	ns
	TST	1.22	14	ns	1.28	14	ns
	S.E.	0.75	14	ns	1.88	14	ns
Control							
	SOL	1.26	8	ns	1.18	7	ns
	WASO	0.42	8	ns	0.07	8	ns
	TST	0.28	8	ns	0.84	8	ns
	S.E.	0.14	8	ns	0.49	8	ns

In table 20 means and standard deviations of subjective and objective sleep parameters collected during PSG nights are presented. As already discussed a full data set was available for 35 participants.

Table 20 - Mean and SD for Objective and Subjective Sleep Continuity Variables at Baseline and End of Treatment for the 3 Groups

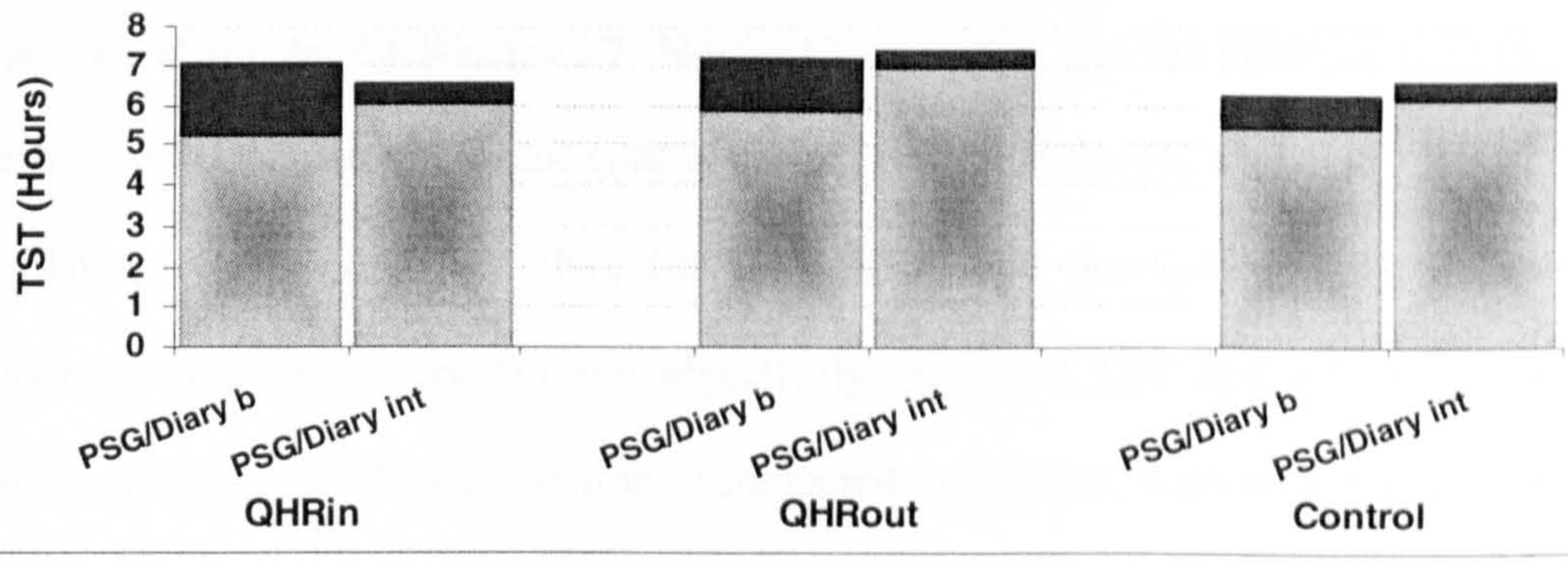
	QHRin (n=13)		**QHRout (n=14)**		**Control (n=8)**	
	PSG	**Diary**	**PSG**	**Diary**	**PSG**	**Diary**
SOL						
Baseline	27.7(30)	63.1(95)	25.8(28)	83.1(127)	26.3(25)	21.0(16)
End Treatment	33.9(35)	40.5(65)	14.1(14)	15.2(15)	18.9(15)	38.5(31)
WASO						
Baseline	54.1(45)	105.9(107)	49.3(39)	70.4(68)	62.5(32)	103.7(45)
End Treatment	70.6(58)	71.1(79)	57.9(42)	43.0(51)	62.2(28)	89.0(59)
TST						
Baseline	421.7(45)	314.6(120)	431.1(65)	355.0(130)	376.5(27)	328.5(51)
End Treatment	393.2(76)	362.2(73)	416.3(58)	427.1(62)	401.5(70)	376.2(102)
S.E.						
Baseline	.83(.08)	.65(.24)	.84(.09)	.69(.23)	.81(.09)	.73(.11)
End Treatment	.79(.13)	.78(.16)	.85(.08)	.88(.10)	.82(.07)	.75(.15)

Inspection of table 20 suggests that the discrepancy between subjectively reported and objectively measured sleep continuity (sub-ob discrepancy) is wider at baseline than at end of treatment.

Taking into consideration that, when only a few PSG nights were recorded, TST and S.E. showed less variability than SOL and WASO (Wohlgemuth, Edinger, Fins and Sullivan; 1999) only TST and S.E. were formally analysed.

Figures 39 and 40 illustrate such sub-ob discrepancies for the TST and S.E. values. The whole rectangle represents the mean PSG defined TST, the lighter (bottom) portion represents the mean diary value. The darker (upper) portion shows the discrepancy between PSG and estimate of TST.

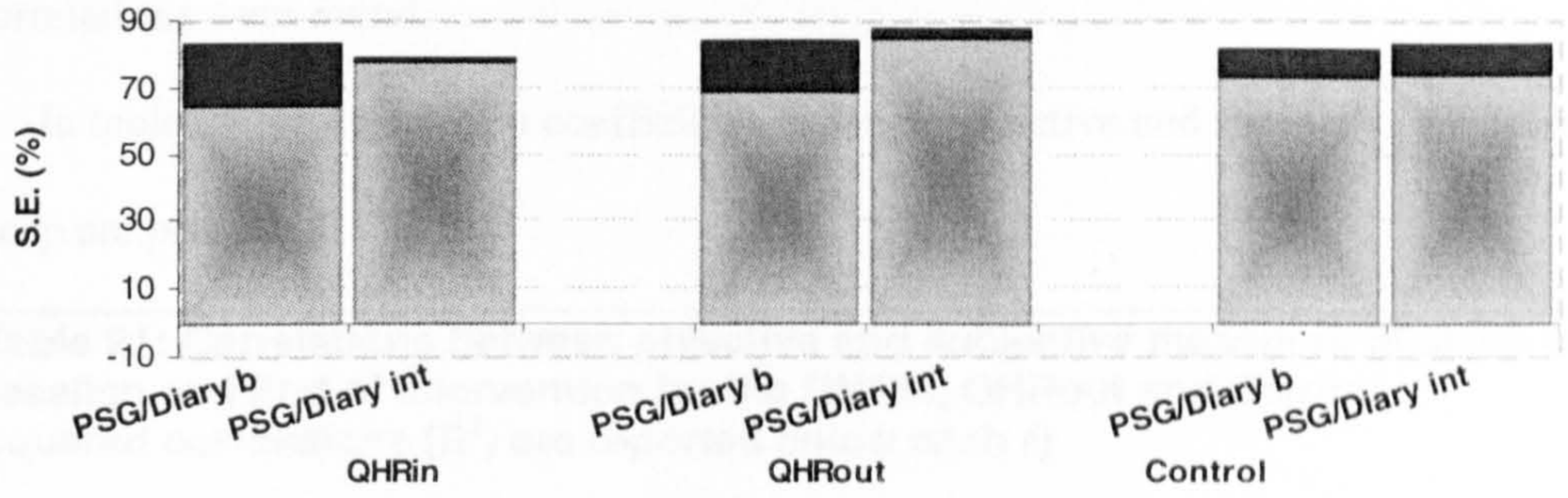
Fig. 39 Subjective and Objective TST at baseline (b) and End of Intervention (int) for the 3 Groups (darker portion indicates the sub-ob discrepancy)



For example, inspection of figure 39 shows that for the QHRin at baseline the discrepancy between PSG and estimate of TST (darker portion of the rectangle) is almost 4 times the discrepancy at the end of the intervention. For the QHRout the discrepancy at baseline is almost 3 times the discrepancy at end of intervention.

Similar observations can be made for the QHRin and the QHRout data on S.E. (fig. 40): the sub-ob discrepancy at the end of the intervention is minimal for the QHRin and for the QHRout as compared to those at baseline. The sub-ob discrepancy for the control group remained, in contrast, unchanged.

FIG. 40 Subjective and Objective S.E. at Baseline (b) and End of Intervention (int) for the 3 groups (darker portion indicates the sub-ob discrepancy)



Wilcoxon Matched Pairs Signed-Ranks Tests showed that there were significant differences between the TST sub-ob discrepancy at baseline and end of intervention for the QHRin [z=2.34, N-ties=13, p=0.019] and the QHRout [z=3.04, N-ties=14, p=0.002] but not for the control group [z=0.50, N-ties=8, n.s.]. Similarly, statistically significant S.E. differences between the sub-ob discrepancies at baseline and end of intervention were found for the QHRin [z=2.7, N-ties=12, p=0.007] and the QHRout [z=3.07, N-ties=14, p=0.002] but not for the control group [z=0.42, N-ties=8, n.s.].

These results confirmed that, for the QHRin and the QHRout, the difference between subjectively reported and objectively measured TST and S.E. values were smaller at the end of the intervention as compared to baseline. With regard to the control group no changes in the sub-ob discrepancy were detected.

5.4.7.2 Associations between Objective and Subjective Measures of Sleep Continuity Variables.

As pointed out in the previous section subjective and objective sleep continuity outcomes contrast sharply and it is, therefore, important to assess if these variables correlate with each other. Spearman's rank order correlations (Rho) were carried out on

sleep diary and PSG data separately for each of the three groups but no significant correlations were found.

In table 21 the correlation coefficients between objective and subjective measures of sleep are presented.

Table 21: Correlations between objective and subjective measures of sleep at Baseline and End of Intervention for the QHRin, QHRout and Control (squared correlations (R^2) are reported below each r)

		2nd Night *Baseline* *Diary - PSG*				*Last Night of Intervention* *Diary - PSG*			
		S.E.	SOL	WASO	TST	S.E.	SOL	WASO	TST
QHRin									
N=13	r	0.55	0.51	0.19	0.46	0.48	0.14	0.29	0.47
	R^2	0.30	0.26	0.03	0.21	0.23	0.05	0.08	0.22
QHRout									
N=14	r	-0.10	0.30	-0.21	0.65	0.40	0.00	0.23	0.52
	R^2	0.01	0.09	0.04	0.42	0.16	0.00	0.05	0.27
Control									
N=8	r	0.56	0.50	0.50	0.40	0.62	0.60	0.18	0.60
	R^2	0.31	0.25	0.25	0.16	0.38	0.36	0.03	0.36

None of the above correlations was significant. The squared correlations show that the proportion of variance in common between the two variables is between 0% and 42%. It should be noted that correlations appear to be variable and unstable; this is not surprising given that correlations are heavily dependent on power (i.e. sample sizes) and sample sizes were very small.

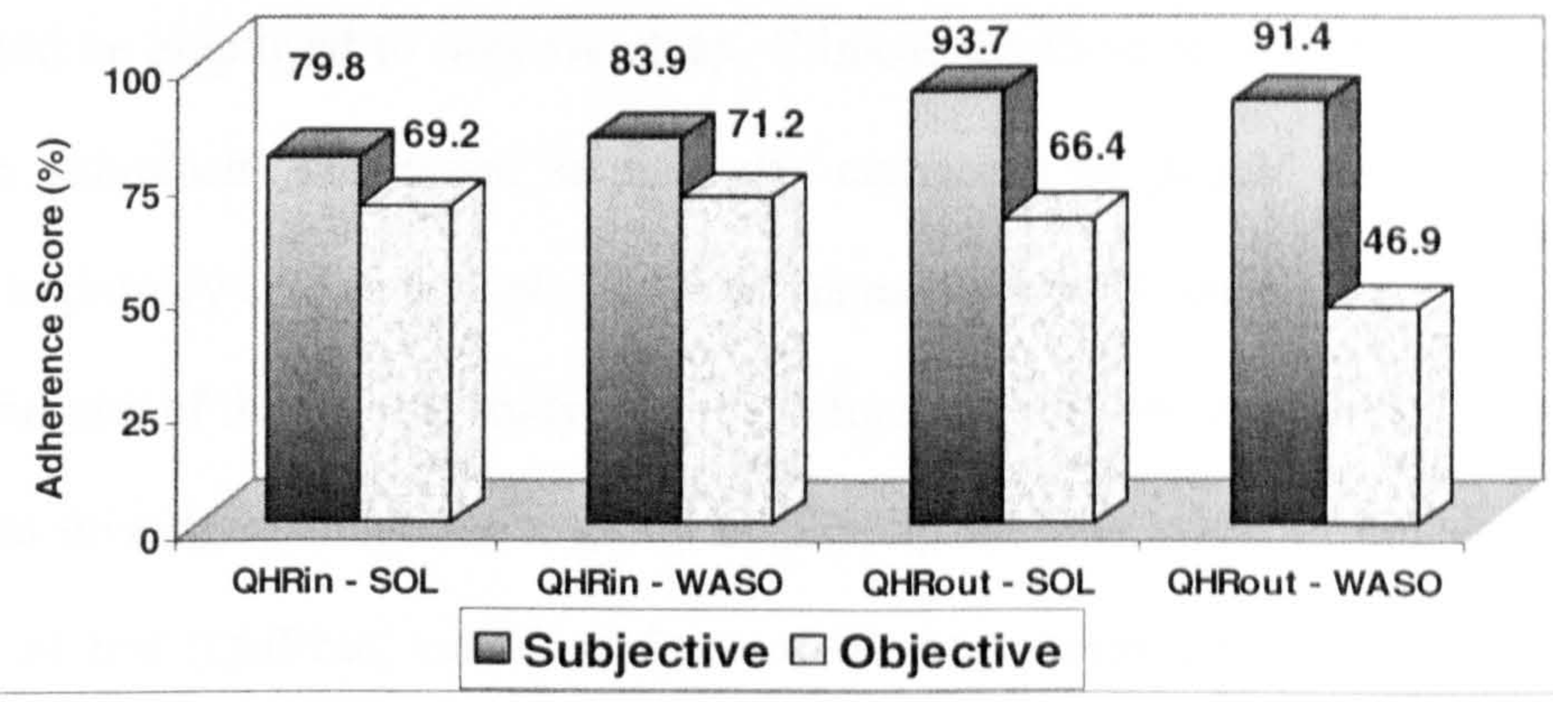
5.4.8 Adherence with the Quarter of an Hour Rule

In this section subjectively and objectively measured adherence with the QHR are reported descriptively.

Subjective measures of adherence were collected during the three weeks of intervention. However, the weekly mean adherence scores were comparable and, therefore, only the first week was considered so as to have a mean of comparison with

objectively measured adherence (one week data) (fig.41). It should be noted that the discrepancy between subjectively reported and objectively measured adherence is wider for the QHRout group than the QHRin. Paired t-tests revealed statistically significant differences between subjective and objective adherence [$t(13)=5.09$, $p<0.0005$] for the QHRout but not for the QHRin.

Fig. 41 - Subjective and Objective Adherence Scores for the QHRin and QHRout Groups



The group SOL change scores from baseline to end of treatment were 9 minutes for QHRin and 38 minutes for QHRout and the WASO change scores were 50 minutes for QHRin and 77 minutes for QHRout. These data give an indication that statistically significant improvements in sleep parameters are obtained even if adherence to the behavioural intervention is not complete. Indeed, the objective data suggest that on average roughly 50% adherence to the rule is sufficient to produce significant changes.

It should be noted that inspection of the raw data on adherence indicated that participants either adhered or did not, making the data more categorical (i.e. adhered, not adhered) than linearly distributed (histograms in appendixes 32a and 32b). Given that correlation cannot be used for categorical data (e.g. gender, yes/no) it was not deemed correct to correlate adherence data to sleep data.

5.5 – Discussion

Following an analysis of the biological, behavioural and cognitive perspectives of insomnia and related therapies it appeared that a closer examination of the single components of stimulus control was warranted and the present experiment was designed. It employed subjective and objective measures of sleep with the aim of investigating whether a simplified behavioural intervention for insomnia, namely the QHR, could be employed to improve sleep. Clinical significance and response curves were also examined. The target sample was composed of people presenting with insomnia in primary care and people recruited via media advertisements.

A second aim of the present study was to explore mechanisms of effect of the QHR. In order to investigate this issue a group of participants was asked to implement the QHR out of bed (QHRout, canonical form of stimulus control) while another group implemented it in bed (QHRin, counter to stimulus control and potentially enhancing sleep incompatible associations). It was reasoned that should the QHR work because it re-establishes the connection between the bedroom environment and falling asleep then the QHRin condition would either not impact on sleep or impact negatively. In addition, sleep related questionnaires were administered to participants at baseline and end of treatment so as to assess if the QHR interventions were associated with changes in cognitive activity (e.g. sleep effort, planning) and arousability.

Finally this study examined the relationship between adherence to treatment and treatment response. The feasibility of employing actigraphy with the added feature of light detection to measure adherence objectively to a behavioural treatment for insomnia was also explored.

5.5.1 - Summary of Results

Seven specific predictions were made. These were based on theoretical considerations pertaining the link between insomnia and poor stimulus control, and the main findings reported in sleep literature.

The first prediction that, following the QHRout, participants' estimates of SOL and WASO would decrease and S.E. would increase was supported. Furthermore, a trend suggesting TST increase was found.

The second prediction was partly supported: the QHRin increased S.E. and trends suggesting an increase in TST and a decrease in WASO were found. However, SOL was not improved following the QHRin.

The third prediction, that the QHR interventions would improve estimates of sleep as compared to the control group, was also partially supported. At the end of the intervention the QHRout group's WASO was shorter, S.E. was greater and trends suggesting longer TST and shorter SOL than those of the control group were found. With regard to the QHRin group trends suggesting longer TST, greater S.E. and shorter WASO than those of the control groups were found.

The fourth prediction, that the QHR interventions would reduce the PSQI total score, was supported. As expected the control group's PSQI total score remained the same.

The fifth prediction was that the QHR interventions would improve perception of sleep quality, restedness and alertness in the morning. Trends indicated that, following either the QHRin or the QHRout, participants considered their quality of sleep better and they felt more rested and alert than at baseline. The control group, in contrast, did not change their ratings of sleep quality, restedness or alertness.

The sixth prediction, that the QHR interventions would meet criteria for clinically significant changes as compared to the control group, was supported. Between 33 and 57% of individuals assigned to the QHR interventions achieved clinically significant changes at post-treatment. Clinically significant improvements (SOL, WASO, TST and S.E.) were evident from the first week of intervention and the magnitude of effect was bigger for the QHRout procedure than the QHRin.

The seventh prediction, that subjective and objective measures of sleep would correlate, was not supported.

The eighth prediction, that treatment response and adherence would be positively correlated could not be tested because adherence data tended to be categorical.

With regard to the examination of sleep related cognitions and arousability, the detection of possible changes might have been hindered by low power. Nonetheless it was found that both QHR interventions resulted in reports of decreased sleep effort, fewer sleep pattern problems and less cognitive and somatic arousal. Additionally the QHRout decreased active problems solving and somatic and sensory engagement. In contrast no changes were found for the control group.

Finally neither the QHR nor the control conditions produced changes in objective sleep continuity or sleep architecture variables. However, and importantly, the discrepancy between subjective and objective measures of sleep continuity diminished following the intervention in the QHR groups but not in the control group.

These findings will be examined and interpreted in turn.

5.5.2 Interpretation of Results

5.5.2.1 Subjective Sleep Continuity Data

The prediction that following the QHR interventions SOL and WASO would decrease and TST and S.E. increase, was fully supported with regard to the QHRout but only partially so for the QHRin. Indeed, following the intervention, the QHRout group took less time to fall asleep, was awake for shorter periods during the night, showed a trend suggesting they slept for longer and increased the time actually asleep while in bed. In contrast, following the intervention, the QHRin took the same time to fall asleep as it did at baseline. Similar to the QHRout, the QHRin group increased the amount of time actually asleep when in bed and trends indicated that the QHRin was less awake during the night and slept longer. Importantly both QHRin and QHRout groups decreased their PSQI total score. The control group's baseline sleep estimates and PSQI total score remained unchanged at the end of treatment.

An important point to make in interpreting these data relates to having detected trends rather than significant effects for TST (both QHRin and QHRout) and WASO (QHRin). Although these results might be due to the QHR interventions not impacting positively on sleep, it is plausible that statistical significance at $\alpha \leq 0.05$ was not achieved due to the Bonferroni correction being very conservative or to the low power to detect significance due to modest sample sizes. However, given the explorative nature of this study, it was felt important reporting trends suggesting that further research is worthwhile.

The findings that the QHRout intervention produced significant changes in SOL and WASO corroborated Tokarz and Laurence's (1974) findings that delivering only the

situational components of the stimulus control package improved sleep. Furthermore, these results are not surprising taking into account that, in this research, the QHRout was posited to encapsulate the essence of the conditioning aspects of stimulus control. Indeed, as discussed in the introduction, stimulus control has consistently improved these two sleep parameters in students, GP recruited patients and older adults (Espie et al, 1989; Lacks et al, 1983a, 1983b, Morin et al, 1988; Puder et al 1983; Sanavio et al, 1990). The present results, therefore, lend support to the idea that the QHRout epitomises stimulus control for insomnia.

Similarly to Davies et al's (1986) report, that counter-stimulus control improved WASO, a trend suggesting that the QHRin improved WASO was found. However, unexpectedly and contrary to Zwart and Lisman's (1979) findings with counter-stimulus control, the QHRin did not impact on SOL. Obviously, the QHRin is only one of the six instructions of counter-stimulus control and, therefore, it is possible that the positive outcomes reported by those authors were not due specifically to the instruction 'sit up in bed if unable to sleep' (QHRin). If this was the case, however, the QHRin should have resulted in no positive outcomes in both cases: at sleep onset and during the night. This would also have been consistent with stimulus control theory as the bedroom environment rather than becoming associated with sleep would signal wakefulness.

It could be speculated that people enjoyed spending some time reading in bed and, therefore, retired to bed earlier than at baseline, with the intention of reading rather than sleeping. However, examination of TIB showed similar values at baseline and end of intervention (8hr & 7min vs. 8hr & 8min). Another possibility is that the 'cosiness' of reading in bed at the beginning of the night was a (positive) reinforcer for staying awake (elicited response). In the middle of the night, in contrast, having to read may have been unpleasant (negative reinforcer) which, in turn, elicited falling asleep quickly. In other

words, falling asleep quickly became a way to stop something unpleasant. Of course taking into account the sample size, it is possible that the power to detect differences was too low (Kazdin and Bass, 1989).

The finding that S.E. increased following the QHR interventions, is consistent with results showing that significant changes in S.E. values were obtained in CBT trials (e.g. Backhaus, Hohagen, Voderholzer and Riemann, 2001, Morin et al, 1994). Unfortunately a direct comparison with stimulus control is difficult to make because during the 1970s and the 1980s (when most studies on single therapies were carried out) S.E. was not reported. Calculation of S.E. from Morin et al's (1994) stimulus control findings (S.E.= 0.69 at baseline and 0.84 at end of treatment), suggests that the present results are similar to those obtained in previous studies with the entire stimulus control package. It is noteworthy that S.E. depends on TIB and TST values and, as discussed above, following the QHR interventions TST increased while TIB remained unchanged: it is, therefore, unsurprising that S.E. increased accordingly.

The present findings open the possibility that conditioning operates on two different, and perhaps additive, dimensions: an external environmental dimension and a cognitive/internal (to the individual) dimension. This possibility would help explain the findings that the QHRout is more efficacious than the QHRin: by getting out of bed faulty conditioning is broken on both levels and appropriate conditioning is re-established. It could be argued that the QHRout impacted positively on sleep for at least three reasons. First, it fosters the association between bedroom and sleep by making the bedroom an eliciting stimulus for falling asleep. Second, it helps breaking the association between being in bed and both negative (e.g. rumination about lack of sleep not sleeping) and/or constructive (planning the day ahead) cognitions. This possibility was suggested by Bootzin and colleagues as a possible explanation of findings that

counter stimulus control instructions resulted in sleep improvements (Bootzin et al, 1991; 2000). Third, it makes falling asleep a behaviour that stops an aversive event (i.e. getting out of bed) from happening or continuing (Bootzin et al, 1972). The QHRin, however, may work only on two levels. For negative reinforcement reasons: falling asleep quickly if awake at night stops an aversive event (i.e. having to sit up in bed and read a book). This would help explaining why the QHRin worked during the night (when sitting up reading is most likely unpleasant) but not at bedtime (when reading a book in bed can be a pleasant activity). In addition, as in the case of the QHRout, having to read breaks the habit to lie in bed thinking and/or worrying.

5.5.2.2 The PSQI Total Score

One of the outcome measures of this study, the PSQI total score, decreased following the QHR interventions while the control group's PSQI did not indicating that individuals in the QHR groups, but not in the control group, had a more positive overall perception of their sleep quantity and quality following intervention. Admittedly, the PSQI at end of intervention did not indicate normal sleep (PSQI<=5). As already proposed in study two this could be because the PSQI at baseline was higher (13 for the QHRin and 14 for the QHRout) than the mean score (10.38) of the normative individuals with insomnia of the Pittsburgh study (Buysse et al, 1989). This difference is especially considerable if the fact that the individuals in this trial had to be sleep medication free is taken into consideration (hence PSQI score ranged from 0 to 18 rather than 0 to 21). It is also important to consider that most treatment studies do not achieve a PSQI score below six and such an outcome might be a (strict) clinical end-point.

Importantly, this result provides further evidence to that gathered via sleep diaries that the QHR interventions were efficacious at improving sleep difficulties.

5.5.2.3 Sleep Quality Data

Following the QHR interventions, trends indicating improvements in perception of sleep quality, restedness and alertness emerged. Sleep continuity improvements were coupled to feeling more refreshed and alert in the morning and to considering the quality of sleep better as compared to baseline. In view of Vincent, Penner and Lewycky's (2006) findings that sleep quality and duration of sleep time were the most important predictors of a patient's perception of having 'improved a lot' after completion of CBT-I, these results are encouraging. Following the QHR interventions people rated their quality of sleep higher as compared to baseline and, as discussed in the previous section sleep duration was increased by about one hour: the two most important predictors of 'My sleep has improved a lot', as defined by Vincent et al's (2006) recent study, were both significantly improved.

With regard to alertness and feeling rested the following comment is noteworthy. The ICSD-R diagnosis of insomnia includes a 'complaint of decreased functioning during wakefulness'. It is likely that the perceived degree of alertness and restedness impact on the overall sense of being able to function well during the day. Morin and Azrin (1988) found that stimulus control decreased self and significant others' ratings of sleep problems' interference with daily functioning. Unfortunately perception of daily functioning (e.g. ability to concentrate, fatigue, irritability) was not measured in this study.

Alternatively, it could be that qualitative aspects of sleep were rated higher than at baseline because participants perceived fewer sleep difficulties (e.g. shorter SOL, longer

TST). Indeed, Neitzer-Semler and Harvey (2005) found that people who were told that actigraphy revealed they had had a bad night would report functioning less well during the day as compared to people told they had had a good night, independently of real sleep continuity.

5.5.2.4 Clinical Changes Associated with the Quarter of an Hour Rule

Clinically significant changes were observed in 33 to 57% of individuals assigned to the QHR interventions and about one third of them returned to normalcy. These results are consistent with Lacks and Powlishta's (1989) claim that about half of people with insomnia show reliable changes following insomnia treatment and that about one third return to normative level of functioning. The present results are also in line with findings from meta-analytic reviews that around 40 to 60% of individuals respond clinically to CBT treatments (Irwin et al, 2006; Morin et al, 1994; Murtagh and Greenwood 1995).

Another important parameter for clinical significance of results is the magnitude of effect. Using Cohen's (1992) scheme, the effect sizes for WASO, TST, S.E. and PSQI for the control group were zero. These results suggest that self-monitoring per se has no impact on these sleep continuity parameters and on the PSQI. Interestingly, a small effect was found for SOL. It should be remembered that SOL deteriorated (increased) in the control group during the intervention phase. Unfortunately the design of the study does not allow exploration of this finding. It could be speculated that self-monitoring made participant focus on their inability to fall asleep and, in so doing, increased worry and sleep effort. These, in turn, impacted negatively on SOL.

In contrast, large effect sizes for PSQI and S.E. and TST were found for both the QHRout (2.3, 0.8 and 1.1 respectively) and the QHRin (1.7, 0.8 and 0.8 respectively).

The TST effect sizes for both QHR interventions were in the large range while those typically reported at end of CBT interventions are in the medium range (Morin et al, 1994=0.42; Murtagh et al, 1995=0.49, Smith et al, 2002=0.46). However, this result might not be surprising if the following is taken into consideration. Both sleep restriction and stimulus control, which are typically included in CBT packages, by curtailing sleep opportunity (amount of time the patient can be in bed) often result in modest TST increases during the treatment phase whereas the QHR does not limit the amount of time to be spent in bed (providing one is not in bed awake for more than a quarter of an hour). Following three weeks of QHR interventions TST was increased by around 60 minutes, in CBT trials, instead, TST is generally increased by only a modest 20-30 minutes at the end of the insomnia treatment with further gains of around 30 minutes at follow-up (Morin et al, 1994; Murtagh et al, 1995).

With regard to WASO medium to large effects sizes, comparable to those reported by meta-analyses of the whole stimulus control package (Morin et al, 1994) and those of meta-analytic studies on behavioural treatment for insomnia (Irwin, et al, 2006; Morin, et al, 1994; Montgomery and Dennis, 2004; Murtagh and Greenwood 1995; Pallesen, et al, 1998; Smith, Perlis, et al, 2002) were found.

Furthermore, the QHRout effect size for SOL was also consistent with meta-analytic findings (medium to large range); in contrast the QHRin effect size for SOL was small. This finding is not surprising given that the average SOL improvement was only 9 minutes.

An important finding was that the magnitude of effects obtained with the QHRout were bigger than those obtained with the QHRin. This result is not dissimilar to that obtained by Davies et al (1986). They found that counter-stimulus control was only moderately effective reducing SOL by only about 30%. Admittedly in their study there

was not a direct comparison between stimulus control and its counter but the magnitude of effects for counter-control was smaller that those typically obtained with stimulus control (Morin et al, 1994; Morin, Mimeault et al, 1999; Murtagh et al, 1995). In order to interpret the finding, that the magnitude of effect was larger for the QHRout, the learning theory perspective is useful. It could be argued that the QHRin, which requires sitting up in bed and reading, is not as effective at facilitating new associations between the bedroom and sleep as the QHRout.

With regard to the response curve, these clinically significant changes were evident from the first week of intervention. It is important, however, to remember that the present study does not allow the assessment of the optimal dose needed to detect improvements, rather it suggests that one single dose of therapy is sufficient to improve sleep parameters. The few minimal telephone contacts following the intervention sessions might have been necessary to maintain improvements or superfluous but this was not tested. In addition the present design does not permit to determine if more sessions would have enabled a larger number of individuals to reach clinically significant changes. Nonetheless these findings give further evidence to Edinger, Wohlgemuth, Radtke and Marsh's (2004) findings that one CBT session had greater efficacy in decreasing WASO than either two or eight sessions .

It is important to note that the present results represent response to the acute treatment phase and whether treatment gains would be maintained, enhanced or would disappear after last contact with the researcher is unknown.

5.5.2.5 Sleep Related Questionnaires

Several interesting findings derived from questionnaires responses. First, baseline endorsement of sleep disruptive behaviour score of the SBRS-R was unchanged at the

end of the intervention. This could be due to lack of power due to small samples. Nonetheless, this finding was not surprising with regard to the QHRin because it required 'remaining in bed when awake' (q. 11) and 'reading in bed' (q. 1). Given that adherence data evidenced good adherence with the QHRin, scores on questions 11 and 8 were likely to increase. Additionally, the QHRin did not require discontinuing behaviours such as watching T.V. (q. 2), taking naps (q. 8), or switching the light off as soon as entering the bed (q. 10). Although the QHRout did not comprise instructions regarding all sleep disruptive behaviours measured in the scale, it did require 'getting out of bed if not asleep in 20 minutes (q. 12) and 'not lying awake in bed if not asleep' (q. 11). It was, therefore, expected to find a change in score following intervention. Indeed inspections of mean values suggested a decrease in sleep disruptive behaviours (supported by repeated measure t-test).

Interestingly, responses to the SDQ evidenced that sleep patterns of the QHRin and QHRout groups had improved following the intervention while those of the control group remained unchanged. These results suggest that despite the fact that QHR interventions did not directly attempt to regularise sleep schedules, participants started to do so themselves. It could be speculated that experiencing sleep improvements by having a routine to follow when not asleep, made participants seek more routine-like sleep habits. Alternatively, this could be the result of sleep monitoring; this interpretation is, however, unlikely because the control group did not show such change.

Analyses of the GSES and SDQ questionnaires indicated that, following the intervention, sleep effort, cognitive arousal and somatic arousal were decreased in the QHRin and the QHRout. These findings are not surprising: having a specific activity to perform if not asleep might have reduced cognition regarding inability to sleep and

consequently effortfully trying to fall asleep (Espie et al, 2006). In turn, this enabled cognitive and somatic de-arousal.

In order to interpret these results it is important to remember that although these questionnaires assess several distinct constructs (worries about not sleeping, sleep effort and sensory and somatic engagement), they are different aspects of a broader construct, that is cognitive activity. Indeed, the questionnaires were found to correlate. Although the QHR interventions did not challenge or address cognition directly, it is likely that they did so implicitly. After all, the QHR intervention implies that 'it is ok giving up trying to sleep' (read if you cannot sleep). It could, therefore, be speculated that behavioural instructions per se, changed sleep cognitions. On the other hand, it is plausible that noticing sleep improvements changed attitudes toward sleep.

It might seem surprising that a behavioural intervention, intending to establish a connection between the bedroom environment and falling asleep, influences such aspects of cognitive activity. However, the QHR requires performing and engaging with one simple, emotionally neutral, activity (i.e. reading something light) which may inhibit self-generated and sleep related and emotionally laden, mental activity. Indeed, previous research evidenced that under light cognitive load sleep onset was shortened (Ansfield, Wegner and Bowser, 1996; Haynes et al, 1981) in people with sleep problems.

It is important to note that the design of this study does not permit to rule out that other generic psychological explanations such as self-efficacy, mastery, placebo effects or demand characteristics (e.g. social desirability, reactivity) might have been responsible for the changes evidenced by this battery of questionnaires.

Before concluding this section, it is important to mention that improvement in sleep continuity was evidenced by subjective sleep diary, and that questionnaires are another

form of self-report. It is, therefore, likely that they follow a similar gradient: if the sleep diary evidences improvements sleep related questionnaires are also likely to depict improvement.

5.5.2.6 Objective Sleep Continuity Data

As discussed in the previous sections, subjective measures of sleep indicated sleep improvements following the QHR intervention. However, when sleep was measured objectively, no significant changes in sleep continuity and sleep architecture parameters were found. Another finding of the present study was that the discrepancy between subjectively reported and objectively measured sleep variables at baseline decreased in the QHR groups but not in the control group. These results are consistent with previous findings that insomnia treatment produces positive outcomes when sleep continuity is measured subjectively without necessarily resulting in changes in objective sleep (see Stepanski, 2000 for a review). In order to provide an interpretation of these findings several considerations must be made.

First, the logical possibility that participants may have presented with paradoxical insomnia (SSM) can be ruled out by baseline PSG evidencing sleep maintenance and/or sleep onset difficulties in most participants. Estimates of sleep difficulties certainly suggested more severe insomnia than the PSG defined sleep implied, however, this is not an atypical finding in the insomnia literature (Stepanski, 2000). Importantly, no PSG improvements were detected at end of intervention. These findings could be interpreted as evidence that the QHR interventions were not actually efficacious in improving sleep i.e. PSG defined sleep did not change following intervention.

Another explanation can be proposed by taking into account that the primary/psychophysiological insomnia diagnostic criteria characterise insomnia as a

difficulty in initiating /maintaining sleep at least three nights a week. These criteria suggest that insomnia does not necessarily occur every night and clinical evidence indicates that night-to-night variability and unpredictability of sleep are considered classic features of insomnia. Indeed, aetiological models of insomnia recognize these characteristics as central to the configuration of insomnia (Espie, 2002; Morin, 1993) and these characteristics have been corroborated by empirical findings (Coates et al, 1981; Edinger et al, 1991, 1997; Vallieres, Ivers, Bastien, Beaulieu-Bonneau and Morin, 2005). It could be argued that the QHR interventions decreased sleep difficulties but did not eliminate completely the possibility of 'bad' nights. Indeed, estimates of sleep suggested reduction of sleep problems following the QHR intervention, but only one third of individuals returned to normalcy: that is sleep difficulties were diminished but still present. Given nightly variability, recording PSG only one night at baseline and one at the end of the intervention might not have permitted detection of changes reliably.

This possibility finds support in investigations of the stability of PSG measures in insomniacs and normal sleepers both at home and in laboratory settings. For example, one recording night resulted in inadequate stability (reliability) of measurement because of unpredictable nightly variations in both normal and poor sleepers (Wohlgemuth et al, 1999; Bootzin, Bell, Halbisch, Kuo, Wyatt, et al, 1995). In view of these findings, Wohlgemuth et al (1999) suggested averaging multiple nights of recording with the number of recording nights varying depending on the sleep variable under examination: SOL and WASO being the most susceptible variables to nightly variations and TST the least. Similarly, Means et al (2003) found evidence of variability across nights for both poor and normal sleepers (the inter-night correlations ranged between 0.37 and 0.64 for the insomnia group and between 0.35 and 0.47 for normal sleepers).

The present findings, that following the QHR PSG sleep did not change, could, therefore, be due to the fact that one single night of PSG evaluation did not provide stability due to nightly variations.

A third possible explanation is that the QHR interventions did not produce sleep changes. Rather, therapeutic outcomes evidenced by estimates of sleep were due to factors such as desire to please the experimenter or simply having the attention of a therapist or thinking that someone finally took the sleep problem seriously.

It is also plausible that participants improved their time perception ability and this resulted in better estimates of time asleep and awake. This would explain the decrease in sub-ob discrepancies following the intervention. Harvey's (2002) model incorporates errors in time perception as an important feature in the experience and maintenance of insomnia. However, it is difficult to relate the QHR interventions to better perception of time passing. The self-monitoring process is, at face value, the best candidate to promote better time perception and Fichten, Creti, Amsel, Bailes and Libman (2005) suggested that self-monitoring could be of benefit by demonstrating that the sleep problem is not as severe as believed. Despite completing a sleep diary each morning for 5 weeks, the control group did not change their estimates of sleep continuity and it seems, therefore, unlikely that self monitoring alone was the reason for a decrease in the sub-ob discrepancies in the active treatment groups. One way to enhance accuracy of time passing is provided by matching, several times, one's feeling of time elapsed with actual elapsed time. It could be argued the QHR interventions increased accuracy because every time participants got up to read they checked the time. However, participants were explicitly asked not to change their clock watching behaviour and to keep completing their diary in the way they did at baseline. Nonetheless, the present study could not directly control for clock monitoring and this remains a possible

explanation, although an unlikely one given that people with insomnia have a tendency to clock monitor and do not seem to benefit from it (Harvey and Schmidt, 2000). Another possibility is that participants in the QHR interventions knew they were being monitored via actigraphy. Although the rationale for wearing an actiwatch did not mention monitoring adherence or sleep, participants might have worked this out and, as a result, might have started to clock monitor so as to provide sleep estimates matching those detected by actigraphy.

A further possibility, that the subjective experience of change is only moderately related to the physiological changes measured by PSG (Engle-Friedman et al, 1992), seems to provide a better alternative.

Several studies have highlighted that patients with insomnia routinely report more severe sleep disturbance than evidenced by traditional PSG measures. In contrast, normal sleepers are generally more accurate in estimating their sleep latency (Bonnet and Arand, 1997; Edinger, Fins, et al, 2000; Means et al, 2003; Mercer et al, 2002; Schramm et al, 1995). For example, Schramm et al (1995) found that normal sleepers sub-ob discrepancies were significantly smaller than those displayed by people with insomnia when PSGs were recorded in a sleep laboratory setting. Similarly, Mercer et al (2002) found that people with insomnia showed a greater discrepancy than good sleepers between PSG-defined sleep and sleep diary reports during home recordings.

In the present study: the sub-ob discrepancies for both TST and S.E. were statistically significantly smaller, for both the QHRin and the QHRout, at the end of the intervention than at baseline. In contrast the control group sub-ob discrepancy did not change. Taking into consideration previous research showing that the sub-ob discrepancies were greater for people with insomnia as compared to normal sleepers, the present results could be, tentatively, interpreted as a shift towards normalcy

following the QHR interventions. The next question, then, would be why the sub-ob discrepancy should be greater in people with insomnia as compared to normal sleepers.

The sub-ob discrepancy, evidenced in people complaining of poor sleep, has been proposed to be due to an important feature of insomnia: elevated levels of fast EEG activity. Indeed, a number of studies employing EEG spectral analysis have reported evidence of elevated beta and gamma activity in people presenting with insomnia as compared to normal sleepers. For example, Merica and Gaillard (1992) performed EEG power spectral analysis of the PSGs of 26 people complaining of insomnia and 28 normal controls. They found that fast EEG frequencies (14.7-30 Hz) dropped more slowly and to a lesser extent in people with insomnia as compared to controls. Similarly Merica et al (1998) found greater fast EEG activity in people with insomnia as compared to controls. Perlis, Merica et al (2001) found evidence of greater relative power (i.e. power within a frequency band divided by the sum of power of all bands) of activity in the 14-45 band in people with insomnia as contrasted to controls and people with depression.

Furthermore, such fast EEG activity has been found to be correlated with subjective-objective TST discrepancies (Jacobs and Benson, 1993; Krystal, Edinger, Wohlgemuth and Michaels, 2002; Merica et al. 1998; Nofzinger et al, 2000; Perlis, Merica et al, 2001; Perlis, Smith, Andrews, Orff and Giles, 2001). For example Krystal et al (2002) found that greater fast EEG frequencies were related to greater TST, S.E. and WASO sub-ob discrepancies. Similarly, Perlis, Merica et al (2001) found that greater NREM fast frequencies correlated with sub-ob TST discrepancies and tended to correlate with sub-ob SOL discrepancies (Perlis, Smith et al, 2001).

The next question is why fast EEG activity should impact on the perception of sleep and wakefulness. According to Perlis et al's (1997) model, cortical arousal (high

frequency EEG activity) enables elevated levels of sensory and information processing and increased formation of long term memory. Information processing, in turn, makes it more difficult to distinguish sleep from wakefulness. Enhanced long-term memory, may interfere with the person's perception of sleep onset. Normally, the mesograde amnesia related to sleep onset does not allow recall of information from periods immediately prior to sleep and this enables the individual to 'know' they were asleep (Perlis, Smith et al, 2001). However, an enhanced recall and retrieval of information is likely to blur the ability to distinguish between sleep and wakefulness.

A well construed study by Mercer et al (2002) lent support to this hypothesis. Normal sleepers and people with insomnia were presented with an auditory stimulus while they were asleep. Interestingly, during PSG defined sleep (stage 2) people with insomnia, differently to normal sleepers, reported that they were awake prior to hearing the stimulus. This finding suggests that they perceived stage 2 sleep as wakefulness. The investigators proposed that nocturnal mentation while awake might be similar to mentation during PSG defined sleep. Importantly, Mercer and colleagues also found that people with insomnia were as accurate as normal sleepers in estimating the time elapsed between presentations of 2 probes. However, unlike normal sleepers, they underestimated how much of such time they were asleep. This finding suggests that poor sleepers' overestimation of time awake was due to perceiving stage 2 sleep as wakefulness rather than to having a distorted time perception per se.

Interestingly, Jacobs and Benson (1993) employed power spectral analysis of EEG activity in a treatment study. They found that, although beta activity remained higher than the matched normal sleepers control group, it decreased following CBT-I. It should be noted that in their study both diary and PSG defined SOL were improved following 10-week intervention. More recently similar findings were obtained by Cervena and

colleagues following an 8-week CBT program (Cervena, Dauvilliers, Espa, Touchon, Matousek, Billiard, and Besset; 2004).

It is important to note that the findings discussed above in relation to sub-ob discrepancies are not in contrast with Harvey's (2002) argument that insomnia is in part a disorder of interpretation. Indeed, the research above provides evidence of misinterpretation: misinterpretation of sleep and wakefulness rather than misperception of passage of time. People with insomnia may misperceive the amount of time they are awake because during PSG sleep defined they are still engaging with stimuli.

The present research did not employ power spectral analysis. However, some indirect evidence for decreased arousal at bed time is provided by sleep related questionnaires: decreased sleep effort, problem solving, cognitive and somatic arousal and somatic and sensory engagement were evidenced at the end of the QHR interventions as compared to baseline.

In sum, lack of changes in PSG defined sleep does not necessarily mean that the QHR intervention was not efficacious in improving sleep. The present results could be due to measurement unreliability (one night PSG recording) or to the fact that conventional sleep staging is not the best tool to measure sleep and sleep improvements. Importantly, the decrease in sub-ob discrepancy, following the QHR interventions, suggests that people may be better at distinguishing PSG defined sleep from wakefulness and it could be speculated that the QHR enabled extinction of classically conditioned cortical arousal (Perlis et al, 1997) i.e. decreased fast EEG activity.

5.5.2.7 Subjective and Objective Sleep Continuity: Association

No significant correlations between sleep continuity data measured subjectively and objectively (i.e. estimated and PSG SOL, estimated and PSG WASO, etc.) were found. This lack of association might be due to the small number of participants in each group which did not allow sufficient power to detect associations. However, very modest associations between PSG and sleep diary data are not uncommon in the literature. For example, Means et al (2003) looked at consistency in estimates of TST with PSG over four PSG nights. They found only a moderate degree of consistency between diary TST and PSG TST. In contrast, Bastien and colleagues found no significant associations between PSG defined SOL, WASO, TST and S.E. and those subjectively reported in either good sleepers or people with insomnia (Bastien, Fortier-Brochu, Rioux, LeBlanc, Daley and Morin, 2003).

5.5.2.8 Adherence with the Quarter of an Hour Rule

The finding that treatment response was not linearly correlated with adherence was unexpected. As discussed in the introduction, adherence to treatment has rarely been measured and, moreover, it has typically been measured only at the end of treatment or at follow up. Riedel and Lichstein (2001) measured adherence daily and found that greater adherence to a consistent 'bed-time' and 'wake-time' was associated with better outcome. However Vincent and Hameed (2003) failed to find a significant association between adherence to a consistent sleep schedule and outcome. Rather, in their study, therapist-rated adherence and spousal-rated adherence were significantly associated with outcome. Unfortunately their study did not permit the assessment of adherence to

components of CBT other than sleep schedule. Consequently outcome could have been associated to other components of CBT.

In the present study, around 80% of participants in the QHRin and around 65% of those in the QHRout adhered to the rule between 60 and 100 % of times according to objective adherence data. Reported adherence was much higher: around 85% of participants in the QHRin and 95% of those in the QHRout reported applying the rule between 60 and 100% of the time. Both sets of data (objective and subjective) indicate that there was adequate adherence with the behavioural procedure and this contrasts with results from previous research (Chambers, 1992; Harvey, Inglis et al, 2002; Schramm et al, 1995). For example, Chambers (1992) found that only around 30% of patients consistently adhered to behavioural components. With regard to stimulus control, Harvey, Inglis et al (2002) found that, at one year follow-up, less than 50% of their sample adhered to sleep scheduling (that is sleep restriction and stimulus control) and in Schramm et al's (1995) study stimulus control was reported to be adhered to "very rarely". Although adherence rates were higher in the present study, this could be due to measurements being taken during the acute treatment phase and whether adherence rates would remain high at follow up is unknown.

As already mentioned participants tended to adhere to the QHR most of the time or not to adhere at all. This dichotomy makes the adherence data more similar to a categorical (i.e. adhered/not adhered) than a linear model. A correlation coefficient, as a numerical measurement of linear association, becomes rather meaningless. Importantly, lack of variance in adherence score also makes it difficult to suggest what amount of adherence is necessary to obtain sleep improvements. On the other hand the finding that sleep improvements were achieved following the QHR interventions despite QHR not

being implemented 100% of the time suggests that complete adherence with the QHR intervention might not be necessary in order to achieve positive clinical outcomes.

On the other hand the fact that people tended to adhere most of the times or not at all is interesting in its own right. Chambers (1992) pointed out, that people might not adhere to instructions because they are poorly understood and/or remembered. Perhaps good adherence was obtained in this study because of the simplicity of mastering and remembering the QHR. Participants had to follow only one instruction, they had a simple flow-chart showing how to correctly implement the QHR and, during the 4 telephone contacts, the researcher checked that the QHR was implemented correctly. One comment made often by participants regarded the difficulty of applying the rule and that some times "it just seemed impossible to get up and read". This comment highlights that difficulty might not only relate to understanding but also to motivation and commitment to follow instructions. It, also, suggests that support and encouragement are important ingredients to 'boost' treatment adherence.

One possibility for the finding that some people did not adhere is that they were unmotivated to read. It would be incorrect to assume that all insomnia patients are literate to the extent that reading at bedtime would be an enjoyable experience. In addition, the QHRout condition might have been particularly aversive: for example fear of disturbing one's spouse (by getting up and returning to bed several times) or a cold sitting-room (although use of a blanket was suggested).

The next chapter regards the theoretical and clinical implications deriving from the present research. In addition issues limiting the present research and informing future studies are presented.

Chapter Six

General Issues and Future Research

In the previous section tentative explanations for the obtained results were given. In this section first the theoretical and clinical considerations are extracted and then the issues limiting this piece of research considered. These considerations are, in turn, used to inform future research needs regarding the QHR.

6.1 Theoretical Implications

The QHR was proposed to epitomise the stimulus control package for insomnia devised by Bootzin (1972) by employing learning theory concepts. The present results evidenced that the QHR was as efficacious as the whole stimulus control in improving estimates of sleep and imply that the QHR may be an essential component of the whole package.

Employing the QHR in two different locations (i.e. out of bed and in bed) permitted exploration of the mechanisms of effects of the QHR. First of all, the finding, that both the QHRout and, to a lesser extent, the QHRin improved sleep, suggest the mechanism of effects of the QHR cannot be attributed solely to falling asleep having returned under the stimulus control of the bed and bedroom environment.

It could be, as suggested by Bootzin (1972) that the QHR interventions become a 'punishment' for wakefulness in bed. This seems a plausible explanation as the majority of participants, when asked about implementing the QHR interventions, reported that sitting up in bed to read and having to get out of their bed during the night was unpleasant. This hypothesis may also help explain why the QHRout produced better

outcomes than the QHRin. Getting out of bed may be more unpleasant than sitting up in bed and, therefore, it might work better in eliciting a response to stop the occurrence of a negative event. In other words the person falls asleep more rapidly so as not to have to apply the rule (i.e. getting out of bed and reading, sitting up in bed with the light on and reading).

Another possibility is that the faulty conditioning hypothesised to underpin insomnia is both to the bedroom itself and to cognitive activity in bed. The finding that following the QHR interventions aspects of cognitive activity decreased substantiates this possibility. Indeed, sleep effort, cognitive arousal, and engagement with external stimuli all decreased. It is noteworthy that stimulus control has previously been thought to operate by decreasing cognition at night (e.g. Hughes and Hughes, 1978; Turner and Ascher, 1979b; Zwart and Lisman, 1979). For example, Zwart and Lisman (1979) proposed that stimulus control and its counter disrupted the connection between the bedroom environment and arousability due to mentation and negative emotion (e.g. worry, frustration). The six participants in Turner and Ascher's study (1979) pointed out that stimulus control helped to break up lying in bed and thinking and that it gave them a means of asserting self-control over the bedtime cognitions by simply getting out of bed. However, this contention was speculative and had received no empirical confirmation. The results obtained by analysing the battery of questionnaires employed in the present study, in contrast, provided some support to this hypothesis.

The present findings obviously fall short of demonstrating a cause effect relationship. While it is plausible that sleep effort and cognitive activity at night decrease in response to sleep improvements, it is also possible that putting effort into the process of falling asleep support and sustain sleep problems (Espie et al, 2006). The researcher speculated that the QHR, by providing participants with something to do that

was not cognitively taxing, decreased both worries about sleep and effort to fall asleep. Espie's (2002) psychobiological model of insomnia proposes that 'letting go' of effort to fall asleep enables an optimal level of de-arousal, which, in turn, facilitates 'letting go of wakefulness' typical of normal sleep. Likewise, Morin (1993) proposed that methods indirectly preventing/stopping emotional and cognitive load in bed were perhaps more efficient that methods drawing attention to mentation. Indeed, rumination theories suggest that one way of reducing negative and repetitive thinking, about a topic relevant to the individual, is distracting attention away from the recurrent thought (Martin and Tesser, 1996; Nolen-Hoeksema, 1991). It could be speculated that this is exactly what is at play in the QHR: the person disengages from attending to the sleep process and from effortfully trying to fall asleep. In addition the QHR faces the person with a paradox (similar to paradoxical intention: Fogle and Dyall, 1983; Broomfield and Espie, 2003)): the person wants 'desperately' to fall asleep and the QHR asks them to give up trying and, instead, read for as long as they need to.

The findings that the magnitude of effect of the QHRout was greater than that of the QHRin could be indicating that the QHRout operates both at an external (e.g. overt activities in bed) and internal (i.e. thinking) level, thus breaking the connection between the bedroom and any sleep incompatible (behavioural and/or cognitive) activity. In contrast the QHRin disrupts only cognitive activity by giving something to do (reading) instead of thinking. Both conditions may facilitate breaking the connection between cognitive activity and the bedroom environment, and at a classical conditioning level break the pairing between the bedroom and anxiety and frustration (reduced cues associated with arousal). The finding that QHR interventions decreased the sub-ob discrepancy supports this possibility. As discussed in the previous chapter, the sub-ob discrepancy has been hypothesised to arise from being cortically alert during NREM

sleep. In addition, the QHRout may also remove the external stimuli (e.g. TV, book, mobile) signalling the presence of a pleasurable activity (e.g. watching a favourite soap opera) if wakefulness is maintained.

Whereas each of these possibilities seems reasonable, further research is obviously needed to determine their relative merits.

The present research highlighted the possibility of measuring adherence to a behavioural treatment objectively via actigraphy and light. This is an important design advance given that retrospective reports of adherence present problems of validity. It is common for participants to confuse adherence behaviour with outcome or for adherence reports to be influenced by degree of improvement (Kazantzis, Deane and Ronan, 2000).

6.2 Clinical Implications

This was the first study showing that a simplified form of stimulus control for insomnia, which takes only 30 minutes to be delivered, produced clinically significant acute therapeutic gains.

The finding that after a single session, lasting 30 minutes, participants improved their sleep is promising and opens a variety of possibilities: the QHR could be a promising first line intervention for insomnia. At present, sleep hygiene is a commonly utilized treatment option despite little evidence of its efficacy as a standalone therapy for insomnia (Stepanski and Wyatt, 2003). Its popularity is most probably due to the fact that it comprises a list of instructions that can be handed in to patients or talked through in a few minutes. The QHR could be delivered, for example, by health professionals in GP practices. Indeed, one study has already shown that GPs themselves

could and were favourable to deliver a stimulus control treatment requiring three sessions (Baillargeon, Demers and Ladoucer, 1998).

Additionally, the fact that the QHR comprises only one instruction, easy to understand and remember, opens clinical applications targeting people who find it difficult to remember and/or to assimilate how to implement more complicated treatments such as multi component CBT, or find it difficult to adhere to elaborated packages due to lack of motivation (e.g. due to depression).

The attrition in the present study was very low in comparison to that in other trials. Perlis, Alloia et al (2000) averaged attrition rates in 20 studies and the average drop out rate was 15.3% with some studies reaching as high as a 42% attrition rate. They suggested that attrition might be due to protocol demands as some individuals found the insomnia interventions too taxing. In the present study only one person dropped out during baseline and none of the people who started the intervention discontinued it. Admittedly 20% of individuals (half of them from the control group) did not undergo the end of intervention PSG. Such a low attrition rate might suggest that the QHR intervention is not only easy to deliver but that is also well accepted and well tolerated by people with insomnia. This is a great strength of this insomnia intervention. Indeed it has been pointed out that notwithstanding the efficacy of a given treatment, if it is too time consuming, or too difficult and not acceptable to patients it is likely to be of little clinical use (Bell, 2001; Morin and Wooten, 1996).

Importantly 26 of the 44 participants who completed the study were people who sought treatment (asking to their GPs) and this group appears to be more refractory to treatment than people actively recruited for clinical research (Stepanski et al, 1989). Although the severity of sleep disturbance is comparable, individuals seeking treatments display higher levels of psychological distress than those responding to recruitment

advertisements. Harvey and Tang (2003) pointed out that findings involving treatment seeking patients by GPs are likely to be the most generalizable to a real world sample followed by patients recruited via advertisements.

Finally the finding that the QHR implemented in bed is efficacious may provide an insomnia intervention for people who have problems getting out of bed (for example elderly people with mobility problems) or patients who find it impossible to comply with getting out of bed. On the other hand, the QHRin was found to have only moderate effectiveness as compared to the QHRout. This finding corroborated Davies and colleagues' (1986) findings with counter-control and suggests that the QHR should require participants to go to another room if not asleep within a quarter of an hour. As proposed by Bootzin and Epstein (2000), only those individuals finding difficult to get out of bed for mobility or other issues (e.g. cold room, pain, fear of disturbing bed partner) should implement the QHR in bed.

As already discussed, the present research evidenced an important application of actigraphy, with the added feature of light detection, as an objective measurement of adherence. Measuring adherence objectively could help a clinician understand if the therapy is not producing the expected effects because of poor adherence with the behavioural instructions. In addition, patients can be shown their nightly adherence, and reasons for non-adhering can be worked through together with the therapist.

6.3 Limitations of the Present Study

These sections discusses several methodological considerations which preclude drawing strong conclusions from this experimental trial. Future research addressing these issues is suggested in the next section (section 6.4).

First, from a clinical perspective, the biggest limitation is the absence of follow-up. Follow-ups are very important to ascertain if therapeutic gains are short lived or will be maintained over time. Although previous research has found that psychological and behavioural therapies for insomnia produced stable changes in sleep pattern over time, with gains maintained up to 12, 24 and 36 months following end of treatment (e.g. Backhaus et al, 2001; Espie, Inglis Tessier and Harvey, 2001; Morin, Colecchi et al, 1999; Sanavio et al, 1990), the lack of follow-up limits the conclusions of the present investigation to the time frame of the study, and thus prevents us from making any comments on sustained effects. However, this was an experimental trial, designed to test the feasibility of a simplified behavioural intervention for insomnia and to shed light on mechanisms of effect.

It is important to remember that means and variability were influenced by the fact that some participants had only sleep onset insomnia, some had only sleep maintenance insomnia and some others met both sets of criteria (mixed insomnia). Although there were no statistical differences among the three groups on averaged measures of SOL and WASO during baseline, the groups did comprise different numbers of participants who had clinically defined mixed, onset or sleep maintenance insomnia. On the one hand, this suggests that positive results are especially meaningful because the sample, like so much of the clinical population was heterogeneous in their presentation of insomnia symptoms. On the other hand this might have had some influence on the treatment outcomes.

With regard to generalisability, the trial included well-screened, healthy, reasonably well-educated and well functioning individuals. Additionally, only people who were not taking sleep medications and who did not present with concurrent psychopathologies (e.g. depression) were recruited. Although this may limit the generalisability to other

samples the experimental nature of the trial (which tested a novel therapy for insomnia) justifies setting more strict criteria. Obviously more studies are needed to determine if similar therapeutic outcomes can be obtained in other samples (e.g. people using hypnotic drugs, people with insomnia and depression).

Another pitfall of the present research is the small sample size. Small sample size might have reduced the power of the statistical analyses to detect potential differences among the conditions. In order to avoid a type II error a sample size of minimum 27 participants per group to detect a large treatment difference and minimum 70 per group to detect smaller differences have been recommended (Kazdin and Bass, 1989). Furthermore, a bigger sample size would permit analysis of the impact of the QHR on people presenting with one insomnia complaint (i.e. only SOL, only WASO, mixed).

In addition, this study is limited by lack of a true control group that is equivalent to a pill placebo in pharmacological studies. However, this is a limitation common to most psychological research and one that is difficult to address. Nonetheless not having a 'pure' placebo makes it difficult to determine what percentage of the variance in outcomes is due to specific therapeutic ingredients (i.e. stimulus control) the measurement process (self-monitoring), credibility of treatment rationales, or to non-specific factors (therapist's attention, patient's expectation).

In the present study the impact of self-monitoring was controlled for, by having a control group whose only task was to monitor daily their sleep for the whole duration of the study. As already discussed in the introduction, credibility of the treatment rationale is a potentially powerful healing aspect of treatment (Baskin, Tierney, Minami and Wampold, 2003): if the rationales are not equivalent the credibility of the treatment and resulting expectations of participants might differ. However, the differential outcomes obtained with the QHRin and the QHRout are unlikely to be due to credibility of the

rationale: the treatment groups' responses to a credibility questionnaire showed no significant differences at pre-treatment regarding the credibility of the treatment. However, the control group was aware of not receiving treatment.

With regard to structural equivalence (Howard, Kopta, Krause and Orlinsky, 1986), in the sense of dose of treatment, this was ensured by providing participants with equal number of therapist-patient contacts of equal length. Importantly both the QHR and control groups were given the same number, of equivalent length, contact. These features of experimental control reduced experimenter bias and controlled for differential therapist's attention. Nonetheless, it is appreciated that non-verbal behaviour might have played an important role in treatment outcomes.

The possibility that differential therapist expectancies could account for the different magnitude of therapeutic outcomes should also be taken into account. Although the therapist was obviously aware of the participant treatment allocation, the experimental methodology followed a rigorous written protocol: the protocols used to deliver QHRin and QHRout were identical apart from the instructions differentiating the QHRout ('go to another room and read') from the QHRin ('sit up in bed and read').

Another methodological flaw of the present research was that the researcher served both as the designer of the study, the therapist and the evaluator of the treatment outcomes: this could have given raise to biases. Although sleep diary, PSG and questionnaires were employed to assess treatment outcome, some judgement on the part of the scorer was still involved. In the present study, however, an independent technologist blind to treatment allocation scored the PSG data. With regard to diary data, questionnaires responses were inputted into a spreadsheet at the end of the study by the researcher who programmed the spreadsheet to calculate the total scores for each questionnaire.

Despite these methodological limitations, the positive outcomes obtained following the QHR intervention and magnitude of effects warrant further investigation.

6.4 Future Research

The present research suggested that the QHR is a promising intervention for insomnia warranting further research. However, the limitations (due to the exploratory experimental nature of the trial) discussed in the previous section do not permit firm conclusions and indicate that future research should address the following issues.

First, this was the first trial involving only one simplified component of stimulus control and the present findings, although promising, need to be replicated by future investigations and in different centres.

Second, due to its exploratory nature, the present study comprised only small samples and lack of significant differences between the QHRin and QHRout might have been due to power issues. Future research should endeavour to employ larger samples. Importantly, since the stability of results is essential for clinical appreciation of the validity of a psychological treatment for insomnia, future research should include a follow up so as to ascertain if improvements obtained with the QHR are maintained over time.

Third, although the dose-response analyses suggested that therapeutic gains were achieved after only one week of QHR implementation, whether one session is sufficient to maintain such gains or some more contacts, even of a few minutes, are necessary to sustain and consolidate changes remain unclear. Future research with different doses (e.g. similar to Edinger et al's (2004) study) should be devised to determine the optimal number of sessions.

Fourth, research aimed at evaluating the incremental benefit of adding other components to the QHR treatment would help the understanding of the contributions of single components to total effectiveness of multicomponent CBT.

Fifth, future research could investigate the feasibility of using the QHR to help to withdraw from medication. For example, Riedel, Lichstein et al (1998) investigated the efficacy of stimulus control in medicated and non-medicated people with insomnia and found that stimulus control was well received in medicated participants and resulted in better outcome as compared to the only medication group. Given the simplicity and limited time necessary to administer the QHR, it could be an important aid to withdrawal programs.

Sixth, cross-sectional research has consistently evidenced that several areas (e.g. daytime functioning, mood and distress) of health related quality of life (HRQoL) are poor among people with insomnia. It seems important to include measures of these domains because patients are generally seeking insomnia treatment because of the real (or perceived) negative effects of insomnia on their daily lives. In this regard, the most often used measurement of HRQoL in sleep research is the SF-36 (Ware, Gandek, et al, 1994). Importantly the only study to date measuring changes in HRQoL following CBT-I showed that CBT-I improved both sleep quality and HRQoL in older adults (Dixon, Morgan, Mathers, Thompson and Tomeny, 2006). Their study also highlighted that the SF-36 is a valid instrument to measure such changes.

Furthermore, participants randomised to the control group were aware that they were not receiving therapy and, therefore, this group did not represent a placebo condition. Future research, could, for example, simply ask participants to 'sit up in bed if not asleep within a quarter of an hour' to explore if the QHR break the connection between wakefulness and cognitive activity. Alternatively it could ask participants to

'go to another room and sit on a chair' to explore if the mechanisms of effect of the QHR are to do purely with learning to associate the bedroom with falling asleep. Just sitting there, would presumably not alter internal states such as rumination. A counter demand condition could also represent a feasible control group.

In addition, the present study highlighted the importance of recording PSG for multiple nights. In particular, Wohlgemuth et al's (1999) study suggested averaging 4-5 nights to obtain reliable measures. Multiple PSG nights are seldom recorded due to the considerable cost, inconvenience and burden on participants but, the night-to-night variability questions the validity of results obtained by only one recording night.

Finally, the findings that sub-ob discrepancies were reduced following the QHR intervention confirmed the importance of carrying out power spectral analysis in addition to standard PSG. A few studies have found higher beta and gamma activity in people presenting with insomnia and that such activity was correlated with subjective-objective TST discrepancies (Perlis et al, 2001). Future research should consider exploring the impact of the QHR on fast EEG activity. Power spectral analysis will be performed on the PSG data collected in this research in the near future.

6.5 Concluding Comments

This study was, of course, preliminary and future research is required to replicate its positive findings and to address the limitations discussed in the previous section. Nonetheless, it provides the first evidence from a randomised controlled trial that the QHR, a simplified intervention extracted from the stimulus control package, is a promising insomnia treatment. Indeed, the QHR produced statistically and clinically

significant sleep changes, comparable to those obtained by more sophisticated and complex CBT packages, within the first week of implementation.

Importantly, the QHR required only 30 minutes face-to face contact and a few telephone contacts lasting less than 10 minutes. It is quick to administer, participants found it easy to understand how to apply the QHR and adherence to the rule was high. These findings have important clinical implications for prevention. Recent meta-analyses (Cole and Dendukuri, 2003; Riemann and Voderholzer, 2003) have suggested that insomnia is a key risk factor for first episode of depression, and chronic insomnia is associated with increased utilisation of health services (Ford and Kamerow, 1989). Early intervention focussing directly on sleep disturbances which is easy to administer and to understand may prove particularly cost-effective.

Furthermore, this study highlighted the importance of investigating critical components of CBT so as to understand whether multi-component programs are superior to mono-therapies.

The QHR reduced cognition at bedtime in that sleep effort, engagement with sensory factors and worry were diminished following the intervention. Furthermore, both the canonical QHRout and its counter (QHRin) impacted positively on sleep. Theoretically these findings lend support to the hypothesis that the mechanisms of effect of the QHR are not solely due to poor situational stimulus control. Rather, classically conditioned cortical arousal might also play a role in insomnia and the QHR indirectly, by asking people to read, breaks the connection between the bed and cognitive engagement.

Admittedly, following the QHR improvements were evidenced by participants' estimates of their sleep but not by PSGs. Obviously this could be due to the QHR not impacting on sleep. However, by taking into account that sub-ob discrepancies

decreased following intervention, it can be argued that conventional PSG staging might not be the best tool to evidence changes. Indeed previous research suggested that beta, alpha and gamma activity (as measured by power spectral analyses) better correlate with estimates of sleep than PSG staging.

The home-based portable polysomnograms were found to be an excellent alternative to laboratory polysomnograms. They were well tolerated by participants, involved minimal disruption to evening activities and the sleep environment, and allowed assessment of sleep naturally in the home environment. This might prove a good alternative to laboratory recordings if multiple nights are to be recorded.

Very importantly this study also provided evidence of the feasibility of measuring adherence to a behavioural treatment objectively via actigraphy with the added feature of light detection.

To conclude, this was the first randomised controlled trial in insomnia research employing one single element of stimulus control needing only thirty minutes to be delivered. The QHR produced statistically and clinically significant outcomes, it suggested that mono-therapies are worth investigating because they could provide a cost-effective alternative to more complex multi-component CBT-I. Theoretically the QHR suggested that stimulus control might operate both on operant and classical conditioning levels and might indirectly break the pattern of ruminating in bed. The QHR is, therefore, a line of research worth pursing on both theoretical and clinical grounds.

References

References

- Alperson, J., & Biglan, A., (1979). Self-administered treatment of sleep onset insomnia and the importance of age. *Behavior Therapy*, 10: 347-356
- American Academy of Sleep Medicine, (2005). *ICSD-2 - The international classification of sleep disorders: diagnostic and coding manual.* Westchester: Illinois
- American Psychiatric Association, *(2000). Diagnostic and statistical manual of mental disorders (4th Edition Text Rev.)* (DSM-IV Rev). Washington DC: APA
- American Sleep Disorder Association Atlas Task Force. EEG arousals: scoring rules and examples. *Sleep*, 15: 174-184
- American Sleep Disorder Association, (1997). *ICSD-R - The international classification of sleep disorders: diagnostic and coding manual.* Rochester: Minnesota
- Ancoli-Israel, A., & Roth, T., (1999). Characteristics of insomnia in the United States: results of the 1991 national sleep foundation survey. I. *Sleep,* 22(suppl. 2), S347-S353
- Anderson, M.W., Zendell, S.M., Rosa, D.P., Rubinstein, M.L., Herrera, C.O., Simons, O., Caruso, O., & Spielman, A.J., (1988). Comparison of sleep restriction therapy and stimulus control in older insomniacs: an update. *Sleep Research*, 17:144
- Ansfield, M.E., Wegner, D.M., & Bowser, R., (1996). Ironic effects of sleep urgency. *Behaviour Research Therapy*, 34(7), 523-531
- Backhaus, J., Hohagen, F., Voderholzer, U., Riemann, D., (2001). Long-term effectiveness of a short-term cognitive behavioural group treatment for primary insomnia. *European Archive of Psychiatry and Clinical Neuroscience,* 251: 35-41
- Backhaus, J., Junghanns, K., Broocks, A., Riemann, D. & Hohagen, F., (2002). Test-retest reliability and validity of the Pittsburgh Sleep Quality Index in primary insomnia. *Journal of Psychosomatic Research,* 53, 737-740
- Baillargeon, L., Demers, M., & Ladouceur, R., (1998). Stimulus-control: nonpharmacological treatment for insomnia. *Canadian Family Physician*, 44, January, 73-79
- Baskin, T.W., Tierney, S.C., Minami, T., & Wampold, B.E., (2003). Establishing specificity in psychotherapy: a meta-analysis of structural equivalence of placebo controls. *Journal of Consulting and Clinical Psychology*, 71(6), 973-979
- Bastien, C.H., & Morin C.M., (2000), Familial incidence of insomnia. *Journal of Sleep Research*, 9, 49-54

- Bastien, C.H., Fortier-Brochu, E., Rioux, I., LeBlanc, M., Daley, M., & Morin, C.M., (2003). Cognitive performance and sleep quality in the elderly suffering from chronic insomnia. Relationship between objective and subjective measures.
- Bernstein, I.L., (1978). Learned taste aversion in children receiving chemotherapy. *Science*, 200: 1302-1303
- Bixler, E.O., Kales, A., Soldatos, C.R., Kales, J.D., & Healy, B., (1979). Prevalence of sleep disorders in the Los Angeles metropolitan area. *American Journal of Psychiatry*, 136: 1257-1262
- Bolles, R.C., (1970). Species-specific defense reactions and avoidance learning. *Psychological Review*, 77: 32-48
- Bonnet, M.H. & Arand, D.L., (1997). Physiological activation in patients with sleep state misperception. *Psychosomatic Medicine*, 59, 533-540
- Bonnet, M.H., & Arand, D.L., (1995). 24 hour metabolic rate in insomniacs and matched normal sleepers. *Sleep*, 18: 581-588
- Bonnet, M.H., (1980). Sleep, performance and mood after the energy-expenditure equivalent of 40 hours of sleep deprivation. *Psychophysiology*, 17(1): 56-63
- Bonnet, M.H., McNulty, T.B., & Arand, D.L., (1993). 24-hour metabolic rate in matched normals and insomniacs. *Sleep Research*, 22, 175
- Bootzin, R.R. & Nicassio, P.M., (1978). Behavioural Treatment for insomnia. In M Hessen, R.M. Eisler & P.M. Mittler Eds, *Progress in behaviour modification*, Vol. 6 (pp.1-45). New York: Academic Press
- Bootzin, R.R., & Epstein, D.R., (2000). Stimulus control. In K.L. Lichstein and C.M. Morin (Eds), *Treatment of late life insomnia* (pp. 167-206). USA: Sage Publications
- Bootzin, R.R., (1972). Stimulus control treatment for insomnia. The Proceedings, 80th Annual Convention
- Bootzin, R.R., (1975). A comparison of stimulus control instructions and progressive relaxation training in the treatment of sleep onset insomnia. Unpublished manuscript, North Western university, USA
- Bootzin, R.R., Bell, I.R., Halbisch, R., Kuo, T.F., Wyatt, J.K., Rider, S.P., & Manbers, R., (1995). Night to night variability in measures of sleep and sleep disorders: a six night PSG study. *Sleep Research*, 24: 121-128
- Bootzin, R.R., Epstein, D., & Wood, J.M., (1991). Stimulus control instructions. In P. Hauri, editor, *Case studies in insomnia*, Plenum, New York, pp 3526-3538

- Borkovec, T.D., & Costello, E., (1993). Efficacy of applied relaxation and CBT in the treatment of generalised anxiety disorder. *Journal of Consulting and Clinical Psychology*, 61, 611-619.
- Borkovec, T.D., (1979). Pseudo-(experiential) insomnia and idiopathic (objective) insomnia: theoretical and therapeutic issues. Advances in Behavior Research and Therapy, 2: 27-55
- Borkovec, T.D., (1982). Insomnia. *Journal of Consulting and Clinical Psychology*, 50: 880-895
- Borkovec, T.D., Grayson, J.B., O'Brien, G.T., & Weerts, T.C., (1979). Relaxation treatment of pseudo-insomnia and idiopathic insomnia: an EEG evaluation. *Journal of Applied Behavioral Analysis*, 12: 37-54
- Bouchard S., Bastien, C., & Morin, C., (2003). Self-efficacy and adherence to cognitive behavioural treatment of insomnia. *Behavioral Sleep Medicine*, 1, 187-199
- Brassington, G.S., King, A.C., & Bliwise, D.L., (2000). Sleep problems as a risk factor for falls in a sample of community dwelling adults aged 64-99. *Journal of the American Geriatric Society*, 48: 1234-1240
- Broomfield, N.M., & Espie, C.A., (2003). Initial insomnia and paradoxical intention: an experimental investigation of putative mechanisms using subjective and actigraphic measurement of sleep. *Behavioural and Cognitive Psychotherapy*, 31, 313-324
- Broomfield, N.M., & Espie, C.A., (2005). Towards a valid reliable measure of sleep effort. *Journal of Sleep Research*, 14: 401-407
- Buysse, D.J., (2003). They see you when you're sleeping, they know when you're awake: commentary on 'wrist' actigraph versus self-report in normal sleepers: sleep schedule adherence and self-report validity' by Collen E. Carney, Laura E. Lajos, and William F. Waters. *Behavioral Sleep Medicine*, 2(3), 144-147
- Buysse, D.J., Reynolds III, C.F., Monk, T. H., Berman, S.R. & Kupfer, D.J., (1989). The Pittsburgh Sleep Quality Index: a new instrument for psychiatric practice and research. *Psychiatric Research*, 28, 193-213
- Carney, C.E., Lajos, L.E., & Waters, W.F., (2003). Wrist actigraph versus self-report in normal sleepers: sleep schedule adherence and self-report validity. *Behavioral Sleep Medicine*, 2(3), 134-143
- Carpenter, J.S. & Andrykowski, M.A., (1998). Psychometric evaluation of the Pittsburgh Sleep Quality Index. *Journal of Psychosomatic Research*, 45(1), 5-13
- Carskadon M.A., & Dement, W.C., (1981). Cumulative effects of sleep restriction on daytime sleepiness. *Psychophysiology*, 18: 107-113

- Carskadon, M.A., Dement, W.C., Mitler, M.M., Guilleminault, C., Zarcone, V.P., & Spiegel, R., (1976). Self-reports versus sleep laboratory findings in 122 drug-free subjects with complaints of chronic insomnia. *American Journal of Psychiatry*, 133: 1382-1388
- Cervena, K., Dauvilliers, Y., Espa, F., Touchon, J., Matousek, M., Billiard, M., & Besset, A., (2004). Effect of cognitive behavioural therapy for insomnia on sleep architecture and sleep EEG power spectra in psychophysiological insomnia. *Journal of Sleep Research*, 13, 385-393
- Chambers, M.J., (1992), Therapeutic issues in the behavioural treatment of insomnia. *Professional Psychology: Research and Practice*, 23(2), 131-138
- Chambers, M.J., (1994). Actigraphy and insomnia: a closer look. *Sleep*, 17(5): 405-408
- Chambless, D.L., Tran, G.Q., & Glass, C.R., (1997). Predictors of response to cognitive-behavioral group therapy for social phobia. *Journal of Anxiety Disorder*, 11, 221-240
- Chesson, A.L., Anderson, W.M., Litner, M., Davila, D., Hartse, K. et al, (1999). Practice parameters for the non-pharmacological treatment of chronic insomnia. *Sleep*, 22: 1128-1233
- Cirignotta, F., Mondini, S., Zucconi, M., Lenzi, P.L. & Lugaresi, E., (1985). Insomnia: an epidemiological survey. *Clinical Neuropharmacology*, 8 (S1): 49-54
- Coates T.J., George, J.M., Killen, J.D., Marchini, E., Hamilton, S., & Thorensen, C.E., (1981). First night effect in good sleepers and maintenance insomniacs when recorded at home. *Sleep*, 1981, 4: 293-298
- Cohen, J., (1988). Statistical power analysis for the behavioural science (2nd ed.). Hillsdale, NJ: Laurence Erlbaum Associates
- Cohen, J., (1992.). A power primer. *Psychological Bulletin*, 112, 155-159
- Cole, M.G, & Dendukuri, N., (2003). Risk factors for depression among elderly community subjects: A systematic review and meta-analysis. *American Journal of Psychiatry* ,160: 1147-1156
- Crosbie, J., (1993). Interrupted time-series analysis with brief single-subject data. *Journal of Consulting and Clinical Psychology*, 61(6), 966-974
- Crowne, D.P., & Marlowe, D., (1960). A new scale of social desirability indipendent of psychopathology, *Journal of Consulting Psychology*, 24(4): 349-354
- Currie, R.S., Wilson, G.K., Pontefract, J.A. & deLaplante, L., (2000). Cognitive-behavioral treatment of insomnia secondary to chronic pain. *Journal of Consulting and Clinical Psychology, 68(3)*, 407-416

- Daan, S., Beersma, D.G.M., & Borbely, A.A., (1984). Timing of human sleep: recovery process gated by a circadian pacemaker. *American Journal of Physiology*, 246: 161-178
- Davies, R., Lacks, P., Storandt, M. & Bertelson, A.D., (1986). Counter control treatment of sleep maintenance insomnia in relation to age. *Psychology and Aging*, 1(3), 233-238
- Dement, W.C. & Vaughan, C., (1999). *The promise of sleep*. Delacorte Press, New York
- DeValck, E., Cluydts, R., & Pirrera, S., (2004). Effect of cognitive arousal on sleep latency, somatic and cortical arousal following partial sleep deprivation. *Journal of Sleep Research*, 13: 295-304
- Devilly, G.J., & Borkovec, T.D., (2000). Psychometric properties of the credibility/expectancy questionnaire. *Journal of Behavior Therapy and Experimental Psychiatry*, 31, 73-86
- Dijk, D.J., & Czeisler, C.A., (1995). Contribution of the circadian pacemaker and the sleep homeostat to sleep propensity, sleep structure, electroencephalographic slow waves, and sleep spindle activity in humans. *Journal of Neuroscience*, 15, 3526-3538
- Dixon, S., Morgan, K., Mathers, N., Thompson, J. & Tomeny, M., (2006). Impact of cognitive behavioural therapy on health-related quality of life among adult hypnotic users with chronic insomnia. *Behavioral Sleep Medicine*, 4(2): 71-84
- Dorsey, C.M., & Bootzin, R.R., (1997). Subjective and psychophysiologic insomnia: an examination of sleep tendency and personality. *Biological Psychiatry*, 41: 209-216
- Dunbar, M., Ford, G., Hunt, K., & Der, G., (2000). A confirmatory factor analysis of the hospital anxiety and depression scale: comparing empirically and theoretically derived structures. *British Journal of Clinical Psychology*, 39, 79-94
- Edinger, J.D. & Sampson, W.S., (2003). A primary care 'friendly' cognitive behavioral insomnia therapy. *Sleep*, 26, 177-182
- Edinger, J.D., & Fins, A.J., (1995). The distribution and clinical significance of sleep time misperceptions among insomniacs. *Sleep*, 18: 232-239
- Edinger, J.D., & Means, M.K., (2005). Cognitive-behavioral therapy for primary insomnia. *Clinical Psychology Review*, 25, 539-558
- Edinger, J.D., Fins, A.I., Glenn, D.M., Sullivan, R.J., Bastian, L.A., Marsh, G.R., Dailey, D., Hope, T.V., Young, M., Shaw, E., & Vasilas, D., (2000). Insomnia and the eye of the beholder: Are there clinical markers of objective sleep disturbances among adults with and without insomnia complaints? *Journal of Consulting and Clinical Psychology*, 68: 586-593
- Edinger, J.D., Fins, A.I., Sullivan, Jr., R.J., Marsh, G.R., Dailey, D.S., Hope, T.V., Young, M., Shaw, E., Carlson, D. & Vasilas, D., (1997). Sleep in the laboratory and

sleep at home: comparisons of older insomniacs and normal sleepers. *Sleep,* 20(12), 1119-1126

- Edinger, J.D., Glenn, D.M., Bastian, L.A., Marsh, G.R., Daile D., Hope, T.V., Young, M., Shaw, E. & Meeks, G., (2001). Sleep in the laboratory and sleep at home II: Comparisons of middle-aged insomnia sufferers and normal sleepers. *Sleep*, 24(7), 761-769
- Edinger, J.D., Marsh, G.R., McCall, V.M., et al (1991). Sleep variability across consecutive nights of home monitoring in older mixed DIMS patients. *Sleep*, 14: 13-17
- Edinger, J.D., Wohlgemuth, W.K., (1999). The significance and management of persistent primary insomnia: the past, present and future of behavioural insomnia therapies. *Sleep Medicine Reviews*, 3(2), 101-118
- Edinger, J.D., Wohlgemuth, W.K., Radtke R.A., Marsh, G.R. & Quillian, R.E., (2001). Does cognitive-behavioural insomnia therapy alter dysfunctional beliefs about sleep? *Sleep,* 24(5), 591-599
- Edinger, J.D., Wohlgemuth, W.K., Radtke, R.A., & Marsh, G.R., (2004). Dose response effects of behavioral insomnia therapy: final report. *Sleep*, 27(AS): A265
- Engle-Friedman, M., Bootzin, R.R., Hazlewood, L. & Tsao, C., (1992). An evaluation of behavioural treatments for insomnia in the older adult. *Journal of Clinical Psychology*, 48(1), 77-90
- Espie, C.A. & Wicklow, A., (1999). Cognitive therapy for insomnia. In Shapiro, C. & Sloan, E.P. (Eds), *Sleep in Psychiatry*. UK: Cambridge University Press
- Espie, C.A., (1991). *The psychological treatment of insomnia*. John Wiley and Sons Ltd: Chichester-England
- Espie, C.A., (2000). Assessment and differential diagnosis. In K.L. Lichstein and C.M. Morin (Eds), *Treatment of late life insomnia* (pp. 81-108). USA: Sage Publications
- Espie, C.A., (2002). Insomnia: conceptual issues in the development, persistence and treatment of sleep disorder in adults. *Annual Review of Psychology*, 53, 215-243
- Espie, C.A., Brooks, D.N. & Lindsay W.R., (1989). An evaluation of tailored psychological treatment of insomnia. *Journal of Behavioural Therapy and Experimental Psychiatry,* 20(2), 143-153
- Espie, C.A., Broomfield, N.M., MacMahon, K.M.A., Macphee, L.M., & Taylor, L.M., (2006). The attention-intention-effort pathway in the development of psychophysiologic insomnia: a theoretical review. *Sleep Medicine Review*, 10: 215-245
- Espie, C.A., Inglis, S.J. & Harvey L., (2001). Predicting clinically significant response to cognitive behaviour therapy for chronic insomnia in general medical practice: analyses

of outcome data at 12 months post-treatment. *Journal of Consulting and Clinical Psychology*, 69(1), 58-66

- Espie, C.A., Inglis, S.J., Harvey, L., & Tessier, S., (2000). Insomniacs' attributions: psychometric properties of the dysfunctional beliefs and attitudes about sleep scale and the sleep disturbance questionnaire. *Journal of Psychosomatic Research*, 48, 141-148
- Espie, C.A., Inglis, S.J., Tessier, S.J., & Harvey L., (2001). The clinical effectiveness of cognitive behaviour therapy for chronic insomnia: implementation and evaluation of a sleep clinic in general medical practice. *Behaviour Research and Therapy*, 39, 45-60
- Espie, C.A., Lindsay, W.R., Brooks, D.N., Hood, E.M. & Turvey, T., (1989). A controlled comparative investigation of psychological treatments for chronic sleep-onset insomnia. *Behavior Research and Therapy*, 27: 79-88
- Fichten C.S., Creti, L., Amsel R., Bailes, S., & Libman, E., (2005). Time estimation in good and poor sleepers, *Journal of Behavioral Medicine*, 28(6): 537-553
- Fogle, D.O., & Dyall, J.A., (1983). Paradoxical giving up and the reduction of sleep performance anxiety in chronic insomnia. *Psychotherapy Theory, Research and Practice*, 20: 21-30
- Foley, D.J., Monjan, A.A., Brown, S.L., Simonsick, E.M., Wallace, R.B., et al, (1995). Sleep complaints among elderly persons: an epidemiologic study of three communities. Sleep, 18: 425-433
- Ford, D.E., & Kamerow, D.B., (1989). Epidemiologic study of sleep disturbance and psychiatric disorders. *JAMA*, 262: 1479-1484
- Frangakis, C.E., & Baker, S.G., (2001). Adherence sub sampling designs for comparative research: estimation and optimal planning. *Biometrics*, 57, 89-908
- Franklin, R.D., Gorman, B.S., Beasley, T.M.& Allison, D.B., (1996). Graphical display and visual analysis. In Franklin, R.D., Allison, D.B. & Gorman, B.S., *Design and analysis of single case research.* New Jersey: Lawrence Erlbaum Associates
- Freedman, R., (1986). EEG power in sleep onset insomnia. *Electroencephalography and Clinical Neurophysiology*, 63: 408-413
- Gallup Organisation, (1995) *Sleep in America.* Princeton, NJ: Gallup
- Gilovich, T., (1991). How we know what isn't so. New York: Free Press
- Gislason, T., & Almqvist, M., (1987). Somatic diseases and sleep complaints. An epidemiologic study of 3.201 Swedish men. *Acta Medica Scandinavica*, 221: 475-481
- Gross, R.T. & Borkovec, T.D., (1982). Effects of cognitive intrusion manipulation on sleep-onset latency of good sleepers. *Behaviour Therapy*, 13: 112-116

- Hall, M., Buysse, D.J., Nowell, P.D., Nofzinger, E.A., Houck, P., Reynolds, C.F., & Kupfer, D.J., (2000). Symptoms of stress and depression as correlates of sleep in primary insomnia. *Psychosomatic Medicine,* 62(2): 227-230
- Harvey, A.G., & Schmidt, D.A., (2000). Clock monitoring in the maintenance of insomnia. *Sleep*, 24 (Suppl.): 334-335
- Harvey, A.G., & Tang, K.Y., (2003). Cognitive behaviour therapy for primary insomnia: can we rest yet? *Sleep Medicine Reviews*, 7(3), 237-262
- Harvey, A.G., (2000a). Sleep hygiene and sleep onset insomnia. *Journal of Nervous Mental Diseases,* 188: 53-55
- Harvey, A.G., (2000b). Pre-sleep cognitive activity: a comparison of sleep onset insomniacs and good sleepers. *British journal of Clinical Psychology*, 39: 275-286
- Harvey, A.G., (2002a). A cognitive model of insomnia. Behaviour Research and Therapy, 40, 869-893
- Harvey, A.G., (2002b). Trouble in bed: the role of pre-sleep worry and intrusions in the maintenance of insomnia. *Journal of Cognitive Psychotherapy*, 16(2), 161-177
- Harvey, A.G., (2002c). Identifying safety behaviors in insomnia. *Journal of Nervous Mental Disorder,* 190: 16-21
- Harvey, K.J. & Espie, C.A., (2004). Development and preliminary validation of the Glasgow Content of Thoughts Inventory (GCTI): a new measure for the assessment of pre-sleep cognitive activity. *British Journal of Cognitive Psychology*, 43: 409-420
- Harvey, L., Inglis, S.J., & Espie, C.A., (2002). Insomniacs' reported use of CBT components and relationship to long-term clinical outcome. *Behaviour Research and Therapy,* 40, 75-83
- Hauri, P., (1981). Treating psychophysiological insomnia with biofeedback. *Archives of General Psychiatry*, 38: 752-758
- Hauri, P.J., & Wisbey, J., (1992). Wrist actigraphy in insomnia. *Sleep*, 15(4): 293-301
- Haynes, S.N., & O'Brien, W.H., (1990). Functional analysis in behaviour therapy. *Clinical Psychology Review*, 10: 649-668
- Haynes, S.N., Adams, A., & Franzen, M., (1981). The effects of pre-sleep stress o n sleep-onset insomnia. *Journal of Abnormal Psychology*, 90: 601-606
- Haynes, S.N., Adams, A.E., West, S., Kamens, L. & Safranek, R., (1982). The stimulus control paradigm in sleep-onset insomnia: a multimethod assessment. *Journal of Psychosomatic Research*, 26(3), 333-339
- Haynes, S.N., Follingstad, D.R., & McGowan, W.T., (1974). Insomnia: sleep patterns and anxiety level. *Journal of Psychosomatic Research*, 18, 69-74

- Haynes, S.N., Price, M.G. & Simons, J.B., (1975). Stimulus control treatment of insomnia. *Journal of Behaviour Therapy and Experimental Psychiatry*, 6, 279-282
- Hoelscher, T.J. & Edinger, J.D., (1988). Treatment of sleep maintenance insomnia in older adults: sleep period reduction, sleep education and modified stimulus control. *Psychology and Aging*, 3(3), 258-263
- Hoffestein, V., & Szalai, J.P., (1993). Predictive value of clinical features in diagnosing obstructive sleep apnea. *Sleep*, 16: 118-122
- Howard, K.I., Kopta, S.M., Krause, M.S., & Orlinsky, D.E., (1986). The dose-effect relationship in psychotherapy. *American Psychologist*, 41: 159-164
- Hughes, R.C., & Hughes, H.H., (1978). Insomnia: effects of EMG biofeedback relaxation and stimulus control. *Behavioral Engineering*, 5:67-72
- Hygge, S., & Ohman, A., (1978). Modelling processes in the acquisition of fear: vicaious electrodermal conditioning to fear-relevant stimuli. *Journal of Personality and Social Psychology*, 36: 271-279
- Irwin, M.R., Cole, J.C., & Nicassio, P.M., (2006). Comparative meta-analysis of behavioral interventions for insomnia and their efficacy in middle aged adults and in older adults 55+ years of age. *Health Psychology*, 25: 3-14
- Jacobs, G.D., Benson, H. (1993). Home-based central nervous system assessment of a multifactor behavioural intervention for chronic sleep-onset insomnia. *Behaviour Therapy*, 24, 159-174
- Jasper, H.H., (1958). The ten-twenty electrode system of the International Federation. *Electroencephalographic Clinical Neurophysiology*, 10: 371-375
- Jean-Louis, G., Kripke, D.F., Cole, R.J., Assmus, J.D., & Langer, R.D., (2001). Sleep detection with an accelerometer actigraph: comparison with Polysomnography. *Physiology Behavior*, 72: 21-28
- Kales, A., & Kales, J.D., (1984). *Evaluation and Treatment of insomnia*. Ney York: Oxford University Press
- Kales, A., Caldwell, A.B., Preston, T.A., Healey, S., & Kales, J.D., (1976). Personality patterns in insomnia. *Archive of General Psychiatry*, 33: 1128-1134
- Katz, D.A. & McHorney, C.A., (1998). Clinical correlates of insomnia in patients with chronic illness. *Archives of International Medicine*, 158: 1099-1107
- Kazarian, S.S., Howe, M.G., & Csapo, K.G., (1979). Development of the Sleep Behavior Rating Scale. *Behavior Therapy*, 10: 412-417

- Kazdin, A.E., & Bass, D., (1989). Power to detect differences between alternative treatments in comparative psychotherapy outcome research. *Journal of Consulting Clinical Psychology*, 57: 138-147
- Kazdin, A.E., & Krouse, R., (1983). The impact of variations in treatment rationales on expectancies for therapeutic change. *Behavior Therapy*, 14, 657-671
- Kazdin, A.E., (1977). Assessing the clinical or applied importance of behaviour change through social validation. *Behavioural Medicine*, 1: 427-452
- Kazdin, A.E., (1979). Therapy outcome questions requiring control of credibility and treatment generated expectancies. *Behavior Therapy*, 10, 81-93
- Kazdin, A.E., (1980). *Research design in clinical psychology*. New York: Harper and Row
- Keppel, G., & Wickens, T.D., (2004). Design and analysis: a researcher's handbook (4th Ed.). Upper Saddle River, NJ:Pearson.
- Kim, K., Uchiyama, M., Okawa, M., Liu, X., & Ogihara, R., (2000). An epidemiological study of insomnia among Japanese general population. *Sleep*, 23: 41-47
- Kohn, L., & Espie, C.A., (2005). Sensitivity and specificity of the insomnia experience: a comparative study of psychophysiologic insomnia, insomnia associated with mental disorders and good sleepers. *Sleep*: 28(1): 1-9
- Kramer, M., (2000). Hypnotic medication in the treatment of chronic insomnia: non nocere! Doesn't anyone care? *Sleep Medicine Review*, 4: 529-541
- Kripke, D.F., (2000). Chronic hypnotic use: deadly risks, doubtful benefit. *Sleep Medicine Reviews*, 4: 5-20.
- Krystal, A.D., Edinger J.D., Wohlgemuth, W.K. & Marsh G.R., (2002). NREM sleep EEG frequency spectral correlates of sleep complaints in primary insomnia subtypes. *Sleep*, 25(6), 630-640
- Krystal, A.D., Edinger, J.D., Wohlgemuth, W.K., & Michaels, C., (2002). A within-subject study of the relationship of non-REM EEG spectral amplitude and the agreement between subjective and objective assessments of sleep time. *Sleep*, 25 (Abstracts supplement): A33-A34
- Kuisk, L.A., Bertelson, A.D., & Walsh, J.K., (1989). Presleep cognitive hyperarousal and affects as factors in objective and subjective insomnia. *Perceptual and Motor Skills*, 69, 1219-1225.
- Kushida, C.A., Chang, A., Gadkary, C., Guilleminault, C., Carrillo, O., & Dement, W.C., (2001). Comparison of actigraphic, polysomnographic, and subjective assessment of sleep parameters in sleep disorders patients. *Sleep Medicine*, 2(5), 389-396

- Lacks, P. & Powlishta, K., (1989). Improvement following behavioural treatment for insomnia: clinical significance, long-term maintenance and predictors of outcome. *Behaviour Therapy*, 20, 117-134
- Lacks, P., Bertelson, A.D., Gans, L. & Kunkel, J., (1983a). The effectiveness of three behavioural treatments for different degrees of sleep onset insomnia. *Behaviour Therapy*, 14, 593-605
- Lacks, P., Bertelson, A.D., Sugerman, J. & Kunkel, J., (1983b). The treatment of sleep maintenance insomnia with stimulus control techniques. *Behaviour Research and Therapy*, 21(3), 291-295
- Lamarche, C.H., & Ogilvie, R.D., (1997). Electrophysiological changes during the sleep onset period of psychophysiological insomniacs, psychiatrics insomniacs and normal sleepers. *Sleep*, 20: 724-733
- Lawrence, P.S., & Tokarz, T., (1976). A comparison of relaxation training and stimulus control. Paper presented at *Association for the Advancement of Behavior Therapy*, New York
- Leger, D., Guilleminault, C., Dreyfus, J.P., Delahaye, C., & Paillard, M., (2000). Prevalence of insomnia in a survey of 12.778 adults in France. *Journal of Sleep Research*, 9: 35-42
- Lichstein K.L., & Rosenthal, T.L., (1980). Insomniacs' perception of cognitive versus somatic determinance of sleep disturbance. *Journal of Abnormal Psychology*, 89: 105-107
- Lichstein, K.L. & Riedel, B.W., (1994). Behavioural Assessment and treatment of insomnia: a review with an emphasis on clinical application. *Behaviour Therapy*, 25, 659-688
- Lichstein, K.L., & Fischer, S.M., (1985). Insomnia. In M. Hersen and A.S. Bellack (eds). *Handbook of clinical behavior therapy with adults*. NY: Holt, Reinehart and Winston
- Lichstein, K.L., & Morin, C.M, (2000). Treatment overview. In Treatment of late-life insomnia, K.L. Lichstein & C.M. Morin (eds), Sage Publications: California
- Lichstein, K.L., (1988), Sleep compression treatment of an insomnoid. *Behaviour Therapy*, 19, 625-632
- Lichstein, K.L., Durrence, H.H., Taylor, D.J., Bush, A.J. & Riedel, B.W., (2003). Quantitative criteria for insomnia. *Behavior Research and Therapy*, 41, 427-445
- Lichstein, K.L., Riedel, B.W., Wilson, N.M., et al, (2001). Relaxation and sleep compression for late-life insomnia. A placebo-controlled trial. *Journal of consulting Clinical Psychology*, 69: 227-239

- Martin, L.L. & Tesser, A., (1996). Some ruminative thoughts. In Wyer R.S. (Ed), *Advances in social cognition* (vol. IX, 1-47). NJ: Erlbaum
- McGhie, A. & Russell, S.M., (1962). The subjective assessment of normal sleep. *Journal of Mental Science*, 108: 642-654
- McGrogan, N., Braithwaite, E. & Tarassenko, L., (2001). Biosleep: a comprehensive sleep analysis system. *Proceedings of EMBC*
- McGrogan, N., Braithwaite, E., & Tarassenko, L., (2001). Biosleep: a comprehensive sleep analysis system. *Proceedings of EMBC*
- Means, M.K., Edinger, J.D., Glenn, D.M., & Fins, A.I., (2003). Accuracy of sleep perceptions among insomnia sufferers and normal sleepers. *Sleep Medicine*, 4: 285-296
- Mendelson, W.B., James, S.P., Garnett, D., Sack, D.A., & Rosenthal, N.E., (1986). A psychophysiological study of insomnia. *Psychiatry Research*, 19: 267-284
- Mercer, J.D., Bootzin R.R., & Lack L.C., (2002). Insomniacs' perception of wake instead of sleep. *Sleep*, 25(5): 564-571
- Merica, H., & Gaillard, J.M., (1992). The EEG of the sleep onset period in insomnia: a discriminant analysis. *Physiology and Behavior*, 52: 199-204
- Merica, H., Blois, R. & Gaillard, J.M., (1998). Spectral characteristics of sleep EEG in chronic insomnia. European Journal of Neuroscience, 10: 1826-1834
- Monk, T.H., Buysee, D.J., Rose, L.R., Hall, J.A., & Kupfer, D.J., (2000). The sleep of healthy people. Chronobiology International, 17(1): 49-60
- Monroe, L.J., (1967). Psychological and physiological differences between good and poor sleepers. *Journal of Abnormal Psychology*, 72: 255-264
- Montgomery, P., & Dennis, J., (2004). A systematic review of non-pharmacological therapies for sleep problems in later life. *Sleep Medicine Reviews*, 8, 47-62
- Morgan, K., (2003). Daytime activity and risk factors for late-life insomnia. *Journal of Sleep Research*, 12: 231-238
- Morgan, K., Dixon, S., Mathers, N., Thompson, J. & Tomeny, M., (2004). Psychological treatment for insomnia in the regulation of long-term hypnotic drug use. *Health Technology Assessment*, 8(8): III
- Morgan, K., Dixon, S., Mathers, N., Thompson, J. & Tomeny, M., (2003). Psychological treatment for insomnia in the management of long-term hypnotic drug use: a pragmatic randomised controlled trial. *British Journal of General Practice*, 53(497): 923-928
- Morgan, K., Thompson, J., Dixon, S., Tomeny, M., & Mathers, N., (2003). Predicting longer-term outcomes following psychological treatment for hypnotic-dependent chronic insomnia. *Journal of Psychosomatic Research*, 54: 21-29

- Morin C.M., Colecchi C., Stone, J., Sood R. & Brink, D., (1999). Behavioural and pharmacological therapies for late-life insomnia: a randomised controlled trial. *JAMA*, 281(11), 991-999
- Morin, C.A., Colecchi, C.A., Ling, W.D. & Sood, R.K., (1995). Cognitive behaviour therapy to facilitate benzodiazepine discontinuation among hypnotic-dependent patients with insomnia. *Behaviour Therapy*, 26, 733-745
- Morin, C.M. & Azrin, N.H., (1987). Stimulus control and imagery training in treating sleep maintenance insomnia. *Journal of Consulting and Clinical Psychology*, 55(2), 260-262
- Morin, C.M. & Azrin, N.H., (1988). Behavioural and cognitive treatments of geriatric insomnia. *Journal of Consulting and Clinical Psychology*, 56(5), 748-753
- Morin, C.M. & Espie, C.A., (2003). *Insomnia: a clinical guide to assessment and treatment*. Kluwer Academic/Plenum Publisher, New York
- Morin, C.M., & Wooten, V., (1996). Psychological and pharmacological approaches to treating insomnia: critical issues in assessing their separate and combined effects. *Clinical Psychological Review,* 16: 521-542
- Morin, C.M., (1993). Insomnia: psychological assessment and management. New York: Guilford Press
- Morin, C.M., (2005). Psychological and behavioural treatment for primary insomnia. In *Principles and Practice of Sleep Medicine*. Edited by M.H. Kryger, T. Roth & W.C. Dement. Eslevier Saunders: Philadelphia PA
- Morin, C.M., Bastien, C., & Savard, J., (2003). Current status of cognitive-behavior therapy for insomnia: evidence for treatment effectiveness and feasibility. In *Treating Sleep Disorders: Principles and Practice of Sleep Medicine*. Edited by M.L. Perlis & K.L. Lichstein. Wiley and Sons: New Jersey
- Morin, C.M., Culbert, J.P., & Schwartz, S.M., (1994). Nonpharmacological interventions for insomnia: a meta-analysis of treatment efficacy. *American Journal of Psychiatry*, 151, 1172-1180
- Morin, C.M., Hauri, P.J., Espie, C.A., Spielman, A.J., Buysse, D.J., & Bootzin, R.R., (1999). Nonpharmacological treatment of chronic insomnia. *Sleep*, 22(8), 1134-1156
- Morin, C.M., Kowatch, R.A., Barry, T. & Walton, E., (1993). Cognitive-behaviour therapy for late-life insomnia. *Journal of Consulting and Clinical Psychology*, 61(1), 137-146
- Morin, C.M., Mimeault, V., & Gagne', A., (1999). Nonpharmacological treatment of late-life insomnia. *Journal of Psychosomatic Research*, 46(2), 103-116

- Morin, C.M., Savard, J., & Blais F.C., (2000). Cognitive therapy. In K.L. Lichstein and C.M. Morin (Eds), *Treatment of late life insomnia* (pp. 207-230). USA: Sage Publications
- Morin, C.M., Stone J., Trinkle, D., Mercer, J. & Remsberg, S., (1993). Dysfunctional beliefs and attitudes about sleep among older adults with and without insomnia complaints. *Psychology and Aging*, 8(3), 463-467
- Morley, S., (1987). Single case methodology in behavior therapy. In SJ Lindsay andGE Powell (eds), *A handbook of clinical adult psychology*. London: Gower Press
- Murtagh, D.R.R., & Greenwood, K.M., (1995). Identifying effective psychological treatments for insomnia: a meta-analysis. *Journal of Consulting and Clinical Psychology*, 63(1), 79-89
- National Institute of Health State Of the Science Conference Statement, (2005). Manifestations and management of chronic insomnia in adults. June, 13-15, 2005. Sleep, 28(9): 1049-1057
- Neitzer-Semler, C., & Harvey, A.G., (2004). Monitoring of sleep-related threat in primary insomnia: development and validation of the sleep associated monitoring inventory (SAMI). *Psychosomatic Medicine*, 66: 242-250
- Nelson, J., & Harvey, A.G., (2003). Pre-sleep imagery under the microscope: a comparison of patients with insomnia and good sleepers. *Behavior Research and Therapy*, 41, 273-284
- Nicassio, P.M., Mendlowitz, D.R., Fussell, J.J. & Petras, L., (1985). The phenomenology of the pre-sleep state: the development of the pre-sleep arousal scale. *Behaviour Research and Therapy*, 23(3), 263-271
- Nofzinger, E.A., Price, J.C., Metzer, C.C., Buysse, D.J., Villemagne, V.L., Miewald, J.M., Sembrat, R.C., Steppe, D.A., & Kupfer, D.J., (2000). Towards a neurobiology of dysfunctional arousal in depression: the relationship between beta EEG power and regional cerebral glucose metabolism during NREM sleep. *Psychiatry Research-Neuroimaging*, 98(2): 71-91
- Nolen-Hoeksema, S., (1991). Responses to depression and their effects on the duration of depressive episodes. *Journal of Abnormal Psychology*, 100: 569-582
- Norton, G.R. & De Luca, R.V., (1979). The use of stimulus control procedures to eliminate persistent nocturnal awakenings. *Journal of Behaviour Therapy and Experimental Psychiatry*, 10, 65-67
- Ohayon, M.M., & Hong, S.C., (2002). Prevalence of insomnia and associated factors in South Korea. *Journal of Psychosomatic Research*, 53: 593-600

- Ohayon, M.M., & Partinen, M., (2002). Insomnia and global sleep dissatisfaction in Finland. Journal of Sleep Research, 11: 339-346
- Ohayon, M.M., (2002). Epidemiology of insomnia: what we know and what we still need to learn. *Sleep Medicine Reviews*, 6(2), 97-111
- Pallesen, S., Nordhus, I.H., & Kvale, G., (1998). Non-pharmacological interventions for insomnia in older adults: a meta-analysis of treatment efficacy. *Psychotherapy Theory Research and Practice Training*, 35: 472-481
- Pardey, J., Roberts, S., Tarassenko, L. & Stradling, J., (1996). A new approach to the analysis of the human sleep/wakefulness continuum. *Journal of Sleep Research*, 5, 201-210
- Perlis, M., Aloia, M., Millikan, A., Boehmer, J., Smith, M., Greenblatt, D., & Giles, D., (2000). Behavioral treatment of insomnia: a clinical case series study. *Journal of Behavioral Medicine*, 23(2), 149-161
- Perlis, M.L., Giles, D.E., Mendelson, W.B., & Bootzin, R.R., (1997). Psychophysiological insomnia: the behavioural model and a neurocognitive perspective. *Journal of Sleep Research, 6*, 179-188
- Perlis, M.L., Giles, D.E., Mendelson, W.B., Bootzin, R.R., & Wyatt, J.K., (1997). Psychophysiological insomnia: the behavioural model and a neurocognitive perspective. *Journal of Sleep Research*, 6(3): 179-188
- Perlis, M.L., Kehr, E.L., Smith, M.T., Andrews, P.J., Orff, H. & Giles, D.E., (2001). Temporal and stagewise distribution of high frequency EEG activity in patients with primary and secondary insomnia and in good sleeper controls. *Journal of Sleep Research*, 10(2): 93-104
- Perlis, M.L., Merica, H., Smith, M.T. & Giles, D.E., (2001). Beta EEG activity and insomnia. *Sleep Medicine Review*, 5(5), 365-376
- Perlis, M.L., Pigeon, W.R., & Riemann, D., (2006). The treatment of chronic insomnia: state of the science. *Sleep Medicine Reviews* (submitted)
- Perlis, M.L., Smith, M.T., & Pigeon, W.R., (2005). Aetiology and pathophysiology of insomnia. In *Principles and Practice of Sleep Medicine*. Edited by M.H. Kryger, T. Roth & W.C. Dement. Eslevier Saunders: Philadelphia PA
- Perlis, M.L., Smith, M.T., Andrews, P.J., Orff, H., & Giles, D.E., (2001). Beta/gamma EEG activity in patients with primary and secondary insomnia and good sleeper controls. *Sleep*, 24(1), 110-117

- Perlis, M.L., Smith, M.T., Orff, H.J., Andrews, P.J., & Giles, D.E., (2001). The mesograde amnesia of sleep may be attenuated in subjects with primary insomnia. *Physiology and Behavior*, 74(1-2): 71-76
- Pollack, C.P., Tryon, W.W., Nagaraja, H., & Dzwonczyk, R., (2001). How accurately does wrist actigraphy identify the states of sleep and wakefulness? *Sleep*, 24(8), 957-965
- Prochaska, J.O., DiClemente, C.C., & Norcross, J,C., (1992). In search of how people change. Applications to addictive behaviors. *American Psychologist*, 47: 1102-1114
- Puder, R., Lacks, P. Bertelson, A.D. & Storandt, M., (1983). Short term stimulus control treatment of insomnia in older adults. *Behaviour Therapy*, 14, 424-429
- Rechtschaffen, A., & Kales, A. (Eds), (1968). *A manual of standardised terminology: techniques and scoring system for sleep stages of human subjects.* Los Angeles, UCLA Brain Information Service
- Richardson, G.S., & Roth, T., (2001). Future directions in the management of insomnia. *Journal of Clinical Psychiatry*, 62(Suppl. 10), 39-45
- Riedel, B.W., & Lichstein, K.L., (2000). Insomnia and daytime functioning. *Sleep Medicine Reviews*, 4: 277-298
- Riedel, B.W., & Lichstein, K.L., (2001). Strategies for evaluating adherence to sleep restriction treatment for insomnia. *Behavior Research and Therapy*, 39, 201-212
- Riedel, B.W., Lichstein, K.L., Peterson, B.A., Epperson, M.T., Means, M.K. & Aguillard, R.N., (1998). A comparison of the efficacy of stimulus control for medicated and non-medicated insomniacs. *Behaviour Modification*, 22(1), 3-28
- Riemann, D., & Voderholzer, U., (2003). Primary insomnia: A risk factor to develop depression? *Journal of Affective Disorders*, 76: 255-259
- Roberts, S., & Tarassenko, L., (1992). New method of automated sleep quantification. *Medical and Biological Engineering and Computing*, 30(5): 509-517
- Roth, T., & Ancoli-Israel, S., (1999). Daytime consequences and correlates of insomnia in the United States: results of the national sleep foundation survey. II. *Sleep*, 22(suppl. 2), S354-S358
- Russell, J. & Lader, M., (1992). *Guidelines for the prevention and treatment of benzodiazepine dependence*. Mental Health Foundation, London
- Sadeh, A. & Acebo, C., (2002). The role of actigraphy in sleep medicine. *Sleep Medicine Reviews*, 6(2), 113-124
- Salkovskis, P.M., (2002). Empirically grounded clinical interventions: cognitive-behavioural therapy progress through a multi-dimensional approach to clinical science. *Behavioral and Cognitive Psychotherapy*, 30: 3-9

- Sanavio, E., (1988). Pre-sleep cognitive intrusions and treatment of onset-insomnia. *Behaviour Research and Therapy,* 26(6), 451-459
- Sanavio, E., Vidotto, G., Bettinardi, O., Rolletto, T. & Zorzi, M. (1990). Behaviour therapy for DIMS: comparison of three treatment procedures with follow-up. *Behavioural Psychotherapy*, 18, 151-167
- Schramm, E., Hohagen, F., Backhaus, J., Lis, S. & Berger, M., (1995). Effectiveness of a multicomponent group treatment for insomnia. *Behavioural and Cognitive Psychotherapy,* 23, 109-127
- Seligman, M.E.P., (1971). Phobias and preparedness, *Behavior Therapy*, 2: 307-320
- Semler, C.N., & Harvey, A.G., (2005). Misperception of sleep can adversely affect daytime functioning in insomnia. *Behaviour Research and Therapy*, 43(7): 843-856
- Shettleworth, S.J., (1972). Constraints on learning. In Lehrman, D.S., Hinde, R.A., & Shaw, E., (eds), *Advances in the study of behavior*, vol. 4, New York: Academic Press
- Shocat, T. Umphress, J, Israel, A.G. and Ancoli-Israel, S. (1999) Insomnia in primary care patients. Sleep; 22: Suppl. 2: S359-S365
- Simon, G. & VonKorff, M., (1997). Prevalence, burden and treatment of insomnia in primary care. American Journal of Psychiatry, 154: 1417-1423
- Singleton, N., Bumpstead, R., O'Brien, M., Lee, A., & Meltzer, H., (2001). Psychiatric morbidity among adults living in private households, 2000. London: HMSO
- Sloan, E.P., Hauri, P., Bootzin, R., Morin, C., Stevenson, M., & Shapiro, C.M., (1993). The nuts and bolts of behavioral therapy for insomnia. *Journal of Psychosomatic Research*, 37(S1), 19-37
- Smith MT, Perlis, ML, Park A, Giles DE, Pennington JA, Buysee, D. (2002). Behavioral treatment vs pharmacotherapy for Insomnia - A comparative meta-analyses. *American Journal of Psychiatry*; 159: 5-11
- Smith, L.J., Nowakowski, S., Soeffing, J.P., Orff, H.J., & Perlis, M.L., (2003). The measurement of sleep. In: *Treating Sleep Disorders: principles and practice of behavioural sleep medicine*. Ed. M.L. Perlis & K.L. Lichstein, John Wiley& Sons, Inc., New Jersey, NJ
- Smith, M.T., Perlis, M.L., Park, A., Smith, M.S., Pennington, J., Giles, D.E. & Buysse, D.J., (2002). Comparative meta-analysis of pharmacotherapy and behaviour therapy for persistent insomnia. *American Journal of Psychiatry*, 159, 5-11
- Smith, M.T., Smith, L.J., Nowakowski, S., & Perlis, M.L., (2003). In *Treating Sleep Disorders: Principles and Practice of Sleep Medicine*. Edited by M.L. Perlis & K.L. Lichstein. Wiley and Sons Ltd: New Jersey

- Spielman, A., Caruso, L., & Glovinsky, P., (1987). A behavioural perspective on insomnia treatment. *Psychiatrics Clinics of North America*, 10: 541-553
- Spielman, A.J., Saskin, P., & Thorpy, M.J., (1987). Treatment of chronic insomnia by restriction of time in bed. *Sleep*, 10, 45-56
- Bell, L., 2001). What predicts failure to engage in or drop out from treatment for bulimia nervosa and what implications does this have fro treatment? *Clinical Psychology and Psychotherapy*, 8(6): 424-435
- Kazantzis, N., Deane, F.P., & Ronan, K.R., (2000). Homework assignments in cognitive and behavioural therapy: a meta-analysis. *Clinical Psychology: Science and Practice*, 7: 189-201
- Spilker, B., (1991). *Guide to clinical trials*. New York, Raven
- Stepanski, E., Koshorek, G., Zorick, F., Glinn, M., Rohers, T., & Roth, T., (1989). Characteristics of individuals who do or do not seek treatment for insomnia. *Psychosomatics*, 30: 421-427
- Stepanski, E.J., & Wyatt, J.K., (2003). Use of sleep hygiene in the treatment of insomnia. *Sleep Medicine Reviews*, 7(3), 215-225
- Stepanski, E.J., (2000). Behavioral therapy for insomnia. In M.H. Kryger, T. Roth, & W.C. Dement Eds. *Principles and Practice of Sleep Medicine*. Philadelphia, PA: W.B. Saunders Company, 647-656
- Sterling, T.D., Rosenbaum, W.L., & Weinkam, J.J., (1995). Publication decisions revisited: the effect of the outcome of statistical tests on the decision to publish and vice-versa. *American Statistician*, 49: 108-112
- Sugarman, J.L., Stern, J.A., & Walsh, J.K., (1985). Daytime alertness in subjective and objective insomnia: some preliminary findings. *Biological Psychiatry*, 20: 741-750
- Tokarz, T., & Lawrence F, (1976). *An analysis of temporal and stimulus factors in the treatment of insomnia.* Paper presented at the Eighth Annual Meeting of the Association for the Advancement of Behavior Therapy. Chicago
- Toler, C.H., (1978). The treatment of insomnia with relaxation and stimulus control instructions among incarcerated males. *Criminal Justice and Behaviour*, 5(2), 117-130
- Turner, R.M. & Ascher, L.M., (1979a). Controlled comparison of progressive relaxation, stimulus control, and paradoxical intention therapies for insomnia. *Journal of Consulting and Clinical Psychology*, 47(3), 500-508
- Turner, R.M. & Ascher, L.M., (1979b). A within subject analysis of stimulus control therapy with severe sleep-onset insomnia. *Behaviour Research and Therapy*, 17, 107-112

- Urdan, T.C., (2001). *Statistics*. Lawrence Earlbaum Association, Publishers, Mahhawa: NJ
- Vallieres A., Belanger, L., Bastien, C.H., Espie, C.A., & Morin, C.M., (2005). Are psychophysiological insomniacs sleeping better away from home? *Sleep* (28): A231
- Vallieres, A., Ivers, H., Bastien, C.H., Beaulieu-Bonneau, S., & Morin, C.M., (2005). Variability and predictability of chronic insomnia. *Journal of Sleep Research*, 14: 447-453 2005
- Vincent, N., Penner, S., & Lewycky, S., (2006). What predicts patients' perceptions of improvement in insomnia? *Journal of Sleep Research*, 15: 301-308
- Vincent, N.K., & Hameed, H., (2003). Relation between adherence and outcome in the group treatment of insomnia. *Behavioral Sleep Medicine*, 1(3), 125-139
- Ware, J.E., Gandek, B., et al, (1994). The SF-36 Health Survey: development and use in mental health research and the IQOLA project. *International Journal of Mental Health*, 23: 49-73
- Webb, W.B., & Schneider-Helmert, D., (1988). Awakenings: subjective and objective relationships. *Perceptual and Motor Skills*, 59: 63-70
- Webb, W.B., (1988). An objective behavioural model of sleep. *Sleep*, 11(5), 488-496
- Wicklow, A., & Espie, C.A., (2000). Intrusive thoughts and their relationship to actigraphic measurement of sleep: towards a cognitive model of insomnia. *Behaviour Research and Therapy*, 38, 679-693
- Wohlgemuth, W.K., Edinger, J.D., Fins, A.I., & Sullivan Jr, R.J., (1999). Stability of PSGH measures in elderly insomniacs and normals both at home and in the lab. *Psychophysiology*, 36:233-244
- Yves, E., Morin, C., Cervena, K., Carlander, R., Beset, A., & Billiard, M., (2003). Family studies in insomnia. *Sleep*, 26, A304
- Zigmond, A.S., & Snaith, R.P., (1983). The hospital anxiety and depression scale. *Acta Psychiatrica Scandinavia*, 67, 361-370
- Zwart, C.A. & Lisman, S.A., (1979). Analysis of stimulus control treatment of sleep-onset insomnia. *Journal of Consulting and Clinical Psychology*, 47(1), 113-118

List of Tables and Figures

Appendix 1: Diagnostic criteria for psychophysiological insomnia (ICSD-II)

Psychophysiological Insomnia – Diagnostic criteria

a. The patient's symptoms meet the criteria for insomnia.
b. The insomnia is present for at least one month.
c. The patient has evidence of conditioned sleep difficulty and/or heightened arousal in bed as indicated by one or more of the following
    i. Excessive focus on and heightened anxiety about sleep
    ii. Difficulty faling asleep in bed at the desired bedtime or during planned naps, but no difficulty falling asleep during other monotonous activities when not intending to sleep
    iii. Ability to sleep better away from home than at home
    iv. Mental arousal in bed characterised either by intrusive thoughts or a perceived inability to volitionally cease sleep-preventing mental activity
    v. Heightened somatic tension in bed reflected by a perceived inability to relax the body sufficiently to allow the onset of sleep
d. The sleep disturbance is not better explained by another sleep disorder, medical, or neurological disorder, mental disorder, medication use, or substance use disorder.

Appendix 3: Sleep Diary

SLEEP DIARY: We are interested in finding out what normal sleepers do differently from poor sleepers both during a typical night and a night when sleep does not come that easily. Plese fill in the diary each morning upon awakening. Thanks very much!! Marina Malaffo

If you want more information you can contact me on:

0141 2113902 (University of Glasgow, Psychological Medicine, Gartnavel Royal Hospital)

Please circle as appropriate: I am generally (last 6 months) a **GOOD / POOR** Sleeper

I am: M / F My occupation: **Student / Employed / Unemployed / Retired** Age: .....

Diary entries started: ...../...../2004							
What time did you rise from bed this morning?							
What time did you wake up this morning?							
Did you wake up because of: 1: Alarm clock/asked to be waken up 2: Noise or light 3: Just woke up							
What time did you go to bed last night?							
What time did you switch off the light intending to sleep?							
How long did you take to fall asleep?							
If you had difficulties falling asleep, what did you do? (e.g. just lie in bed trying, read a book, listened to the radio, made myself a hot drink)							
How many times did you wake up during the night?							
How long were you awake, in total, during the night after you first fell asleep?							
What did you do when you woke up during the night if you could not fall asleep immediately? (e.g. just lie there, read a book, listened to the radio, made myself a hot drink)							
Did you take any nap yesterday? If yes for how long?							
How much units of alcohol did you take yesterday?							
Measuring the quality of your sleep:	*****	****	****	****	****	****	****
How was your sleep? 0=very bad; 1=bad; 2=so so; 3=good; 4= very good							
How rested do you feel this morning? 0=not at all; 1=little; 2=so so; 3=quite; 4=very rested							
How alert do you feel this morning? 0=not at all; 1=little; 2=so so; 3=quite; 4=very alert							

Appendix 4: The PSQI

Questionnaire A **ID:**

The following questions relate to your sleep habits **during the past month** only. Your answers should indicate the most accurate reply for the majority of days and nights in the past month. Please answer all the questions.

1. During the past month, when have you usually gone to bed at night?

Usual bed time: . . . . . .

2. During the past month, how long (in minutes) has it usually taken you to fall asleep each night? **Number of minutes: . . . . . .**

3. During the past month, when have you usually got up in the morning?

Usual getting up time: . . . . . .

4. During the past month, how many hours of actual sleep did you get at night? This may be different to the number of hours you spend in bed.

Hours of sleep per night: . . . . . .

5. During the past month, how often have you had trouble sleeping because: you:

	Not during the past month	Less than once a week	Once or twice a week	Three or more time a week
a. Cannot get to sleep within 30 minutes				
b. Wake up in the middle of the night or early morning				
c. Have to get up and use the bathroom				
d. Cannot breathe comfortably				
e. Cough or snore loudly				
f. Feel too cold				
g. Feel too hot				
h. Had bad dreams				
i. have pain				

j. Other reasons, please describe them and how often it happened: . . . . . .

. . . . . .

6. During the past month, how would you rate your sleep quality overall? (Please circle your choice)

Very Good Fairly good Fairly bad Very bad

7. During the past month, how often have you taken medicines (prescribed or 'over the counter') to help you sleep? (Please circle your choice)

Not during the past month	Less than once a week	Once or twice a week	Three or more time a week

8. During the past month, how often have you had trouble staying awake while driving, eating meals, or engaging in social activities? (Please circle your choice)

Not during the past month	Less than once a week	Once or twice a week	Three or more time a week

9. During the past month, how much of a problem has it been for you to keep up enough enthusiasm to get things done? (Please circle your choice)

No problem at all	Only a very slight problem	Somewhat of a problem	A very big problem

10. Do you have a bed partner or a room mate?

No bed partner or room mate	Partner or room mate in other room	Partner or room mate in same room but not same bed	Partner in same bed

If you have a room mate or bed/partner ask him/her how often in the past month you have:

	Not during the past month	Less than once a week	Once or twice a week	Three or more time a week
a. Loud snoring				
b. Long pauses between breaths while asleep				
c. Legs twitching or jerking while asleep				
d. Episodes of disorientation or confusion during sleep				

e. Other restless while you sleep: please describe and write down how often: . . . . . .

. . . . . .

Appendix 5: The Sleep Diary (study 2 and 3)

SLEEP DIARY: BASELINE / INTERVENTION ID: . . . . . .

This sleep diary helps both us and yourself to find out your sleep pattern and how you feel about your sleep. **In order to get a precise picture of your night-by-night sleep and your feelings about it, it is essential that you fill the diary in every morning.** Please, remember that there is not a right or wrong answer but just the way you slept each night.

Diary entries started: DATE:							
What time did you rise from bed this morning?							
What time did you wake up this morning?							
What time did you go to bed last night?							
What time did you switch off the light intending to sleep?							
How long (mins) did you take to fall asleep?							
If you had difficulties falling asleep, what did you do? (for example: just lie in bed, read a book, think, …)							
How many times did you wake up during the night?							
How long were you awake, **in total**, during the night after you first fell asleep?							
What did you do when you woke up during the night if you could not fall asleep immediately?							
Did you take any nap yesterday? If yes for how long?							
How much units of alcohol did you take yesterday?							
Did you take any sleeping tablets? (Y / N)							
Measuring the quality of your sleep:	*****	****	****	****	****	****	****
How was your sleep? 0=very bad; 1=bad; 2=so so; 3=good; 4= very good							
How rested do you feel this morning? 0=not at all; 1=little; 2=so so; 3=quite; 4=very rested							
How alert do you feel this morning? 0=not at all; 1=little; 2=so so; 3=quite; 4=very alert							

Appendix 6: The HADS

ID: Date: . . . . . .

QUESTIONNAIRE B

Please read each item below and circle the reply which comes closest to how you have been feeling in the past week. Don't take too long over your replies, your immediate reaction to each item will probably be more accurate than a long, thought-out response.

1. I feel tense or 'wound up'

Most of the time — A lot of the time

From time to time, occasionally — Not at all

2. I still enjoy the things I used to enjoy

Definitely as much — Not quite so much

Only a little — Hardly at all

3. I get a sort of frightened feeling as if something awful is about to happen

Very definitely and quite badly — Yes, but not too badly

A little, but it does not worry me — Not at all

4. I can laugh and see the funny side of things

As much as I always could — Not quite so much now

Definitely not so much now — Not at all

5. Worrying thoughts go through my minds

A great deal of the time — A lot of the time

Not too often — Very little

6. I feel cheerful

Never — Not often

Sometimes — Most of the time

7. I can sit at ease and feel relaxed

Definitely — Usually

Not often — Not at all

8. I feel as if I am slowed down

Nearly all the time — Very often

Sometimes — Not at all

9. I get a sort of frightened feeling like 'butterflies' in the stomach

Not at all — Occasionally

Quite often — Very often

10. I have lost interest in my appearance

Definitely — I don't take as much care as I should

I may not take quite as much care — I take just as much care as ever

11. I feel restless as if I have to be on the move

Very much indeed — Quite a lot

Not very much — Not at all

12. I look forward with enjoyment to things

As much as I ever did — Rather less than I used to

Definitely less than I used to — Hardly at all

13. I get sudden feelings of panic

Very often indeed — Quite often

Not very often — Not at all

14. I can enjoy a good book or radio or television programme

Often — Sometimes

Not often — Very seldom

Please check that you have answered all the questions. Thank you.

Appendix 7: Monitoring the use of the 'Quarter of an hour rule':

We want to evaluate how difficult it is to follow this rule. Please fill out the following table.

For each day put your answer in the box:

a. **If you followed the rule write 'Y', if you did not write 'N'.**

b. **If the question is not applicable to you write 'N/A'**

c. **When you are asked 'How many times..?', write the number applying to you.**

Please remember that there is not a right or wrong answer but only what happened each night.

Weeks: 1 / 2 / 3								
RULE	**TODAY'S DATE**							
When you first went to bed:								
1. Did you take less than a quarter of an hour to fall asleep once you switched off the light?								
2. **If not**, did you go to another room?								
3. **If you went to another room**, after about how many minutes did you do so?								
4. In order to follow the rule, how many times altogether did you have to go up before falling asleep?								
6. If you had to get up more than once, after how long did you do so?	2nd time							
	3rd time							
	4th time							
After you fell asleep:								
7. If you awoke during the night, did you fall back asleep within a quarter of an hour?								
8. **If you did not fall back asleep** within a quarter of an hour, did you go to another room?								
9. **If you went to another room**, after about how many minutes did you do so?								
10. In order to follow the rule, how many times altogether did you have to get up during the night?								
11 If you had to get up more than once, after how long did you do so?	2nd time							
	3rd time							
	4th time							
12. **Please describe how it was to follow the rule by circling the number that applies to you**								
	(1= not difficult at all,	1	1	1	1	1	1	1
	2= a little difficult,	2	2	2	2	2	2	2
	3= difficult,	3	3	3	3	3	3	3
	4= very difficult,	4	4	4	4	4	4	4
	5= extremely difficult)	5	5	5	5	5	5	5

Appendix 8: Flowchart QHRout

Preparation for The 'QUARTER OF AN HOUR RULE'

Decide which room you are going to if you cannot fall asleep within 15 minutes

Room

. . . . . .

↓

Think about a couple of activities you would want to carry out in the chosen room if you don't fall asleep within 15 minutes

Activities

. . . . . .

. . . . . .

↓

Put the material needed for your chosen activities in the room where you are going to carry them out

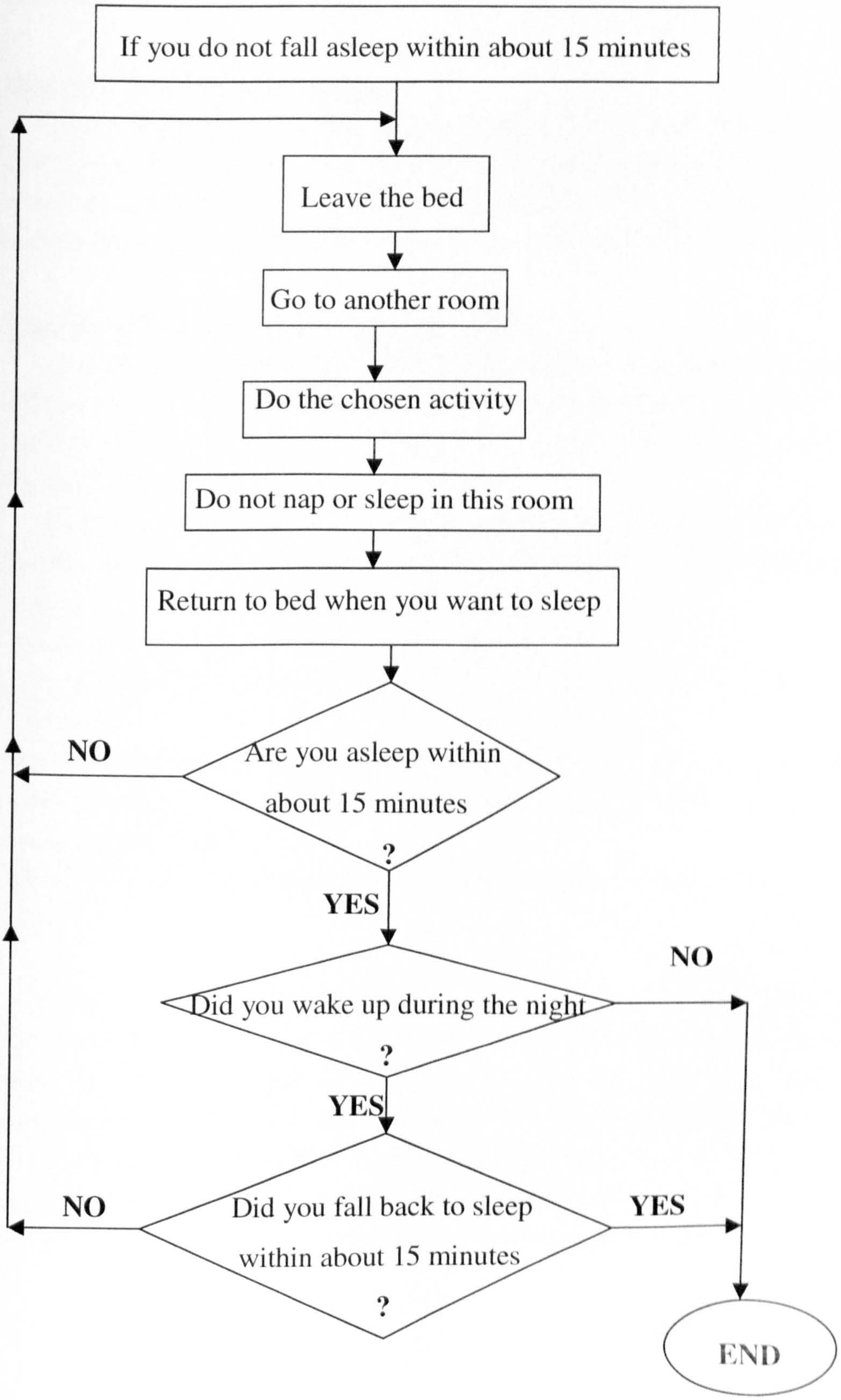
↓

Make sure the room is prepared so that you will be comfortable enough in it if awake during the night (e.g. warm, with something to drink)

↓

END

The 'QUARTER OF AN HOUR RULE'



Appendix 9: Participants' information sheet

This information sheet will help you to understand why we are doing this research and what it will involve. Please take some time to read it and feel free to contact me if something is not clear (my contact numbers are listed below).

What is the purpose of this study?

The aim of this study is to explore how a sleep intervention called 'the quarter of an hour rule' affects sleep. The study investigates how you feel about your sleep before, during and after the sleep intervention, but it also investigates your brain activity during the night. The equipment (called EEG) we have to measure brain activity is easy to use at home and is completely safe.

What do I have to do if I take part in this study?

If you decide to participate you would, first of all, be asked some questions about your sleep. In addition you will have one questionnaire to fill in. These would enable the researcher to find out if your sleep problems are the kind of problems we are investigating in this research study. This will take approximately 10 minutes.

If your sleep difficulties are of the types studied in this research and you wanted to participate in the study, you would be involved in the research for 6 to 8 weeks. During this weeks you would have to:

. meet with the researcher twice for about 45 minutes each,

. meet another 5 times (for about 5 minutes each) so as to be given or return the EEG equipment

. record your brain activity, using the EEG home recording equipment provided, for 6 nights

. wear an 'actiwatch' (very similar to a wrist watch), for the whole period of the study, so as to measure your body movements

. fill in a sleep diary each morning for the whole duration of the study (about 5 minutes every day)

During the first meeting with the researcher you will have the opportunity to ask any questions you wish to about the study, you will then be asked you to sign a consent form to participate in the study. The researcher will then tell you how to fill in the 'sleep diary', how to use the EEG recording equipment and how to use the actigraph. It might seem a lot of information to take in: for this reason you will be given a short and easy memo about what you are requested to do.

During the second meeting, the researcher will explain to you the 'quarter of an hour rule' intervention for insomnia and you will be asked to start implementing it from that very first night.

How much time will be required of me?

All together to fill in the sleep diary each morning, meet with the researcher, use the EEG home recording equipment, you will be required to devote around 7 hours of your time over a period of 6 to 8 weeks.

Benefits to you in participating

Participating in this study will help you to understand your sleep pattern better. In addition, it is hoped that the sleep intervention will help you to improve your sleep, although this cannot be guaranteed. Finally the information gathered with this research will be helpful to devise better treatment for insomnia.

Will my answers be covered by confidentiality?

All information about you collected during this research will be kept strictly confidential. The only people who will have access to your data are the researcher herself and her supervisor. Your name will not be kept together with your data (an ID number will be used instead).

What if I change my mind?

You can decide to withdraw from the study at any time without having to explain your decision.

Thank you very much for taking the time to read this information. I would be very pleased if you decide to participate in this research study.

If there is something you are not clear about, or if you wish more information, please do not hesitate to contact:

- *Ms Marina Malaffo, Department of Psychological Medicine, University of Glasgow*
  Tel: 0141 211 3902 or 07745811303, e-mail: 0219144m@student.gla.ac.uk

If you would like to speak to an independent advisor about any concerns you may have regarding this study please contact:

- *Ms. Heidi-Louise Kelly (Research Fellow) The sleep clinic, Department of Psychological Medicine, University of Glasgow*
  Tel: 0141 211 3943 , e-mail: Hk16b@clinmed.gla.ac.uk

Appendix 10: Consent Form

An investigation of sleep patterns pre and post insomnia intervention

CONSENT FORM

1. I have read and understood the 'Information Sheet' containing information regarding the study I am going to participate in.
2. The researcher explained further any points I asked to. I have been given a contact telephone number so as to be able to discuss points at a later time should I wish to.
3. In addition I had the opportunity to discuss any points and doubts with an independent source.
4. I understand that I can decide to withdraw from this study at any moment without having to explain my reasons for wishing to do so.
5. I have been assured that any information I provide is strictly confidential and that should the study be published the names of the participants to this study won't be available to third parties.

I, ... agree to participate in this study.

(Name and Surname in print)

Signature: ..... Date: .....

Appendix 11: EEG Recording INSTRUCTIONS

EEG Recording : **INSTRUCTIONS**

a. You have to wear and record your EEG on two consecutive nights (Monday and Tuesday nights).

b. Setting up your EEG Recording equipment.

Step 1: Plug 4 electrodes into the Trackit Poly channels:

One electrode plugged in 'NEUT'	
One electrode plugged in 'REF'	
One electrode plugged in '2a'	
One electrode plugged in '1a'	

Step 2: Attach the 4 electrodes to yourself

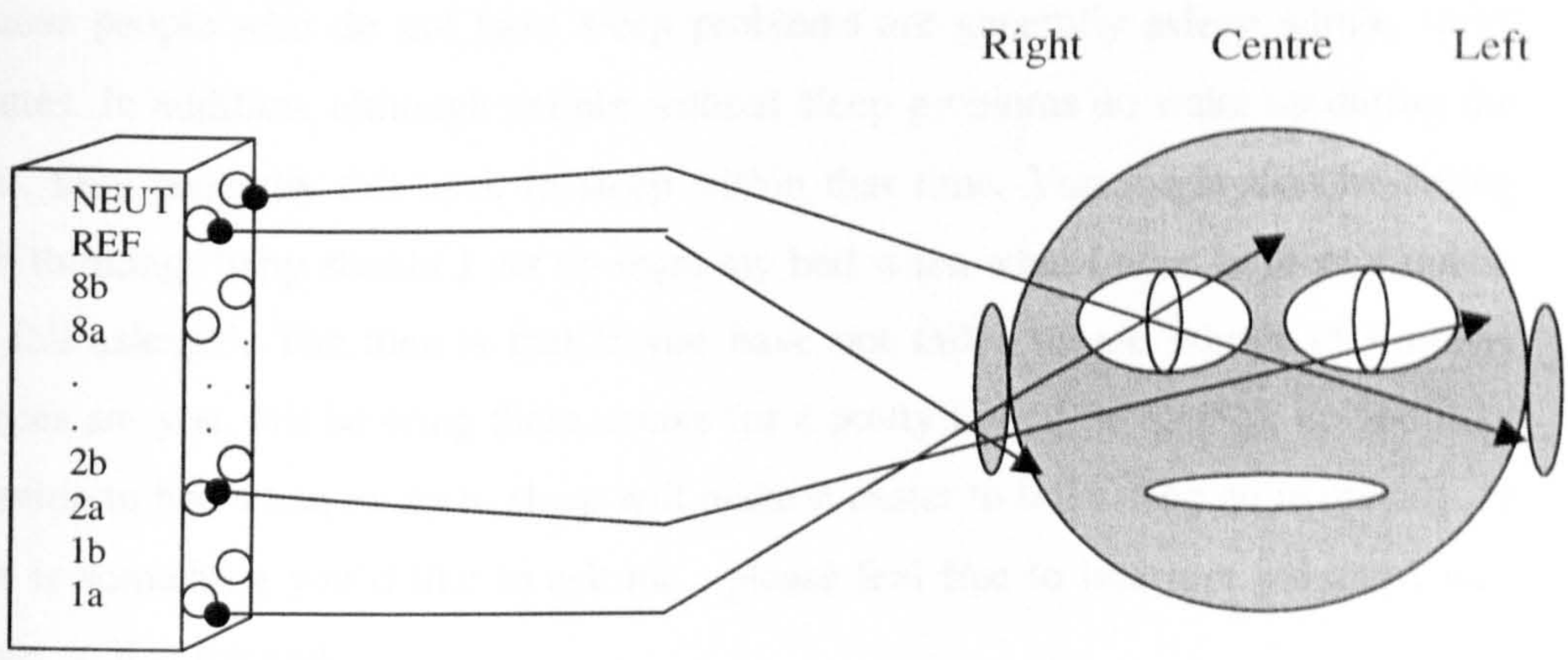
'NEUT' goes behind your left hear

'REF' goes behind your right ear

'2a' goes on the left temple

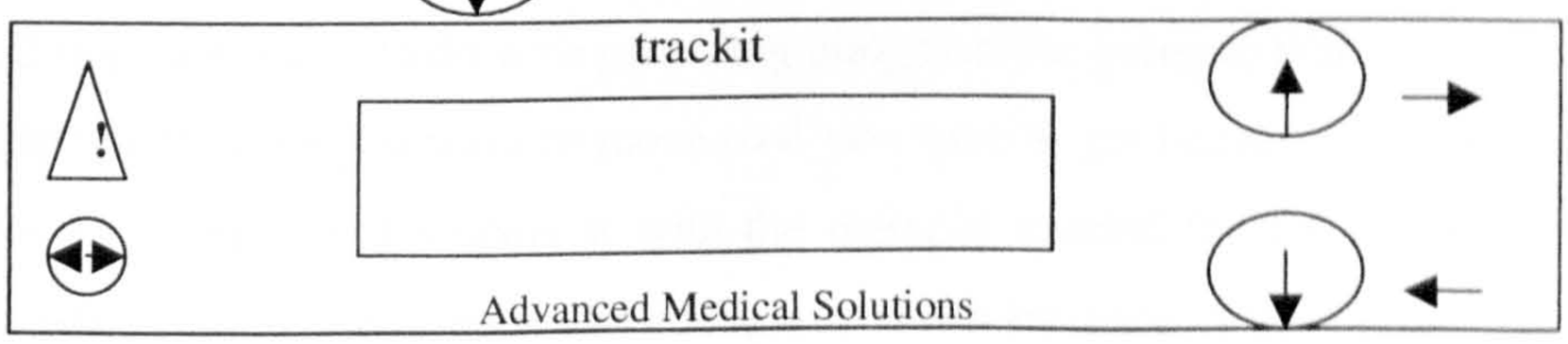
'1a' goes in the centre of your forehead

(Before attaching the electrodes to yourself please remember to rub the skin with the alcohol wipe provided)



Step 3: Press the arrow ' ↑ ' when you switch off the light (attempting to sleep)

Step 4: Press the arrow ' ↓ ' when you wake up in the morning (ready to get up)



APPENDIX 12: THE QHR

PROTOCOL 'QUARTER OF AN HOUR INTERVENTION'

Today we are going to learn a procedure which hopefully will help you sleep better and your task will be to implement and follow it from tonight. It is called the 'quarter of an hour rule' because you'll have to get up from your bed and do something in another room if you cannot fall asleep within around 15 minutes of trying to sleep. In addition you will have to get up and do something in another room if you wake up during the night and cannot fall asleep again within 15 minutes. Although it might sound odd that you have to get up from your bed when all you want to do is to stay in bed and try to fall asleep, I am asking you to try to keep following this procedure. Please keep following it for the requested 3 weeks even if you do not feel it is working. As you have probably already experienced in other things in your life, it takes some time for changes to happen.

What You might be thinking: 'Why 15 minutes?'. This time has been chosen because people who do not have sleep problems are generally asleep within 10/15 minutes. In addition, although people without sleep problems do wake up during the night, they generally fall back to sleep within that time. You might also be sitting there thinking: 'why should I get up from my bed when what I want is to stay in bed and fall asleep?'. The idea is that if you have not fallen asleep within 15 minutes chances are you will be lying there awake for a pretty long time: getting up and then returning to bed when ready to sleep will make it easier to fall asleep quite quickly. If there is something you'd like to ask me , please feel free to interrupt me and I will answer to it at the end.

The procedure is very simple and today we are going to learn and rehearse it till you feel comfortable about putting it into practice. In addition you will also have a memo at home with you if you want to go through it again.

We can divide the 'quarter of an hour rule in 3 different steps:

The first 2 steps are really to do with preparing things before going to bed.

1. Decide which room you want to move to if you have to get because not asleep within 15 minutes and prepare it with the material needed for the activities. Also make sure you will be warm enough as it can be quite chilly during the night. You could also prepare something to drink (e.g. hot milky drink, herbal tea, a juice) but make sure it doesn't contain caffeine or alcohol. We can write

it on the memo with a pencil and if you realise you'd like to change to another room just make the change.

2. Decide a couple of activities you might like (and is feasible) to carry out in another room if you cannot fall asleep within 15 minutes. Also think a couple of activities (they can be the same ones) you would like to do if you wake up in the middle of the night. We can write the activities on your memo and, again, if you'd like to change them just write down the new activities.

You can do this preparation stage when you want to but at least an hour before going to sleep. When do you think it will a good time to prepare the room and material for you?

3. Go to bed as usual. However if you are not asleep within 15 minutes from the moment when you have decided to sleep get up and go to the room chosen to carry out one of the activities. You do not need to keep looking at your clock to see if 15 minutes has passed as generally we have quite a good sense about this. In this other room carry out what you have chosen and when you feel ready to sleep again move back to your bedroom. If you are not asleep within 15 minutes just repeat this step. Eventually you will be asleep within 15 minutes.
4. If during the night you wake up and cannot fall back to sleep within 15 minutes get up and go to the room.

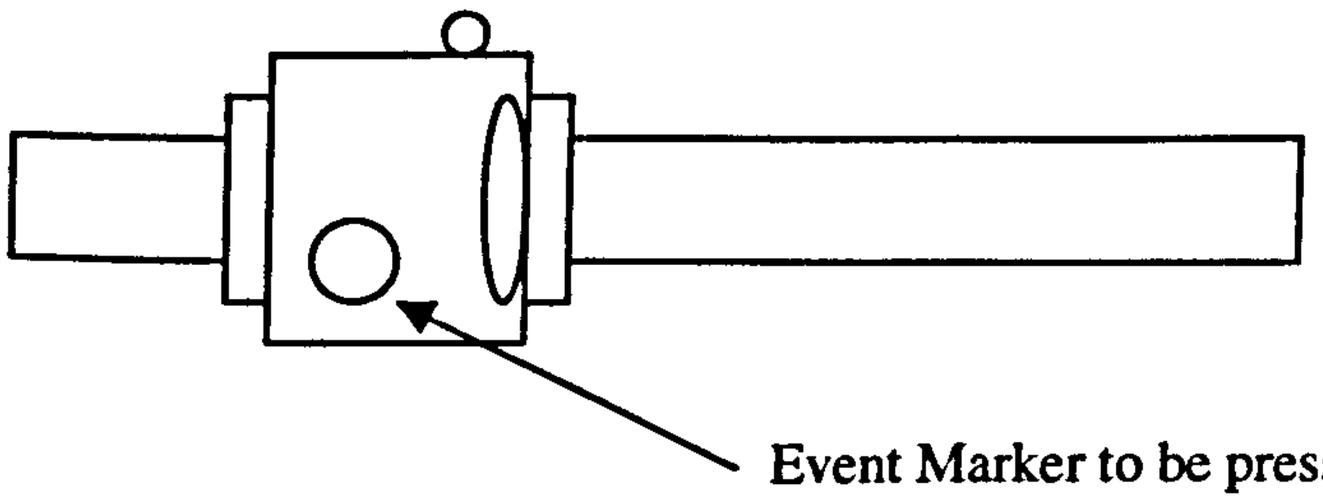
Appendix 13: Actigraghy instruction

2. Movement recording (ACTI-WATCH) : INSTRUCTIONS

a. You have to wear your acti-watch 24 hours a day for the duration of the experiment The only time when you should remove the watch is when you are performing 'wet activities' (e.g. shower, bathing, swimming, washing-up). This is because the watch is not waterproof. Please remember to put your acti-watch back on as soon as you finish 'wet activities'.

b. Press the event marker on the acti-watch face when you switch off the light (and attempt to sleep). Press it again when you wake up in the morning (ready to get up).

c. If during the night you get up (e.g. to get a drink or go to the bathroom) please press the event marker again. Once you get back to bed and try to sleep again, please press the event marker again.



Event Marker to be pressed when attempting to sleep and again when getting up.

3. Sleep Diary : INSTRUCTIONS

Please remember to fill in your daily sleep diary every morning, possibly when you wake up or while having breakfast, for the duration of the research. Remember to answer all questions. As agreed I will contact you during the morning to get your answers.

Appendix 14: Q1 - Sleep Behaviour Self Rating Scale-R

Date: . . . . . ID: . . . . . **basel/interv**

Questionnaire One

Please indicate how often you have done the following things in **your bed before falling asleep** or while **in your bedroom during the last week.** Complete the questionnaire by considering what you would do in an average week.

BEHAVIOUR	**NEVER**	**RARELY**	**SOME TIMES**	**OFTEN**	**VERY OFTEN**
Read a book or a magazine					
Watch TV					
Listen to the radio					
Have a conversation with someone					
Speak on the telephone/mobile					
Eat or drink					
Smoke					
Please also answer the following questions	----------	----------	----------	----------	----------
I take naps during the day or evening					
I feel sleepy when I go to bed					
I switch the light off as soon as I get into bed					
I spend a lot of time lying awake in bed at night					
If I cannot get to sleep within approximately 20 minutes I get out of bed and move to another room until I feel sleepy again					
I set myself a regularly rising time each morning					
If I have a bad night's sleep I still get up at my usual time					

Appendix 15: Q2 - The Glasgow sleep effort scale

Questionnaire Two

The following seven statements relate to your night-time sleep pattern in the past week. Please indicate by circling one response only how true each of the statement is for you.

1. I put too much effort into sleeping at night when it should come naturally

   Very much To some extent Not at all

2. I feel I should be able to control my sleep at night

   Very much To some extent Not at all

3. I put off going to bed at night for fear of not being able to sleep

   Very much To some extent Not at all

4. I worry about not sleeping if I am in bed at night and cannot sleep

   Very much To some extent Not at all

5. I am no good at sleeping at night

   Very much To some extent Not at all

6. I get anxious about sleeping before I go to bed at night

   Very much To some extent Not at all

7. I worry about the long term consequences of not sleeping at night

   Very much To some extent Not at all

Appendix 16: Q 3 - Sleep disturbance questionnaire

Questionnaire Three

On the nights when you didn't sleep well last week, the problem seemed to be:

	Never True	Seldom true	Sometimes true	Often true	Very often true
1. I cannot get into a comfortable position in bed					
2. My mind keeps turning things over					
3. I cannot 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 bed time					
8. I try too hard to get to sleep					
9. My body is full of tension					
10. I am unable to unempty 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 statement is most relavant to you? Item number: . . . . . .

Are there any other factors associated with your poor sleep pattern? If so, please write a short note in this space: . . . . . .
. . . . . .
. . . . . .
. . . . . .

Appendix 17: Q 4 - Glasgow Intrusive thoughts inventory

Questionnaire Four

Here are some thoughts that people have when they cannot sleep. Please indicate by placing a tick in the appropriate box how often over the past 7 nights the following thoughts have kept you awake.

	Never	Sometimes	Often	Always
1. Things in the future				
2. How tired/sleepy you feel				
3. Things that happened that day				
4. How nervous/anxious you feel				
5. How mentally awake you are				
6. Checking the time				
7. Trivial things				
8. How you cannot stop your mind from racing				
9. How long you have been awake				
10. Your health				
11. Ways you can get to sleep				
12. Things you have to do tomorrow				
13. How hot/cold you feel				
14. Your work/responsibilities				
15. How frustrated/annoyed you feel				
16. How light/dark the room is				
17. Noises you hear				
18. Being awake all night				
19. Pictures in your mind				
20. The effects of not sleeping well				
21. Your personal life				
22. How thinking too much is the problem				
23. Things in your past				
24. How bad you are at sleeping				
25. Things to do to help you sleep				

Appendix 18: Q 5 - Pre-sleep arousal scale

Questionnaire five

Please describe how intensely you experienced each of the symptoms mentioned below as you attempted to sleep during the last week.

	Not at all	Slightly	Moderately	A lot	Extremely
Heart racing, pounding or beating irregularly					
A jittery, nervous feeling in your body					
Shortness of breath or laboured breathing					
A tight, tense feeling in your muscles					
Cold feeling in your hands, feet or your body in general					
Have stomach upset (knot or nervous feeling in stomach, heartburn, nausea, gas, etc.)					
Perspiration in palms of your hands or other parts of your body					
Dry feeling in mouth or throat					
Worry about falling asleep					
Review or ponder events of the day					
Depressing or anxious thoughts					
Worry about other problems other than sleep					
Being mentally alert or active					
Cannot shut off your thoughts					
Thoughts keep running through your head					
Being distracted by sounds, noise in the environment (e.g. ticking of clock, traffic)					

Appendix 19: Q 6 - DBAS-10

Questionnaire 6

Statements reflecting people's beliefs and attitudes about sleep are listed below. Please indicate to what extent you personally agree or disagree with each statement right now. There is no right or wrong answer. For each statement, place a mark (x) along the line wherever your personal rating falls. Try to use the whole scale, rather than placing your marks at one end of the line.

Example: If I sleep too much I don't perform as well the next day

Strongly Disagree — Strongly Agree

1. I need 8 hours of sleep to feel refreshed and function well during the day

Strongly Disagree — Strongly Agree

2. When I don't get the proper amount of sleep on a given night, I need to catch up on the next day by napping or on the next night by sleeping longer

Strongly Disagree — Strongly Agree

3. After a poor night's sleep, I know that it will interfere with my daily activities on the next day

Strongly Disagree — Strongly Agree

4. When I feel irritable, depressed or anxious during the day, it is mostly because I did not sleep the night before

Strongly Disagree — Strongly Agree

5. When I feel tired, have no energy, or just seem not to function well during the day, it is generally because I did not sleep well the night before

Strongly Disagree — Strongly Agree

6. I am concerned that chronic insomnia may have serious consequences on my physical health

Strongly Disagree Strongly Agree

---

7. I am worried that I may lose control over my abilities to sleep

Strongly Disagree Strongly Agree

---

8. When I sleep poorly on one night, I know it will disturb my week schedule for the whole week

Strongly Disagree Strongly Agree

---

9. When I have trouble getting to sleep, I should stay in bed and try harder

Strongly Disagree Strongly Agree

---

10. I get overwhelmed by my thoughts at night and often feel I have no control over this racing mind

Strongly Disagree Strongly Agree

Appendix 20: Credibility – expectancy

ID: _____ Date:_____

Please indicate how much you believe, RIGHT NOW, that the therapy will help you reduce your sleeping problems, by circling the voice relevant to you. There is not right or wrong answer, just the way you think and feel.

Part A: - ANSWER IN TERMS OF WHAT YOU <u>THINK RIGHT NOW</u>

1. How logical does the therapy offered to you seem?

   Not at all logical Somewhat logical Logical Very logical

2. How successful do you think this treatment will be in reducing your sleep problems?

   Not at all useful Somewhat useful Useful Very useful

3. How confident would you be in recommending this treatment to a friend who experiences sleep problems?

   Not at all confident Somewhat confident Confident Very confident

Part B: - ANSWER IN TERMS OF WHAT YOU <u>REALLY AND TRULY FEEL</u>

4. How much do you really feel that the therapy will help you to reduce your sleeping difficulties?

   Not at all A little Quite Very much

Appendix 21: Monitoring the use of the 'Quarter of an hour rule' in bed:

We want to evaluate how difficult it is to follow this rule. Please fill out the following table.

For each day put your answer in the box:

a. **If you followed the rule write 'Y', if you did not write 'N'.**

b. **If the question is not applicable to you write 'N/A'**

c. **When you are asked 'How many times..?', write the number applying to you.**

Please remember that there is not a right or wrong answer but only what happened each night.

Weeks: 1 / 2 / 3 **RULE** **TODAY'S DATE**							
When you first went to bed:							
1. Did you take less than a quarter of an hour to fall asleep once you switched off the light?							
2. **If not**, did you sit up in bed?							
3. **If you did sit up in bed**, after about how many minutes did you do so?							
4. In order to follow the rule, how many times altogether did you have to sit up before falling asleep?							
6. If you had to sit up more than once, after how long did you do so?	2nd time						
	3rd time						
	4th time						
After you fell asleep:							
7. If you awoke during the night, did you fall back asleep within a quarter of an hour?							
8. **If you did not fall back asleep** within a quarter of an hour, did you sit up in bed?							
9. **If you did sit up in bed**, after about how many minutes did you do so?							
10. In order to follow the rule, how many times altogether did you have to sit up during the night?							
11 If you had to sit up more than once, after how long did you do so?	2nd time						
	3rd time						
	4th time						
12. **Please describe how it was to follow the rule by circling the number that applies to you**							
(1= not difficult at all,	1	1	1	1	1	1	1
2= a little difficult,	2	2	2	2	2	2	2
3= difficult,	3	3	3	3	3	3	3
4= very difficult,	4	4	4	4	4	4	4
5= extremely difficult)	5	5	5	5	5	5	5

Appendix 22: PROTOCOL 'QHR out of bed INTERVENTION'

PROTOCOL 'QHR out of bed INTERVENTION'

Today we are going to learn a procedure which hopefully will help you sleep better and your task will be to implement and follow it from tonight. It is called the 'quarter of an hour rule' because you'll have to get out of your bed, go to another room and read the book I will give you today if you cannot fall asleep within around 15 minutes of trying to sleep. In addition you will have to get out of your bed and read the book if you wake up during the night and cannot fall asleep again within 15 minutes. Although it might sound odd that you have to get to another room when all you want to do is to lie in your bed and try to fall asleep, I am asking you to try to keep following this procedure. Please keep following it for the requested 3 weeks even if you do not feel it is working. As you have probably already experienced in other things in your life, it takes some time for changes to happen.

What You might be thinking: 'Why 15 minutes?'. This time has been chosen because people who do not have sleep problems are generally asleep within 10/15 minutes. In addition, although people without sleep problems do wake up during the night, they generally fall back to sleep within that time. You might also be sitting there thinking: 'why should I get out of my bed when what I want is to lie in bed and fall asleep?'. The idea is that if you have not fallen asleep within 15 minutes chances are you will be lying there awake for a pretty long time: going to another room and then returning to bed again when ready to sleep will make it easier to fall asleep quite quickly. If there is something you'd like to ask me, please feel free to interrupt me and I will answer to it at the end.

The procedure is very simple and today we are going to learn and rehearse it till you feel comfortable about putting it into practice. In addition you will also have a memo at home with you if you want to go through it again.

We can divide the 'quarter of an hour rule in 2 different steps:

The first step is really to do with preparing things before going to bed.

1. Make sure the book you will have to read if not asleep in approximately a quarter of an hour is nearby your bed is in the room you will go if you cannot sleep. Also make sure you will be warm enough as it can be quite chilly during the night. For example have a cardigan or a little blanket to put round your

shoulder. Remember also to make sure that the actiwatch (during the week you will be wearing one) is not covered by your sleeve as it need to be able to pick up light from the lamp.

2. Go to bed as usual. However if you are not asleep within 15 minutes from the moment when you have decided to sleep get out of your bed, go to the room you have chosen to go and start reading your book. You do not need to keep looking at your clock to see if 15 minutes has passed as generally we have quite a good sense about this. Keep reading until you feel ready to sleep again: when this time arrives go back to your bed, lie down and sleep. If you are not asleep within 15 minutes just repeat this step. Eventually you will be asleep within 15 minutes.
3. If during the night you wake up and cannot fall back to sleep within 15 minutes sit up and read your book.

Appendix 22b: PROTOCOL 'QHR in bed INTERVENTION'

Today we are going to learn a procedure which hopefully will help you sleep better and your task will be to implement and follow it from tonight. It is called the 'quarter of an hour rule' because you'll have to sit up in your bed and read the book I will give you today if you cannot fall asleep within around 15 minutes of trying to sleep. In addition you will have to sit up in your bed and read the book if you wake up during the night and cannot fall asleep again within 15 minutes. Although it might sound odd that you have to sit up in your bed when all you want to do is to lie in your bed and try to fall asleep, I am asking you to try to keep following this procedure. Please keep following it for the requested 3 weeks even if you do not feel it is working. As you have probably already experienced in other things in your life, it takes some time for changes to happen.

What You might be thinking: 'Why 15 minutes?'. This time has been chosen because people who do not have sleep problems are generally asleep within 10/15 minutes. In addition, although people without sleep problems do wake up during the night, they generally fall back to sleep within that time. You might also be sitting there thinking: 'why should I sit up in my bed when what I want is to lie in bed and fall asleep?'. The idea is that if you have not fallen asleep within 15 minutes chances are you will be lying there awake for a pretty long time: sitting up and then lying down again when ready to sleep will make it easier to fall asleep quite quickly. If there is something you'd like to ask me, please feel free to interrupt me and I will answer to it at the end.

The procedure is very simple and today we are going to learn and rehearse it till you feel comfortable about putting it into practice. In addition you will also have a memo at home with you if you want to go through it again.

We can divide the 'quarter of an hour rule in 2 different steps:

The first step is really to do with preparing things before going to bed.

1. Make sure the book you will have to read if not asleep in approximately a quarter of an hour is nearby your bed (possibly on your bed-side table). Also make sure you will be warm enough as it can be quite chilly during the night. For example have a cardigan or a little blanket to put round your shoulder. Remember also to make sure that the actiwatch (during the week you will be wearing one) is on the wrist on the side where the table lamp is.
2. Go to bed as usual. However if you are not asleep within 15 minutes from the moment when you have decided to sleep sit up in your bed and start reading

your book. You do not need to keep looking at your clock to see if 15 minutes has passed as generally we have quite a good sense about this. Keep reading until you feel ready to sleep again: when this time arrives just switch on the light again, lie down and sleep. If you are not asleep within 15 minutes just repeat this step. Eventually you will be asleep within 15 minutes.

3. If during the night you wake up and cannot fall back to sleep within 15 minutes sit up and read your book.

Appendix 23a

Preparation for The 'QUARTER OF AN HOUR RULE'

Decide which room you are going to if you cannot fall asleep within a quarter of an hour

Room . . . . . .

↓

Put the book in the room where you are going to go if you are not asleep within a quarter of an hour

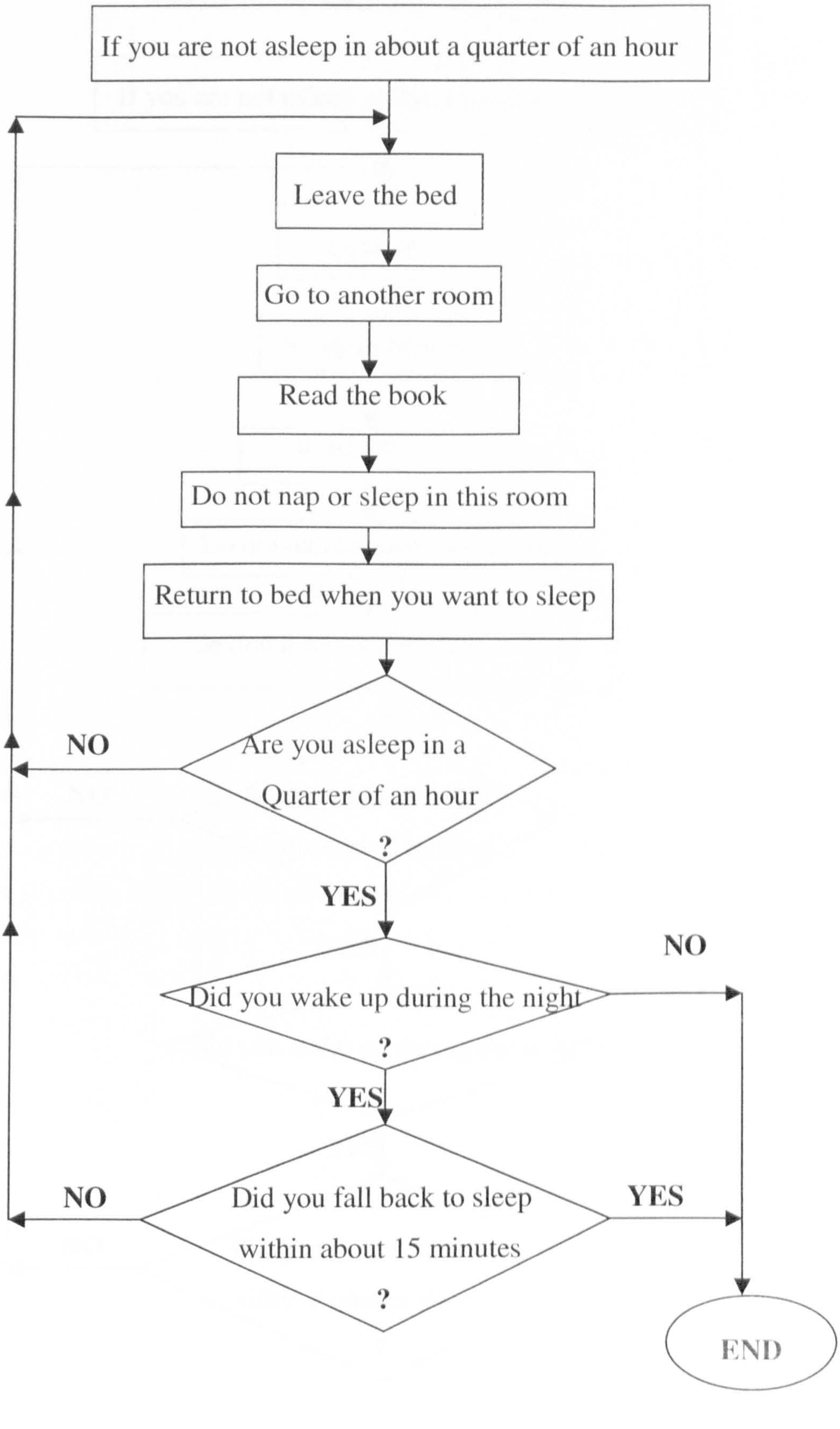
↓

Make sure the room is prepared so that you will be comfortable enough in it if awake during the night (e.g. warm, with something to drink)

↓

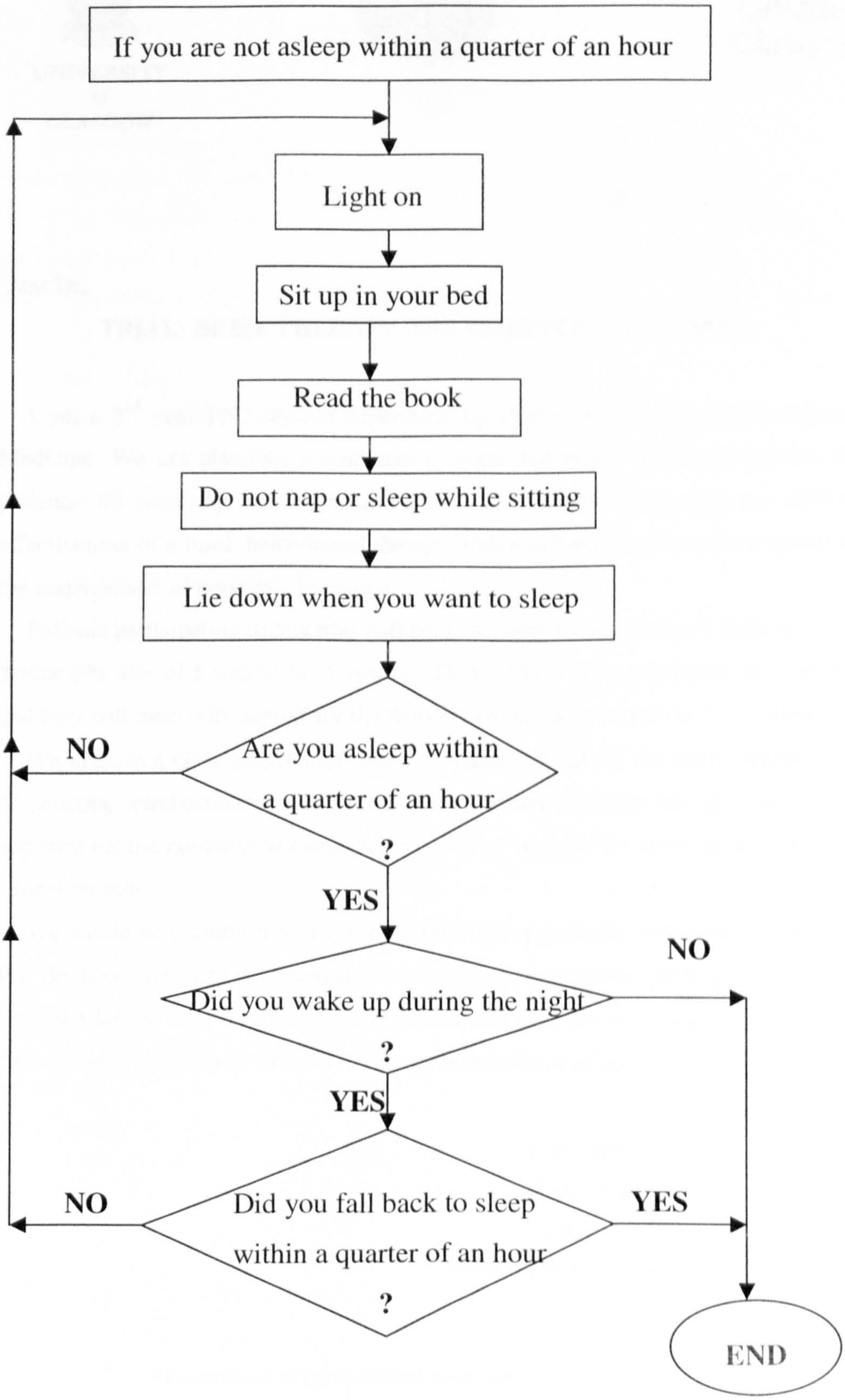
END

The 'QUARTER OF AN HOUR RULE'



Appendix 23b: Flowchart the QHRin

The 'QUARTER OF AN HOUR RULE'



Appendix 24: Information GP

UNIVERSITY of GLASGOW

SLEEP RESEARCH GROUP

NHS Greater Glasgow

10 January 2004

Dear Dr,

TRIAL: BRIEF THERAPY FOR PERSISTENT INSOMNIA

I am a 2nd year PhD student supervised by Prof Colin Espie at Psychological Medicine. We are planning a randomised controlled trial (which will involve 50 patients: 40 receiving active treatment and 10 controls) comparing the clinical effectiveness of a brief, behavioural therapy with a self-monitoring control group in the management of persistent insomnia.

Patients participating in this trial will fill in a sleep diary and wear a light detector device (the size of a watch) for 5 weeks. Their EEG will be monitored for 4 nights and they will meet with myself for the delivery of the intervention on 2 occasions.

We enclose a GP's information sheet containing details of the study, recruitment of patients, reimbursement and so on. NHS Greater Glasgow has granted ethical approval for the randomised controlled trial which will run for approximately for the 18next months.

We would be grateful if you could discuss this opportunity with your colleagues. We do hope you will be interested in having your practice participating in this clinical trial on brief treatment for insomnia and propose to contact your practice manager in 2/ 3 weeks to find out if you are interested or otherwise.

Yours sincerely,

Marina Malaffo

Tel 0141 211 3902

email: 0219144m@student.gla.ac.uk

Department of Psychological Medicine, Academic Centre,

Gartnavel Royal Hospital, 1055 Great Western Road, Glasgow G12 0XH

Trial: Simplified CBT for Insomnia

A research project, funded by Greater Glasgow Primary Care NHS Trust, entitled 'Does a simplified treatment for insomnia improve polysomnographic sleep of patients treated in primary care?' is currently being conducted by Marina Malaffo (PhD student), Department of Psychological Medicine, under the supervision of Prof Colin Espie. We are recruiting 50 patients with insomnia from GP practices. The study will run until September 2005 approximately.

The intervention takes the form of 1 individual session where patients are given education and behavioural techniques to improve their sleep patterns. Patients' sleep will be assessed both via interviews and questionnaires and PSG (night EEG). Support is given by The Sleep Research Team at the University of Glasgow. This includes two Clinical Psychologists.

What the GP has to do:

Simply fill in a referral form and a reimbursement form and send it to the Sleep Clinic. We will send you a sleep assessment and intervention outcome of the referred patient in due course.

Referred patients with insomnia will be randomly allocated to 1 of 2 groups (3:1 ratio):

- **Active Treatment Group:** will receive full assessment of their sleep (using PSG as well as interview and sleep diary) + simplified CBT for insomnia
- **Control Group:** will receive full assessment of their sleep (including PSG)

What the patient is required to do:

- Consent to participate in the study
- Fill in a daily sleep questionnaire for 5 weeks
- 4 nights of EEG monitoring in their own house (the researcher will attach electrodes in the evening and go back in the morning to remove them)
- If allocated to active treatment group, meet with the researcher 2 times for about 30 minutes each to learn the intervention

What the patient will gain from participating in the insomnia trial:

- A full assessment of their sleep
- A better understanding of their sleep patterns
- If allocated to the active group, a good chance of learning to sleep better

If you have any queries, contact: **Marina Malaffo Tel: 0141 211 3902 e-mail: 0219144m@student.gla.ac.uk**

Many thanks for your help.

Marina Malaffo

(Supervised by Professor Colin A. Espie)

PAGE NUMBERING AS ORIGINAL

Appendix 25: Referral Criteria for GP and Referral Form

Simplified CBT for insomnia trial

REFERRAL CRITERIA

Patients must :

- ☐ be between 18 and 70 years of age in good general health
- ☐ have problems getting to sleep (at least 30 minutes) and/ or staying asleep (awakenings during the night)
- ☐ have sleep problems at least 4 nights per week
- ☐ having had sleep problems for at least 6 months

Patients are unsuitable if they have one or more of the following characteristics:

1. Depression and/ or psychotic illness and/ or dementia
2. Be currently taking hypnotic medication for sleep disturbance

IF UNSURE: Refer the patient.

HOW ARE REFERRRALS MADE?

Send a referral form to: Marina Malaffo, Southern General Hospital, Department of Neurosurgery, Sackler Foundation, 1345 Govan Rd, Glasgow G51 4TF

We will then contact the referred patient informing him/her of the insomnia trial.

If you have any queries, contact: **Marina Malaffo Tel: 0141 232 7699 e-mail: 0219144m@student.gla.ac.uk**

Many thanks for your help.

Professor Colin A. Espie, University of Glasgow

(on behalf of the *Sleep* research group)

SLEEP Research 'Quarter of an Hour rule' REFERRAL FORM

Patient's NAME: _____ **D.O.B.** _____

ADDRESS: _____ **TEL:** _____

_____

_____

Id. No. _____

Brief description of problem (nature and duration):

_____

_____

_____

_____

_____

_____

Known or possible contributory factors:

_____

_____

(Confirmation that) the patient IS NOT currently prescribed sleeping medication ☐

(Confirmation that) the patient IS NOT currently severely depressed ☐

(TICK TO CONFIRM)

GP's printed name _____

GP's Signature _____ **Date** _____

Practice: _____

Completed referral forms should be mailed to

Marina Malaffo, Southern General Hospital, Department of Neurosurgery, Sackler Foundation of Psychobiological Research, 1345 Govan Road, Glasgow G51 4TF

Appendix 26: Letter to consultant referring patient to sleep clinic

26th May 2004

Dear Dr ,

Name patient (DoB)

As already communicated to you, Prof Colin Espie will not be able to see the above named patient whom you recently referred to the Sleep Clinic.

However, our Sleep Research Group is currently running an insomnia clinical trial funded by Greater Glasgow Primary Care NHS Trust. We would, therefore, be happy to contact your patient to propose participating in the clinical trial. If the patient consents to it, his/her sleep will be fully assessed (including PSG) and a copy of the assessment result would be sent to you. In addition the patient might be randomised into an insomnia intervention.

If we do not hear from you within the next three weeks, we will assume that you are satisfied for us to contact the above named patient directly. We will send him/her an information leaflet explaining the research trial and a consent form to be filled in and returned to us if the patient is willing to take part in the clinical trial.

Please find here enclosed an information sheet regarding the clinical trial. Should you have any other patient whom you wish to refer for the study or would you like more information about the insomnia trial, please do not hesitate to contact us.

Yours sincerely,

Marina Malaffo

(PhD Student supervised by Prof Colin Espie)

Department of Psychological Medicine,

Academic Centre, Gartnavel Royal Hospital, 1055 Great Western Rd, Glasgow G12 OXH

Tel 0141 211 3902 Email 0219144m@student.gla.ac.uk

Trial: Simplified CBT for Insomnia

A research project, funded by Greater Glasgow Primary Care NHS Trust, entitled 'Does a simplified treatment for insomnia improve polysomnographic sleep of patients treated in primary care?' is currently being conducted by Marina Malaffo (PhD student), Department of Psychological Medicine, under the supervision of Prof Colin Espie. We are recruiting 50 patients with insomnia from GP practices. The study will run until September 2005 approximately.

The intervention takes the form of 1 individual session where patients are given education and behavioural techniques to improve their sleep patterns. Patients' sleep will be assessed both via interviews and questionnaires and PSG (night EEG). Support is given by The Sleep Research Team at the University of Glasgow. This includes two Clinical Psychologists.

What the GP has to do:

Simply fill in a referral form and a reimbursement form and send it to the Sleep Clinic. We will send you a sleep assessment and intervention outcome of the referred patient in due course.

Referred patients with insomnia will be randomly allocated to 1 of 2 groups (3:1 ratio):

- **Active Treatment Group:** will receive full assessment of their sleep (using PSG as well as interview and sleep diary) + simplified CBT for insomnia
- **Control Group:** will receive full assessment of their sleep (including PSG)

What the patient is required to do:

- Consent to participate in the study
- Fill in a daily sleep questionnaire for 5 weeks
- 4 nights of EEG monitoring in their own house (the researcher will attach electrodes in the evening and go back in the morning to remove them)
- If allocated to active treatment group, meet with the researcher 2 times for about 30 minutes each to learn the intervention

What the patient will gain from participating in the insomnia trial:

- A full assessment of their sleep
- A better understanding of their sleep patterns
- If allocated to the active group, a good chance of learning to sleep better

Appendix 27: Letter to participants for Information Pack

Date

Dear ..,

Your medical practice is participating in a study into the assessment of and the non-pharmacological treatment of sleep difficulties being conducted here at the University of Glasgow. Your GP, Dr ...I, referred you with a view to participate in the study as you have been troubled by sleeping difficulties for some time.

The enclosed information sheet gives you details of the study and includes a consent form which, if you would like to be considered for this study, you should complete and return to us in the freepost envelope here enclosed. Two questionnaires are also enclosed: please fill them in and send them back with your consent form if you want to be considered.

I will phone you in a couple of weeks to find out if you received the material and if you have any questions.

We look forward to hearing from you.

Yours sincerely,

Marina Malaffo
(supervised by Prof Colin Espie)

Department of Psychological Medicine
Based At
Southern General Hospital, Department of Neurosurgery, Sackler Foundation of Psychobiological Research, 1345 Govan Road, Glasgow G51 4TF
Tel 0141 - 232 7699

Appendix 27a: Participant's Information Pack

UNIVERSITY
of
GLASGOW

Treating insomnia

If you have trouble sleeping,
then the **Sleep Clinic** could help.

Appendix: cont.

Some background information

- Insomnia is a common problem: at least one in ten adults has a problem getting to sleep or staying asleep. A sleep intervention, called 'the quarter of an hour rule' should help you to improve your sleep. **The quarter of an hour rule does not rely on using pills, rather it helps to mould your sleep back into a proper shape.** This, then, is a research study. It is funded be Glasgow Primary Care NHS trust. Our aim is to see if the quarter of an hour rule helps people to get over their insomnia by sleeping better at the end of a treatment programme.
- In order to carry out this study we need to have some people who get the treatment and some other people who do not get it. Therefore, if you decide you want to take part in this study, you may not receive the quarter of an hour rule. This is the only way we can compare the results properly. We would be pleased if you would consider taking part.

Do I have to take part?

- Taking part in the research is completely voluntary. It is up to you to decide whether or not to take part and if you decided to take part and then wanted to withdraw from the study, you are free to do so at any time and without having to explain the reasons for your decision. This will not affect the care you receive.

Name:(Mr/Mrs/Miss/Ms)_____

_____

Address _____

_____

Postcode _____

Day Tel Number:_____

Evening Tel Number:_____

- I am interested in taking part in the 'quarter of an hour' research study, and agree to be contacted by the main researcher.
- **I understand that I am not obliged to take part, may withdraw from the study at any time and this will not affect my care in any way.**
- Signed _____

  Date _____

Send to: Marina Malaffo, The Sleep Clinic
Academic Centre, Psychological Department
Gartnavel Royal Hospital
1055 Great Western Road
Glasgow G12 0XH

- Thank you for reading this information. If there is anything you are not clear about or if you have any questions you can **call the researcher, Marina Malaffo on 0141 211 3902** (available 9.30 to 4.30 Tuesday to Friday).
- If you would like some independent advice, you can contact Dr Leanne Fleming at Psychological Medicine, University of Glasgow, telephone 0141 211 3943. Dr Fleming is not involved in any way with this project so can deal with any queries before during or after the project completely independently.

Professor Colin A. Espie, University of Glasgow
(on behalf of the *Sleep Clinic* research group)

This research is funded by
Greater Glasgow Primary Care NHS Trust

What is involved in taking part

If you decided to take part, there would be a number of stages to your involvement. First of all, you would be asked to complete some short questionnaires so as to assess whether your sleep problems are exactly those examined in this research.

The researcher would then phone you at home to tell you whether you were suitable for the study, and if so, to check if you would like to take part in the study.

If you were suitable for the study, and you still wanted to take part, you would be involved in the study for 5 weeks.

You would be asked to:

1. wear an 'actigraph' (similar to a wrist watch) on your wrist for 2 weeks. In this way your sleep and wake patterns can be measured. The actigraph measures body movements and detects light and stores information in a tiny microprocessor inside. The information recorded in the actigraph will then be read using a computer.
2. fill in a simple sleep diary each morning for the duration of the study (5 weeks): this takes only 5 minutes. The sleep diary will help us to monitor your sleep (e.g. when you go to sleep, how long it takes you to fall asleep) on a nightly basis.

Appendix. Cont.

3. collect your 'brain waves' (EEG) during 4 nights: 2 nights at the beginning of the study and 2 nights at the end of the study. It will measure your sleep (e.g. when you dream, when you sleep deeply and when you sleep lightly). The EEG recording equipment is portable and easy to use in your own home. The researcher will have to set up the EEG recording equipment and attach the electrodes to your head. This means that in these evenings you will meet with the researcher, either at your home or at the Southern General Hospital (whatever you prefer). Setting up the EEG equipment will take around 20 minutes, and thereafter you will carry on with your evening activities and go to sleep as usual. In the morning you will disconnect the EEG equipment by your self or, if you prefer, the researcher will meet with you and disconnect it for you.

After your first assessment **you <u>may</u> be** offered the quarter of an hour treatment. This is a matter of chance since we have to pick up people at random. If you are not picked at this stage you will still receive the full sleep assessment. Taking part in the study would involve attending 2 half an hour meetings, one per week and one 15 minutes telephone contact. These meetings would be at the surgery, the Southern General Hospital or the researcher's office at Glasgow University The researcher would take you through the quarter of an hour programme a step at a time. You would get notes to take home which would help you to remember what has been said and to put what you have learned into practice.

What are the benefits to taking part?

- We hope that you would be helped by whichever treatment you receive for your insomnia. However, this cannot be guaranteed. The information we receive from this study may help us to treat future patients with insomnia better.

If I do decide to take part what happens next?

- Fill-in and return the tear-off slip at the end of this leaflet and send it to

Marina Malaffo , The Sleep Clinic
Psychological Medicine, Academic Centre
Gartnavel Royal Hospital
1055 Great Western Road
Glasgow G12 0XH

The researcher will then telephone you at home over the next few days to answer any questions you may have about the study and make sure you still want to take part. This study has been approved by the Greater Glasgow NHS Research Ethics Committee and this form is usual practice for research studies.

Appendix cont.

- Thank you for reading this information. If there is anything you are not clear about or if you have any questions you can **call the researcher, Marina Malaffo on 0141 211 3902** (available 9.30 to 4.30 Tuesday to Friday).
- If you would like some independent advice, you can contact Dr Leanne Fleming at Psychological Medicine, University of Glasgow, telephone 0141 211 3943. Dr Fleming is not involved in any way with this project so can deal with any queries before during or after the project completely independently.

Professor Colin A. Espie, University of Glasgow
(on behalf of the *Sleep Clinic* research group)

This research is funded by
Greater Glasgow Primary Care NHS Trust

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GLASGOW

SLEEP RESEARCH
LABORATORY

Appendix 28: THE GOOD SLEEP GUIDE

DURING THE EVENING

1. Daily exercise is a good way to encourage sleep. Try some light exercise early in the evening, such as walking or swimming, as this will make your body ready for sleep later on.

2. Avoid drinking coffee or tea or eating chocolate in the evening because they all contain caffeine, which makes you more alert. A hot milky drink is a good alternative. Also don't drink alcohol as a nightcap – it usually upsets sleep.

3. Wind down during the course of the evening. Try not to do anything that is mentally stimulating within 90 minutes of bedtime. Give yourself time to relax and prepare for sleep – try having a warm bath or listening to some soothing music.

4. Do not sleep or doze in the armchair because this can upset your night time sleeping patterns. Keep all your sleep for bedtime.

5. Feeling worried or anxious or just having an active mind can cause a sleepless night. Try to put the day to rest. Take time to think it through and write down any worries or concerns or loose ends in a notebook. Say goodbye to them before you go to bed – you can deal with them more effectively in the morning.

6. Make sure your bed and bedroom are comfortable for sleep. Your room should be well ventilated, not too cold or too warm.

AT BEDTIME

1. Try to get up at the same time every day, even at weekends, use an alarm clock if you need to. This helps establish your sleep pattern to follow a regular routine.

2. Go to bed when you are "sleepy tired" and not before. Try to notice when you are ready to sleep.

3. Try to avoid reading or watching TV in bed as this can stimulate the brain, sometimes making it difficult to relax. Keep these waking activities for another room.

4. Put the light out when you get into bed and try to ensure your room is as dark as possible. This helps signal to your body that it's time for sleep.

5. Let yourself relax. Tell yourself that "sleep will come when it's ready". Enjoy relaxing even if you don't at first fall asleep. If you are not good at relaxing, try to learn a relaxation method.

6. Do not try to fall asleep – let sleep find you. Sleep is not something you can switch on automatically. Trying too hard can switch sleep off.

IF YOU HAVE PROBLEMS GETTING TO SLEEP

1. Remember that sleep difficulties are quite common, and are not as damaging as you might think. Most people cope quite well even after a disturbed night's sleep.

.2. Try not to get upset or frustrated or to think about the next day. Avoid watching the clock, as this will only make the time pass more slowly.

3. Instead, if you are awake for more than 15 minutes, you just might not be ready for sleep. Try getting up, going into another room, and accepting it philosophically.

4. Do something relaxing while you are up like listening to music or reading.

5. Go back to bed again when you feel "sleepy tired". Remember you may have to repeat this rising and returning to bed several times a night at first.

6. Whenever you experience difficulties sleeping try to follow these tips. A good sleeping pattern may take a number of weeks to establish. Be confident that you will achieve this by working through

"The Good Sleep Guide"!

This guide has been prepared by Professor Colin A. Espie, Director of the University of Glasgow Sleep Research Laboratory, Southern General Hospital, Glasgow
http://www.gla.ac.uk/sleepresearch
The Good Sleep Guide is recommended by the British Sleep Sciety

Appendix 29: PARTICIPANT CONSENT FORM (study 3)

'Does a simplified treatment for insomnia improve the polysomnographic sleep of patients treated in Primary Care?'

Name (Mr./ Mrs./ Miss/ Ms) _____

Address _____

_____

Postcode _____

Telephone Number _____ (day)

_____ (evening)

I confirm that I have read the information sheet for the above study, I understand its content and I have received my own copy.

I have also had the opportunity to ask any questions about taking part to the study (and what it will involve) with the research worker.

I understand that I am free to withdraw from the study at any time, without having to give a reason. I also understand that if I withdraw, my care and treatment will be not affected in any way.

I understand that all information collected during the study, including questionnaires, sleep diaries and sleep data from the actiwatch and the EEG recorder, will be treated confidentially.

I hereby consent to participate in the above study.

Signed _____ Date _____

GP Name _____

GP Address _____

Department of Psychological Medicine

Academic Centre, Gartnavel Royal Hospital, 1055 Great Western Road, Glasgow G12 OXH

Based at Southern general hospital, department of Neurosurgery, Sackler Foundation of Psychobiological Research, 1345 Govan Rd, Glasgow

Appendix 30: Home visit Protocol

HOME VISIT SAFETY PROTOCOL FOR PSG STUDIES

(01/09/2004)

Home based PSG research projects require researchers to undertake home visits. Health, safety and welfare of research and clinical staff are regulated by the Health and Safety at Work etc Act 1974 which also requires workers themselves to take reasonable care of their own safety.

This procedure has been formulated in order to remind researchers to adopt a common sense approach to each situation so as to take reasonable care of themselves and to be responsible for their own safety.

You and your supervisor should discuss this protocol and adapt it to your specific project in order to make sure that it is conducted as safely as possible. Remember also that 'Management of Aggression (Breakaway) courses' are made available on a regular basis within the Trust.

WHAT to do before offering a home visit appointment

- 1. Before agreeing with a participant a home visit, it is important to check within yourself whether you feel comfortable with that particular participant (e.g. mannerism, GPs notes, means of recruitment). Inform your supervisor of participants you do not feel comfortable with.
- 2. It is also important to find out if there is anything unsafe about the neighbourhood the person lives
    - **If the person or the neighbourhood could pose risks, it is important to attend the visit accompanied by another person (e.g. a friend, a colleague)**

When an appointment is offered (irrespectively of whether one or two people go to the participant's home) the following must be in place:

1. You **must have a mobile phone** with you and ensure it is switched on. If you do not have one, ask your supervisor to be provided with one. Remember to check that it is charged and has calling credit.

2. **Inform** a nominated person (family member, partner, friend, colleague), ***where* you are going** (full address/name and telephone of the person being visited) **and *when*** (day/date/time and approximately when the visit should finish).
3. Contact the nominated person prior to entering the premises and again immediately after leaving the participant's home. Obviously your contact must be available for the time period you are paying the home visit and must understand the importance of hearing back from you. Make sure your contact person has your mobile phone number.
4. You should carry a personal attack alarm – one will be available from your supervisor.
5. Do not forget that you might be at risk also while walking to the participant's home or entering the close, especially if not well lit.

WHAT THE NOMINATED PERSON SHOULD DO IF YOU DON'T CONTACT HIM/HER WITHIN 15 MINUTES FROM THE TIME AGREED

- Phone you on your mobile phone and/or at the patient's home number.
- If he/she is unable to speak to you, they should inform the Police and your supervisor
- If you answer the phone and are in a threatening situation, please state **"My phone does not have battery left, cannot speak just now".**

This will act as an ***alert signal.*** In this case the nominated person should inform

the Police and your supervisor.

During the home visit:

1. Ensure that your phone and attack alarm are within easy reach.
2. If you feel threatened in any way even before you enter the building, you should leave. Make your excuses and leave if anything worries you.
3. If you are asked to leave by your participant you should go without hesitation.
4. All significant incidents, in particular those of violence or aggression both physical and verbal including inappropriate behaviour must be recorded in line with the Trust and University's incident reporting procedure.

Appendix 31 : Results of one-way ANOVA on the baseline scores of the battery of questionnaires

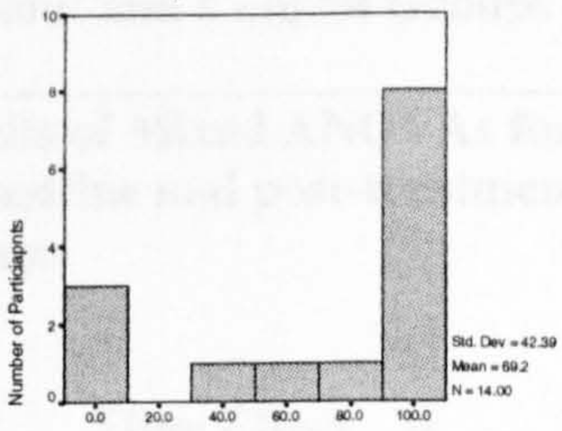
Results of One-way ANOVA on the baseline scores of sleep related questionnaires for the QHRin, QHRout and Control Groups

Mean Scores	F (2,36)	p	Bonferroni corrected t-tests
Sleep Behaviour Self Rating Scale-R	1.9	n.s.	
The Glasgow Sleep Effort Scale	0.5	n.s	
Sleep Disturbance Questionnaire.			
physical tension	3.7	0.04	n.s.
sleep pattern problems	0.7	n.s.	
cognitive arousal	0.8	n.s.	
sleep effort	1.9	n.s.	
Glasgow Content of Thoughts Inventory			
active problem solving	1.2	n.s.	
sleep and wakefulness	1.4	n.s.	
somatic and sensory engagement	5.3	0.01	QHRin vs QHRout p= 0.01
Pre-Sleep Arousal Scale			QHRin vs Control p= 0.009
cognitive arousal	0.1	n.s.	
somatic arousal	2.5	n.s.	
DBAS-10			
immediate negative consequences	0.7	n.s.	
long term negative consequences	0.4	n.s.	
need for control	1.2	n.s.	

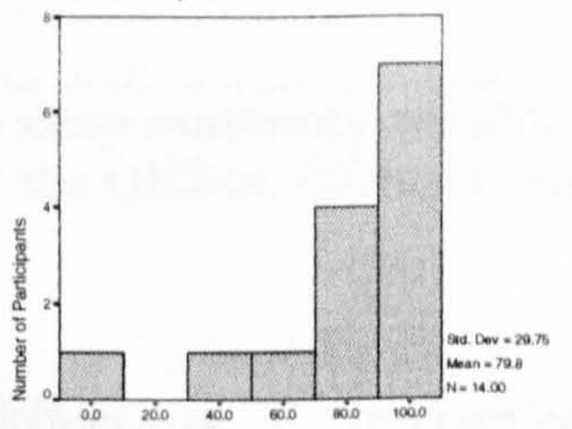
Appendix 32 - Histograms: AdherenceRaw Data

Appendix 32a- Number of Participants in the QHRin group according to SOL and WASO adherence score measured objectively and subjectively

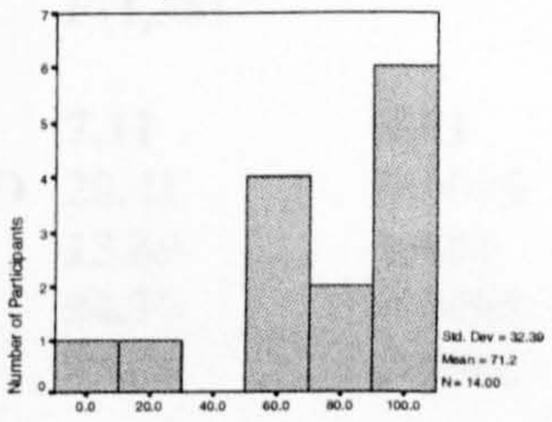
QHRin objective adherence score at SOL



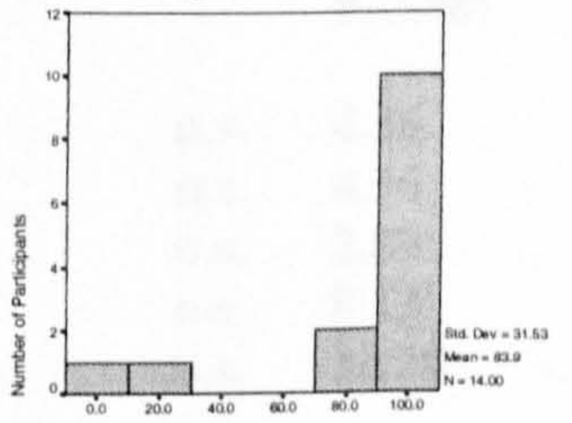
QHRin subjective adherence score at SOL



QHRin objective adherence score at WASO

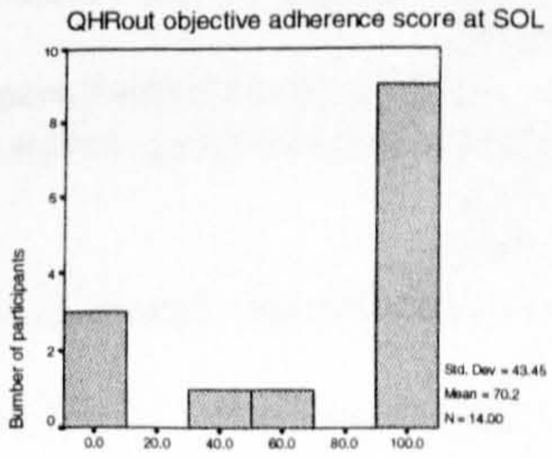


QHRin subjective adherence score at WASO

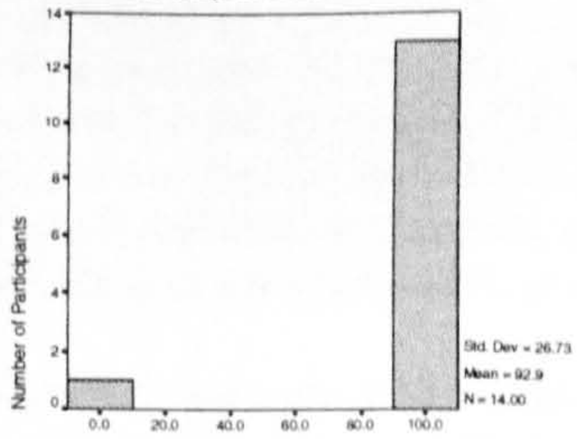


Appendix 32b - Number of Participants in the QHRout group according to SOL and WASO adherence score measured objectively and subjectively

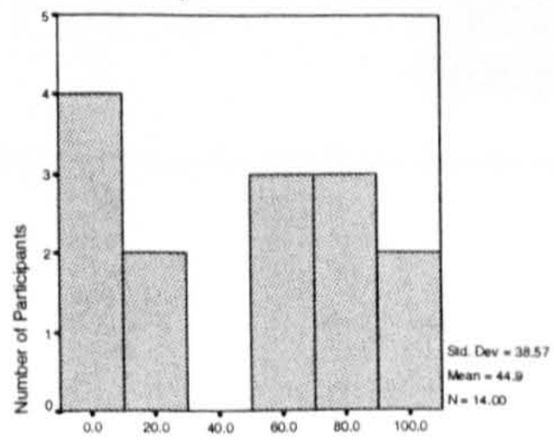
QHRout objective adherence score at SOL



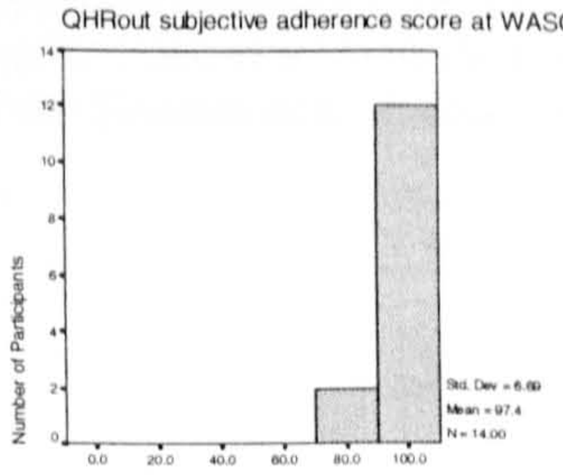
QHRout subjective adherence score at SOL



QHRout objective adherence score at WASO



QHRout subjective adherence score at WASO



Appendix 33: Results of Mixed ANOVAs for subjective sleep continuity variables and PSQI on baseline and post-treatment scores for the QHRin, QHRout and Control Groups

Results of Mixed ANOVAs for subjective sleep continuity variables and PSQI on baseline and post-treatment scores for the QHRin, QHRout and Control Groups

	Time Effect F(1,38)	p	Group Effect F(2,38)	p	Interaction F(2,38)	p
SOL	7.11	0.01	0.81	n.s.	4.36	0.02[(1)]
WASO	20.41	0.0005	1.36	n.s.	4.56	0.02[(2)]
TST	13.89	0.001	2.32	n.s.	2.89	0.07
S.E.	24.79	0.0005	1.78	n.s	8.13	0.001[(3)]
PSQI	46.04	0.0005	2.00	n.s.	13.38	0.0005[(4)]

Post-Hoc Corrections

[(1)] Bonferroni Corrected: QHRout Baseline vs Post-Treatment t(14)=3.99, p=0.001
Uncorrected t-test: QHRout vs Control at Post-Treatment t(25)=2.14, p=0.04

[(2)] Bonferroni Corrected: QHRout Baseline vs Post-Treatment t(14)=5.26, p<0.0005
QHRout vs Control at Post-Treatment t(25)=3.63, p=0.001
Uncorrected t-test: QHRin vs Control at Post-Treatment t(24)=2.34, p=0.03

[(3)] Bonferroni Corrected: QHRout Baseline vs Post-Treatment t(14)=5.52, p<0.0005
QHRout vs Control at Post-Treatment t(25)=3.27, p=0.003
QHRin Baseline vs Post-Treatment t(13)=3.68, p=0.003
Uncorrected t-test: QHRin vs Control at Post-Treatment t(24)=2.51, p=0.02

[(4)] Bonferroni Corrected: QHRout Baseline vs Post-Treatment t(14)=6.68, p<0.0005
QHRout vs Control at Post-Treatment t(25)=3.95, p=0.001
QHRin Baseline vs Post-Treatment t(13)=4.19, p=0.001
QHRin vs Control at Post-Treatment t(24)=3.54, p=0.003