Stress Reactivity in Individuals with Non-REM Parasomnias, Insomnia and Good Sleep

Clinical Research Portfolio

Volume I

(Volume II bound separately)

Sarah Elizabeth Young

Submitted in part fulfilment of the requirements for

Degree of Doctorate in Clinical Psychology

Faculty of Medicine Graduate School

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Chapter Two: Major Research Project

CHAPTER ONE: SYSTEMATIC LITERATURE REVIEW

A Systematic Review of Biofeedback Interventions as a Treatment for Insomnia

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Abstract

Physiological, cognitive and cortical arousal can interfere with sleep initiation. Any relaxation technique may help to reduce arousal levels and so facilitate sleep, and biofeedback is a particular type of self-regulation procedure. Both electromyograph (EMG) biofeedback and electroencephalograph (EEG) biofeedback have been investigated as treatments for insomnia. This review aimed to evaluate the evidence for the efficacy of biofeedback as a treatment for insomnia. Studies were identified by searching electronic databases, the reference sections of relevant articles and the contents pages of a key journal. For articles to be selected, it was necessary for the main target problem to be insomnia and for a group design to have been employed. The outcome measures had to at least include sleep log data. Twelve studies were reviewed and discussed in terms of their methodological quality and research findings. There is evidence that different procedures targeting physiological, cognitive and cortical arousal have shown promise but the mechanisms driving the observed reductions in insomnia symptoms are not yet clear. Many of the studies reviewed are decades old and the methodological quality of several of the studies is low according to today's standards. The results of this review suggest that more research in this area is warranted in order to better understand the mechanisms underlying various biofeedback protocols and also to perhaps better understand the interaction of arousal and sleep processes.

Introduction

Insomnia is a common problem in the general population. Ohayon (2002) reported the prevalence of insomnia diagnoses, according to DSM-IV (Diagnostic and Statistical Manual of Mental Disorders, 4th edition; American Psychiatric Association, 1994) criteria, to be estimated at 6% with about a third of the general population presenting with at least one symptom. Prevalence generally increases with age and prevalence rates are higher in women than in men and in individuals with other medical or psychiatric diagnoses. In both the DSM-IV and ICSD-2 (International Classification of Sleep Disorders, 2nd edition; American Academy of Sleep Medicine, 2005) diagnostic classification systems, insomnia is defined by difficulties in initiating or maintaining sleep, or sleep that is non-restorative in nature, which results in impairments on daily functioning. However the systems differ in their approach to categorising subtypes. DSM-IV adopts a much broader approach than ICSD-2, which includes a greater number of subtypes. For example, the DSM-IV diagnosis of Primary Insomnia would be applied to all insomnia not induced by substances or due to a psychiatric or medical condition, whereas the ICSD-2 system would divide this same group into more specific diagnoses e.g. Psychophysiological Insomnia, Idiopathic Insomnia, Paradoxical Insomnia. Chronic insomnia (defined as >1 month duration in DSM-IV and >6 months in ICSD-2) can have considerable, negative impact on an individual's quality of life as well as on health care services (Simon & VonKorff, 1997).

The process of initiating sleep involves reducing physiological arousal levels, as indicated by, for example, heart rate or muscle tone (Budzynski, 1973; Ogilvie, 2001). If physiological arousal does not sufficiently reduce, then it can interfere with sleep initiation. It has been well documented that individuals with insomnia show higher levels of somatic

arousal compared to normal sleepers (Bonnett & Arand, 1996, 1998; Adam et al., 1986; Stepanski et al., 1989, 1994; Haynes et al., 1974; Haynes et al., 1981; Monroe, 1967; Freedman & Sattler, 1982; Riemann et al., 2010). Cognitive arousal may also interfere with both the initiation and the maintenance of sleep e.g. intrusive (often negative) thoughts, and excessive worry about sleep difficulties. (Morin, 1993; Perlis et al., 1997; Harvey, 2002). Indeed, some studies have reported that cognitive arousal interferes with sleep initiation to a greater degree than physiological arousal (Lichstein & Rosenthal, 1980; Robertson et al., 2007). Harvey (2002) suggests that excessive worry about the consequences of insomnia can trigger autonomic arousal and distress. This then leads to an attentional bias towards the sleep difficulty and its perceived consequences, and so to further worry. The important role of selection attentional processes and sleep effort has been discussed in depth by Espie et al. (2006). Importantly, however, as Perlis et al. (1997) have suggested, cortical arousal is a useful construct for insomnia which serves to explain the interaction between brain physiological processes and mental states. Recent work by Cortoos et al. (2011) helpfully discusses not only 'types' of hyperarousal in insomnia patients in comparison to good sleepers, but also addresses the variability of arousal status which characterises insomnia.

Any relaxation technique may help to reduce arousal levels and so facilitate sleep, and biofeedback is a particular type of self-regulation procedure whereby relaxation skills are learned through operant conditioning. In this way, biofeedback training enables individuals to develop awareness of, and learn how to control, internal physiological processes that tend to occur outside of normal awareness (Budzynski, 1973). As the term suggests, biofeedback involves the measurement of some aspect of biological activity, which is then fed back to the individual, so that they are continually aware of the current level of that activity.

Feedback is usually by sound, with tones that vary as the level of activity changes, or visually using a display.

There are various types of biofeedback, each focusing on learning to control a specific process e.g. muscular activity, heart rate, brain activity or skin conductance. Two types of biofeedback have been investigated as treatments for insomnia: electromyograph (EMG) biofeedback and electroencephalograph (EEG) biofeedback, or neurofeedback. EMG biofeedback uses surface electrodes to monitor electrical activity of a skeletal muscle. The electrodes are often placed on the frontalis muscle, which is located in the forehead, and a noise is produced which varies in tone or frequency to indicate the level of muscular activity. The aim is often to decrease the pitch of this noise and so learn to decrease muscle tension (Coursey et al., 1980). The hypothesis behind this intervention in relation to insomnia is that by reducing muscle tension, physiological arousal will reduce, which will help with sleep initiation.

Neurofeedback uses the EEG to measure and instantly feed back (visually or auditorily) the electrical activity of the brain (a proxy for cortical arousal), which occurs across a range of frequencies (see below).

Beta (≥13 Hz): Faster, smaller brainwaves – associated with being alert, intellectual activity and concentration.

Alpha (8-12 Hz): Slower & larger than beta waves – associated with being more relaxed and less alert.

Theta (4-8 Hz): Slower and larger than alpha waves - associated with a 'daydream-like' state, deep relaxation and with the transition between awake and sleep states.

Delta (0.5-3.5 Hz): Slowest, largest brainwaves – associated with being asleep.

Hammond (2006)

Brain activity of each frequency band are usually present at all times, however, different frequencies are dominant at different levels of awareness (Hammond, 2006). Therefore, neurofeedback can be used to inhibit or to reinforce specific EEG frequencies. Instant feedback relating to patterns of brain activity (of which we are not normally aware) is given and self-regulation is again acquired through operant conditioning (Cortoos et al., 2006). Theta biofeedback, for example, focuses on theta waves, typical of stage 1 sleep and of the transition between wakefulness and sleep. Their presence indicates being in a very relaxed state. The goal of theta feedback is to increase theta activity, and so relaxation, which will facilitate sleep initiation (Hauri, 1981). Another type of neurofeedback is sensorimotor rhythm (SMR) feedback. SMR is a low amplitude brainwave rhythm (in the 12-14 Hz moderate to fast range), associated with immobility during wakefulness, and localised in the sensorimotor cortex (Cortoos et al., 2006). SMR is thought to be associated with stage 2 sleep and individuals with insomnia seem to produce less SMR during wakefulness than good sleepers (Jordan et al.,1976 as cited in Hauri, 1981). So it has been hypothesised that increasing SMR during wakefulness may improve sleep (Hauri, 1981). There is some evidence that for individuals with high resting muscle tension, who find it difficult to relax, prior EMG biofeedback training is necessary in order for neurofeedback training to be effective. However this did not seem to be necessary in individuals who were already easily able to reach a state of relaxed muscle tension (Sittenfield et al., 1972 as cited in Budzynski, 1973)

The aim of this review is to evaluate the evidence for the efficacy of biofeedback as a treatment for insomnia. Articles investigating the efficacy of any type of biofeedback were searched for and considered for inclusion.

Method

Search Strategy and Search Terms

The following electronic databases were searched from the earliest records available until July 2011: Ovid Medline, All EBM Reviews, Embase, CINAHL Plus, PsycINFO and Psychology and Behavioural Sciences Collection. The search terms used were: 'biofeedback', 'neurofeedback', 'theta-feedback', 'insomnia*' and 'sleep*'. Truncation was used, indicated by *, to find different word endings. Searches were combined using the Boolean commands 'AND' and 'OR', and duplicates were removed. The search was supplemented by searching reference sections of selected papers as well as of relevant review papers which had been identified in the database search (Ebben & Spielman, 2009; Lundh, 1998; Morin, Culbert & Schwartz, 1994; Morin et al., 1999; Spielman, Caruso, & Glovinsky 1987). A search was also carried out of the online contents pages of all issues of the journal *Applied Psychophysiology and Biofeedback* (previously *Biofeedback and Self-Regulation;* 1976 – June 2011).

Selection Criteria

Inclusion and exclusion criteria were employed to select appropriate articles:

Inclusion Criteria

- Main target problem of insomnia.
- Group design
- Outcome measures to include at least one of the following: sleep-onset latency (SOL), wake after sleep onset (WASO), total sleep time (TST) or no. of awakenings.

Exclusion Criteria

- Single case studies, review articles
- Non English language
- Participants without sleep difficulties
- Primary target problem other than insomnia with sleep difficulties being a secondary problem.

Methodological Quality Rating

Studies were evaluated using a modified version of the Clinical Trials Assessment Measure (CTAM; Tarrier & Wykes, 2004; Figure 1.1). The CTAM is a measure which was designed to assess the methodological rigour of randomised controlled trials of psychological treatments. Its content was influenced by the CONSORT guidelines (Moher et al., 2001), a review of trial assessment scales as well as expert opinion. The CTAM comprises 15 items relating to: the participant sample, method of allocation to groups, assessment method and measures, experimental control, analysis of results and the description of treatment, including use/adherence to protocol. For the purpose of this review, the 'assessment' section was slightly modified to make it more applicable to the sleep research studies being reviewed, but without altering the weighting of the scoring system. The weighting system was designed to take into account previous evidence regarding the influence of research methodology on outcome. For example, a maximum of 10 points are given to items relating to the participant sample and a maximum of 32 points are given to items relating to 'assessment' (see Figure 1.1 for further detail about the allocation of points). Item scores are added to reach a maximum of 100. The CTAM has been shown to demonstrate good inter-rater reliability (0.96), adequate internal consistency (Cronbach's alpha = 0.69) and good

criterion validity when compared with other quality assessment scales (Tarrier & Wykes, 2004).

Fifty percent of the articles were rated by an independent rater, also a Trainee Clinical Psychologist. Initially, 92.5% agreement was achieved. Differences in ratings were then discussed until 100% agreement was reached. Using an arbitrary rating system, studies were then classified according to their quality rating score as: very low, low, moderate, high or very high quality.

0 – 20 = Very low

21 – 40 = Low

41 – 60 = Moderate

61 – 80 = High

81 – 100 = Very high

Figure 1.1: Clinical Trials Assessment Measure (CTAM; Tarrier & Wykes, 2004) – Modified

Author(s):		
Title:		
Year:	Journal:	
Reviewer:		
Trial Design Area	Item	Score
Sample (maximum = 10)	Type of Sample: convenience sample (2); geographic cohort (5); highly selective sample (0) [Convenience sample: e.g. clinic attendees, referred patient; Geographic cohort: all patients eligible in a particular area; Highly selective: e.g. volunteers]	
	Sample Size: at least 27 participants in each group or based on adequate power calculations (5)	
Allocation (maximum = 16)	True randomisation or minimisation allocation (10)	
	Process of randomisation described (3)	
	Process of randomisation carried out independently from the trial research team (3)	
Assessment (for the main outcome) (maximum = 32)	Assessment carried out by independent assessors (10)	
	* Sleep log data (at least one of sleep-onset latency (SOL), wake after sleep onset (WASO), total sleep time (TST) or no. of awakenings) or other standardised subjective assessments (3) and/or... Subjective physiological data e.g. polysomnography or EEG (3)	
	Assessments carried out blind to treatment allocation (10)	
	Methods of rater blinding adequately described (3)	
	Rater blinding verified (3)	
Control groups (maximum = 16)	TAU is a control group (6) ... and/or a control group that controls for non-specific effects or other established or credible treatment (10)	
Analysis (maximum = 15)	Analysis appropriate to design and type of outcome measure (5)	
	Analysis includes all participants randomised (intention to treat) (6) ... and adequate investigation and handling of drop-outs from assessment if attrition rate exceeds 15% (4)	
Active Treatment (maximum = 11)	Treatment adequately described (3)	
	Treatment protocol or manual used (3)	
	Adherence to the treatment protocol quality assessed (5)	
	Total Score =	

Where criterion not reached for any question score = 0
Total maximum score = 100
* Modified item

Results

Search Results

The electronic database search generated 534 articles. Fourteen articles were selected on the basis of their title or abstract and full text articles were requested. Thirteen full text articles were available for review, 3 of which were subsequently excluded (Appendix 1.2, articles 2-4). Ten articles were selected for inclusion and their references then examined, identifying two further articles. Review articles which had been identified in the database search were examined for further potentially relevant articles. The contents pages of all issues of the journal *Applied Psychophysiology and Biofeedback were also searched. H*owever, no further articles were identified. A flowchart illustrating the search process is presented in Figure 1.2. The reasons for the exclusion of articles which had been selected for full text review are outlined in Appendix 1.2. Twelve articles, summarised in Table 1.1, were finally included in the review.

Figure 1.2: Flowchart of Literature Search Process

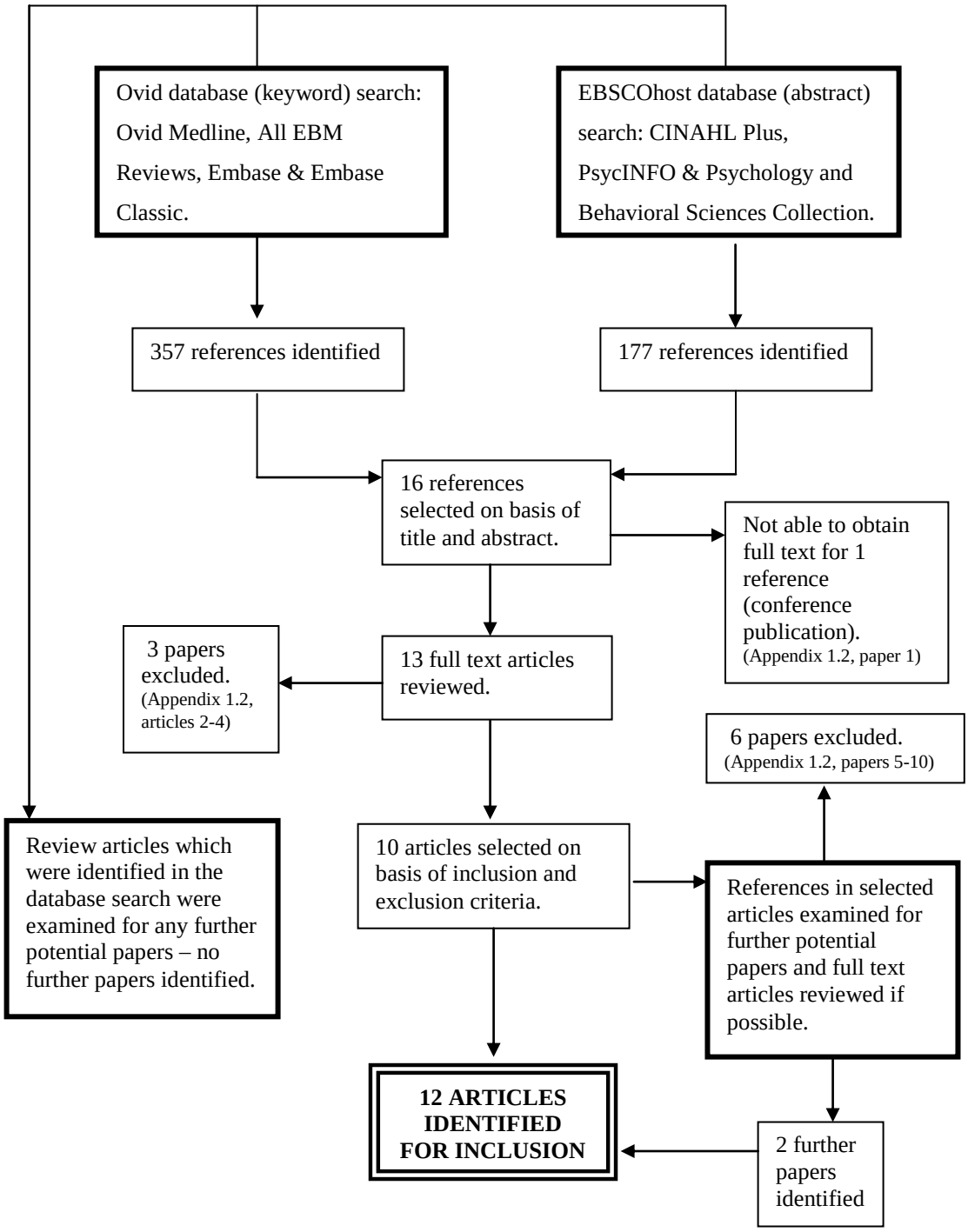

Table 1.1: Summary of Selected Articles

Study	**Quality Rating** (max = 100)	**Participants**	**Groups**	**Treatment Length & Follow-up**	**Main Outcome Measures**	**Summary of Main Findings**
Cortoos et al. (2010a)	69 High	17; sleep-onset or -maintenance insomnia (referred patients) (12 healthy sleepers) Mean age: 41.5 in neurofeedback group; 43.8 in EMG group Duration: >6months	Neurofeedback EMG biofeedback Healthy control	20 sessions over 8 weeks. No follow up.	Home Sleep Log: Total Sleep Time (TST**)**, Sleep onset latency (SOL), Wake after sleep onset (WASO), Sleep efficiency (SE). Polysomnography: TST, SOL, WASO, SE, Percentage of sleep time spend in different stages of sleep.	Both treatment groups showed a decrease in sleep onset latency. Significant increase in total sleep time after neurofeedback only.
Coursey et al. (1980)	42 Moderate	22; chronic sleep-onset insomnia Duration: at least 2 years, average 14 years Mean age: 38.6	EMG biofeedback Autogenic training Electrosleep therapy	Sessions (35-40mins), twice weekly for 6 weeks. Daily home practice: EMG group 2x daily (10mins); autogenic 3x daily (5-8mins) Electrosleep: 5x weekly treatment, no home practice. 1 month follow up.	Home Sleep Log: SOL, SE Sleep-EEG: SOL, TST, SE, WASO, % REM/Delta sleep.	EEG sleep records and home sleep log showed meaningful improvement in 3 biofeedback participants, 2 autogenic participants and none of the electrosleep participants. The biofeedback group showed significantly more improved patients than the electrosleep group.

Study	**Quality Rating** (max = 100)	**Participants**	**Groups**	**Treatment Length & Follow-up**	**Main Outcome Measures**	**Summary of Main Findings**
Freedman & Papsdorf (1976)	67 High	18; sleep onset insomnia (volunteers) Duration: at least 6 months Age 17-39 (median 23)	EMG biofeedback Progressive relaxation Placebo 'relaxation'	6 (30-min) sessions over 2 weeks. Daily home practice (20-min) 2 month follow-up interview.	Home Sleep Log: SOL Polysomnography: SOL	Biofeedback and relaxation both significantly improved sleep onset latency but did not significantly differ from each other.
Hauri (1978)	46 Moderate	37; chronic, severe insomnia. Referred insomnia patients.	EMG biofeedback EMG/theta feedback SMR feedback TAU control	15 - 60 sessions No follow up at time of reporting	Home Sleep Log: TST, SOL, no. awakenings Laboratory: SOL, SE, no. awakenings, % of sleep stages.	TST and SOL significantly improved in EMG biofeedback group according to home but not lab data. SMR group showed improved SOL in home data and SE in lab data. No significant improvements in EMG/theta or TAU control.
Hauri (1981)	49 Moderate	46; chronic, serious insomnia (referred patients & media recruited volunteers) Duration 2-40 years (mean 8 years)	EMG biofeedback EMG/theta feedback SMR feedback No-treatment control	15 – 62 sessions (mean 24.8) 9 months follow up	Home Sleep Log: TST, SOL, no. awakenings Laboratory: SE, SL, no. of awakenings, % of different sleep stages, no. of body movements.	No significant differences between the 3 treatment groups. Specific types of biofeedback may be effective in the treatment of specific types of insomnia.
Hauri et al. (1982)	49 Moderate	16; chronic insomnia	Theta feedback SMR feedback	About 6 EMG sessions as well as about 26 theta or SMR sessions, over 13 weeks on average (sessions 2-3x per week) 9 months follow up	Home Sleep Log: TST, SOL, no. awakenings Laboratory: SE, SL, no. of awakenings, % of different sleep stages, no. of body movements.	Theta & SMR feedback groups showed improved sleep in sleep log data. Sleep lab data indicated that tense/anxious participants benefited from theta feedback only, while those who were relaxed at baseline benefited only from SMR.

Study	**Quality Rating** (max = 100)	**Participants**	**Groups**	**Treatment Length & Follow-up**	**Main Outcome Measures**	**Summary of Main Findings**
Haynes, Sides & Lockwood (1977)	40 Low	24; Moderate sleep-onset insomnia (uni & media recruited volunteers) Duration: average 7.1 years Mean age: 34.2	EMG biofeedback Relaxation instructions Control (relaxation with no instructions)	6 (30-min) bi-weekly sessions Daily home practice at bedtime. 3 & 12 months follow up	Home Sleep log: SOL, TST, no, of awakenings	Biofeedback and relaxation equally effective in treating sleep onset insomnia, both significantly better than conrol in self-report sleep measures.
Hughes & Hughes (1978)	37 Low	36; insomnia Duration: at least 4 months	EMG biofeedback Pseudo-biofeedback Relaxation Stimulus control training	Biofeedback and pseudo-biofeedback: 8 (40-min) sessions, 4 for relaxation and 2 for stimulus control training. Daily home practice (bedtime) 1 year follow up	Home Sleep log: SOL	SOL decreased in all four groups, no significant differences between groups.
Nicassio, Boylan & McCabe (1982)	36 Low	40; Severe insomnia (volunteers) Duration: average 11.2 years Age 17-64 (mean 43.5)	EMG biofeedback Pseudo-biofeedback Progressive relaxation No-treatment control	10 (30-min) sessions over 6 weeks & daily home practice (twice daily) 6 months follow up	Home Sleep Log: SOL, TST, no. awakenings, sleep quality Spouse/roommate reports	Reported SOL decreased in progressive relaxation and EMG biofeedback. However, neither more effective than placebo group. Improvements maintained at follow-up
Sanavio (1988)	42 Moderate	24; persistent psychophysiological insomnia (referred patients) Duration: approx 10 years (1 – 25 years) Clinically referred patients	EMG biofeedback (high & low cognitive arousal) Cognitive treatment (high & low cognitive arousal)	6 (35-min) sessions over 2 weeks 3 and 12 months follow up	Home Sleep Log: SOL, TST, no. awakenings Spouse/roommate reports	Both treatments significantly improved SOL, TST & sleep quality. Neither treatment more beneficial than the other or for high/low cognitive arousal groups.

Study	**Quality Rating** (max = 100)	**Participants**	**Groups**	**Treatment Length & Follow-up**	**Main Outcome Measures**	**Summary of Main Findings**
Sanavio et al. (1990)	46 Moderate	40; persistent insomnia (both sleep onset & maintenance) Duration: 5-25 years (mean 11.8) Clinically referred participants	EMG biofeedback Cognitive treatment Stimulus control & relaxation Waiting list control	6 (50-60-min) sessions over 2 weeks & daily home practice 1 & 3 year follow ups	Home Sleep Log: SOL, WASO, TST Spouse/roommate reports	Sleep improved in all 3 treatment groups. No changes in waiting list control. No significant differences between treatments. Maintained at follow-up.
VanderPlate & Eno (1983)	28 Low	24; mild insomnia; volunteer undergraduate female students Duration: 3 months – 3 years (mean 1.4 years)	EMG biofeedback EMG pseudo-feedback Self-monitoring Waiting list	10 (15-min) sessions & daily home practice 2 months follow up	Home Sleep Log: SOL, TST	Both biofeedback & pseudo-feedback groups showed reduced SOL, no difference between groups. Biofeedback but not pseudo-feedback showed significantly reduced EMG levels.

Abbreviations
Electromyograph (EMG); Electroencephalograph (EEG); Sensorimotor rhythm (SMR); Treatment as usual (TAU)

Total Sleep Time (TST); Sleep onset latency (SOL); Wake after sleep onset (WASO); Sleep efficiency (SE)

Pittsburgh Sleep Quality Index (PSQI; Backhaus et al., 2002); Insomnia Severity Index (ISI; Bastien et al., 2001); Quality of Life Inventory (QOLI; Frisch et al., 2005)

Using the modified CTAM (Figure 1.1), two articles received high quality rating scores, six received moderate scores and four received low scores (Table 1.1). The mean score was 46.

Studies comparing the efficacy of EMG biofeedback and relaxation as treatments for insomnia will initially be reviewed, starting with the earliest. The earliest studies included a placebo relaxation condition but not a placebo biofeedback condition. Studies which introduced a placebo biofeedback condition will then be considered, followed by studies comparing EMG biofeedback with cognitive treatments. The review will then move on to consider studies looking at the efficacy of neurofeedback. Some studies compared EMG biofeedback with neurofeedback and others looked at the efficacy of different neurofeedback protocols.

Studies comparing EMG biofeedback to relaxation

Freedman & Papsdorf (1976) compared EMG biofeedback with instructed progressive relaxation as well as a placebo 'relaxation' group in which instructions were given to perform a set of simple exercises originally intended to help low back pain. Instructions were administered verbally to each group by the experimenter. Participants were informed about all three procedures before being randomly assigned to a group. Participants took part in 6 treatment sessions over 2 weeks and were asked to practice each evening at home (the biofeedback group practiced at home without the machine). SOL was measured subjectively using sleep logs and objectively by all-night EEG recordings. Volunteer participants, recruited from advertisements placed around a university campus and in the media, spent two consecutive nights in the sleep laboratory at baseline and post-treatment (recordings made on the 2nd night only). The two-month follow up consisted of subjective measures only. Biofeedback and relaxation both significantly improved sleep onset latency,

but did not significantly differ from each other. A significant correlation was found between subjective and objective SOL suggesting good reliability. However, it was reported that the baseline physiological data did not significantly correlate with the baseline SOL, suggesting that there may have been factors other than physiological arousal responsible for decreasing SOL. Sleep-onset times in the lab were independently and blindly re-scored with high reliability found. Freedman & Papsdorf's study scored relatively highly on the quality rating scale compared to the majority of other studies reviewed (see Table 1.1). This study was one of only two to receive 'High' scores according the arbitrary rating system.

Haynes et al. (1977) also found both EMG biofeedback and relaxation alone to significantly reduce SOL compared to a control group, and again the two treatments did not significantly differ from each other. Similar to the study by Freedman & Papsdorf, participants were volunteers recruited via newspaper advertisements and from university. About half of the participants were university students and the mean age of participants was 29.3 years. In this study, control group participants were told to relax but not given any further instructions on how to do so. Unlike the previously described study, in which instructions were given verbally, recorded instructions were played to all groups which included the same rationale about the benefits of relaxation. Conversation between experimenter and participants was kept minimal. It was reported that the two-week baseline phase in which sleep logs were kept was also intended to control for possible reactive effects of self-monitoring. The groups did not differ at baseline in terms of sleep variables. The improvements in self-report sleep measures seen post-intervention were maintained at 1 year, however several participants were lost to follow-up. A limitation of this study is that only subjective sleep measures were employed. Additionally, it received a 'low' score on the quality rating scale. Tarrier & Wykes (2004), in their analysis of randomised controlled trials of cognitive behaviour

therapy for schizophrenia, found evidence that studies with poorer methodology tended to over-estimate the positive benefits of the treatment.

Coursey et al. (1980) compared EMG biofeedback to a different type of relaxation therapy, autogenic therapy (as described by Schultz & Luthe, 1959), as well as electrosleep therapy. Like the study by Haynes et al., training took place over a six week period (in comparison to the shorter 2 week period of the Freedman & Papsdorf study). However, unlike Haynes et al.'s study, the therapist was present throughout sessions and discussion took place about individual progress, home practice and any problems encountered. The participants in this study were, on average, older and reported difficulties with insomnia for a longer duration than in the two previously described studies (see Table 1.1). Although the method of recruitment is not described, it was reported that participants were required to have previously sought help due to their insomnia, a criteria that was not required in the above described studies. Participants in this study reported previously trying multiple treatments.

Autogenic therapy has more of a cognitive component than the progressive relaxation used in the two studies described above. Participants focused on simple, repetitive phrases suggesting feelings of warmth and heaviness e.g. "my right arm is heavy". At the same time they attended to the feelings experienced in the corresponding body part. Electrosleep therapy was employed as a placebo control group to the biofeedback group, something that had not been done in the above described studies. Electrosleep resembled biofeedback since electrodes were attached to the forehead and a therapist was present, however, it did not involve active engagement in relaxation. Instead it involved administration of low-current electrical stimulation. The daily home practice varied between groups (see Table 1.1) but both groups carried out the final practice before bed. A portable EMG feedback device was

provided for home practice, something which wasn't available to participants in the Freedman & Papsdorf study. This greatly increased the amount of biofeedback practice that participants were able to engage in (twice daily for 6 weeks at home). Daily sleep logs were kept from 2-5 weeks pre-treatment and for 2 weeks post-treatment. All-night EEG recordings were made on 4 consecutive nights (out of 5 in the sleep laboratory) 1 week pre- and 1 month post-treatment. Both EEG & sleep logs showed significantly reduced SOL in 50% (3/6) of biofeedback participants, 2/6 autogenic participants and none of the electrosleep participants with the biofeedback group showing significantly better improvement than the electrosleep group. Since improvements were not seen in all groups, it seems less likely that they were due to placebo or expectation. Objective EEG recordings corroborated the subjective data. It was reported that the participants who did not respond to treatment at all were experiencing more serious and ongoing life stress at the time of the study than those who did respond. However it was also noted that this stress was not evident in EMG levels indicating that this stress did not seem to be interfering with sleep due to muscle tension. The study by Coursey et al. received a score of 42 on the rating scale, which lies at the lower end of the 'medium' category.

Hughes & Hughes (1978) compared EMG biofeedback with pseudo-biofeedback (a placebo in which false feedback was given), relaxation as well as stimulus control training. They found that self-reported SOL decreased in all four groups with no significant differences between groups, including the pseudo-feedback placebo group. The four groups were designed to be as similar as possible to each other. The biofeedback and pseudo-biofeedback groups were identical except for the actual/false feedback. The studies by Freedman & Papsdorf and Haynes et al. employed a relaxation group placebo but not a biofeedback placebo. Electrosleep was introduced as a placebo by Coursey et al., and whilst

it was similar in procedure to EMG biofeedback, it was not as similar as the pseudo-feedback employed in this study, which was a strength. The relaxation group listened to taped instructions and the stimulus control group were given instructions during the first session relating to their sleep behaviour and their progress was discussed and encouraged during subsequent sessions. All sessions started with a review of the previous week's sleep chart. This individual review and discussion may have been an important part of the intervention, and may be comparable to the opportunity that participants in Coursey et al.'s study had to discuss their progress.

Daily sleep logs were kept for two weeks at baseline and again post-treatment. The number of training sessions varied between groups and depended on the complexity of the treatment (see Table 1.1). However, participants in all groups received substantially less training than participants in Coursey et al.'s study and the stimulus control training group received only two training sessions. Similar to participants in the above described studies, they were told to apply what had learned in the evening to help them sleep, although biofeedback equipment was not provided. All four groups showed reduced SOL, but did not differ significantly from each other. Improvements were maintained at 1 year follow-up (conducted by telephone), however contact was only made with 12 participants at this time. Like in the above studies, participants were volunteers, they were not required to have sought help previously (as with Coursey et al.) and the reported duration of symptoms was not reported although it may have been as little as four months (see Table 1.1). Objective laboratory data was not collected and the study received a 'low' quality rating score (similar to Haynes et al.).

EMG activity was not significantly reduced and so did not seem to explain the significant reduction in SOL. Also, EMG scores did not significantly differ across groups indicating that the modest reduction that was found was not due to the effects of a particular treatment. Medication was ruled out as a possible confounding variable. Hughes & Hughes suggested that the introduction of a bedtime routine may be a nonspecific effect of the treatments which could have influenced the results. Cognitive activity that may have previously interfered with sleep-onset could have been reduced through involvement in the routines engaged in by participants as part of their treatment. Indeed this may be true of the other studies reviewed. It was also suggested that expectation of improvement could have resulted in less worry about insomnia. It has been well documented that worry about insomnia interferes with sleep (Espie, 2002; Harvey, 2002).

In a similar study, Nicassio et al. (1982) compared EMG biofeedback with pseudo-feedback and progressive relaxation, as well as a no-treatment control group. The results were similar to those of Hughes & Hughes (1978) in that EMG biofeedback was found to be equally as successful as relaxation alone in improving sleep, and neither treatment was significantly more effective than the pseudo-feedback placebo group. The participants in Nicassio et al's study were older and are likely to have had insomnia for considerably longer (see Table 1.1; however mean duration was not reported by Hughes & Hughes). Participants in all groups took part in a greater (and equal) number of training sessions, and more frequent home practice compared to the Hughes & Hughes study. Additionally, subjective sleep log data was substantiated by spouse/roommate reports however, again there was no polysomnographic data collected. Again, this study scored 'low' on the quality rating scale. Improvements were maintained at 6-months on follow-up.

EMG activity was found not to be significantly associated with SOL reduction, suggesting that it may not have been a reduction in physiological relaxation that was responsible for the reduced SOL. Also supporting this is the fact that the false-biofeedback placebo treatment also resulted in reduced SOL. As in the study by Hughes & Hughes, the placebo was intended to control for expectation of improvement. It could be that a high expectation for improvement may have impacted on the results of both studies. Cognitive processes may have had a role in the improved sleep reported. It was suggested by Nicassio et al. that engaging in the treatment at bedtime could perhaps focus attention away from cognitive intrusions which may otherwise have delayed sleep-onset. Perhaps the daily home practice aspect of the treatments is important for this reason.

Neither of the studies by Nicassio et al. or Hughes & Hughes employed objective laboratory data unlike the studies by Freedman & Papsdorf and Coursey et al. There is evidence that individuals with insomnia are often inaccurate when self-reporting sleep latency, with a tendency to over-estimate it (Freedman & Papsdorf, 1976; Monroe, 1967). However, Freedman & Papsdorf reported that participants' estimates of SOL became more consistent with objective data as treatment progressed.

In the previously described studies, EMG level was not found to be significantly related to insomnia or sleep improvements suggesting that learned relaxation may not have taken place. VanderPlate and Eno (1983) carried out a study with the aim of further investigating the efficacy and specific effects of EMG biofeedback for insomnia and to do so when learned relaxation does in fact occur. They compared EMG biofeedback, an identical pseudo-biofeedback group, a self-monitoring group and a waiting list control. The treatment protocol was not sufficiently described to allow comparison with the other studies.

The self-monitoring control was included to investigate whether the self-monitoring aspect of treatments alone led to improved sleep. Both the biofeedback and pseudo-biofeedback group reported significantly reduced SOL relative to the two controls with no significant difference between these groups (maintained at 2 months follow-up). SOL in the self-monitoring or waiting list control groups did not improve. Findings relating to the non-specific effects of the placebo group are consistent with Hughes & Hughes and Nicassio et al.'s studies. However, the findings also suggest that the self-monitoring aspect of the interventions is not responsible for the improved sleep. Biofeedback but not pseudo-feedback was found to significantly reduce EMG levels suggesting that the participants in this group had actually learned to relax (Hughes & Hughes found no difference between these groups in EMG level suggesting that this learning may not have taken place). However, no relationship was found between EMG level and insomnia. This study provides further evidence that reduced SOL following biofeedback may not be due to reduced muscle tension. VanderPlate and Eno suggest that expectancy may play a role. Results of this study, however, should be interpreted with caution. Like many of the previously discussed studies, the sample was small. This study, however, employed an all female, volunteer undergraduate student sample with relatively mild insomnia compared to other studies, therefore the results may not generalise to the wider insomnia population. The self-report data was not corroborated with objective laboratory data and the quality rating of this article was the lowest of the studies reviewed (see Table 1.1).

Studies comparing EMG-biofeedback & Cognitive Treatment

Pre-sleep arousal can be both physiological and cognitive in nature, and most likely a combination of both. Morin (1993) outlines an interacting sub-systems model of insomnia in which cognitive processes such as worry, unrealistic expectations or rumination over

possible consequences of sleep loss may become dysfunctional and result in hyperarousal (via maladaptive habits and their consequences). In this model, hyperarousal can be emotional, cognitive and physiological in nature and in turn leads to further dysfunctional cognitions. Physiological and cognitive hyperarousal are likely to interact in both directions with an increase or decrease in one, similarly affecting the other. Another example of this interaction is given by Morin et al. (2003) who report that individuals with insomnia perceive life stressors as more stressful compared to those without sleep difficulties, which results in increased pre-sleep arousal and poorer sleep.

Sanavio (1988) hypothesised that individuals who experienced either excessive physiological arousal or excessive cognitive arousal may benefit from a treatment specifically addressing that type of arousal. Sanavio compared EMG biofeedback with a cognitive treatment which included paradoxical intention, thought stopping and cognitive restructuring components. The Pre-sleep Intrusive Cognitions Inventory (PICI; Rolletto et al., 1983, in Sanavio, 1988) was administered to identify individuals with either high or low pre-sleep cognitive arousal. Six high and six low arousal participants were then randomly assigned to each treatment group and a 7 day baseline was established from sleep logs. Treatments were standardised using video and written information and scripts were provided for home practice. Both treatment groups reported significantly improved SOL, TST & sleep quality which was maintained at 3 and 12 months. However, there were no significant differences between treatments and neither treatment was more beneficial than the other for high and low cognitive arousal groups, thus not supporting the hypothesis. A strength of this study was that participants were referred patients reporting insomnia for a duration of approximately 10 years. Limitations include the use of a very small sample, the short treatment period and the quality rating which just made the ‘moderate’ classification

(see Table 1.1). Spouse/roommate checks showed adequate reliability with the sleep log data, although no polysomnography or EEG recordings were carried out.

In a subsequent study, Sanavio et al. (1990) investigated the effects of three different types of treatment: EMG biofeedback, cognitive treatment (including paradoxical instructions, thought-stopping and cognitive-restructuring) and stimulus control therapy (targeting maladaptive sleeping habits and also incorporating a progressive relaxation component). These treatments were compared to a waiting list control. The rationale behind stimulus control therapy is that poor sleep hygiene may have led to going to bed becoming associated with arousal instead of sleep cues. All three treatment groups reported improved SOL, WASO, sleep quality and restedness two months post-training. Of note is that only the cognitive treatment group reported significantly improved TST. Similar to the previous study, there were no overall significant differences between treatments and no change was reported by controls. These effects were maintained at one and three year follow-up. Sanavio et al. chose not to restrict participants to those with only sleep-onset difficulties for the reason that this may not be reflective of the general insomnia population. All participants were required to be clinically referred patients and to report both sleep-onset and –maintenance difficulties with a duration of at least 5 years. Video and written information was used to standardise the treatments and manuals & scripts were provided for home practice. Like the Sanavio (1988) study, the sample was small and no laboratory data was gathered to corroborate the subjective data, however spouse/room-mate data were gathered and agreement with the self-report data was high.

Studies comparing EMG-biofeedback & Neurofeedback

Hauri (1978) compared three different types of biofeedback: EMG biofeedback, EMG-theta feedback, and SMR feedback, as well as a treatment as usual (TAU) control with the intention of investigating whether any effects were due to either non-specific effects of biofeedback training or to a specific biofeedback parameter. Participants were randomly assigned to one of the four groups. The EMG theta group first received EMG training similar to the EMG biofeedback group before commencing the theta feedback training. Feedback was in the form of clicking sounds. SMR feedback was given by a yellow light which varied with SMR and a sound which indicating when SMR activity had reached a certain level. The TAU group received an appropriate "best current treatment" e.g. psychotherapy, stimulus control therapy or medication. The number of training sessions greatly varied between participants (15-60) according to individual progress. Additionally, training procedures and interactions between technicians and participants were not standardised either. Sleep was evaluated in the laboratory for 3 nights pre- and post-treatment. Daily sleep logs were kept throughout the training period and for 2 weeks post-treatment. TST and SOL significantly improved in the EMG biofeedback group according to subjective, home data but not lab data. The SMR group showed improved SOL in home data and improved SE in the lab data as well as to lengthen time in stage 2 sleep. No significant improvements in EMG/theta or TAU control. The EMG/theta group were reported to have been less thoroughly trained in EMG than EMG alone group, which may explain the differing results between these two groups. Differences between the laboratory and home data suggest problems with reliability or validity. Hauri suggested that individual differences may impact on the method of biofeedback most likely to be beneficial i.e. differences in levels of psychological or physiological arousal or sleep spindle activity during stage 2 sleep. When this was investigated post hoc, it was found that when

appropriate biofeedback was provided (by chance due to randomisation), it was indeed more successful.

However, it was noted by Hauri that there was some concern about there being variation over time in the enthusiasm held by technicians for particular therapies. This was addressed by monitoring this and attempting to increase enthusiasm for therapies as considered appropriate through discussion and the provision of reading material. Participants in this study were referred patients with chronic, severe insomnia, which is a strength. However limitations include the lack of standardisation in treatment which was evident. The study received a moderate quality rating (Table 1.1). Also of note is that although participants did learn to produce more SMR in the lab, they did not become aware of any feelings associated with SMR activity, which was reported as frustrating. SMR feedback is a more complicated form of biofeedback in terms of participant understanding as well as training provision.

In a further study by Hauri (1981), EMG biofeedback, EMG/theta feedback and SMR feedback were again compared but this time with a no-treatment group to control for change occurring over time, or due to life events. Interest at this time was not just in looking at relaxation as the end point but in trying to establish mechanisms by which change took place. Participants, of which some were referred insomnia patients and all reporting symptoms for at least 2 years (Table 1.1), were randomly allocated to groups. Polysomnography was carried out over 3 nights in the laboratory pre-training, immediately post-training and at 9 months follow up. This time, neither of the three treatment groups showed any more improved sleep than the control group and there were no significant differences between the three groups.

Also investigated was whether the treatment each participant was randomly assigned to was clinically appropriate or inappropriate according to their specific type of insomnia and whether this affected outcome. This was similar to the approach taken by Sanavio (1988). Both EMG and EMG/theta biofeedback was considered appropriate for individuals whose insomnia was thought to be due to high arousal levels. However, some individuals already had low arousal levels, and for these individuals, SMR training was considered appropriate. It was hypothesised that these individuals may have difficulties inducing sleep due to having a weaker sleep system, evident in poorly formed sleep spindles, and that this may be targeted by SMR training. Unlike in Sanavio's (1988) study, the results indicated that only those who received appropriate biofeedback showed significantly improved sleep. Relaxation training helped those who found it difficult to relax, but those who were able to relax easily were more likely to be helped by SMR feedback. These results indicate that the effects were not likely to be due to a placebo effect. The technician carrying out the training was blind to whether participants were receiving appropriate or inappropriate feedback. Similar to the 1978 study, the data indicated that EMG training alone may be more successful than EMG/theta training and it is possible that this is due to the EMG portion of the training being more thorough when it was the sole type of training administered.

Hauri et al. (1982) replicated the 1981 study because of identified methodological shortcomings: the post hoc nature of the findings relating to appropriate/inappropriate biofeedback on outcome, and the differences in the equipment used in EMG/theta feedback compared with SMR feedback (i.e. auditory versus visual feedback as well as differences in the way the equipment was affected by artifacts). However, both studies were given identical moderate quality ratings (Table 1.1). The recruitment, selection and assessment of participants remained the same. However, participants were then randomly assigned to

either a theta or SMR feedback group, with the groups being matched for sex and age. All participants received some EMG feedback training at the outset. The same type of equipment was used for both groups with a coloured light, which varied in brightness, being used to provide the feedback. The same explanation was provided to participants in both groups and the technicians were thought to be unbiased in their expectations. Data were gathered from 2 weeks of home sleep logs and 2 nights in the laboratory (out of a consecutive 3 nights) pre-training, immediately post-training and again 9 months later.

The results generally replicate those of the original study. The subjective data showed improved TST and SOL in both the theta and SMR feedback groups. However, the laboratory data indicated that neither of the groups had significantly improved sleep over the course of the study and that neither theta nor SMR feedback was significantly more successful than the other. Participants were randomly allocated to groups so some may have received biofeedback training appropriate to their particular type of insomnia while others may have received a more inappropriate treatment. Looking at the subjective sleep log data alone, appropriateness did not seem to be relevant with improvements being reported when both appropriate and inappropriate training was administered. This finding is similar to the findings by Sanavio (1988) which included subjective data only. However, the objective laboratory data indicated that when the feedback was appropriate (i.e. theta feedback was deemed appropriate for participants who were muscularly tense, and SMR feedback was deemed appropriate for those who were not), SE & SOL significantly improved. Sleep did not improve when the feedback was of the inappropriate type.

A limitation of this study is that the results did not all reach significance, however the low numbers would suggest it may not have been adequately powered. The results add to the

evidence that arousal levels are relevant to which type of biofeedback is likely to be most beneficial. Relaxation training methods seem most suitable for those with high arousal levels and SMR feedback most suitable for those already able to relax sufficiently.

All the studies reviewed so far were published between 1976 and 1990. However, a more recent study has since emerged (Cortoos et al., 2010a) and a further study was known to be in press at the time of writing (Hammer et al., 2011). Cortoos et al. investigated neurofeedback, although rather than focusing on frequencies associated with sleep-onset, as Hauri did, Cortoos et al. decided to focus on several frequencies relating to sleep and cognitive processes. This change in protocol was reported to be influenced by more recent research findings and theories including the neurocognitive model (Perlis et al., 1997). The neurofeedback group in this study was trained to increase SMR, whilst inhibiting theta and high beta activity. Also influencing this change in protocol was Cortoos et al.'s observation that research suggests that, for a proportion of insomnia patients, treatments targeting cognitive and physiological arousal does not result in substantial enough improvements in sleep. A focus on cortical arousal was therefore thought to be warranted.

The biofeedback training in this study varied from that of previous studies in another aspect also as it took place at participants' homes over an internet connection. The researchers named this variation 'tele-neurofeedback' and 'tele-biofeeback'. Participants were trained to use the equipment and attach the electrodes themselves. Training took place according to a strict timetable and the therapist phoned participants prior to each session to check that there were no technical difficulties. The equipment gave visual feedback as part of training.

A good sleeper control group was also included in order to compare baseline sleep measures.

Sleep logs were completed for two weeks prior to training and during training. Polysomnography was recorded in the sleep laboratory pre- and 2 weeks post-training. The results indicated that the neurofeedback group was more successful at improving sleep according to both objective and subjective data than the EMG biofeedback group. Both types of biofeedback led to decreased objective SOL, however most participants reported insomnia of the sleep-maintenance type. Only the neurofeedback group showed improved objective TST and the neurofeedback group showed greater improvement in objective WASO. The neurofeedback group showed improvement in all subjective measures, while the EMG biofeedback group did not show improved sleep logs.

The neurofeedback group showed greater improvements compared to the EMG biofeedback group. This contrasts with the previous research findings discussed. Hauri (1981) found no significant differences between EMG biofeedback, EMG/theta feedback and SMR feedback groups. As discussed, the neurofeedback protocol used by Cortoos et al. is different to the SMR training used by Hauri in previous studies, targeting cognitive processes rather than sleep onset. This approach had never before been used in an insomnia intervention study.

Subjective and objective data indicated that the insomnia participants in this study were more cognitively aroused but not more physiologically aroused than the healthy sleeper controls. This may have therefore influenced results i.e. that the intervention targeting physiological arousal was less successful. All of the subjective sleep variables improved in the neurofeedback group only indicating that nonspecific effects of the training alone were not responsible for the improvements. Previous biofeedback studies took place in a laboratory setting, often with a therapist. It is possible that the home setting may have impacted on the results. The participants in this study were referred insomnia patients

reporting symptoms for a minimum of 6 months. This study scored highly on the quality rating scale, obtaining the highest score amongst the studies reviewed (Table 1.1). Like all of the previous biofeedback studies, the small sample size may affect power.

The tele-feedback procedures reduce the time commitment for participants but, obviously the home environment is not a controlled environment and the number of distractions or disruptions experienced by participants is not known. Cortoos suggested that further investigation to compare neurofeedback in the lab with tele-neurofeedback would be useful.

Discussion

The aim of this review was to evaluate the evidence for the efficacy of biofeedback as a treatment for insomnia. Twelve articles were reviewed and included articles investigating the efficacy of EMG biofeedback and various neurofeedback protocols. Various biofeedback procedures were compared to each other and to relaxation and cognitive therapies as well as a variety of control groups.

Several studies found EMG biofeedback to significantly improve SOL but to be no more effective than relaxation. This finding was reported by Freedman & Papsdorf (1976) in their high quality rated study employing both subjective and objective data; Haynes et al. (1977) in their low quality rated study which employed subjective data only; and also in Hughes & Hughes' (1978) study which also employed only subjective data and was rated as low quality. However participants in all three studies were relatively young volunteers from the general population and university campuses and the results may not generalise to a wider and more chronic insomnia population. Hughes & Hughes' study included a pseudo-biofeedback group as a placebo with an almost identical procedure to the EMG biofeedback group. Nicassio et al. (1982) also employed a similar placebo group and the findings were similar with EMG biofeedback, pseudo-feedback and relaxation groups all reporting reduced subjective SOL. Participants in Nicassio et al.'s study reported more chronic insomnia and the subjective data was corroborated by spouse/roommate reports and the participants. VanderPlate & Eno (1983) also found pseudo-feedback to improve subjective SOL, although it is of note that this study received the lowest quality rating of those reviewed. Since these placebo groups, which

employed procedures almost identical to their corresponding EMG biofeedback procedures, also reported improved (subjective) sleep, it seems likely that non-specific effects of the interventions are impacting on the outcome.

Whilst results indicate that engaging in EMG biofeedback can lead to reduced sleep latency, it seems that this may not be due the intended effects of biofeedback i.e. learning how to reduce muscular tension or to relax by whatever means. In general, EMG level was not found to be significantly related to insomnia or sleep improvements (Freedman & Papsdorf, 1976; Haynes et al., 1977; Hughes & Hughes, 1978; Nicassio et al., 1982). It is of note that these studies are roughly thirty years old, and other than the Freedman & Papsdorf study, do not score highly on the modified CTAM. This suggests they are not meeting today's standards in terms of their methodological quality. The data suggest that improvements in sleep variables may not have been due to learned relaxation occurring but to some nonspecific effects of treatment. The findings of the studies which employed pseudo-biofeedback further implicate that non-specific effects may have impacted on the results.

So what are these non-specific effects that may be influencing the results and resulting in improved sleep? VanderPlate & Eno (1983) included a self-monitoring control group in their study and concluded that the self-monitoring aspect of the interventions is not responsible for the improved sleep. However, the previously described limitations of this study should be noted. There seems to be a substantial amount of variation in protocol between studies e.g. in the number of treatment sessions, delivery of training and interaction with the therapist. One thing they all have in common is daily home practice before bedtime. It has been suggested that this bedtime routine could be an important factor. It is

possible that engaging in some practice which focuses attention away from maladaptive cognitive processes could prevent the high arousal that might otherwise occur and interfere with the onset of sleep. Expectation of improvement could also be responsible for improved sleep. It has been suggested that this expectation could result in less worry about insomnia and its consequences which are thought to play a part in the maintenance of sleep difficulties (Espie, 2002). It is possible that the bedtime routine, expectation of improvement and resulting reduced worry could all play a part in reducing engagement in sleep effort, which can be responsible for maintaining sleep difficulties (Espie et al., 2006).

Sanavio (1988) hypothesised that cognitive treatment may be more successful for individuals with high cognitive arousal impacting on their sleep and that EMG biofeedback may be more successful for individuals with low cognitive (and therefore physiological) arousal. Although sleep improvements were observed following both treatments, there were no significant differences between treatments and neither treatment was more beneficial than the other for high and low cognitive arousal groups. Perhaps a limitation of this study, not previously discussed is the dualistic approach to the study design. Whilst it may be true that some individuals experience either physiological or cognitive pre-sleep arousal as dominant to the other, given the work by Morin (1993; et al., 2003) described above, it is difficult to conceptualise the possibility of treating one form of hyperarousal without also treating the other. It therefore is not surprising that both treatments were equally as successful for both high and low cognitive arousal groups.

Studies investigating the efficacy of Neurofeedback procedures as an intervention for insomnia were also reviewed. Protocols varied in the type of brain activity they targeted with the older studies targeting sleep onset processes (SMR and theta feedback; Hauri 1978,

1981; Hauri et al., 1982) and more recently, Cortoos et al. (2010) introduced a protocol which targeted brain activity related to cognitive processing.

Hauri (1981) found EMG biofeedback, EMG/theta feedback and SMR feedback to be equally successful at improving sleep when the groups as a whole were compared. However, when individuals were considered post hoc, the results indicated that EMG and EMG/theta biofeedback was successful when participants' insomnia was due to high arousal and SMR training was successful when participants' insomnia was not due to high arousal but instead likely to be due to a weaker sleep system. These results contrast with the findings of Sanavio (1988) who did not find support for an appropriate/inappropriate treatment hypothesis, although Sanavio's study was concerned with cognitive arousal, assessed by a self-report questionnaire as opposed to the more objective EMG data collected in Hauri's (1981) study.

Hauri et al.'s (1982) subsequent replication study compared theta with SMR feedback. Both treatment groups showed some improved sleep however neither of the groups had significantly improved sleep over the course of the study and neither protocol was significantly more successful than the other. The objective, but not subjective, data provided further evidence to the hypothesis that particular biofeedback protocols are more appropriate for particular types of insomnia. Training, in which the end-point was relaxation, was more appropriate for those with high arousal levels and SMR feedback seemed more suitable to those already able to relax sufficiently.

Cortoos' et al. (2010a) found their neurofeedback protocol which targeted cognitive processing to be more successful at improving sleep than EMG biofeedback in their high

quality rated study of clinical insomnia patients. A similar neurofeedback protocol had previously been found to improve attentional processes in a non-sleep disordered sample (Egner & Gruzelier, 2001, 2004 as cited in Cortoos et al., 2010a). Cortoos et al.'s study provides evidence that cortical arousal may influence sleep processes in some individuals. Indeed, the results are discussed with reference to Perlis et al.'s (1997) neurocognitive model which suggests that cortical hyperarousal may interfere with normal information processing, which leads to disruptions in sleep. It is important to note that the participants in Cortoos et al.'s sample did not appear to have insomnia caused by high physiological arousal at bedtime. This may, in part, explain why the EMG biofeedback training was less successful in this study. Indeed, different biofeedback protocols may be more or less suitable for different insomnia presentations as suggested by Hauri (1981) and Hauri et al.'s (1982) findings.

Altogether, the studies reviewed suggest that biofeedback may provide a promising treatment for insomnia patients. Different biofeedback protocols have shown promise in different studies and this may be due to the large variation in procedures, protocols, participant characteristics and insomnia subtypes. There is evidence that different procedures targeting physiological, cognitive and cortical arousal have shown promise but the mechanisms driving the observed reductions in insomnia symptoms are not yet clear. Many of the studies reviewed are decades old and the methodological quality of several of the studies is low according to today's standards. For a time, researchers appeared to lose interest in biofeedback as a possible treatment for insomnia and this may have been due to the complicated and time consuming procedures as well as the fact that more simple interventions were found to be just as effective. However, the study by Cortoos et al. shows renewed interest in biofeedback as a treatment for insomnia and technological advances

appear to be able to allow such an intervention to be more easily accessible. Certainly, more research in this area is warranted in order to better understand the mechanisms underlying the intervention and also to perhaps better understand the interaction of arousal and sleep processes.

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CHAPTER TWO: MAJOR RESEARCH PROJECT

Stress Reactivity in Individuals with Non-REM Parasomnias, Insomnia and Good Sleep

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Summary

Sleepwalking and sleep terrors, known as NREM (non-rapid eye movement) parasomnias are a relatively un-studied group of sleep disorders. The purpose of this study was to learn how people with these sleep disorders respond to life stress. The aim was to investigate how their responses to a stressful situation compared to individuals with insomnia and good sleep. Thirty-eight participants were recruited from the general population and attended the University of Glasgow Sleep Centre to take part. To measure stress, heart data was recorded whilst participants took part in baseline, stressor (a difficult mathematical task) and recovery phases. Participants also rated how stressful they found the experience. In general, group differences were not found, however this may be partly due to the small number of people who took part. The results indicated that the sleepwalker/sleep terror group reacted to stress in a similar way to good sleepers. In general, it was the insomnia group but not the NREM group whose data differed from good sleepers. However, both the insomnia and NREM groups exhibited a relatively higher (though not statistically significant) resting heart rate at baseline compared to the good sleeper group, suggesting they may be slightly more stressed in general. The findings indicated that further exploration is warranted which may have potentially important implications for the development of treatments for sleepwalking and sleep terrors.

Abstract

To date, there is little research into either stress reactivity or the specificity of psychological characteristics in particular forms of sleep disorder. NREM parasomnias are a relatively un-studied group of sleep disorders. The purpose of this study was to gain greater insight into how people with NREM parasomnias respond to 'threat' and to life situations. In particular, the aim was to investigate how their responses to a psychological stressor compared to individuals with insomnia and to good sleepers by measuring autonomic arousal, as well as subjective appraisals of stress. Baseline levels of autonomic arousal were intended to provide insight into daytime arousal levels at the trait level. Participants (N = 38) were recruited from the general population and attended the University of Glasgow Sleep Centre to take part. Autonomic arousal was measured via continuous electrocardiogram (ECG) recordings of heart rate (HR) and cardiac vagal tone (CVT) whilst participants took part in baseline, stressor (a difficult mathematical task) and recovery phases. In general, group differences were not found, however this may be partly due to the small sample size and corresponding lack of power to detect differences. The results indicated that the NREM group reacted to stress in a similar way to good sleepers. In general, it was the insomnia group but not the NREM group whose data differed from good sleepers. However, both the NREM parasomnia and Insomnia groups exhibited a relatively higher (though not statistically significant) resting baseline HR compared to the good sleeper group, suggesting a higher level of underlying sympathetic arousal. The findings of this type of study have potentially important implications for the development of treatment programmes for NREM parasomnias. However, further work needs to be done before any conclusions can be drawn. The study was intended as an exploratory study and the preliminary findings indicate that further exploration is warranted.

Introduction

Parasomnias are "undesirable physical events or experiences that occur during entry into sleep, within sleep or during arousals from sleep". The International Classification of Sleep Disorders, Second Edition (ICSD-2) distinguishes between parasomnias associated with non-REM (non-rapid eye movement; NREM) sleep, parasomnias associated with REM (rapid eye movement) sleep, sleep-wake transition parasomnias as well as 'other parasomnias' which are not classified by the other categories (American Academy of Sleep Medicine (AASM), 2005). NREM parasomnias are the most common type of parasomnia (Wills & Garcia, 2002).

There are three normal states that the nervous system can be in: wakefulness, NREM sleep and REM sleep. Most parts of the nervous system are active across the three states, but in different manners, and transitions between states are complex processes (Mahowald & Bornemann, 2005). NREM parasomnias, also known as disorders of arousal, are incomplete arousals from slow-wave NREM sleep which result in behaviours associated with wakefulness. Since arousal is incomplete, the brain is partially awake and partially remains in NREM sleep. The three main NREM parasomnias are sleep terrors, sleepwalking and confusional arousals. They generally occur during the first third of the night and during episodes behaviour can appear confused and semi-purposeful. Individuals generally do not remember the events, which tend to occur no more than once per night and can last from seconds to minutes (Vaughn & O'Neil, 2007). Confusional arousals involve mental confusion or confusional behaviour characterised by disorientation, slow speech, diminished mentation, impaired cognitive response and inappropriate behaviour. Sleepwalking episodes often begin with the individual sitting up in bed and appearing confused before

walking or engaging in more complex behaviours such as dressing, or even driving. Individuals may be emotionally calm or may be agitated. Violent behaviours are more likely to occur if attempts are made to wake the sleepwalker. Sleep terrors are characterised by intense fear which is evident in the individual's behaviour and autonomic arousal. Individuals tend to suddenly sit up in bed, let out a piercing scream and can be inconsolable as well as unresponsive to external stimuli. (AASM, 2005.)

NREM parasomnias are more common in children than adults. Whereas NREM parasomnias in children appear to often be associated with genetic and developmental factors, persistence (or onset) into adolescence or adulthood has been associated with psychopathology (Kales et al., 1980a; Kales et al., 1980b; Kales et al., 1982; Gau & Soong, 1999; Ohayon, Guilleminault & Priest, 1999). However, many individuals with NREM parasomnias do not present with significant psychopathology and in fact show good psychosocial functioning (Mahowald, 2002; Mahowald & Borneman, 2005; Schenck, Boyd & Mahowald, 1997).

NREM parasomnias are thought to occur as a result of a complex group of priming and precipitating factors in those that are genetically predisposed. NREM parasomnias seem to be more likely to occur in the presence of factors that deepen sleep, fragment sleep or make arousal from sleep more difficult. Factors such as stress, sleep deprivation, medication, alcohol and febrile illness can affect sleep and make the occurrence of NREM parasomnias more likely (Pressman, 2007; Mahowald & Borneman, 2005). It is thought that a trigger is also necessary for a specific event to occur. Triggers include noise, touch, and co-existing sleep disorders such as sleep disordered breathing or periodic leg movements in sleep.

Sleep disturbance and psychopathology commonly co-occur and there is a particularly high comorbidity between insomnia, anxiety and depression (Morin & Ware, 1996) with sleep difficulties being a symptom of both anxiety and depression. The link between insomnia and stress is well documented. Stress can have the effect of increasing arousal levels which can make it more difficult to fall, or stay, asleep (Morin et al., 2003). Stress and anxiety have also been linked to NREM parasomnias (Mahowald & Borneman, 2005; Mahowald & Schenck, 2005; Pressman, 2007; Ohayon, Guilleminault & Priest, 1999). Like anxiety and depression, insomnia and NREM parasomnias are both psychophysiological in nature since they have both psychological and physical aspects.

Previous research has found that individuals with insomnia show greater autonomic arousal compared to normal sleepers (Bonnett & Arand, 1996, 1998; Adam et al., 1986; Stepanski et al., 1989, 1994; Haynes et al., 1974, 1981; Monroe, 1967; Freedman & Sattler, 1982; Riemann et al., 2010), suggesting insomniacs react more readily to stressors at a physiological level. Autonomic arousal occurs as the 'fight or flight' response and has often been used as a measure of stress reactivity (Boyce et al. 2001) since it is an indication of how an individual responds at a physiological level to stressful or threatening events. Since autonomic arousal is closely related to emotion it makes sense that individual differences in autonomic arousal in response to stress may be linked to the development of symptoms of psychopathology. Studies have found individual differences in autonomic arousal to be associated with psychopathology in both adults (e.g. Raine, 1996; Rechlin et al., 1994; Felsten, 2002, 2004) and children (e.g. Boyce et al., 2001). Felsten, for example, reported associations between stress reactivity, neuroticism and a vulnerability to stress and depressed mood.

Also of interest is the appraisal of stress. Morin et al. (2003) found that individuals with insomnia tend to appraise their lives as more stressful than good sleepers even though both groups reported similar levels of stressors. Clinical observations of clients presenting with NREM parasomnias at the Glasgow Sleep Centre suggest a tendency to appear to cope well with challenges and stress, although perhaps superficially. Clinicians reported a tendency for such individuals to appear carefree and unperturbed with regards stressors even when an underlying emotional vulnerability was evident. This pattern of 'normal' psychological functioning during wakefulness in clinical NREM parasomnia populations has also been reported in the literature along with suggestions that these individuals have a capacity to dissociate during wakefulness (Crisp, 1996; Crisp et al., 1990). Klackenberg (1982) reported findings indicating that children with persistent sleepwalking tended to be more inhibited with aggression and have a greater mental defense against anxiety, whilst being well functioning in terms of popularity and behaviour. Individuals who report low levels of stress even when high autonomic arousal is evident may be described as repressors. Weinberger et al. (1979) identified repressors as individuals with low trait anxiety but high defensiveness. They found repressors to respond to stressful situations with high physiological arousal suggestive of anxiety. However, they also showed a tendency to avoid disturbing cognitions, deny cognitive anxiety and report low trait anxiety. Derakshan & Eysenck (1999) concluded from their study that repressors truly believe themselves to be low in trait anxiety and that there may be avoidance or cognitive biases working at a subconscious level to inhibit awareness of anxiety.

Emotional stress can affect sleep quality and can influence sleep physiology as well as various aspects of REM sleep e.g. dream content and recall, latency to REM sleep, and REM sleep duration and density (Vandekerckhaove & Cluydts, 2010). Aspects of REM

sleep are thought to influence the way in which sleep impacts mood the following day. REM sleep is thought to have an important role in overnight emotional processing and affects our autonomic functioning and reactivity the following day (Van der Helm & Walker, 2009; Vandekerckhaove & Cluydts, 2010).

There do not appear to be any experimental studies of stress reactivity in individuals with NREM parasomnias reported in the literature. It was thought that this would be an interesting area to investigate, along with subjective appraisals of stress, in order to gain some insight into how this group responds to 'threat' and to life situations. In addition to looking at how individuals react to stressors physiologically, baseline levels of autonomic arousal in the absence of a stressor would provide insight into daytime arousal levels as a trait-like feature. To date, there is little research into either stress reactivity or the specificity of psychological characteristics in particular forms of sleep disorder. This study is designed to make comparisons between two types of sleep disorder: NREM parasomnias and insomnia.

Aims & Hypotheses

Aims

The primary aim of this study is to investigate stress reactivity in individuals with NREM parasomnias by measuring autonomic arousal at baseline, in reaction to a psychological stressor and during a recovery period. A secondary aim is to investigate individuals' subjective reports of stress across these three phases. A NREM parasomnia group (NREM) will be compared to a good sleeper control group (GS) as well as an insomnia control group (Insomnia). Using another sleep disorder control/comparison group allows better discrimination between findings associated with NREM parasomnias specifically, and those associated with disordered sleep more generally. An insomnia group was chosen since, like NREM parasomnias, insomnia is also psychophysiological in nature. The ICSD-2 diagnostic criteria describe psychophysiological insomnia as insomnia which is caused by heightened arousal and learned associations which prevent sleep (American Academy of Sleep Medicine, 2005).

Hypotheses

Aspects of the study are exploratory in nature and so both hypotheses and supplementary research questions are posed.

(1) Baseline autonomic arousal

a) It is hypothesised that both NREM and Insomnia will show relatively greater autonomic arousal at resting baseline compared to GS.

b) Research question: Is there a difference in autonomic arousal at baseline between NREM and Insomnia?

(2) Stress reactivity

a) It is hypothesised that both NREM and Insomnia will show relatively greater stress reactivity compared to GS.

b) Research question: Is there a difference in stress reactivity between NREM and Insomnia?

(3) Recovery

a) It is hypothesised that both NREM and Insomnia will show a relatively slower return to baseline levels of autonomic arousal compared to GS.

b) Research question: Is there a difference in terms of how quickly NREM and Insomnia return to their baseline autonomic arousal level?

(4) Subjective stress

a) It is hypothesised that Insomnia will report relatively higher levels of stress than both GS and NREM, and that NREM will report the lowest level of stress.

Method

Design

The study employed a mixed design. All participants in the independent groups, NREM, Insomnia or GS, completed baseline, stressor and recovery phases of the experiment, with autonomic arousal being measured continuously. This latter repeated measures aspect of the design permitted mean values for several time points per phase to be compared. Primary dependent variables were: (1) autonomic arousal at resting baseline, (2) stress reactivity i.e. autonomic arousal in response to stress in comparison to the baseline level, (3) how quickly autonomic arousal returns to the baseline level, and (4) subjective, self-report stress ratings. Additionally, the study incorporated a replication of the baseline-stressor-recovery procedure in order to strengthen the study design. However the results of this replication have not yet been examined. Analysis of the physiological data is a detailed and time-consuming process, therefore, it was agreed that priority should be afforded in the first instance to the primary within and between group comparisons (see page 82).

Inclusion and Exclusion Criteria

Inclusion Criteria

Participants in the NREM group were required to meet the International Classification of Sleep Disorders, Second Edition (ICSD-2; AASM, 2005) criteria for either Sleepwalking or Sleep Terrors. To meet criteria for sleepwalking, ambulation must occur during sleep. To meet criteria for sleep terrors, participants must experience a sudden episode of terror during sleep, usually accompanied by a loud scream, autonomic arousal and behaviour associated with intense fear. Either disturbance must not be better explained by any other disorder or substance.

Participants in the insomnia group were required to meet the ICSD-2 general criteria for insomnia. To meet criteria, difficulty initiating or maintaining sleep, or sleep that is non-restorative had to be reported as well as daytime impairment related to the sleep difficulty. The sleep difficulty had to occur despite adequate opportunity for sleep. The full criteria for each disorder is outlined in Appendix 2.2. Good sleepers were required to report no current difficulties sleeping and to be satisfied with their current amount of sleep. All participants were required to be adults 17 years and over.

Exclusion Criteria

Individuals were excluded if they did not meet the inclusion criteria outlined above or if they met criteria for both NREM parasomnia and insomnia. Other exclusion criteria included: evidence of other sleep disorders e.g. sleep apnoea or restless leg syndrome (RLS), neurological disorders e.g. narcolepsy or epilepsy, cardiac problems, pregnancy, substance abuse or medications which can affect autonomic arousal. Individuals with DSM based Axis 1 psychiatric disorders were excluded if their symptoms were reported as severe and/or untreated. Individuals with stable and treated symptoms were included.

Sample Size/Power Calculation

To the knowledge of the researcher, there are no prior studies investigating autonomic arousal in both NREM parasomnia and insomnia groups. A recent study (Espie et al., unpublished abstract) found statistically significant differences in HR and CVT in individuals with insomnia (n=8) compared to normal sleepers (n=9). A medium-large effect size (d = 0.75) was found for HR and a large effect size (d = 1.63) was found for CVT. Since this is a preliminary study, both the effect size and sample size must be estimated. It was hoped that this study would provide a definitive answer of how to power future studies

investigating stress reactivity in more than one psychophysiological sleep disordered group. The power calculation carried out was based on the hypothesis that a significant difference in stress reactivity would be found between groups. This would involve comparing the three groups in terms of their stress reactivity change scores i.e. the difference between autonomic arousal at baseline and in response to the stressor. A total sample size of 69 participants was calculated for a one-way analysis of variance (ANOVA) using a medium-large effect size (f = 0.375, equivalent to d = 0.75, Cohen, 1988; as in the study by Espie et al. described above), a significance level of 0.05 and standard power of 0.8. It was therefore estimated that 23 participants per group should be recruited in order to detect significant differences between the groups.

Participants

Participant Recruitment

Participants were recruited from the general population. Recruitment took place in conjunction with the primary investigator of a parallel study, Katherine Hooker, with whom the screening procedure was shared. Participants contacted the UGSC in response to advertisements on the UGSC website, newspaper articles and posters placed in public spaces (Appendix 2.3). UGSC staff employed a telephone screening interview (Appendix 2.9) to ascertain whether potential participants met inclusion/exclusion criteria. Both studies were also advertised at the Glasgow Science Centre where the primary investigators talked to approximately 180 individuals, over 8 days, about the studies. Forty-six interested individuals completed a screening form. Participants were also identified via an existing database held at the UGSC which has details of participants who have not been suitable for previous research and have agreed to be contacted for future studies, as well as through the parasomnia clinic which is ongoing at the UGSC. The screening information of

approximately 166 potential participants was considered for inclusion. Potential participants were excluded at this point due to the following reasons: not meeting inclusion criteria for any of the groups, reporting medication use or a heart condition, stating that they did not wish to be contacted about this part of the study or living outside of the Glasgow area (unless it had been noted that they still wished to be contacted). Approximately 60 individuals were contacted by telephone about participation by the primary researcher. Additionally, some good sleeper participants were recruited by word of mouth e.g. individuals known to the researcher, UGSC staff and other Trainee Clinical Psychologists.

Participant Numbers

Fifty-five individuals agreed to participate, however 4 later cancelled and 9 did not attend. Forty-two individuals completed the experimental procedure however 3 of these were excluded due to equipment malfunction and one other due to current psychiatric symptoms. Thirty-eight participants were included in the analysis: 15 GS, 12 Insomnia (ICSD-2 General Criteria for Insomnia; AASM, 2005) and 11 NREM. The NREM group comprised of 3 male sleepwalkers, 6 individuals reporting sleep terrors (2 M, 4 F) and 2 female participants reporting both sleepwalking and sleep terrors.

Measures

Measures of Emotional Functioning and Psychopathology

Depression Anxiety Stress Scales (DASS)

The DASS-21 (Appendix 2.4) was administered to provide information at a symptom level in order to describe the groups. The DASS-21 is a short form of Lovibond & Lovibond's (1995) 42-item self report questionnaire which is a reliable and valid measure of the severity of symptoms of Depression, Anxiety and Stress. Each of the

three scales of the DASS-21 contain seven items consisting of a statement relating to a symptom and respondents are asked to indicate how much each statement applied to them over the past week on a scale of 0-3. Scores are summed separately for depression, anxiety and stress. In an independent study, Henry & Crawford (2005) found the DASS-21 to have adequate construct validity and each scale to have high reliability (Cronbach's alpha was 0.88 for Depression, 0.82 for Anxiety, and 0.90 for Stress). The DASS was selected over other measures because of its dimensional nature, which reflects the occurrence of symptoms in the general population, and because it provides separate scores for depression, anxiety and stress. The DASS-21 was chosen over the longer version due to its shorter administration time.

The Mini-International Neuropsychiatric Interview (M.I.N.I.)

The M.I.N.I version 5.0.0 (Sheehan et al., 1998) is a short, structured psychiatric diagnostic interview which has been validated against the Structured Clinical Interview for DSM diagnoses (SCID-P) and the Composite International Diagnostic Interview for ICD-10 (CIDI) as well as against expert opinion. The M.I.N.I was administered when possible psychopathology was indicated during the screening interview or by the DASS-21.

Sleep Measures

Pittsburgh Sleep Quality Index (PSQI)

Quantitative measure of sleep disturbance.

The PSQI (Appendix 2.5; Buysse et al., 1989) was administered to confirm group allocation and to provide a quantitative measure of sleep disturbance in order to describe participants' quality of sleep. The PSQI is a reliable and valid self-rated questionnaire

designed to provide a standardised measure of sleep quality and to discriminate between good and poor sleepers. There are 19 self-rated questions (as well as an additional 5 questions rated by a bed partner which were not used in this study), which are grouped into 7 component scores and then summed to obtain a global PSQI score. Higher scores indicate poorer sleep quality. Buysse et al. report that the PSQI has adequate validity and test-retest reliability and that the seven component scores have a high degree of internal consistency. Backhaus et al. (2002) investigated the psychometric properties of the PSQI for primary insomnia. High test-retest reliability for the overall PSQI global score was found ($r = 0.87$) as well as good internal consistency (Cronbach's alpha = 0.85) and good validity with high correlations found between PSQI and sleep log data.

Sleep Condition Indicator (SCI)

The SCI (Appendix 2.6) is a measure developed at the UGSC which is based on proposed DSM-5 criteria (http://www.dsm5.org/ProposedRevision/Pages/proposedrevision.aspx?rid=65) for a sleep disorder. Participants are asked to report on the pattern, quality, impact and their concern about their sleep over the past month as well as their history of sleep problems. Psychometric data showing good internal consistency has been obtained based on 11,129 individuals from the general population (Cronbach's alpha = 0.89; Espie, Kyle et al., in preparation).

Impact of Poor Sleep

The Impact of Poor Sleep questionnaire (Appendix 2.7), developed at the UGSC, contains items which inquire about the extent that poor sleep has affected six areas of daily functioning. These areas are based on the proposed daytime impact areas of

Insomnia Disorder in DSM-5, the proposed revision of DSM-IV (American Psychiatric Association, 2010).

Autonomic Arousal

Autonomic arousal was measured by recording and analyzing continuous electrocardiography (ECG) output using the Neuroscope™ method (Little et al., 1999), yielding heart rate (HR; beats per minute; bpm) and the non-invasive index of cardiac vagal tone (CVT), the Linear Vagal Scale (LVS; Julu, 1992). HR is continually under the influence of both sympathetic and parasympathetic activity, each having an antagonistic but complementary influence (Sherwood, 2010). Sympathetic activity has the effect of quickening HR while parasympathetic (vagal) tone slows it down. Vagal tone affects HR more quickly (within one heart beat) than sympathetic activity, thus it is vagal tone which is responsible for rapid changes in HR (Little et al., 1999). HR and CVT were recorded continuously throughout all phases of the experimental procedure. Average values were then calculated for each one-minute period of the experimental procedure.

Visual-analogue Stress Scale

A self-report measure of stress was employed at several points throughout the experimental procedure to investigate participants' subjective experience of stress. Each time the stress scale was administered, participants were asked to rate how they felt during the previous few minutes using a visual-analogue scale ranging from 0 (very calm) to 10 (very stressed) (Appendix 2.8). The visual analogue scale has been shown to be a valid measure of perceived stress when compared to scores on the Perceived Stress Scale (PSS; Cohen et al., 1983), a validated measure, in 360 individuals (Lesage & Berjot, 2011).

The Scottish Index of Multiple Deprivation (SIMD)

The SIMD (The Scottish Government, 2009) was employed as a measure of socio-economic status. It provides a relative measure of deprivation by ranking small geographical areas (data zones) based on the domains of: income, employment, crime, education, health, housing and access. SIMD quintile scores were obtained using participants' postcodes. SIMD Quintile 1 represents the most deprived fifth of the data and Quintile 5 the least deprived fifth of the data.

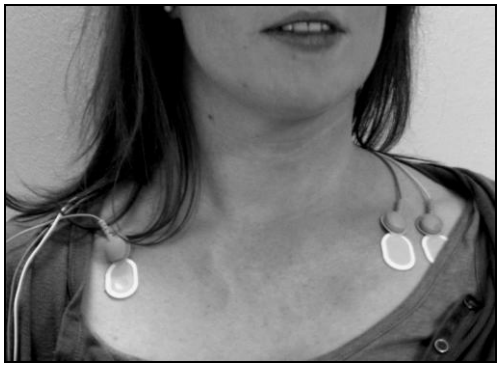
Experimental Procedures

As part of the screening procedure (discussed in the Participant Recruitment section above), participants were screened using a screening interview (Appendix 2.9), and completed a battery of 4 questionnaires: The DASS-21, PSQI, SCI and Impact of Poor Sleep questionnaire. Participants were invited to attend the University of Glasgow Sleep Centre (UGSC) to take part in the study. On arrival it was ensured that participants were familiar with the participant information sheet (Appendix 2.10) which provided information about e.g. the procedure, right to withdraw and confidentiality. Consent was also obtained (Appendix 2.11). The M.I.N.I version 5.0.0 was administered to screen for DSM-based Axis 1 disorders when possible psychopathology was indicated during the screening interview or by the DASS-21.

The same bedroom at the UGSC was used each time which was quiet with standardised temperature and light level to ensure calm, pleasant surroundings. Due to access issues and time constraints, it was not possible for all participants to attend within a similar time period to overcome time of day as a possible confounding variable.

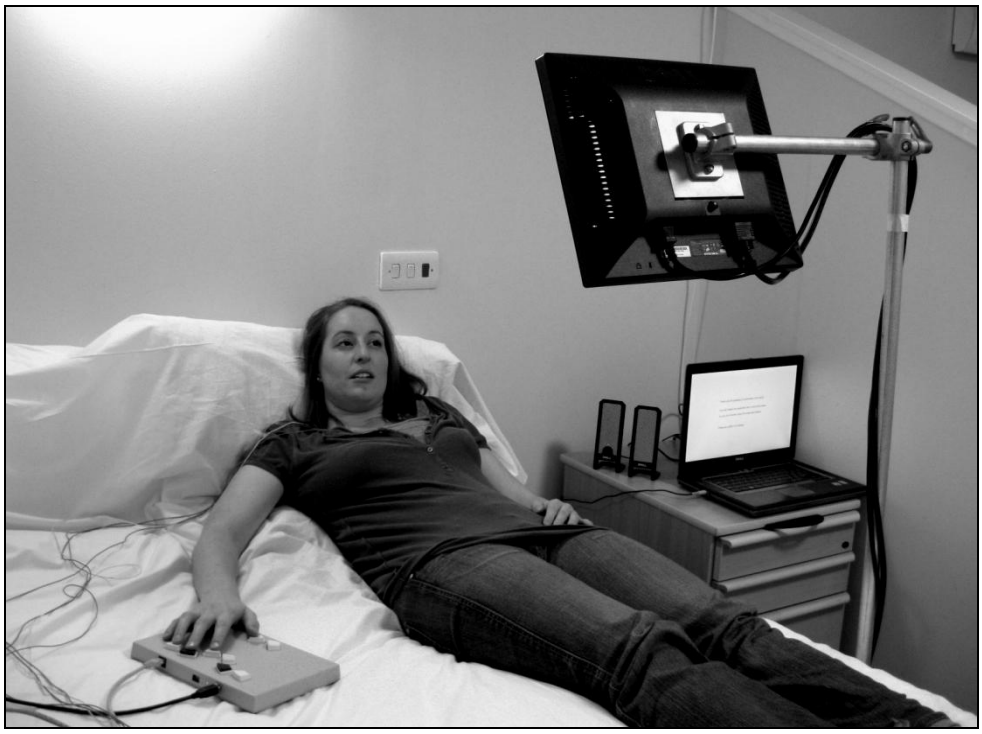
Four adhesive-backed, Ambu wet gel disposable ECG electrodes, with popper lead connections, were used per person and attached according to UGSC standard operational procedure. Skin was first prepared using an alcohol-based wipe and an abrasive pad in order to ensure a good connection. One electrode was placed just below the right clavicle and another on the left torso, above the waist, in order to record activity diagonally across the body. The ground and reference electrodes were placed side by side just below the left clavicle. Each electrode was attached to the Lifelines Trackit Ambulatory EEG recorder, with a poly patient connector unit add on, by a cable. The positioning of three of the four electrodes can be observed in Figure 2.1.

Figure 2.1: Positioning of Three of Four ECG Electrodes



Participants were then directed to the bed where they were asked to lie in a prone position with their head and shoulders supported by pillows so that they could comfortably view a computer screen. Body movements can affect the measures therefore participants were asked to remain as still as possible throughout the procedure and it was important that no postural muscles were engaged. The computer screen was positioned a set distance from the participant on a specially designed metal stand. The participants' dominant hand rested so that they could comfortably access the response button pad. The experimental set-up is illustrated in Figure 2.2.

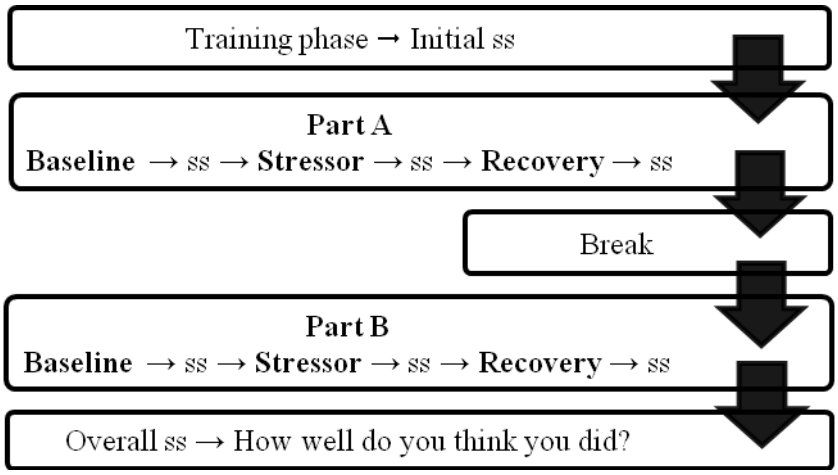
Figure 2.2: Experimental Set-up



Participants were provided with verbal instructions (Appendix 2.12) which included requesting that they remain as still as possible throughout, other than pushing the response buttons on the response button pad. The researcher then left the bedroom and remained in the control room throughout the procedure. A microphone system allowed audio contact between the bedroom and the control room. Stimuli were presented visually on the monitor and participants responded using a button press with four fingers to move through the experiment and to answer multiple choice questions. A short training phase was initially presented to allow participants to practice using the response buttons. The diagram presented to aid training is illustrated in Appendix 2.13. Only when all four training items had been responded to correctly was it possible to proceed. The experimental procedure included a baseline, stressor, and recovery phase, before a break period and then repetition of the three phases. Appropriate instructions were presented visually along with the stimuli

and frequent prompts were provided to instruct participants to press a button in order to move on to the next screen. The stress scale was administered prior to the first baseline phase and again following the completion of each subsequent phase. Participants responded verbally to the visual-analogue scale and their responses were noted by the researcher in the control room. Vocal responses would be likely to affect the ECG data, however the stress scale responses took place before and after the experimental phases. The break provided participants a chance to move about. Participants were able to choose the length of the break and were instructed to press a button when they were ready to commence the second half. On completion of all phases, participants were asked to rate their overall stress as well as how well they thought they had performed in the stressor phases on a visual-analogue scale of 0 – 10. The order of phases and stress scales is illustrated in Figure 2.3.

Figure 2.3: Order of Experimental Phases



Note
ss: subjective stress scale

Baseline and Recovery Phases

Each baseline and recovery phase consisted of a piece of classical music [e.g. Piano Sonata No. 14 'Moonlight Sonata' (Beethoven); Piano Concerto No. 2 (Shostakovich)] which played for five minutes while a picture was displayed on the screen for the same duration. Participants were instructed to listen to the music while looking at the image on the screen.

A different image and piece of classical music was used for each of the four phases, each chosen for their intended relaxing properties. A selection of images and pieces of music were sourced by the researcher and rated for their relaxing properties by both the researcher and another Trainee Clinical Psychologist, who was not involved with the study, as part of the selection procedure. The selected pictures are presented in Appendix 2.14.

Stressor Phase

Participants were presented with a series of multiple choice questions and were asked to read each question and respond with the appropriate response button. They were informed that their responses would be timed and that their task was to answer as many questions correctly as they could, as quickly as possible. The questions were adapted from foundation and general level standard grade mathematics past papers (SQA, 2010). Questions were selected which could be easily adapted to a multiple choice format with four response options. See Appendix 2.15 for examples of the questions used. Several steps were taken to increase the 'stress' of the task. A loud ticking sound was created by recording a metronome and this was played throughout the stressor phase with the aim of heightening the sense of time pressure. Following each response, participants were told if they were correct or incorrect with a large 'tick' or 'cross' being displayed. If they did not respond prior to the time limit (either 20 or 30 seconds per question) then participants were informed they were out of time and too slow (Appendix 2.16). There were 24 questions in each stressor phase and the time taken to complete this phase depended on the speed of responding and ranged from about 5 to about 9 minutes. Each question needed to be either responded to or reach 'time out' to complete the phase.

Experimental Paradigm Development

Stimulus presentation software, SuperLab (Cedrus Corporation, San Pedro, CA), was used to programme the computer to run the experiment. To help establish whether the stimuli were likely to induce the desired stress response, five volunteer participants took part in the development phase which involved completing the experimental procedure. The aim was that the task level should be considered difficult and the success rate neither too high (not stressful) nor too low (participants may give up). Verbal feedback from these participants as well as their subjective stress and test score data (Table 2.1) validated the experimental procedure, suggesting that the procedure was experienced as stressful and that the questions were set at a suitable difficulty level. It was reported that the 'ticking' sound was perceived as increasing the stress experienced and two of the participants commented that they were worried that I would be able to see that they had performed poorly. No changes were made to the protocol following this phase, other than adjusting the volume on the loudspeakers.

Table 2.1 Data from Experimental Paradigm Development Phase

	Subjective Stress Rating (0 – 10)								How well do you think you did? (0-10)	% correct responses during stressor task	
		Part A			**Part B**						
Particip-ant	**Initial Stress**	**After B**	**After S**	**After R**	**After B**	**After S**	**After R**	**Overall Stress**		**Part A**	**Part B**
A	3	2	7	3	1	7	4	7	2	54.2	66.7
B	8	8	8	7	6	7	6	8	5	37.5	63.0
C	1	1	10	1	2	5	2	5	3	-	40.7
D	1	0	7	1	3	8	2	5	5	54.2	51.9
E	2	1	6	2	1	7	2	4	6	54.2	48.2
Mean	**3**	**2**	**8**	**3**	**3**	**7**	**3**	**6**	**4**	**50.0**	**54.1**

Note: B: Baseline phase; S: Stressor phase; R: Recovery phase

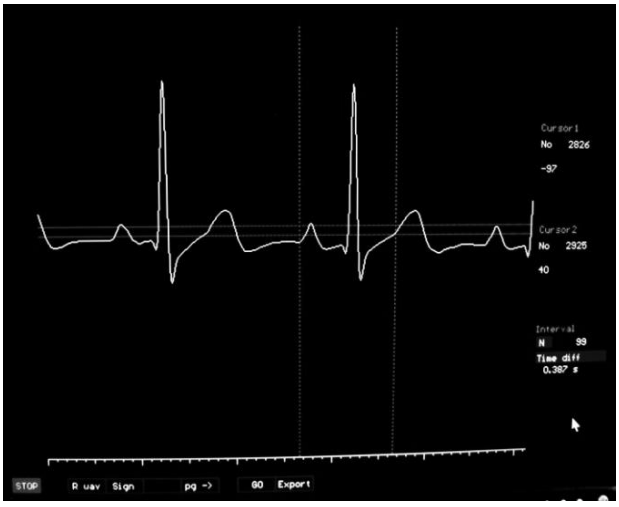
ECG Recording and Data Extraction

The ECG data were recorded using a multi-channel recorder (Trackit™, Lifelines Ltd, UK) according to UGSC standard operational procedure. One channel recorded continuous ECG

waveform data, while a second recorded the timing pulse provided by SuperLab. The recorded data were in the form of EDF (European Data Format) files, which is a standard file format for medical time series data. The experiment had been programmed so that a timing pulse would fire at the beginning and end of each baseline, stressor and recovery phase to mark those time points in the continuous output. Times were also noted by hand by the researcher who could hear, from the control room, each phase begin and end due to the sound (either music or metronome) that accompanied each phase. Spike2 software (Cambridge Electronic Design Limited, Cambridge) was used to establish the start time of each ECG recording as well as the start and finish times of each phase, as marked by the pulses.

HR and CVT were extracted from the EDF data files using Neuroscope™ software (Medifit Instruments Ltd, London). For each participant, a template was selected of a 50 – 100 ms section round a QRS complex (Figure 2.4). The program used that template to identify each R wave throughout the recording allowing the RR intervals to be determined. The RR interval is the measure of the distance between two QRS complexes in an ECG. HR and CVT were calculated from the RR intervals and saved in a VGS file.

Figure 2.4: RR Interval

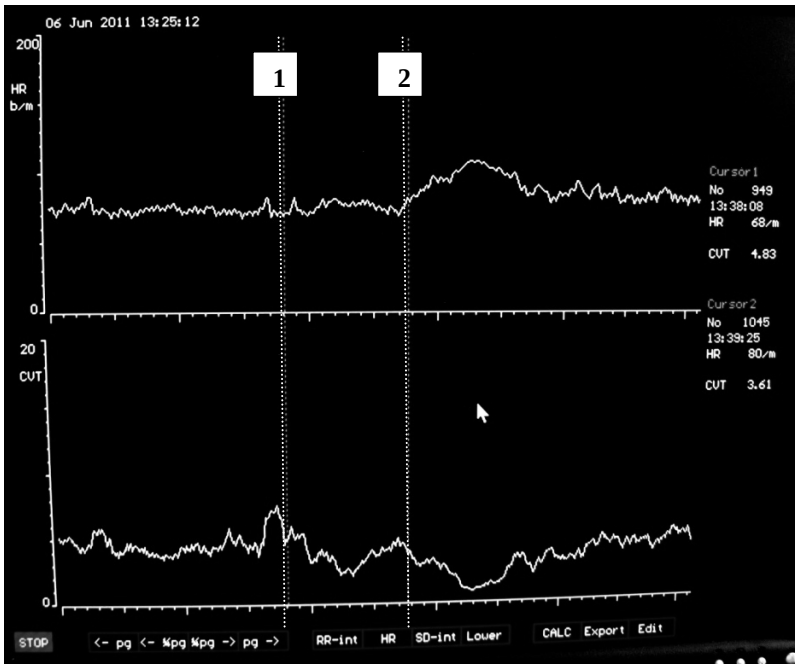


Notes:

- The R wave is the tall peak and the RR interval is the distance between two peaks.
- This image was created by taking a photograph of the computer screen. Although it appears that the x axis is rising, this is in fact only due to the convex nature of the screen.

A second Neuroscope™ program displayed content of the VGS file which allowed visual inspection of the continuous data for HR and CVT (Figures 2.5 & 2.6) in the form of a tachogram where each point corresponds to a heart beat. Mean HR and CVT could then be calculated for specified time periods.

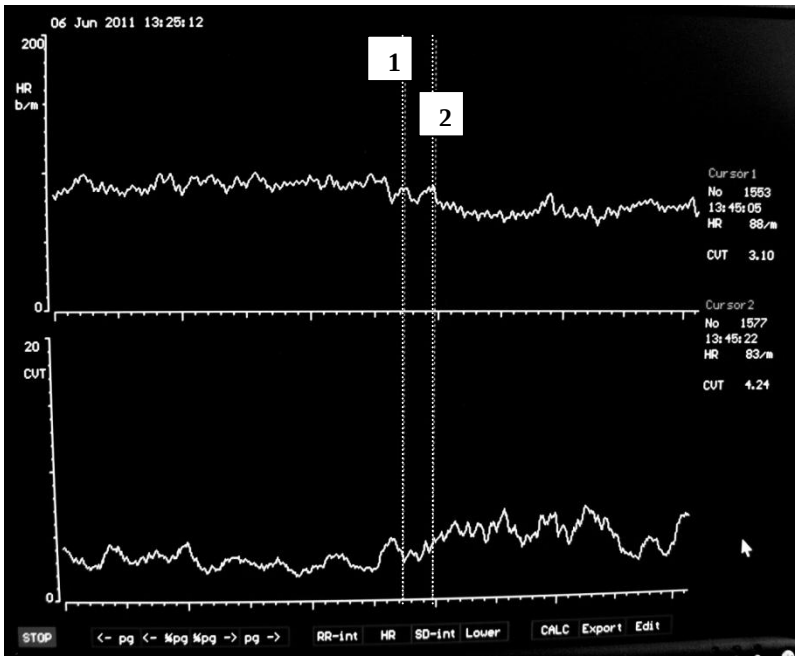
Figure 2.5: Baseline/Stressor Portion of an ECG Output: HR (upper) and CVT (lower)



Notes:

- The x axis represents time (one point per heart beat).
- 1 marks the end of the baseline phase.
- 2 marks the beginning of the stressor phase.
- This image was created by taking a photograph of the computer screen. Although it appears that the x axis is rising, this is in fact only due to the convex nature of the screen.

Figure 2.6: Stressor/Recovery Portion of an ECG Output: HR (upper) and CVT (lower)

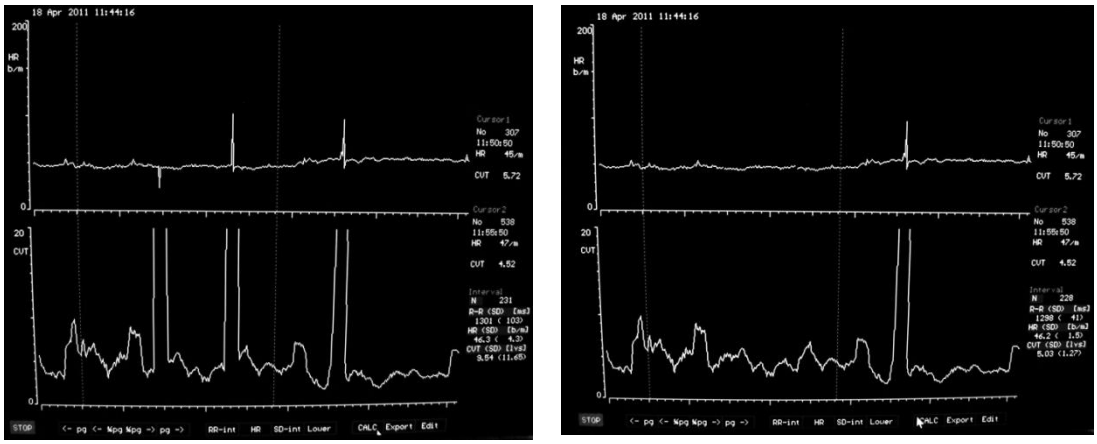


Notes:

- The x axis represents time (one point per heart beat).
- 1 marks the end of the stressor phase.
- 2 marks the beginning of the recovery phase.
- This image was created by taking a photograph of the computer screen. Although it appears that the x axis is rising, this is in fact only due to the convex nature of the screen.

Artifacts in the ECG output were visible for a small number of participants. These may be caused, for example, by participant movement. Suspected artifacts were discussed with a Clinical Scientist at the UGSC to ensure correct differentiation between artifacts and natural anomalies in the output. Artifacts were removed manually using the computer program (Figure 2.7).

Figure 2.7: Removal of Two Artifacts: Before (left) and After (right)



Using cursors, one minute time intervals were selected by hand for each minute of baseline, stressor and recovery phase data. Mean HR and CVT could then be calculated for each minute selected. Due to time constraints and the quantity of data involved, part A of the experimental procedure only has been analysed at this time. A repetition design was used in order to strengthen the study. The rationale was that if effects were found on analysis of data from part A, then the data from part B (the repetition) could be analysed at a later date. Thus, the baseline, stressor and recovery phases discussed from this point will refer to part A of the procedure only. Due to the length of the stressor phase varying from participant to participant, the first five minutes only of the stressor data have been used in the analysis.

Data Analysis

Demographic and procedural variables were investigated as possible confounding factors which would be introduced as covariates in the analysis if appropriate. Mean HR and CVT for consecutive one minute periods during each experimental phase was calculated in order to test the hypotheses. These data were initially plotted on line graphs in order to inspect the data visually and standard error bars were drawn to look at variability. In order to establish the appropriateness of parametric tests, the distribution and variance of the data were explored. To investigate normality, the data were inspected visually using histograms, and values of kurtosis and skewness were calculated and converted to z scores to establish their significance. The results indicated that the HR data were approximately normally distributed, however the CVT data did not approximate a normal distribution. The results of Levene's tests (Appendix 2.17) indicated that homogeneity of variance could be assumed for both HR and CVT data. In light of these findings, the decision was made to use parametric tests to investigate the HR data (since the assumptions of normality and homogeneity of variance were both upheld) and non-parametric equivalent tests to investigate the CVT data since this data did not approximate a normal distribution.

Because this is an exploratory study with specific hypotheses, the decision was made to first use independent t tests (parametric) and Mann-Whitney tests (non-parametric) to compare groups in order to reduce the risk of making a type II error. Where significant results were found, Bonferroni corrections were subsequently employed to reduce the risk of type I error due to multiple comparisons. Where significant results were still found, it was proposed that an appropriate ANOVA (parametric) or Kruskal-Wallis test (non-parametric) would be carried out in order to take a conservative approach and further protect against type I errors.

If results remained significant then any confounding or explanatory variables to do with demographic or procedural factors would be introduced as a covariate. However, this would only be possible with the parametric analyses.

An area under the curve (AUC) analysis was also carried out to further investigate the stress reactivity hypothesis. AUC is a set of mathematical equations which can be used to measure change with reference to the baseline. AUC incorporates both information about the size of responses as well as information about change over time and is a reliable measure of change. Change scores incorporate data from just two data points. However, with AUC, time series data across the stressor phase could be included in the analysis in order to emphasize change over time. AUC is a more sensitive measure of change, and its use was selected for this reason (Fekedulegn et al., 2007). This extra sensitivity is particularly relevant to the HR and CVT data which typically is not expected to vary a great deal in magnitude between participants. Figure 2.10 (page 94) further illustrates AUC.

Effect sizes (Cohen's *d*) were also calculated, using Cohen's (1988) effect size conventions, in order to provide an objective, standardised measure of the size of effects. This enables the direct comparison of different variables within this study as well as the comparison of results with other existing or future studies.

Effect size for Cohen's *d*:	
Small:	0.2
Medium:	0.5

Ethical Approval

Ethical approval was granted by the West of Scotland Research Ethics Service (Appendix 2.18) and Management approval by NHS Greater Glasgow and Clyde Research and Development service in November 2010.

Results

Demographic and Procedural Variables

The age and gender of participants is described in Table 2.2 (a). In the general population, insomnia occurs more frequently in women than men with prevalence increasing with age (Ohayon, 2002). The prevalence of sleepwalking and sleep terrors is thought to decrease with age with similar prevalence rates in both men and women (Ohayon et al., 1999). On visual inspection of the demographic data, the age and gender of participants was found to be in line with what would be expected, given the prevalence rates of insomnia and NREM parasomnias in the general population. On further analysis it was found that age significantly differed according to group membership [H(2) = 11.98, p = 0.001]. Follow-up Mann-Whitney U tests were carried out and a Bonferroni correction applied (0.05/3 = 0.017). The NREM group significantly differed in age from both the GS (U = 36.50, z = -2.39, p = 0.008) and the Insomnia groups (U = 14.50, z = -3.17, p = 0.001). The Insomnia and GS groups did not differ significantly at the 0.017 level (U = 56.50, z = -1.64, p = 0.05). Gender was not found to significantly differ according to group membership [χ^2 (2) = 1.54, p = 0.50], therefore, it was decided that, if appropriate, age alone would be introduced as a covariate.

The percentage of participants in each group who fell into each SIMD quintile are outlined in Table 2.2 (b). A Kruskal-Wallis test indicated that SIMD quintile scores did not significantly differ across groups [H (2) = 3.52, p = 0.17]. Time of day of participation, participant perception of how well they thought they performed in the stressor phase as well as the percentage of correct responses actually obtained were also investigated (Appendix

2.19). These factors were not found to differ significantly across groups and so would therefore not be introduced as covariates in the analysis.

Table 2.2: (a) Age and Gender of Participants, (b) SIMD Quintile Data and (c) Summary of Questionnaire Data

(a)	GS		Insomnia		NREM	
n	15		12		11	
Mean age (SD)	37.53 (14.05)		48.25 (15.85)		26.18 (7.55)	
Min – max age	22 - 68		21 - 68		17 - 45	
% Male (n)	46.7% (7)		25.0% (3)		45.5% (5)	
% Female (n)	53.3% (8)		75.0% (9)		54.5% (6)	
(b)	**%**	**n**	**%**	**n**	**%**	**n**
SIMD Quintile 1	13.3%	2	16.7%	2	36.4%	4
SIMD Quintile 2	13.3%	2	25.0%	3	18.2%	2
SIMD Quintile 3	26.7%	4	8.3%	1	9.1%	1
SIMD Quintile 4	6.7%	1	16.7%	2	27.3%	3
SIMD Quintile 5	40.0%	6	33.3%	4	9.1%	1
(c)	**M**	**SD**	**M**	**SD**	**M**	**SD**
PSQI Global Score	3.87	1.85	11.67	3.85	5.45	1.97
DASS Depression	1.47	1.92	11.67	6.92	7.09	6.60
DASS Anxiety	0.80	1.47	6.17	4.55	5.27	4.92
DASS Stress	4.53	5.58	16.00	8.27	12.36	7.27
SCI Score	3.80	3.76	22.00	6.92	15.64	4.03
Impact of Poor Sleep Score	2.47	3.38	13.42	5.88	12.36	5.7

Questionnaire Data

The PSQI data confirmed group allocation with the Insomnia group, as expected, scoring highest, indicating poorer sleep quality. A significance difference in the PSQI global score was found across groups [$H(2) = 23.95$, $p < 0.001$]. The Insomnia group scored significantly higher on the PSQI Global Score compared to both the GS group ($U = 1.00$, $z = -4.36$, $p < 0.001$) and the NREM group ($U = 6.50$, $z = -3.68$, $p < 0.001$). The NREM and the GS

groups were not found to significantly differ in PSQI global score (U = 47.00, z = -1.87, p = *0.07)* although the good sleepers tended to receive the lowest scores, indicating better sleep quality. Administration of the DASS-21 indicated that that depression, anxiety and stress were experienced to a greater degree in the Insomnia and NREM groups than the GS group, with the Insomnia group receiving the highest mean score for all three. Symptoms of stress and depression appear to be experienced to a greater degree by all three groups than symptoms of anxiety.

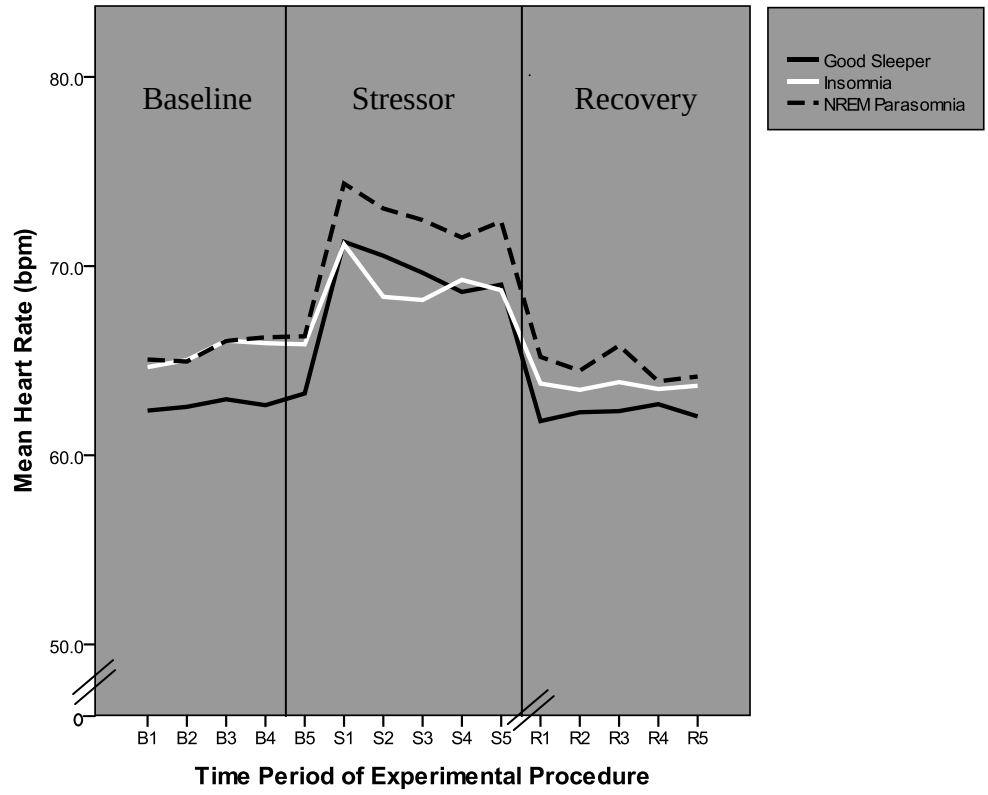
The SCI data indicated that the groups differed significantly in their report of pattern, quality, impact and concern about sleep [$F(2, 35) = 45.73$, $p < 0.001$]. The Insomnia group scored significantly higher on the SCI than both the GS ($p < 0.001$) and NREM ($p = 0.12$) groups. The NREM group also scored significantly higher than the GS group ($p < 0.001$). A significant difference in the Impact of Poor Sleep questionnaire score was found between groups [$F(2, 35) = 20.03$, $p < 0.001$]. The Insomnia group reported a significantly greater impact on daily functioning than the GS group ($p < 0.001$) but not the NREM group ($p = 0.87$). The NREM group reported a significantly greater impact on daily functioning than the GS group.

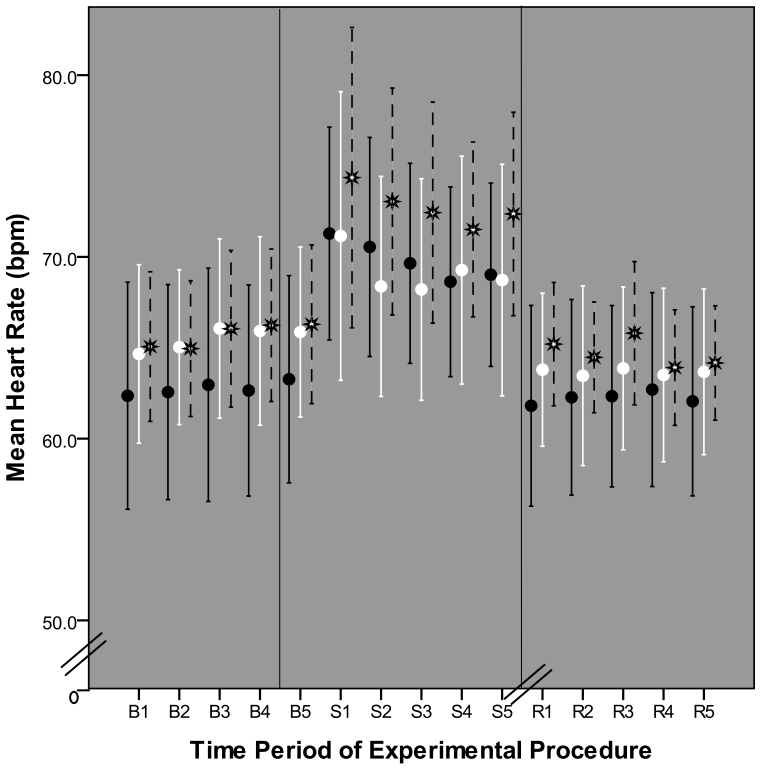
ECG Data

The ECG data for HR and CVT were plotted as line graphs and with standard error bars (Figures 2.8 and 2.9). Large standard error bars indicated that there was a lot of variability in the data. Boxplots were created in order to further investigate this variability (Appendix 2.20) and participants with extreme values in their data were identified. The ECG visual output of these individuals was then re-examined with a Clinical Scientist at the UGSC to check for abnormalities or artifacts that may explain these values, however none were found.

Line graphs were then produced of these individuals' data to investigate whether the pattern of their data was typical of the group. The CVT data of three individuals showed an unusual pattern compared to the rest of the group and a description of these individuals is provided (Appendix 2.21).

Figure2.8: Mean Heart Rate over Consecutive One-minute Periods throughout the Experimental Procedure: Standard Error Bars (+/- 2 SE) Displayed Below

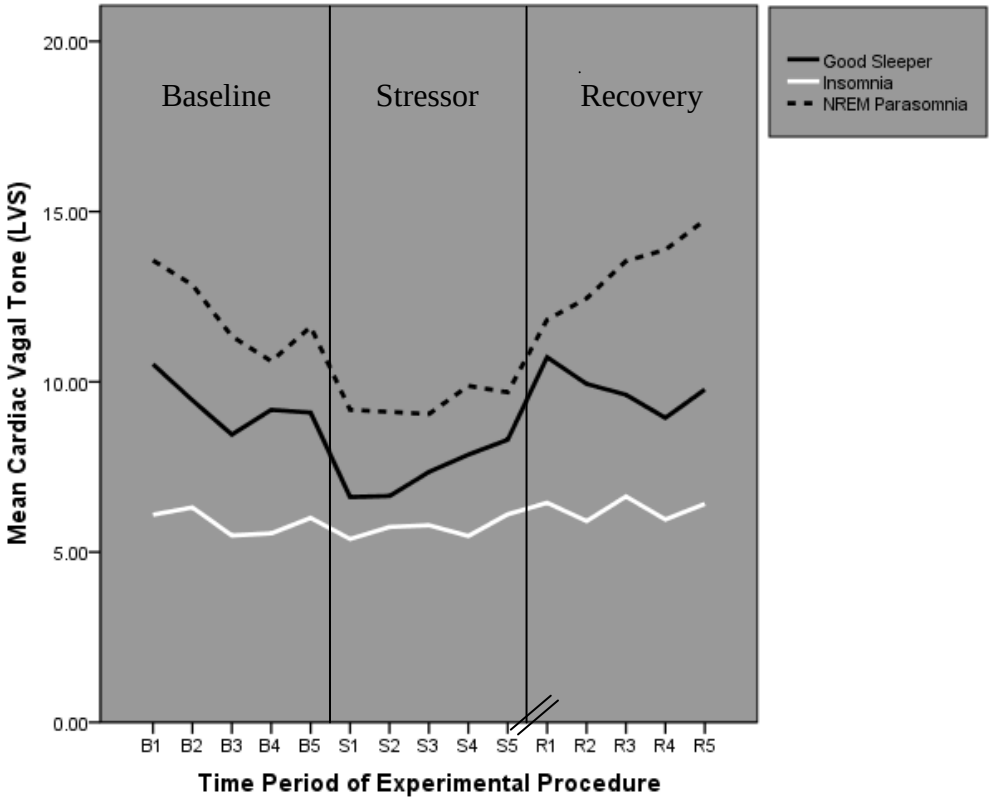


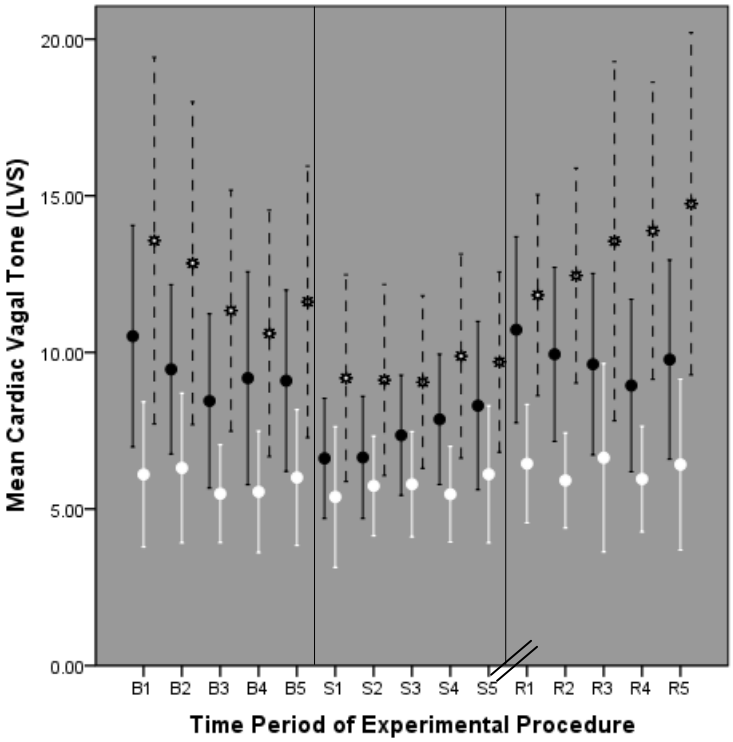


Notes:

- Each point on the x axis represents a one-minute time period.
- B: Baseline
  S: Stressor
  R: Recovery
- Due to the variation in time taken by participants to complete the stressor phase, the first 5 minutes only of this phase are represented.

Figure2.9: Mean Cardiac Vagal Tone over Consecutive One-minute Periods throughout the Experimental Procedure: Standard Error Bars (+/- 2 SE) Displayed Below





Notes:

- Each point on the x axis represents a one-minute time period.
- B: Baseline
  S: Stressor
  R: Recovery
- Due to the variation in time taken by participants to complete the stressor phase, the first 5 minutes only of this phase are represented.

Baseline Autonomic Arousal

Baseline HR

It was hypothesised that both the NREM and Insomnia groups would show relatively greater arousal and therefore relatively higher HR at resting baseline in comparison to good sleepers. Whether there was a difference in HR between the NREM and Insomnia groups had remained a research question.

Visual inspection of the data (Figure 2.8, p89) showed mean HR of all groups remained fairly stable throughout the baseline phase (B). As expected both the NREM and Insomnia groups showed relatively higher resting baseline HR throughout B compared to GS. There did not appear to be a difference between NREM and Insomnia groups. Means and standard deviations for HR for each minute of B are presented in Table 2.3.

Table 2.3: Mean HR (bpm) of each One-minute Period of the Baseline Phase (B)

		B1		**B2**		**B3**		**B4**		**B5**	
Group	n	M	SD	M	SD	M	SD	M	SD	M	SD
GS	15	62.36	11.28	62.56	19.69	62.96	11.59	62.65	10.49	63.27	10.30
Insomnia	12	64.66	7.73	65.03	6.71	66.06	7.77	65.93	8.17	65.87	7.36
NREM	11	65.06	6.13	64.96	5.56	66.05	6.41	66.24	6.24	66.29	6.50

Note: B1 = 1st minute; B2 = 2nd minute etc.

Independent t-tests were carried out to investigate differences in mean baseline HR (using the grand mean of B1 – B5) between groups. On average, participants in the NREM group (M = 65.72, SD = 6.06) had a higher baseline HR compared to good sleepers (M = 62.76, SD = 10.79). This difference was not significant [t (24) = 0.82, p = 0.42; Cohen's d = 0.34, representing a small-medium effect size]. The NREM group (M = 65.72, SD = 6.06) had a similar baseline HR to the Insomnia group (M = 65.51, SD = 7.44), the difference being

non-significant [t (21) = 0.07, p = 0.94; Cohen's d = 0.03, representing a very small effect size]. On average, participants in the Insomnia group (M = 65.51, SD = 7.44) had a higher baseline HR compared to good sleepers (M = 62.76, SD = 10.79) but again, the difference was not significant [t (25) = 0.75, p = 0.46; Cohen's d = 0.30, representing a small effect size]. Since no significant differences were found, the planned mixed between-within subjects analysis of variance was not carried out on the baseline HR data.

Baseline CVT

It was hypothesised that both the NREM and Insomnia groups would show relatively greater arousal and therefore relatively lower CVT at resting baseline in comparison to good sleepers. Whether there was a difference in CVT between the NREM and Insomnia groups had remained a research question.

Visual inspection of the data (Figure 2.9, p90) suggested that for the NREM and GS groups, CVT decreased across the baseline period with the NREM group showing the highest CVT suggesting this group to be least aroused. The Insomnia group, however, showed stable and lower CVT across the baseline phase suggesting that this group showed greater arousal. Means and standard deviations for CVT for each minute of B are presented in Table 2.4.

Table 2.4: Mean CVT (LVS) of each One-minute Period of Baseline Phase (B)

		B1		**B2**		**B3**		**B4**		**B5**	
Group	n	M	SD	M	SD	M	SD	M	SD	M	SD
GS	15	10.52	6.85	9.46	5.24	8.45	5.38	9.18	6.59	9.10	5.61
Insomnia	12	6.10	4.01	6.31	4.13	5.49	2.70	5.55	3.36	6.00	3.75
NREM	12	13.57	9.71	12.84	8.55	11.33	6.39	10.61	6.52	11.62	7.19

Note: B1 = 1st minute; B2 = 2nd minute etc.

Mann-Whitney U tests were carried out to investigate differences in mean baseline CVT (using the grand mean of B1 – B5) between groups. Baseline CVT in the NREM group (Mdn = 10.47) was not significantly higher than the GS group (Mdn = 8.61; U = 64.00, z = -0.96, p = 0.36; Cohen's d = -0.38, representing a small-medium effect size. Baseline CVT in the NREM group (Mdn = 10.47) was significantly higher than the Insomnia group (Mdn = 5.47) at the 0.017 level (U = 27.00, z = -2.4, p = 0.016; Cohen's d = -1.16, representing a very large effect size. A Bonferroni correction had been subsequently applied to adjust the alpha level to 0.017 (0.05/3) since significance was found at the 0.05 level and there had been three planned comparisons. Baseline CVT in GS (Mdn = 8.61) was not significantly higher than Insomnia (Mdn = 5.47; U = 58.00, z = -1.56, p = 0.126; Cohen's d = -0.63, representing a medium to large effect size. Since a significant difference was found, a Kruskal-Wallis Test was carried out on the baseline CVT data and a significant result was found [H(2) = 5.93, p = 0.047].

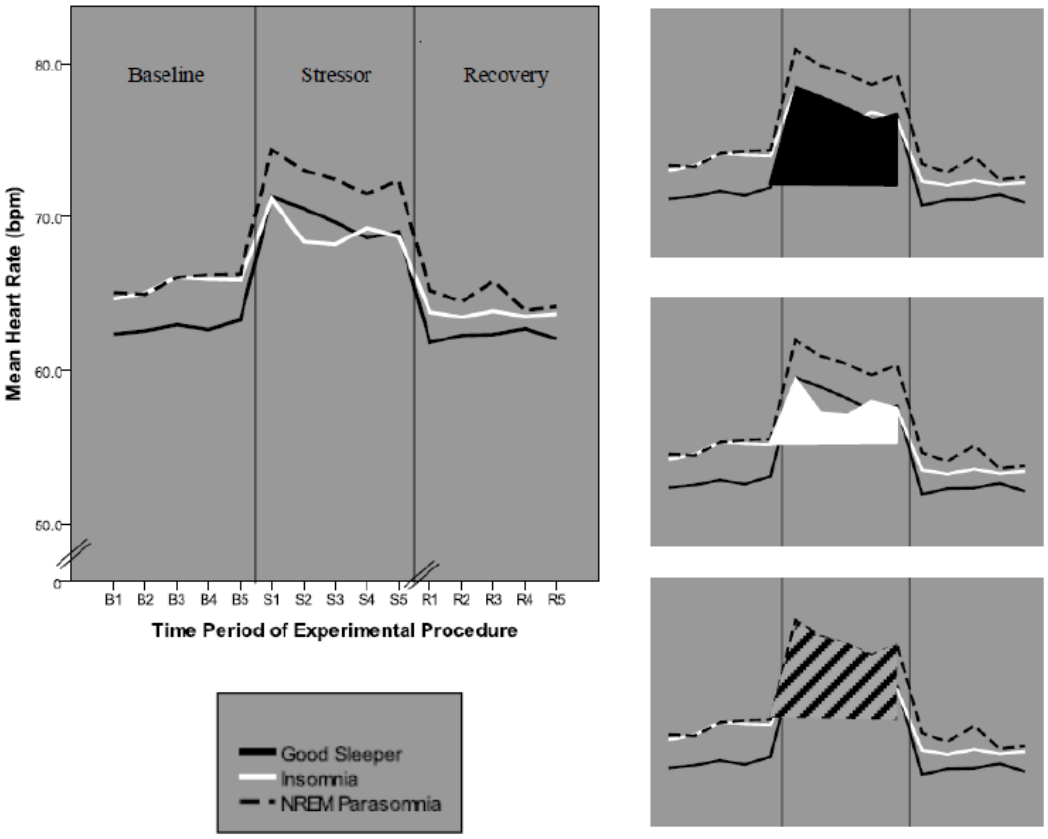
The effect sizes calculated for group interactions of baseline autonomic arousal are presented in Appendix 2.23.

Stress Reactivity

It was hypothesised that both the NREM and Insomnia groups would show relatively greater stress reactivity compared to good sleepers. Whether there was a difference in stress reactivity between the NREM and Insomnia groups had remained a research question. Stress reactivity change scores (Table 2.5, p96) for HR and CVT were obtained by calculating the difference in mean HR and CVT between the final minute of the baseline phase (B5) and the first minute of the stressor phase (S1).

Area under the curve with respect to increase (AUC_I) was also calculated as a measure of change (from B5 to S5) using formulas to calculate the area under the curve as illustrated in the diagram below. AUC_I was calculated to use the baseline (B5) as a reference and ignores the distance from zero. This makes it a sensitive measure of change over time (Fekedulegn, 2007). Figure 2.10 (below) illustrates the areas calculated by AUC_I for the HR data. AUC_I for the CVT data was calculated in a similar manner.

Figure 2.10: Area Under the Curve (B5-S5; HR Data) for GS (upper right), Insomnia (middle right) and NREM (lower right) Groups



Stress Reactivity (HR)

Visual inspection of the HR data (Figure 2.8, p89) suggest that the insomnia group shows a lesser stress reactivity than the NREM and GS groups which appear to have a stress reactivity of a relatively similar magnitude to each other. Independent t-tests were carried out to investigate differences in stress reactivity between groups.

Table 2.5: Stress Reactivity Change Scores (S1 – B5)

		HR		CVT	
Group	n	M	SD	M	SD
GS	15	8.02	5.39	-2.48	4.36
Insomnia	12	5.28	8.03	-0.62	2.93
NREM	11	8.07	6.60	-2.44	3.63

Stress reactivity change scores (Table 2.5) of the NREM group did not differ significantly from stress reactivity of the GS group [t (24) = 022, p = 0.98; Cohen's d = 0.01, representing a very small effect size] or the Insomnia group [t (21) = 0.91, p = 0.38; Cohen's d = 0.38, representing a small-medium effect size]. Stress reactivity of the Insomnia group did not differ significantly from good sleepers [t (25) = 1.06, p = 0.30; Cohen's d = -0.40, representing a small-medium effect size). Since no significant differences were found, the planned one-way between groups ANOVA was not carried out.

AUC_I was then analysed in the same manner as the change score data described above. Stress reactivity (according to AUC_I) of the NREM group (M = 29.23, SD = 17.41) did not differ significantly from the GS group [(M = 29.92, SD = 17.89); t (24) = -0.10, p = 0.92; Cohen's d = -0.04, representing a very small effect size], or the Insomnia group [(M = 14.97, SD = 25.21); t (21) = 1.56, p = 0.13; Cohen's d = 0.66, representing a medium to large effect size]. Stress reactivity of the Insomnia group did not differ significantly from good

sleepers [t (25) = -1.80, p = 0.08; Cohen's d = -0.68, representing a medium to large effect size]. Since no significant differences were found, no further analyses were performed on these data.

Stress Reactivity (CVT)

Visual inspection of the CVT data (Figure 2.9, p90) also showed that the Insomnia group had a smaller in magnitude stress reactivity than both the NREM and GS groups, which again appeared similar in magnitude to each other. This observation was confirmed by calculating stress reactivity change scores (Table 2.5). Mann-Whitney U tests were carried out to investigate differences in stress reactivity (according to change scores) between groups. Stress reactivity in the NREM group (Mdn = -2.44) did not differ significantly from the GS group (Mdn = -1.53; U = 79.00, z = -0.18, p = 0.88; Cohen's d = -0.07, representing a very small effect size), or the Insomnia group (Mdn = -0.41; U = 49.00, z = -1.05, p = 0.32; Cohen's d = -0.45, representing a small-medium effect size). Stress reactivity in the Insomnia group did not differ significantly from good sleepers (U = 7050, z = -0.95, p = 0.35; Cohen's d = -0.37, representing a small-medium effect size). Since no significant differences were found, the planned Kruskal-Wallis Test was not carried out on the stress reactivity CVT data.

AUC_I was then analysed in the same manner as the change score data described above. Stress reactivity (according to AUC_I) in the NREM group (Mdn = -5.14) did not differ significantly from the GS group [(Mdn = -6.51); U = 80.00, z = -0.13, p = 0.92; Cohen's d = 0.05, representing a very small effect size] or from the Insomnia group (Mdn = 0.98; U = 46.00, z = -1.23, p = 0.24; Cohen's d = 0.53, representing a medium effect size). Stress reactivity in the Insomnia group did not differ significantly from the GS group (U = 64.00, z

= -1.27, p = 0.22; Cohen's d = -0.50, representing a medium effect size). Since no significant differences were found, no further analyses were performed.

The effect sizes calculated for group interactions of stress reactivity are presented in Appendix 2.23. As predicted, larger effect sizes were found for group interactions following AUC analysis than with the simple change score data.

Recovery

It was hypothesised that both the NREM and Insomnia groups would show a relatively slower return to baseline levels of autonomic arousal compared to good sleepers. Whether NREM and Insomnia groups differed in terms of how quickly they returned to their baseline autonomic arousal level had remained a research question.

Recovery (HR)

For each participant, the data were examined to ascertain the first time period in which their HR returned to their baseline value or below (Table 2.6). Since HR data were fairly stable throughout the baseline phase, the grand mean of B1 to B5 was calculated as the baseline value. The mean HR for each one-minute period was then examined and the first time period, subsequent to the initial rise in HR at the beginning of the stressor phase, with a mean HR equal to or less than baseline value was noted. Cumulative percentages were also calculated. Three participants in the Insomnia group and one in the GS group reached their baseline HR whilst still in the stressor phase, and at least one participant in each group had not returned to baseline by the end of the recovery phase. On inspection of the cumulative percentages, it could be seen that 75% of the Insomnia group had returned to baseline HR

during the 1st minute of the recovery phase and 91.7% during the 2nd minute. This appeared to represent a generally faster return to baseline HR compared to the NREM and GS groups

Table 2.6: Recovery Phase: Time Period in which Participants Returned to Baseline HR

	GS		Insomnia		NREM	
Time Period 1st Reached Baseline HR	No. Participants	Cumulative %	No. Participants	Cumulative %	No. Participants	Cumulative %
Reached baseline HR during S	1	6.7	3	25	-	-
R1	6	46.7	6	75	5	50
R2	2	60.0	2	91.7	2	70
R3	2	73.3	-	-	1	80
R 4	-	-	-	-	1	90
R 5	1	80	-	-	-	-
Did not reach baseline during R	3	100	1	100	1	100
Total	15		12		10	

Note: Due to equipment failure, recovery phase (R) data were not available for one participant in the NREM group.

Recovery (CVT)

A similar approach was taken to investigate the time period in which CVT first returned to baseline (Table 2.7). Since CVT data showed a steady decline across the baseline phase for the NREM and GS groups, mean CVT for R1 only was used as the baseline value. In all groups, at least four participants reached their baseline CVT whilst still in the stressor phase and at least one participant had not returned to baseline by the end of the recovery phase. Again the Insomnia group appeared to represent a generally faster return to baseline, however this is not surprising since CVT had remained fairly stable across all three phases in this group.

Table 2.7: Recovery Phase: Time Period in which Participants Returned to Baseline CVT

	GS		Insomnia		NREM	
Time Period 1st Reached Baseline CVT	No. Participants	Cumulative %	No. Participants	Cumulative %	No. Participants	Cumulative %
Reached baseline CVT during S	5	33.3	8	66.7	4	40
R1	5	66.7	2	83.3	1	50
R2	-	-	-	-	1	60
R3	-	-	1	91.7	2	80
R4	-	-	-	-	-	-
R5	1	73.3	-	-	-	-
Did not reach baseline during R	4	100	1	100	2	100
Total	15		12		10	

Note: Due to equipment failure, recovery phase (R) data were not available for one participant in the NREM group.

Subjective Stress

It was hypothesised that the Insomnia group would report relatively higher levels of stress than both the GS and NREM groups, and that the NREM group would report the lowest level of stress. On visual inspection of the data (Table 2.8), it could be seen that although the NREM group did report the lowest level of stress the magnitude of this difference was very small and in fact, on average, participants in all three groups reported a similar level of stress throughout the experimental procedure. All three groups reported higher stress levels over the stressor phase than the baseline and recovery phases. Multiple Mann-Whitney U tests were performed for each set of subjective stress data. No significant differences were found between any two groups at any time point ($p \geq 0.11$), confirming the visual inspection of the data. The results of these analyses are presented in Appendix 2.22.

Table 2.8: Mean Subjective Stress Scale Scores Across Experimental Procedure

	Initial Stress		After Baseline Phase		After Stressor Phase		After Recovery Phase		Overall Stress	
	M	SD	M	SD	M	SD	M	SD	M	SD
GS	2.80	2.88	2.07	2.28	6.07	1.94	2.53	1.68	4.33	1.99
Insomnia	2.18	2.27	1.67	1.56	5.83	2.08	2.73	1.68	4.50	1.51
NREM	1.46	1.21	1.46	1.21	5.09	2.17	1.91	0.83	4.18	1.99

Effect Sizes and Statistical Power

The results of this study were generally not found to be statistically significant, however some substantial effect sizes were observed (Appendix 2.23). The small sample size indicated that the study was likely to be underpowered. To investigate this, post hoc calculations were carried out in order to compute achieved power for the baseline and stress reactivity data (Appendix 2.23). The results confirmed that the study was indeed underpowered (achieved power ranging from 0.05 – 0.39), with the exception of the one interaction producing a statistically significant result (NREM-Insomnia for baseline CVT; power of 0.75).

Discussion

NREM parasomnias are a relatively un-studied group of sleep disorders; less being known about this population compared to other sleep disorders, such as insomnia, and there being few treatments offered even to those whose condition causes significant distress or risk to themselves or others. This study was exploratory in nature, the purpose of which was to gain greater insight into how people with NREM parasomnia respond to 'threat' and to life situations.

In particular, the aim was to investigate how their responses to a psychological stressor compared to individuals with insomnia and good sleepers by measuring autonomic arousal, as well as subjective appraisals of stress. Baseline levels of autonomic arousal were also intended to provide insight into daytime arousal levels at the trait level.

Baseline Autonomic Arousal

As expected, both the NREM parasomnia and Insomnia groups exhibited a relatively higher resting baseline HR compared to the good sleeper group, indicating a higher level of underlying sympathetic arousal. The two sleep disorder groups did not differ from each other. HR (all groups) appeared to remain stable throughout the 5-minute baseline period. This contrasted with the baseline CVT data, where the NREM group had the highest values and the insomnia group the lowest, suggesting that the NREM group may be in a more restful state. Both the NREM and the GS groups then appeared to become less relaxed throughout the baseline period, perhaps due to anticipation of the task ahead. The insomnia group had considerably lower CVT which remained fairly stable throughout all phases suggesting a constant level of elevated arousal in relative terms. This finding is in line with

previous research which has found individuals with insomnia to have greater autonomic arousal compared to normal sleepers, both during the day and in the pre-sleep period (e.g. Bonnett & Arand, 1996, 1998; Stepanski et al., 1989, 1994; Haynes et al., 1974, 1981; Monroe, 1967; Freedman & Sattler, 1982; Riemann et al., 2010).

HR can increase due to a reduction in parasympathetic activity (CVT) or an increase in sympathetic activity. Both can happen simultaneously and this is what typically happens in response to stress. In this study, the insomnia group appeared to have CVT that was already so low that it could not have gone much lower in reaction to the stressor; the so-called floor effect. HR did, however, increase during the stressor phase perhaps due to increased sympathetic drive. These data suggest that the insomnia group may go through day-to-day life with a slightly raised level of autonomic arousal as a trait-like feature, in comparison to those without difficulty sleeping. This pattern, however, was not observed in the NREM group. The only statistically significant finding was that baseline CVT in the insomnia group was lower than in the NREM group (representing a very large effect size) The difference between Insomnia and GS baseline CVT represented a medium-large effect size, although a statistically significant difference was not observed. Baseline autonomic arousal differed very little between the NREM and GS groups. However, as described above, medium-large and very large effect sizes were found between the insomnia group and GS and NREM groups respectively. It was the insomnia group which seemed to differ from good sleepers with regards to baseline autonomic arousal and not the NREM group.

Stress Reactivity

Although failing to achieve statistical significance, HR appeared to increase to a greater degree in response to the stressor in the NREM and GS groups. A likely explanation is that

changes in both sympathetic and parasympathetic activity combine to make the increase larger relative to the insomnia group. No significant differences were found between groups for stress reactivity. However as predicted, larger effect sizes were found for group interactions following AUC analysis than with the simple change score data. The results indicated that the NREM group differed little from the GS group in terms of stress reactivity. Nevertheless, medium-large effect sizes were found between the Insomnia and NREM, and Insomnia and GS groups for AUC HR; and medium effect sizes were found between the Insomnia and NREM, and Insomnia and GS groups for AUC CVT. As with the baseline data, it was the insomnia group which differed from the GS group, but in the opposite direction to that predicted. That is, the insomnia group appeared to react less to stress. This is inconsistent with previous research findings which have found reports of increased stress and stress reactivity in individuals with insomnia compared to good sleepers (e.g. Vogontzas et al., 1998; Morin et al., 2003). However, this finding may to be due to the uniformly low CVT level measured in the Insomnia group throughout the procedure, which may have prevented this group from 'reacting' to the stressor with further lowered CVT.

Recovery

Analysis of recovery phase data were difficult to meaningfully analyse due to the method of analysis adopted, i.e. average HR and CVT were calculated for consecutive one-minute periods throughout all phases of the procedure. Complicating this method further for the recovery phase data was the fact that participants took varying lengths of time to complete the stressor phase. To avoid the problem of missing data, it was decided that only data from the first 5 minutes of the stressor phase were included in analysis since these data were complete for all but one of the included participants. However, this method of analysis meant that, for the majority of participants, data between S5 and R1 was not continuous and

contained a 'missing' time period of varying length. This attribute of the data contributed further to the challenge of analysing the recovery data. The analysis that was carried out indicated that participants in the insomnia group generally returned to their baseline level of autonomic arousal (HR and CVT) the most quickly, followed by the NREM group and then the good sleepers. This is not in line with the hypotheses, however, on reflection it is perhaps not surprising since the HR data of the insomnia group increased to a lesser extent than the other two groups and the CVT data appeared not to increase at all in response to the stressor.

If the study were to be repeated, it would be interesting to analyse recovery data using 'running medians' which could have the effect of smoothing the data. This would be a useful technique to use with the time series data, which could help reduce the effects of fluctuations in the data so that trends could be more easily seen. Using this technique, a 'race' could be created in order to investigate how quickly individuals returned to their baseline level and in which order.

Subjective Stress

All three groups reported similar levels of subjective stress, confirming the pilot data that the experiment was stressful, with greater perceived stress being reported during the stressor phase than in either the baseline or the recovery phases. Although the groups did not differ, this data did demonstrate that, at a subjective level, all groups perceived a similar level of stress even where variations in the objective measures were observed. Morin et al. (2003) found that individuals with insomnia tended to appraise their lives as more stressful than good sleepers, even though both groups reported similar levels of stressors. The findings of this study were not in line with Morin et al.'s findings since the Insomnia group did not

subjectively report feeling any more stressed than the other groups. However, this could be partly due to their increased arousal being associated with a lower overall CVT, rather than in reaction to the stressor. The subjective stress data results were not in line with previous research findings that individuals with NREM parasomnias may have a capacity to dissociate or inhibit emotion during wakefulness (Crisp, 1996; Crisp et al., 1990; Klackenberg, 1982). Similarly they were not in line with suggestions that individuals with NREM parasomnias may respond to stressors with high physiological arousal suggestive of anxiety, whilst not being aware of this anxiety at a conscious level, as in Derakshan & Eysenck's (1999) description of 'repressors'. It is also possible that the stressor was not 'stressful' enough, or was not of the type, to observe these effects. It is perhaps of note that the NREM group had a consistently higher mean HR compared to the good sleeper group throughout all phases, which was not reflected in the subjective data.

Participant and Methodological Variables

Effects may also have been partly obscured by participant or methodological variables. Although the age of participants varied between groups, this was in line with what would be expected, given the prevalence rates in the general population. This adds strength to the study in terms of validity. Covariates were investigated and found not to affect the results. A possible confounding variable which was not investigated or controlled for is cardiovascular fitness. It would have been useful to have asked a cardiovascular fitness question as part of the screening interview. Another factor is the influence of ability. Some individuals were likely to have found the procedure more stressful than others in all three groups. The data indicated that both participants' perception of how well they performed during the stressor task and the percentage of questions answered correctly were normally distributed across the study population. Neither of these variables were found to

significantly differ across groups. It was therefore concluded that the influence of ability was not likely to have been a confounding variable. However, it would perhaps have been useful to have also gathered information about participants' level of education. This information, as well as being informative regarding the influence of ability, could have also been used as an indicator relevant to socio-economic status. This demographic was investigated by gathering postcode data and using the SIMD database to calculate a quintile score based on relative deprivation of data zones. The limitation of this method is that deprivation is measured according to geographical area and may not provide a valid measure of socio-economic status at an individual level.

Autonomic arousal is thought to vary over time of day. In general, arousal increases during the morning, stabilises during the afternoon and then decreases again during the evening (Revelle & Loftus, 1992). It was not possible, for practical reasons, for all participants to take part during a specific daytime period. Although time of day of participation was not found to significantly differ across groups, so this may have had limited influence on the experimental data.

Two participants in the insomnia group reported medication use (Citalopram, 20mg) on arrival at the UGSC, which they had not disclosed during the screening process. Antidepressants, and the problems they are prescribed for, are potential confounding variables in the study. One of these individuals had only commenced on the medication a few days previously. Since both participants had travelled to the UGSC and wished to take part, they were run through the procedure. The data of these two participants were subsequently examined. Their physiological data did not contain extreme values or deviate from the typical pattern of responding. Advice was also sought from a psychiatrist about

the potential effect this medication may have on these participants' physiological data. Selective serotonin reuptake inhibitors (SSRIs) like citalopram seem to be less of an issue than other medications regarding potential confounding effects on the physiological data (Rottenberg, 2007). Due to the exploratory nature of this study, and because the doses of citalopram involved were low (and in one of the participants very short term), it was decided that these two participants could be included in the analysis.

Only one participant included in analysis reported significant mental health difficulties. This individual was in the NREM group and met criteria for major depressive episode current and recurrent on the M.I.N.I. This participant reported that her symptoms were currently stable, she was receiving cognitive behavioural treatment at the time of participation and was not taking any medication. On analysis of the physiological data, no extreme values were observed and the data followed the typical observed pattern.

Strengths and Limitations

A strength of this study is that a thorough screening procedure was employed and participants were recruited according to specific diagnostic criteria, inclusion and exclusion criteria. This was supported by analysis of the questionnaire data which confirmed group allocation. Also a strength is that all experimental procedures were piloted and standardised, were carried out according to UGSC standard operational procedure and in the same bedroom and with the same equipment. Participants were provided with identical instructions and explanation which was given in writing, via the participant information sheet, verbally by the researcher with the aid of a script and on-screen during the procedure.

Limitations to this study, which may affect the generalisation of findings, include the participant and methodological variables discussed above. The results of this study were generally not found to be statistically significant, however some substantial effect sizes were observed. The small sample size indicated that the study was likely to be underpowered and post hoc calculations confirmed this to be the case. A total sample size of 69 participants was calculated to be required to detect a large effect size. Thirty-eight participants were included in this study, which is only 55% of the estimated required sample size. Recruitment had been more difficult than expected. Although contact was made with a large number of potential participants, exclusions were made due the strict inclusion/exclusion criteria. Additionally, there were potential participants who, although interested, were not able or did not wish to attend the Southern General Hospital at a later date to participate. The distance participants would have to travel was a factor as was time of day. Due to researcher availability, it was only possible to run participants on certain days, and the sleep laboratory was only available at specific times. Unfortunately these times did not suit some potential participants who were otherwise willing to take part. These restrictions undoubtedly affected the obtained sample size.

This study was intended to be exploratory in nature and a repetition of this study with a larger number of participants would be interesting. However, prior to that, the data from part B, the previously described repetition aspect of the study, should be analysed. It would be informative to observe whether similar effects are observed on analysis of the data from part B. If so, this would indicate the stability or otherwise of the findings thus far.

All three groups did generally differ from each other in a consistent way throughout the procedure. For example, the NREM group had the highest level of CVT, and the Insomnia

group the lowest throughout all phases. There was some crossover in the HR data (particularly with the Insomnia group), although it was observed that the NREM group had consistently higher HR than the GS group. The data from each group also appear to follow similar patterns of response, with the exception of the consistently low CVT in the insomnia group. These observations suggest that further investigation may be worthwhile.

Another limitation is that there was a problem with the timing pulse in a proportion of participants' data occurred for an unknown reason. This meant that, for these participants, analyses had to rely on the hand noted times for phase beginnings and ends. Whilst these times were routinely recorded as accurately as possible, there may have been small fluctuations in the accuracy. It is estimated that for these participants, the possible inaccuracy in the time of phase beginnings and endings is not likely to be more than a few seconds. Also of note is a known design weakness, which is that participants took different lengths of time to complete the stressor phase. This issue was considered during the design of the study but it was not possible to appropriately programme the software to limit the stressor phase to a specific length of time.

Implications

The findings of this type of study have potentially important implications for the development of treatment programmes for NREM parasomnias. However, further work needs to be done before any conclusions can be drawn. The study was intended as an exploratory study and the preliminary findings indicate that further exploration is warranted. The first step would be to analyse the replication data to investigate if the results are replicated. As the results stands, it appears that individuals with NREM parasomnias react

to stressors in a similar manner to good sleepers but may have a slightly higher resting sympathetic activity (similar to those with insomnia).

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CHAPTER THREE: ADVANCED PRACTICE I REFLECTIVE CRITICAL ACCOUNT

Reflections on Working with an Individual with a Diagnosis of Bipolar Disorder

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Submitted in partial fulfilment of the requirements for the degree of Doctorate in Clinical Psychology (D.Clin.Psy)

Abstract

The following reflective account is based upon my experiences of working within a community mental health team. I draw on Gibbs' (1988) model of reflection and Rolfe et al.'s (2001) framework for reflective practice to consider my experience of working with an individual with a diagnosis of bipolar disorder. I consider the thoughts and emotions I experienced at various points whilst working directly with the patient as well as indirectly with other members of a multidisciplinary team in order to gain a more detailed understanding of the client and her difficulties, and to consider effective approaches for assessment and intervention.

CHAPTER FOUR: ADVANCED PRACTICE II REFLECTIVE CRITICAL ACCOUNT

Reflections on a Group Intervention for Asylum Seekers and Refugees, and Issues Relating to Equality

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Abstract

The following reflective account is based upon my experience of working within a specialist trauma service providing mental health care for asylum seekers and refugees with complex difficulties. I draw on structured reflective models (Boud, Keogh and Walker, 1985; Gibbs, 1988) to reflect on my experience of helping to run a group intervention for male survivors of torture. This experience has contributed to my professional development as I have learned skills and become more aware of issues that will help me to plan, manage and run groups in the future. I also consider equalities legislation and the Equality Scheme 2010-2013 and reflect on how this relates to asylum seekers and refugees, and their experience of accessing services.

Appendix 1.2

Excluded Articles

Article	Author	Reason for Exclusion
1	Cortoos et al. (2010b)	Conference publication – full text not obtained.
2	Kazarian et al. (1978)	Not investigating effect of biofeedback on insomnia.
3	Heaton & Rayens (2010)	Participants did not have sleep difficulties.
4	Budzynski (1973)	Outcome measures and results data not reported.
5	Stoyva et al. (1974)	Abstract only.
6	Besner, H.F. (1978)	Abstract only.
7	Good (1975)	Study did not use group design.
8	Montgomery & Besner (1975)	Not able to obtain article.
9	Hauri & Good (1975)	Conference publication – full text not obtained.
10	Sittenfield (1972)	Abstract only.

Appendix 2.1: Guidelines for submission to Journal of Sleep Research

Journal of Sleep Research

Published on behalf of the European Sleep Research Society

Edited by:
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Print ISSN: 0962-1105
Online ISSN: 1365-2869
Frequency: Quarterly
Current Volume: 20 / 2011
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Regular Research Papers
These are of a more usual length 3000-5000 words, with a maximum of 30 references, and will contain original basic or clinical research findings.

Review Papers
Review papers are invited by the Editor. Review papers are intended to be well argued, critical reviews of topical subjects which, will generate debate and provide direction for future research on the topic.

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This should contain a concise title of the article, a shortened version (no more than 50 characters including spaces) for the running head, names of the authors, their affiliations, and the full postal and e-mail address, fax and telephone number of an author to whom correspondence can be addressed.

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Conflict of interests - disclosure of any personal of financial support and author involvement with organization(s) with financial interest in the subject matter of the paper, or any actual or potential conflict of interest-and if no conflict exist, a statement must be included for each author.

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This should be on a separate page, and less than 250 words. It should be followed by up to six key words. It should not be structured. Abbreviations should not be used in the summary.

Main Text

All text must be 1.5- or double-spaced. It should start on a separate page, and include an introduction (approximately 500 words for Regular Research papers) , methods, results and discussion (less than 1000 words for Regular Research Papers). The suggested points of insertion of figures and tables, etc., should be indicated. Authors should avoid abbreviations (except for those commonly understood), long sentences, and many juxtaposed numbers in sentences. Authors should also avoid sending in main text files in Portable Document Format (PDF).

References

References cited in the text should include the author's name and year of publication. Where there are more than two authors, list the first author only, followed by *et al*.

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References to abstracts or letters may be included but these must be stated as such. Unpublished work should only be cited in the text. Only references that have already been published or that are genuinely in press should be included in the reference list.

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Examples of basic references format:

Loomis, A. L., Harvey, E. N. and Hobart, G. Cerebral states during sleep as studied by human brain potentials. *J. Exp. Psychol.*, 1937, 21: 127-144.

Kleitman, N. *Sleep and Wakefulness.* University of Chicago Press, Chicago, 1963 (second edition).

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These should be brief and must include references to sources of financial and logistical support.

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Appendix 2.2: Diagnostic Criteria for Insomnia and NREM Parasomnia Groups

Participants in the insomnia group met the following ICSD-2 criteria:

General Criteria for Insomnia

A. A complaint of difficulty initiating sleep, difficulty maintaining sleep, or waking up too early or sleep that is chronically non-restorative or poor in quality.
B. The above sleep difficulty occurs despite adequate opportunity and circumstances for sleep.
C. At least one of the following forms of daytime impairment related to the night time sleep difficulty is reported by the patient:
    i. Fatigue or malaise
    ii. Attention, concentration, or, memory impairment
    iii. Social or vocational dysfunction
    iv. Mood disturbance or irritability
    v. Daytime sleepiness
    vi. Motivation, energy, or initiative reduction
    vii. Proneness for errors or accidents at work or while driving
    viii. Tension, headaches, or gastrointestinal symptoms in response to sleep loss
    ix. Concerns or worries about sleep

Participants in the NREM parasomnia group met the following ICSD-2 diagnostic criteria for Disorders of Arousal (from NREM Sleep).

Sleepwalking

A. Ambulation occurs during sleep.
B. Persistence of sleep, an altered state of consciousness, or impaired judgment during ambulation is demonstrated by at least one of the following:
    i. Difficulty in arousing the person
    ii. Mental confusion when awakened from an episode
    iii. Amnesia (complete or partial) from an episode
    iv. Routine behaviours that occur at inappropriate times
    v. Inappropriate or nonsensical behaviours
    vi. Dangerous or potentially dangerous behaviours
C. The disturbance is not better explained by another sleep disorder, medical or neurological disorder, mental disorder, medication use, or substance use disorder.

Sleep Terrors

A. A sudden episode of terror occurs during sleep, usually initiated by a cry or loud scream that is accompanied by autonomic nervous system and behavioural manifestations of intense fear.
B. At least one of the following associated features is present:
    i. Difficulty in arousing the person
    ii. Mental confusion when awakened from an episode
    iii. Amnesia (complete or partial) for the episode
    iv. Dangerous or potentially dangerous behaviours
C. The disturbance is not better explained by another sleep disorder, medical or neurological disorder, mental disorder, medication use, or substance use disorder.

Appendix 2.3: Sleep Research Recruitment Poster

Appendix 2.4

DASS21

Name: *Date:*

Please read each statement and circle a number 0, 1, 2 or 3 which indicates how much the statement applied to you *over the past week*. There are no right or wrong answers. Do not spend too much time on any statement.

The rating scale is as follows:

0 Did not apply to me at all
1 Applied to me to some degree, or some of the time
2 Applied to me to a considerable degree, or a good part of time
3 Applied to me very much, or most of the time

1	I found it hard to wind down	0	1	2	3
2	I was aware of dryness of my mouth	0	1	2	3
3	I couldn't seem to experience any positive feeling at all	0	1	2	3
4	I experienced breathing difficulty (eg, excessively rapid breathing, breathlessness in the absence of physical exertion)	0	1	2	3
5	I found it difficult to work up the initiative to do things	0	1	2	3
6	I tended to over-react to situations	0	1	2	3
7	I experienced trembling (eg, in the hands)	0	1	2	3
8	I felt that I was using a lot of nervous energy	0	1	2	3
9	I was worried about situations in which I might panic and make a fool of myself	0	1	2	3
10	I felt that I had nothing to look forward to	0	1	2	3
11	I found myself getting agitated	0	1	2	3
12	I found it difficult to relax	0	1	2	3
13	I felt down-hearted and blue	0	1	2	3
14	I was intolerant of anything that kept me from getting on with what I was doing	0	1	2	3
15	I felt I was close to panic	0	1	2	3
16	I was unable to become enthusiastic about anything	0	1	2	3
17	I felt I wasn't worth much as a person	0	1	2	3
18	I felt that I was rather touchy	0	1	2	3
19	I was aware of the action of my heart in the absence of physical exertion (eg, sense of heart rate increase, heart missing a beat)	0	1	2	3
20	I felt scared without any good reason	0	1	2	3
21	I felt that life was meaningless	0	1	2	3

Appendix 2.5: PSQI (excluding bed partner questions)

Pittsburgh Sleep Quality Index (PSQI)

Instructions: The following questions relate to your usual 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 questions.**

1. During the past month, what time have you usually gone to bed at night? _____
2. During the past month, how long (in minutes) has it usually taken you to fall asleep each night? _____
3. During the past month, what time have you usually gotten up in the morning? _____
4. During the past month, how many hours of actual sleep did you get at night? (This may be different than the number of hours you spent in bed.) _____

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 times 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 to use the bathroom				
d. Cannot breathe comfortably				
e. Cough or snore loudly				
f. Feel too cold				
g. Feel too hot				
h. Have bad dreams				
i. Have pain				
j. Other reason(s), please describe:				
6. During the past month, how often have you taken medicine to help you sleep (prescribed or "over the counter")?				
7. During the past month, how often have you had trouble staying awake while driving, eating meals, or engaging in social activity?				
	No problem at all	Only a very slight problem	Somewhat of a problem	A very big problem
8. During the past month, how much of a problem has it been for you to keep up enough enthusiasm to get things done?				
	Very good	Fairly good	Fairly bad	Very bad
9. During the past month, how would you rate your sleep quality overall?				

Appendix: 2.6

Sleep Condition Indicator

1. Evaluate the pattern of your sleep in the past month	a. *How many minutes has it usually taken you to fall asleep?* b. *How many minutes have you usually been awake during the night (because of your wakenings)?* c. *How many nights each week have been problem nights?*	0-15, 16-30, 31-45, 46-60,61+ 0-15, 16-30, 31-45, 46-60,61+ 0-1, 2, 3, 4, 5-7
2. Evaluate the quality of your sleep in the past month	*What has the quality of your sleep usually been like?*	0 'very satisfactory' to 4 'very unsatisfactory'
3. Evaluate the impact of your sleep on your life in the past month	a. *To what extent has poor sleep affected your mood or well-being in the daytime?* b. *To what extent has poor sleep affected your concentration or productivity?*	0 'not at all' to 4 'very much' 0 'not at all' to 4 'very much'
4. Evaluate your level of concern about sleep in the past month	*To what extent has poor sleep and its consequences been troubling you?*	0 'not at all' to 4 'very much'
5. Evaluate the history of your sleep	*For how many years have you had problems with your sleep?* If you answered 11+ years - *Did you sleep OK as a child?*	'no problem', 0-1 year, 2-5 years, 6-10 years, 11+ years Yes/ no
Use of sleeping aids (currently)	*Do you take sleeping pills prescribed by your doctor?* *Do you take other remedies that you buy at the chemist?* *Do you use alcohol to help you sleep?*	Yes/ no Yes/ no Yes/ no

Scoring

Add scores 1a, 1b, 1c, 2, 3a, 3b, 4, 5 (score range for 0 – 32)

Key

0=Not at all 1=A little 2=Somewhat 3=Much 4=Very much

Appendix: 2.7

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The Impact of Poor Sleep

Over the last month to what extent has poor sleep troubled you in general?

Not at all ____ A little ____ Somewhat ____ Much ____ Very Much ____

More specifically, to what extent has poor sleep affected.....

.....your mood?

Not at all ____ A little ____ Somewhat ____ Much ____ Very Much ____

.....your energy?

Not at all ____ A little ____ Somewhat ____ Much ____ Very Much ____

.....your relationships?

Not at all ____ A little ____ Somewhat ____ Much ____ Very Much ____

.....your ability to stay awake during the day?

Not at all ____ A little ____ Somewhat ____ Much ____ Very Much ____

.....your concentration?

Not at all ____ A little ____ Somewhat ____ Much ____ Very Much ____

.....your ability to get through your work?

Not at all ____ A little ____ Somewhat ____ Much ____ Very Much ____

Appendix 2.8: Visual-analogue Stress Scale

How did you feel during the last few minutes?

0	1	2	3	4	5	6	7	8	9	10
Very Calm										Very Stressed

Please respond by saying the appropriate number out loud…
… then press any button to continue.

Appendix 2.9: Participant Screening Interview: Adapted from GUSC standard screening interview

Participant ref no:	**Today's date:**

Source (to be answered by all participants)

How did you find out about the University of Glasgow Sleep Centre?	
Why have you contacted us?	
Method of initial contact (e.g. mobile, email)	

Personal (to be answered by all participants)

Full Name:	*Date of Birth:*	*Age:*
Telephone (home, mobile):	*Address:*	
Email:		
When is a good time to call?	*Postcode:*	
What GP practice do you attend, and who is the GP you normally see?		
Do you give your consent for us to contact your GP if necessary regarding your health?		

Do you have difficulty sleeping at the moment? (Y/N)
Are you generally satisfied with the amount of sleep you get (Y/N)
Do you work shifts, night shifts?
Are you aware of any other family members with a sleep disorder? *If yes please specify:* *E.g. Do either of your parents have sleep difficulties (now or in the past)?* *What about brothers and sisters?* *What about extended family including grandparents?*

Health (to be answered by all participants)

Do you keep in good health physically? (Y/N)
What physical health problems do you have (if applicable)? *(Could your sleep difficulties be due to any other physical health problems or medication use?)*
What medicines do you take for your physical health? (if applicable)
Roughly, how many units of alcohol do you drink per week? *(Standard (175ml) glass of wine = 2 unit; One pint of standard lager = 2.3 units; Spirit & Mixer = 1 unit)*
Do you take recreational drugs? If so, how often? (e.g. once per week/month etc)
Do you keep in good health mentally? (Y/N)
What mental health problems do you have (if applicable)?
What medicines do you take for your mental health? (if applicable)

Screen for Possible Sleep Disorders Other Than Insomnia.
(to be answered by all participants)

1. Narcolepsy ***a. Do you sometimes fall asleep in the daytime completely without warning? (YES/NO)*** ***If Yes….*** *b. Is it literally impossible to resist 'sleep attacks' during the day?* *c. Do you have collapses or extreme muscle weakness triggered by extreme emotion?* *d. Do you have visual hallucinations, either just as you fall asleep or when you wake in the morning?* *e. Are you paralysed and unable to move when you wake up from your sleep?* *[OFFICE USE ONLY: Possible narcolepsy: 1a¼''TRUE'' AND (1b OR 1c OR 1d OR 1e¼''TRUE'')]*
2. Sleep breathing disorder ***a. Are you a very heavy snorer? (YES/NO)*** ***If Yes…*** *b. Does your partner say that you sometimes stop breathing?* *c. Do you often wake up gasping for a breath?* *d. Are you often excessively sleepy during the day or fall asleep without wanting to?* *[OFFICE USE ONLY: Possible sleep breathing disorder: 2a¼''TRUE'' AND (2b OR 2c OR 2d¼''TRUE'')]*

3. PLMS/ RLS
a. Do your legs often twitch or jerk or can't keep still in bed?(YES/NO)
If Yes…
b. Is it very difficult to get to sleep because of repeated muscle jerks?
c. Do you frequently wake from sleep with sudden jerky movements or with a compulsion to move your legs?
d. Do you simply have to get out of bed and pace around to get rid of these feelings?

[OFFICE USE ONLY: Possible PLMS/ RLS: 3a¼''TRUE'' AND (3b OR 3c OR 3d¼''TRUE'')]

4. Circadian Rhythm Sleep Disorder
a. Do you tend to sleep well but just at the ''wrong times''? (YES/NO)
If Yes…
b. Can you sleep well enough, but only if you stay up very late?
c. Are you in a very sound sleep at normal waking time and could sleep on for hours more?
d. Can you sleep well enough, but only if you go to bed very early?
e. Do you wake very early, bright and alert and no longer sleepy?

[OFFICE USE ONLY: Possible CRSD: 4a¼''TRUE'' AND EITHER (4b AND 4c¼''TRUE'') OR (4d AND 4e¼''TRUE'')]

5. Parasomnia
a. Do you have unusual behaviours, like sleepwalking, associated with your sleep that trouble you or that are dangerous?(YES/NO)
If Yes…
b. Do you sleepwalk frequently and run the risk of injuring yourself or others?
c. Do you have frequent night terrors when you are extremely distressed but not properly awake?
d. Do you act out your dreams and risk injuring yourself or others?
e. Do you have terrible recurring nightmares?

[OFFICE USE ONLY: Possible parasomnia: 5a¼''TRUE'' AND EITHER (5b OR 5c OR 5d OR 5e¼''TRUE''

If YES to b. or c.

- ***Do you currently experience these?(YES/NO)***
- ***How often in the past 6 months?***

Insomnia (if applicable)

Have you always been a poor sleeper? (Y/N)
How long have you had a sleep problem?(yr)
Do you have difficulty falling asleep? (Y/N)
How many nights per week do you have difficulty falling asleep? (out of 7)
How long does it normally take you to fall asleep?(min)
Do you experience sleep disturbance because of waking during the night?(Y/N)
How many nights per week do you have a difficulty with waking up during the night?(out of 7)
How long are you normally awake during the night, in total? (min)
What time do you normally go to bed? (time)
What time do you normally get up?(time)
How long do you normally sleep?(hr/min)

Sleepwalking (if applicable)

Do you sleep walk or carry out any other activities during sleep. Please describe any activities that occur in addition to walking (e.g. eating, sex, undressing/.dressing)?
Is it difficult to arouse you from the sleep state during these events?
Have you experienced mental confusion when awakened from an episode?
Do you have any memory of such events upon awakening?
How often do these events occur in general (e.g. once per week/once per month)? *How many in last 6 months?*
How long ago did you last experience this event?
Approximately how old were you when you first experienced an event like this?
Do you know whether this occurs at the beginning, middle or end of your sleep?

Night Terrors (if applicable)

Do you experience sudden episodes of intense terror during sleep? *Do you engage in any dangerous or potentially dangerous behaviours at these times? (if so, please describe)*
Do you have any memory of such events upon awakening?
Is it difficult to arouse you from the sleep state during these events?
Have you experienced mental confusion when awakened from an episode?
How often do these events occur in general (e.g. once per week/once per month)?
How long ago did you last experience this event?
Approximately how old were you when you first experienced an event like this?
Do you know whether this occurs at the beginning, middle or end of your sleep?

All Sleep Disorders (to be answered by those with a sleep disorder)

Does your sleep disturbance affect how you feel and function during the day (e.g. fatigue, sleepiness, concentration, memory, mood, motivation, irritable, work/social functioning etc.). If yes, specify most salient.

Other Studies (to be answered by all participants)

We are also running a lab based study. Would you be interested in being contacted about this study?
If you are not suitable for any of the studies ongoing at the moment are you happy for your details to be kept on a database so that you may be contacted in the future should a suitable study start?

Appendix 2.10

University of Glasgow Sleep Centre
Sackler Institute of Psychobiological Research
2nd Floor
Neurosurgery Building
Southern General Hospital
Glasgow G51 4TF
Tel: 0141 232 7699

STRESS REACTIVITY IN INDIVIDUALS WITH NON-REM PARASOMNIAS, INSOMNIA AND GOOD SLEEP

PARTICIPANT INFORMATION SHEET

My name is Sarah Young. I am a trainee clinical psychologist from the University of Glasgow working with Professor Colin A Espie, Director of the University of Glasgow Sleep Centre. I would like to invite you to take part in a study looking at how individuals with Non-REM parasomnias (e.g. sleepwalking, night terrors), insomnia and good sleep respond while doing a mental task. I am interested to see how the mind and body responds while doing both easy and difficult tasks by measuring heart rate and asking individuals about their experience. The information gathered is for research purposes only and is a requirement of my training. You will not be identifiable in the results.

Before you decide whether or not to take part, it is important for you to understand why the research is being carried out and what it will involve. Please take your time to read the following information carefully. If there is anything that is unclear or if you would like more information, please let me know. Take your time to decide whether or not you wish to take part.

What is the title of this project?
Stress Reactivity in Individuals with Non-REM Parasomnias, Insomnia and Good Sleep.

Why is the study important?
The current research investigating psychological processes in people with non-REM parasomnias (e.g. sleep walking, night terrors) is limited. Comparing people with non-REM parasomnias to those with insomnia as well as good sleepers will help build on what we do know and may be relevant to developing appropriate treatments in this area.

What are the aims of this study?
The aim of this study is to investigate how people with non-REM parasomnias respond to doing a mental task by measuring their heart rate and asking them to rate how stressful they found the experience. I will compare the responses of individuals with non-REM parasomnias to those with insomnia and good sleep.

Who can take part in this study?
Adults who experience non-REM parasomnias (e.g. sleep walking, night terrors) or insomnia as well as those who consider themselves to be good sleepers.

Individuals will not be able to take part if they experience other sleep disorders e.g. sleep apnoea or restless leg syndrome, neurological disorders e.g. narcolepsy or epilepsy, cardiac problems, pregnancy or medications affecting heart rate. Individuals with mental health problems will be excluded if their symptoms are severe and/or untreated. Individuals with stable and treated symptoms may be included. Those with significant drug or alcohol use will also be excluded.

Do I have to take part?
You do not have to take part in this study. Participation is entirely voluntary. If you do decide to take part you will be given this information sheet to keep and be asked to sign a consent form. If you decide to take part you are still free to come out of the study at any time without giving a reason.

What does participation in this study involve?
If you decide to take part, you will be asked to meet with me at the University of Glasgow Sleep Centre, based in the Southern General Hospital at a time that is convenient to you. You will be given the chance to ask questions and will sign the consent form. You may then be asked questions relating to your psychological well-being before participating in the main part of the study. You will attend the Sleep Centre for approximately 1½ hours.

The main part of the study will take place in one of the bedrooms in the Sleep Centre. You will be asked to lie in a comfortable position on top of the bed and your heart rate will be monitored continuously throughout several phases which will involve both rest periods and phases in which you will carry out a mental task by following instructions on a computer screen. Some parts of the task may be easy and some may be difficult.

Heart rate will be measured using a standard procedure which is frequently carried out at the Sleep Centre. This will involve four small adhesive-backed electrodes being attached to your chest and waist using a gel. You will also be asked to rate how stressful, if at all, you found the experience of the mental task.

Are there any risks involved?
The risks relating to taking part in this study are minimal. The simple monitoring procedure involved is frequently carried out at the Sleep Centre by trained staff.

There is a possibility that some individuals may experience a minor skin irritation due to the attaching of the electrodes. There is also a possibility that a pre-existing, but previously unidentified, mental health problem or cardiac condition may be discovered. If this were to occur, it would be discussed with you and you would be provided with advice, information and referrals as necessary. We would also inform your GP.

What will happen to all of the information?
All of the information collected about you during the research study will be kept ***strictly confidential.*** Personal details (such as your name and address) will not be stored on computer, so that you cannot be recognised from it.

Written feedback will be provided to those who request it following completion of the study.

Who is supervising this study?
My research supervisor, Professor Colin Espie, who works for the University of Glasgow, will supervise me.

Who is paying for this study?
This study is being funded through the University of Glasgow and has been reviewed by a Research Ethics Committee. The committee has approved the research as appropriate.

What if something goes wrong?
If you wish to complain, or have any concerns about any aspect of the way you have been approached or treated during the course of this study, you can contact my supervisor, Professor Colin Espie at the University of Glasgow (Tel: 0141 232 7699), who will be able to advise you on the appropriate complaints procedure, should you wish to use this.

What should I do if I have any questions about this study?
If you would like further details about the study, you can either contact me by phone or email.

What do I do now if I want to take part in this study?
If you decide that you would like to take part in the study you can either contact me on (telephone number) or (email address).

You are under **no obligation** to take part; participation in this study is **completely voluntary.** You do not have to give a reason for not wanting to take part in this study**.**

I would like to take this opportunity to thank you for your time and consideration

Appendix 2.11

University of Glasgow Sleep Centre
Sackler Institute of Psychobiological Research
Southern General Hospital
Glasgow G51 4TF
Tel: 0141 232 7699

Participant Identification Number:

CONSENT FORM

Title of Project: Stress Reactivity in Individuals with Non-REM Parasomnias, Insomnia and Good Sleep

Name of Researcher: Sarah Young

Please initial box

1. I confirm that I have read and understand the information sheet dated March 2011 (version 3) for the above study. I have had the opportunity to consider the information, ask questions and have had these answered satisfactorily. ☐

2. I understand that my participation is voluntary and that I am free to withdraw at any time without giving any reason, without my medical care or legal rights being affected. ☐

3. I understand that data collected during the study, may be looked at by individuals from the University of Glasgow, from regulatory authorities or from the NHS Trust, where it is relevant to my taking part in this research. I give permission for these individuals to have access to my records. ☐

4. I give consent for my GP to be contacted if an undiagnosed medical condition is suspected. ☐

5. I agree to take part in the above study. ☐

_____	_____	_____
Name of Patient	Date	Signature
_____	_____	_____
Name of Person taking consent	Date	Signature

When completed, 1 for patient; 1 (original) for researcher site file.

Appendix 2.12

Verbal Instructions to Participants

- The video camera in the room is not in use.
- The microphone is switched on and I will be able to hear you throughout the experiment from the control room. I will be in the control room while you complete the experiment.
- Able to see the screen properly and read the text clearly? – (Adjust if necessary)
- Follow the instructions on the screen to move through the experiment and respond using the response buttons.
- It is important that you remain as still as possible throughout the experiment. Even small movements will affect the heart rate recordings.
- The only movement you should make is to push the response buttons with your fingers.
- Please find a comfortable position and remain looking at the screen throughout.
- You will begin with a short training phase which will allow you to practice using the response buttons.
- The experiment involves two halves with a break in the middle to allow you to move around. During the break you may move around where you are on the bed or you may get up to walk around. If you wish to walk around, please wait for assistance with the wires before you get up.
- In each half there are 3 phases, and each phase lasts about 5 minutes, one phase in each half may last a little longer depending on how long you take to complete it.
- After each phase you will be asked how you are feeling on a scale of 1-10. Please respond by saying the appropriate number out loud. I will be able to hear you from the control room.
- Part of the experiment will involve you being asked a series of mathematical questions. You are likely to find some quite difficult and some easier. I'm interested to see how well you can do. Your goal is to try to answer as many questions correctly as you can, as quickly as you can.
- Do you have any questions?

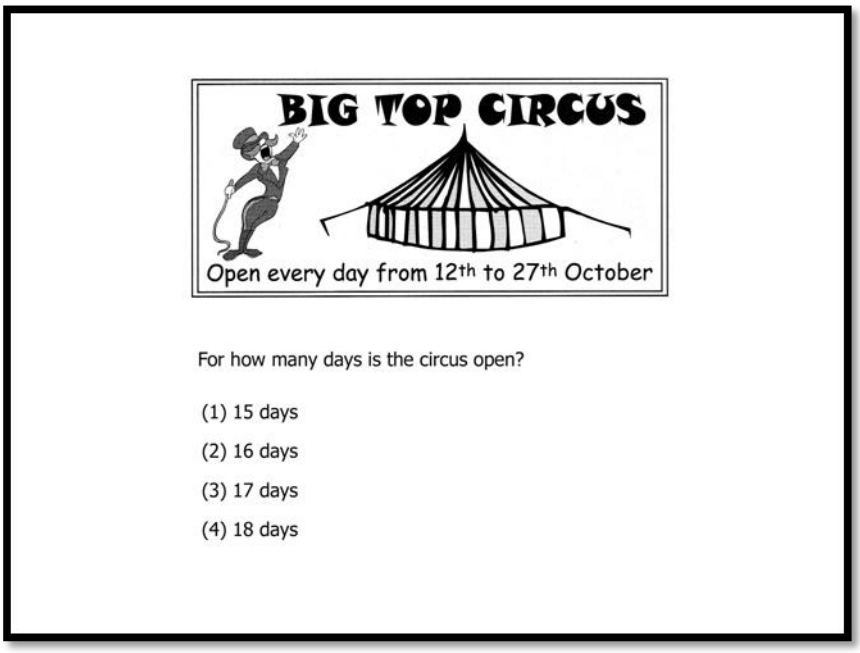
Appendix 2.13

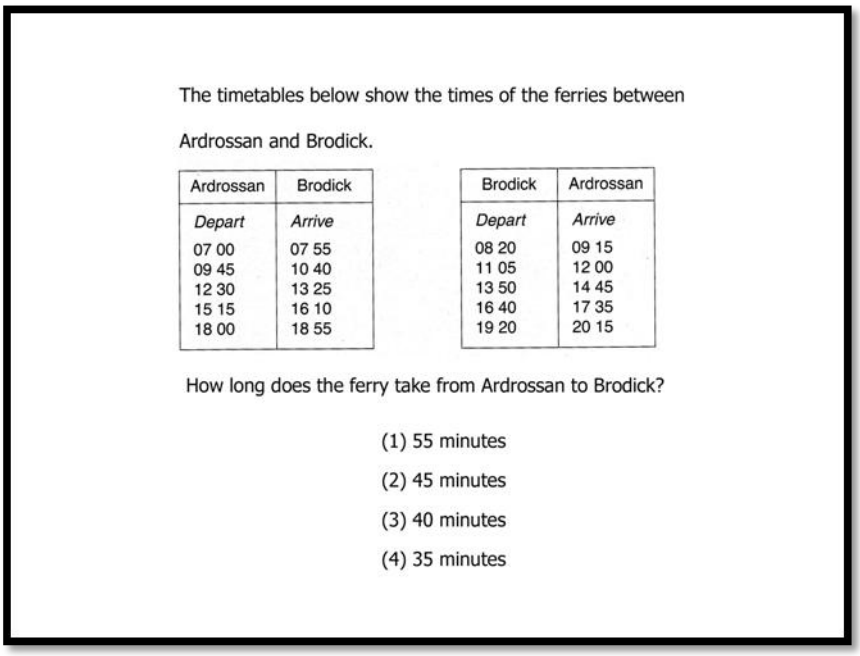
Participant Response Button Training Diagram

Appendix 2.14

Baseline and Recovery Phase Picture Stimuli

Appendix 2.15: Examples of Stressor Phase Stimuli

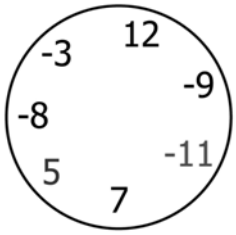




Carry out the following calculation:

13 x 7000

(1) 91 000
(2) 73 000
(3) 210 000
(4) 137 000



The circle above contains seven numbers.

Which three numbers from the circle add up to -10?

(1) 7, -11, -8
(2) -8, -9, 5
(3) 7, -11, -3
(4) -9, -8, 7

This table shows the cost of a room per week in three different hotels.

	COST PER WEEK		
	January	February	March
Hotel Spruce	£230	£240	£255
Hotel Alpine	£215	£235	£250
Hotel Nordic	£190	£220	£240

Fiona pays £470 for a **two week** stay in one of these hotels.

Which hotel did Fiona stay in and in which month?

(1) Hotel Spruce, March

(2) Hotel Nordic, February

(3) Hotel Spruce, January

(4) Hotel Alpine, February

Mr and Mrs Smith and their two children, aged 14 and 10,

are planning a ski holiday. This table shows the prices.

	Cost per person
Adult	£950
Child : over 12	£625
Child : 12 or under	£375

What is the total cost of their holiday?

(1) £2010

(2) £2700

(3) £2300

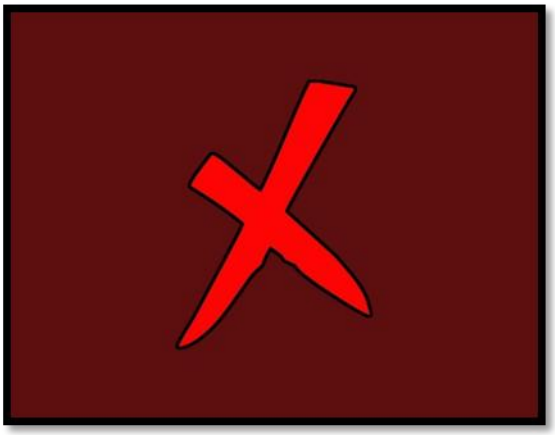
(4) £2900

Appendix 2.16

Stressor Phase Response Messages

Correct Response

Incorrect Response



Too Slow Response

Out of time.
Too slow!

Appendix 2.17

HR Data

Time Period	Levene's Test Result
B1	$F(2, 35) = 1.016$, $p = 0.372$
B2	$F(2, 35) = 1.273$, $p = 0.292$
B3	$F(2, 35) = 1.581$, $p = 0.220$
B4	$F(2, 35) = 1.044$, $p = 0.362$
B5	$F(2, 35) = 0.625$, $p = 0.541$
S1	$F(2, 35) = 0.285$, $p = 0.754$
S2	$F(2, 35) = 0.030$, $p = 0.970$
S3	$F(2, 35) = 0.130$, $p = 0.879$
S4	$F(2, 35) = 0.304$, $p = 0.739$
S5	$F(2, 35) = 0.067$, $p = 0.935$
R1	$F(2, 35) = 0.740$, $p = 0.484$
R2	$F(2, 35) = 0.657$, $p = 0.524$
R3	$F(2, 35) = 0.274$, $p = 0.762$
R4	$F(2, 35) = 0.626$, $p = 0.540$
R5	$F(2, 35) = 0.745$, $p = 0.482$

CVT Data

Time Period	Levene's Test Result
B1	$F (2, 36) = 2.239$, $p = 0.121$
B2	$F (2, 36) = 1.975$, $p = 0.154$
B3	$F (2, 36) = 2.165$, $p = 0.129$
B4	$F (2, 36) = 1.493$, $p = 0.238$
B5	$F (2, 36) = 1.288$, $p = 0.288$
S1	$F (2, 36) = 1.639$, $p = 0.208$
S2	$F (2, 36) = 1.952$, $p = 0.157$
S3	$F (2, 36) = 1.183$, $p = 0.318$
S4	$F (2, 36) = 2.117$, $p = 0.135$
S5	$F (2, 36) = 0.998$, $p = 0.379$
R1	$F (2, 36) = 2.770$, $p = 0.076$
R2	$F (2, 36) = 3.887$, $p = 0.030$
R3	$F (2, 36) = 1.393$, $p = 0.261$
R4	$F (2, 36) = 2.831$, $p = 0.072$
R5	$F (2, 36) = 1.515$, $p = 0.234$

Appendix 2.18

WoSRES
West of Scotland Research Ethics Service

NHS
Greater Glasgow and Clyde

West of Scotland REC 3
Ground Floor – The Tennent Institute
Western Infirmary
38 Church Street
Glasgow G11 6NT
www.nhsggc.org.uk

Professor Colin A. Espie
Professor of Clinical Psychology, and Director, University of Glasgow Sleep Research Laboratory
Gartnavel Royal Hospital
1055 Great Western Road
Glasgow G12 OXH

Date 10 Nov. 10
Your Ref
Our Ref
Direct line 0141 211 2123
Fax 0141 211 1847
E-mail Liz.Jamieson@ggc.scot.nhs.uk

Dear Professor Espie

Study Title: **Stress Reactivity in Individuals with Non-REM Parasomnia, Insomnia and Good Sleep**
REC reference number: **10/S0701/73**

The Research Ethics Committee reviewed the above application at the meeting held on 04 November 2010. Thank you for attending to discuss the study.

Ethical opinion

The members of the Committee present gave a favourable ethical opinion of the above research on the basis described in the application form, protocol and supporting documentation, subject to the conditions specified below.

Ethical review of research sites

The favourable opinion applies to all NHS sites taking part in the study, subject to management permission being obtained from the NHS/HSC R&D office prior to the start of the study (see "Conditions of the favourable opinion" below).

Conditions of the favourable opinion

The favourable opinion is subject to the following conditions being met prior to the start of the study.

Management permission or approval must be obtained from each host organisation prior to the start of the study at the site concerned.

For NHS research sites only, management permission for research ("R&D approval") should be obtained from the relevant care organisation(s) in accordance with NHS research governance arrangements. Guidance on applying for NHS permission for research is available in the Integrated Research Application System or at http://www.rdforum.nhs.uk.

Sponsors are not required to notify the Committee of approvals from host organisations.

Delivering better health

WoSRES
West of Scotland Research Ethics Service

NHS
Greater Glasgow and Clyde

West of Scotland REC 3
Ground Floor – The Tennent Institute
Western Infirmary
38 Church Street
Glasgow G11 6NT
www.nhsggc.org.uk

Professor Colin A. Espie
Professor of Clinical Psychology, and Director
University of Glasgow Sleep Research Laboratory
Gartnavel Royal Hospital
1055 Great Western Road
Glasgow G12 OXH

Date 19th November 2010
Your Ref
Our Ref
Direct line 0141 211 2123
Fax 0141 211 1847
E-mail Liz.Jamieson@ggc.scot.nhs.uk

Dear Professor Espie

Full title of study: **Stress Reactivity in Individuals with Non-REM Parasomnia, Insomnia and Good Sleep**

REC reference number: **10/S0701/73**

Thank you for your letter of 17th November 2010. I can confirm the REC has received the documents listed below as evidence of compliance with the approval conditions detailed in our letter dated 04 November 2010. Please note these documents are for information only and have not been reviewed by the committee.

Documents received

The documents received were as follows:

Document	*Version*	*Date*
Covering Letter		17 November 2010
Participant Information Sheet	2	16 November 2010
Participant Consent Form	2	16 November 2010

You should ensure that the sponsor has a copy of the final documentation for the study. It is the sponsor's responsibility to ensure that the documentation is made available to R&D offices at all participating sites.

10/S0701/73 **Please quote this number on all correspondence**

Yours sincerely

Mrs Liz Jamieson
Committee Co-ordinator

Copy to: Erica Packard, NHS Greater Glasgow and Clyde

Delivering better health

Appendix 2.19

Investigation of Participant and Procedural Variables

The table below indicates the number and percentage of participants in each group who took part in the experiment at different times of day.

Time of Day of Participation

	Good Sleeper		Insomnia		NREM	
	%	n	%	n	%	n
Morning	33.3%	5	25.0%	3	27.3%	3
Afternoon	40.0%	6	66.7%	8	36.4%	4
Evening	26.7%	4	8.3%	1	36.4%	4

Time of day was calculated according to the start time of the ECG recording as follows:

Morning: start time = between 10am and 11.45am

Afternoon: start time = between 11.46am and 4.45pm

Evening: start time = between 4.46pm and 8.30pm

Due to the categorical nature of the data and the small sample size, Fisher's exact test was used to investigate the relationship between time of day and group allocation. No relationship was found ($p = 0.51$).

Participant perception of their performance (How well do you think you did?) and their actual performance (% correct responses) during the stressor phase was also investigated.

Participant Perception of Performance & Actual Performance During Stressor Phase

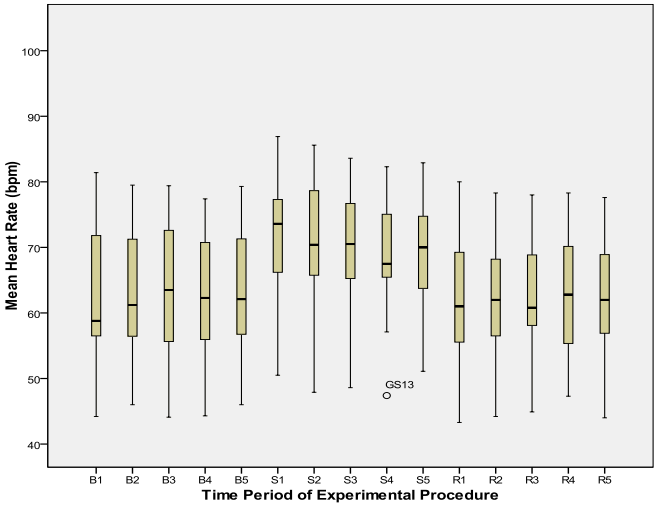
	How well do you think you did? (0-10)		% correct responses during stressor task	
	M	SD	M	SD
GS	5.13	1.73	66.39%	14.47
Insomnia	4.42	2.15	61.81%	23.56
NREM	3.91	2.02	59.85%	18.38

These data were examined for normality and homogeneity of variance. The 'Participant Perception' data were found to be normally distributed, and the groups were found to have equal variances. A one-way between groups ANOVA was therefore carried out. Participant perception of how well they performed in answering the questions during the stressor phase did not significantly differ according to group membership [$F(2, 35) = 1.29$, $p = 0.29$].

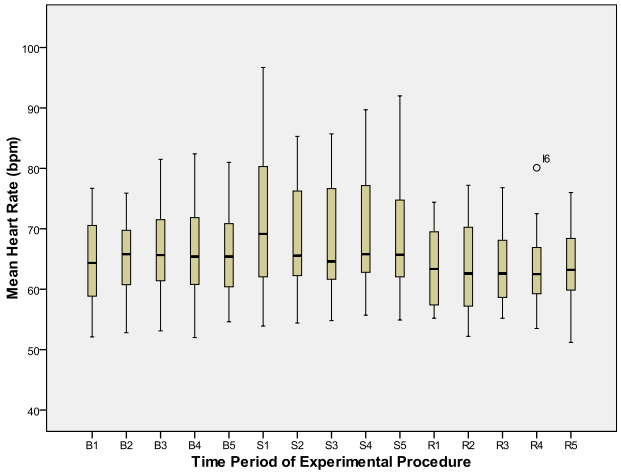
The '% Correct' data were found to be normally distributed, however the variances were found to be significantly different across groups. A Kruskal-Wallis test was therefore carried out. Participant performance in terms of the percentage of correctly answered questions during the stressor phase did not differ significantly across groups [$H(2) = 0.70$, $p = 0.71$].

Appendix 2.20

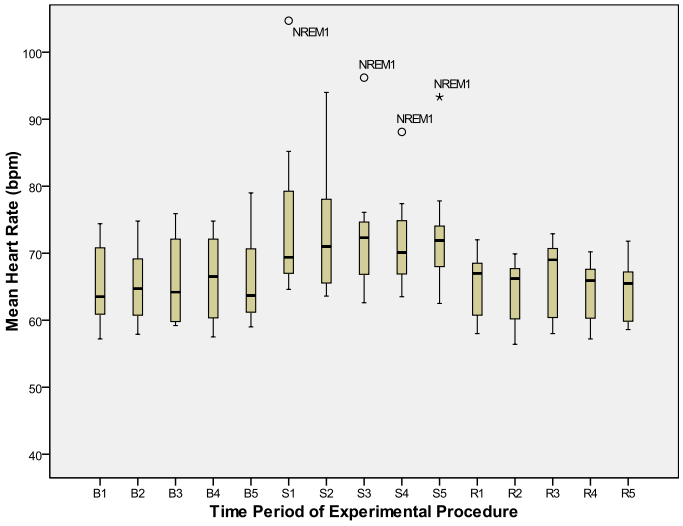
Boxplot of Mean Heart Rate: GS Group



Boxplot of Mean HR: Insomnia Group

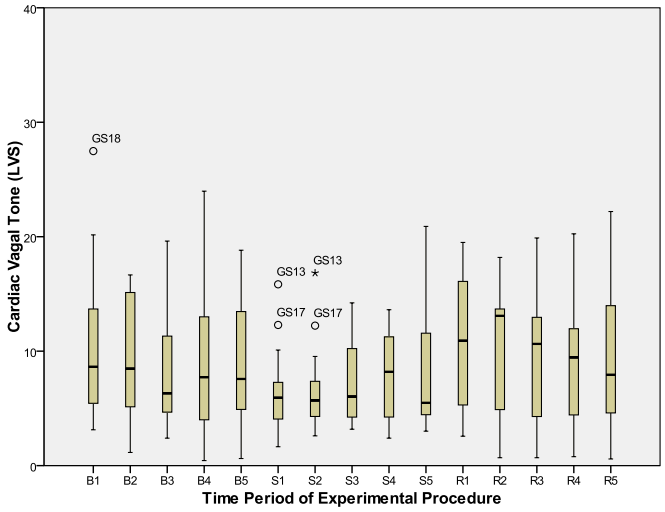


Boxplot of Mean HR: NREM Group

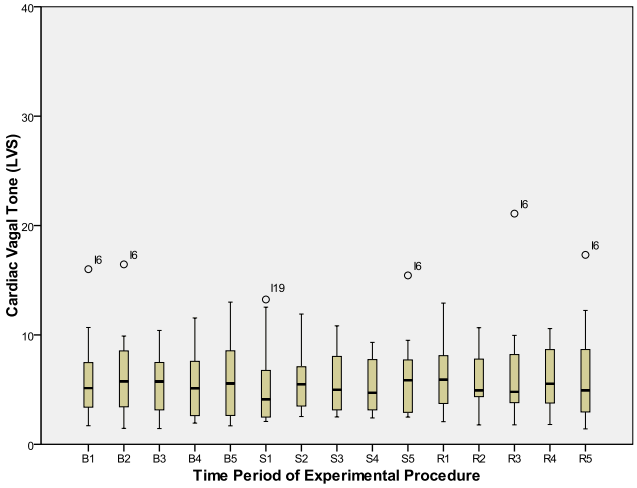


Note:
The boxplots show the range of scores with the box representing the interquartile range and the line in the box representing the median.

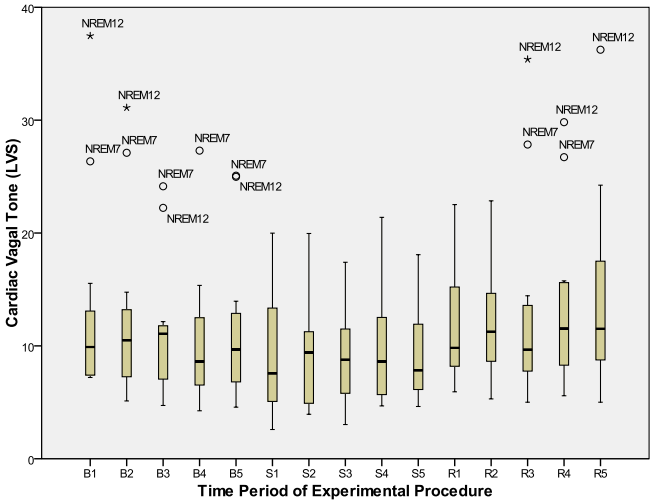
Boxplot of Mean CVT: GS Group



Boxplot of Mean CVT: Insomnia Group



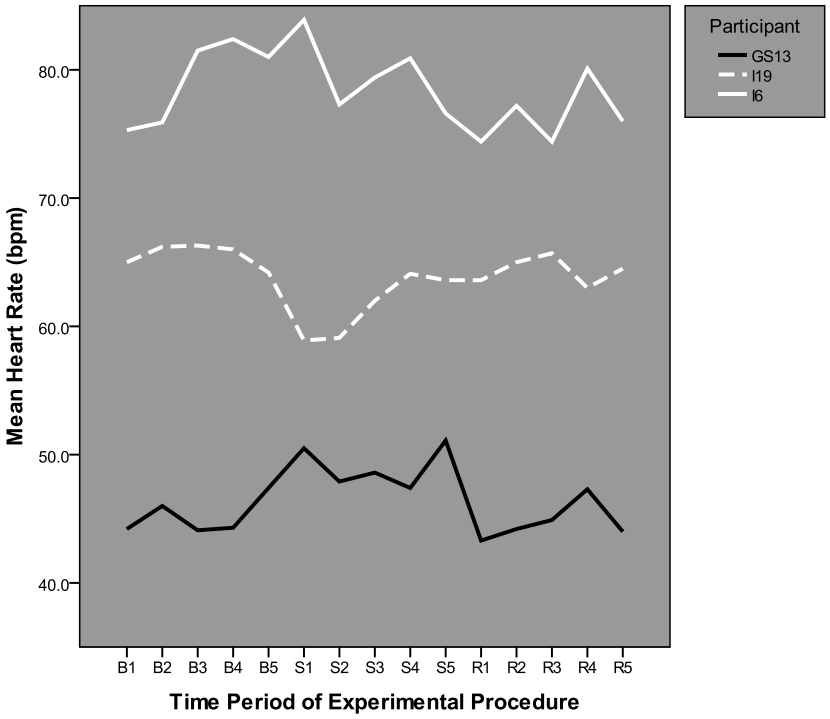
Boxplot of Mean CVT: NREM Group

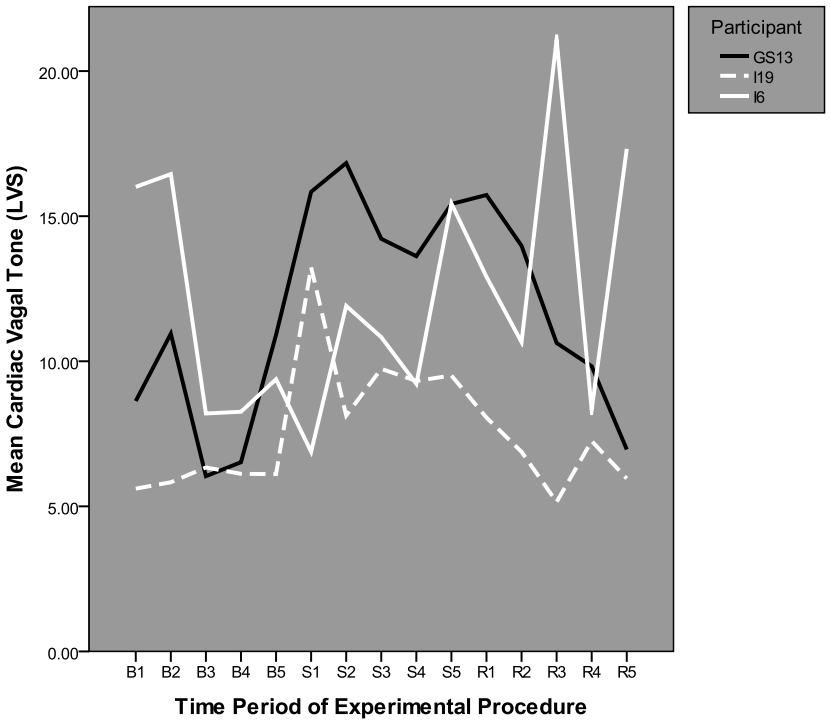


Note:
The boxplots show the range of scores with the box representing the interquartile range and the line in the box representing the median.

Appendix 2.21

<u>Three participants with extreme values whose CVT data does not follow the typical pattern of the group.</u>





Description of the three participants with extreme values whose CVT data does not follow the typical pattern of the group

	Participant GS13 (Good Sleeper)	**Participant I6 (Insomnia)**	**Participant I19 (Insomnia)**
Demographics	Female, 35	Female, 21	Male, 31
Health	No physical or mental health problems reported, no medication.	No physical or mental health problems reported, no medication.	No physical or mental health problems reported, no medication.
Alcohol	28 units per week	4 units per week	None
Global PSQI	2	14	7
DASS	Within normal range	Moderate depression (20) Moderate anxiety (10) Stress normal range	Within normal range
SCI	7	25	9
Impact of Poor Sleep	3	21	8
Description of sleep disorder		Sleep-onset and -maintenance insomnia; difficulties 7 nights per week; sleep problem for 4 years; average 4 hours sleep/night.	Sleep-onset and -maintenance insomnia; difficulties 7 nights per week; sleep problem for 10 years; average 6½ hours sleep/night.
% correct responses during stressor task	66.67%	50.00%	83.33%
How well do you think you did? (0-10)	6	3	6
Subjective Stress (0-10):			
Initial stress	5	0	3
Stress after B	2	0	2
Stress after S	8	7	4
Stress after R	3	3	3
Overall stress	6	4	4

Appendix 2.22

Differences in Subjective Stress between Groups: Mann-Whitney U Test Results

Stress Scale	NREM – GS			NREM – Insomnia			Insomnia - GS		
	U	*z*	*p*	U	*z*	*p*	U	*z*	*p*
Initial Stress	66.50	-0.85	0.41	52.00	-0.57	0.59	78.00	-0.24	0.83
After Baseline Phase	76.50	-0.33	0.76	61.00	-0.32	0.76	89.00	-0.05	0.98
After Stressor Phase	51.50	-1.63	0.11	48.50	-1.10	0.29	78.00	-0.60	0.57
After Recovery Phase	65.00	-0.95	0.35	41.00	-1.36	0.19	72.50	-0.54	0.61
Overall Stress	80.50	-0.11	0.93	59.50	-0.41	0.70	84.50	-0.28	0.80

Appendix 2.23

Summary of Effect Sizes (Cohen's *d*)

Effect size for Cohen's *d*:	
Small:	0.2
Medium:	0.5

Baseline Autonomic Arousal		
Interaction	**HR**	**CVT**
NREM - GS	0.34	-0.38
NREM – Insomnia	0.03	-1.16 *
Insomnia – GS	0.30	-0.63

Stress Reactivity				
	HR		**CVT**	
Interaction	Change Score	AUC	Change Score	AUC
NREM - GS	0.01	-0.04	-0.07	0.05
NREM – Insomnia	0.38	0.66	-0.45	0.53
Insomnia – GS	-0.40	-0.68	-0.37	0.50

* significant result

Achieved Power

Baseline Autonomic Arousal		
Interaction	**HR**	**CVT**
NREM - GS	0.13	0.15
NREM – Insomnia	0.05	0.75
Insomnia – GS	0.12	0.35

Stress Reactivity				
	HR		**CVT**	
Interaction	Change Score	AUC	Change Score	AUC
NREM - GS	0.05	0.05	0.05	0.05
NREM – Insomnia	0.14	0.34	0.18	0.23
Insomnia – GS	0.17	0.39	0.15	0.24

Post hoc calculation of achieved power (independent t tests), given alpha (0.05), sample size and effect size (*d).*

Appendix 2.24: Research Proposal

Introduction

Parasomnias are "undesirable physical events or experiences that occur during entry into sleep, within sleep or during arousals from sleep". The International Classification of Sleep Disorders, Second Edition (ICSD-2) distinguishes between parasomnias associated with Non-REM (NREM) sleep, parasomnias associated with REM sleep, sleep-wake transition parasomnias as well as 'other parasomnias' which are not classified by the other categories (American Academy of Sleep Medicine, 2005). NREM parasomnias are the most common type of parasomnias (Wills & Garcia, 2002).

There are three normal states that the nervous system can be in: wakefulness, NREM sleep and REM sleep. Most parts of the nervous system are active across the three states, but in different manners, and transitioning between states is a complex process (Mahowald & Bornemann, 2005). NREM parasomnias, also known as disorders of arousal, are incomplete arousals from slow-wave NREM sleep which result in behaviours associated with wakefulness. Since arousal is incomplete, the brain is partially awake and partially remains in NREM sleep. The three main NREM parasomnias are sleep terrors, sleepwalking and confusional arousals. They generally occur during the first third of the night and during episodes behaviour can appear confused and semi-purposeful. Individuals generally do not remember the events which tend to occur no more than once per night and can last from seconds to minutes (Vaughn & O'Neil, 2007). Confusional arousals involve mental confusion or confusional behaviour characterised by disorientation, slow speech, diminished mentation, impaired cognitive response and inappropriate behaviour. Sleepwalking episodes often begin with the individual sitting up in bed and appearing confused before walking or engaging in more complex behaviours such as dressing, or even driving.

Individuals may be emotionally calm or may be agitated. Violent behaviours are more likely to occur if attempts are made to wake the sleepwalker. Sleep Terrors are characterised by intense fear which is evident in the individual's behaviour and autonomic arousal. Individuals tend to suddenly sit up in bed, let out a piercing scream and can be inconsolable as well as unresponsiveness to external stimuli. (American Academy of Sleep Medicine, 2005).

NREM parasomnias are more common in children than adults. Whereas NREM parasomnias in children appear to often be associated with genetic and developmental factors, persistence (or onset) into adolescence or adulthood has been associated with psychopathology (Kales, Kales et al., 1980; Kales, Soldatos, et al., 1980; Kales et al., 1982; Gau & Soong, 1999; Ohayon et al., 1999). However, many individuals with NREM parasomnias do not present with significant psychopathology and in fact show good psychosocial functioning (Mahowald, 2002; Mahowald & Borneman, 2005; Schenck et al., 1997).

NREM parasomnias are thought to occur as a result of a complex group of priming and precipitating factors in those that are genetically predisposed. NREM parasomnias seem to be more likely to occur in the presence of factors that deepen sleep, fragment sleep or make arousal from sleep more difficult. Factors such as stress, sleep deprivation, medication, alcohol and febrile illness can affect sleep and make the occurrence of NREM parasomnias more likely (Pressman, 2007; Mahowald & Borneman, 2005). It is thought that a trigger is also necessary for a specific event to occur. Triggers include noise, touch, and co-existing sleep disorders such as sleep disordered breathing or periodic leg movements in sleep.

Sleep disturbance and psychopathology commonly co-occur and there is a particularly high comorbidity between insomnia, anxiety and depression (Morin & Ware, 1996) with sleep difficulties being a symptom of both anxiety and depression. The link between insomnia and stress is well documented. Stress can have the effect of increasing arousal levels which can make it more difficult to fall, or stay, asleep (Morin et al., 2003). Stress and anxiety have also been linked to NREM parasomnias (Mahowald & Borneman, 2005; Mahowald & Schenck, 2005; Pressman, 2007; Ohayon et al., 1999). Insomnia and parasomnias are both psychophysiological conditions since they have both psychological and physical aspects. Anxiety and depression are also examples of psychophysiological conditions although occurring during wakefulness whereas parasomnias and insomnia occur during sleep or incomplete arousal.

Previous research has found that people with insomnia show greater autonomic arousal compared to normal sleepers (Bonnett & Arand, 1996, 1998; Adam et al., 1986; Stepanski et al., 1989, 1994; Haynes et al., 1981, 1974; Monroe, 1967; Freedman & Sattler, 1982; Riemann et al., 2010), suggesting insomniacs react more readily to stressors at a physiological level. Autonomic arousal occurs as the 'fight or flight' response and has often been used as a measure of stress reactivity (Boyce et al. 2001) since it is an indication of how an individual responds at a physiological level to stressful or threatening events. Since autonomic arousal is closely related to emotion it makes sense that individual differences in autonomic arousal in response to stress may be linked to the development of symptoms of psychopathology. Studies have found individual differences in autonomic arousal to be been associated with psychopathology in both adults (e.g. Raine, 1996; Rechlin et al., 1994; Felsten, 2002; 2004) and children (e.g. Boyce et al., 2001). Felsten, for example, reported

associations between stress reactivity, neuroticism and a vulnerability to stress and depressed mood.

There appears to be a gap in the literature with regards experimental studies of stress reactivity amongst people with NREM parasomnias and it would be interesting to investigate this area in order to gain further insight into daytime functioning of this group. In addition to looking at how individuals react to stressors physiologically, baseline levels of autonomic arousal in the absence of a stressor would provide insight into daytime arousal levels as a trait-like feature. To date, there is little research into either stress reactivity or the specificity of psychological characteristics in particular forms of sleep disorder. This study is designed to make comparisons between two types of sleep disorder: NREM parasomnias and insomnia.

Aims and Hypotheses

The aim of this study is to investigate stress reactivity in people with NREM parasomnias by measuring autonomic arousal across three conditions: (1) at baseline, (2) in reaction to a psychological stressor and (3) time taken to return to baseline. People with NREM parasomnias will be compared to two control groups: a primary insomnia control group (PI) and a good sleeper control group (GS). Using a clinical control group in addition to the good sleeper control group will allow comparison of the NREM group to a group of individuals with another sleep disorder. This will allow better discrimination between findings associated with NREM parasomnias specifically, and those associated with disordered sleep more generally. An insomnia group was chosen as the clinical control since, like NREM parasomnias, insomnia is also psychophysiological in nature.

It is hypothesised that the PI group will show increased autonomic arousal at resting baseline, greater stress reactivity and a slower return to baseline in comparison to good sleepers.

It is thought that the NREM group will also show increased autonomic arousal at baseline, greater stress reactivity and a slower return to baseline in comparison to good sleepers since NREM parasomnias are, inherently, (triggered) arousal disorders as well as also being psychophysiological in nature. This prediction for the NREM group, however, is based on rationale rather than evidence and so ultimately remains a research question. The nature of the difference in autonomic arousal between the NREM and PI groups remains a research question. A subjective, self-report measure of stress will also be employed and the nature of how this will differ between groups remains a further research question. The PI group is predicted to report higher levels of stress than good sleepers. Morin et al. (2003) found that insomniacs tend to appraise their lives as more stressful than good sleepers even though both groups reported similar levels of stressors. It will be interesting to investigate how the NREM group's subjective measures of stress compare to the two control groups. Clinical observations of clients presenting with NREM parasomnias at the Glasgow Sleep Centre suggest a tendency to appear to cope well with challenges and stress, although perhaps superficially. Clinicians report individuals to appear carefree and unperturbed with regards stressors although an underlying emotional vulnerability may be evident. It is therefore predicted that individuals in the NREM group may report lower levels of stress than the PI group. Such individuals may be described as repressors. Weinberger et al. (1979) identified repressors as individuals with low trait anxiety but high defensiveness. They found repressors to respond to stressful situations with high physiological arousal suggestive of anxiety. However, they also showed a tendency to avoid disturbing cognitions, deny

cognitive anxiety and report low trait anxiety. Derakshan & Eysenck (1999) concluded from their study that repressors truly believe themselves to be low in trait anxiety and that there may be avoidance or cognitive biases working at a subconscious level to inhibit awareness of anxiety.

In summary, it is hypothesised that both the NREM and the PI group will show indications of stress reactivity on objective physiological measures. It is also hypothesised that the PI, but not the NREM group, will report high levels of stress subjectively.

A parallel questionnaire-based self-report survey study is also being planned (Katherine Hooker). This parallel study will aim to investigate state and trait arousal (pre-sleep arousal and arousal pre-disposition respectively), worry, rumination and emotional inhibition across NREM parasomnia, insomnia and good sleeper groups. Measures of sleep quality, anxiety, depression and stress will also be included as part of the screen. It is hoped that participants in this study will also take part in the parallel survey study by telephone, post or online, prior to their involvement in this study, although it will not be compulsory. This would then allow for descriptions of the three groups in this study, in terms of the above elements, to be produced and compared. If there are any participants who choose not to take part in the parallel study, missing questionnaire data will be sought.

Method

Design

The study will employ a mixed design. Participants will belong to independent groups (NREM, PI or GS) and each group will participate in baseline, stress and return to baseline conditions. The NREM group will be compared to two control groups: the clinical control

(PI) group and good sleepers. The independent variable is the presence or absence of a non-REM parasomnia, insomnia or good sleep according to defined criteria. The dependent variables are: (1) autonomic arousal at resting baseline, (2) stress reactivity i.e. autonomic arousal in response to stress in comparison to baseline, (3) time taken for autonomic arousal to return to baseline, (4) a subjective, self-report measure of stress.

Participants

Participants will meet ICSD-2 criteria for either a NREM parasomnia or insomnia, or meet research diagnostic criteria for good sleep. The prevalence rates in the general adult population for confusional arousals and for sleepwalking are thought to each be approximately 4%. Sleep terrors are thought to occur in up to 3% of the general adult population (Ohayon et al., 1999). Participants in the three groups will be matched for age, gender and socio-economic status (determined by post code). Participants will be recruited from the general population through the media, advertisements and clinical referrals in a manner consistent with the University of Glasgow Sleep Centre's (UGSC) experience and operational procedures. In the past, recruitment in this way at the UGSC has yielded good response rates and spontaneous referrals are also common. Advertisements may be placed, for example, in publications e.g. newspapers or the UGSC newsletter, in public spaces e.g. in hospitals, and on the internet e.g. on the UGSC website. Participants may also be identified via an existing database held at the UGSC which has details of participants who have not been suitable for previous research and have agreed to be contacted for future studies. Additionally, some potential participants may be identified through the parasomnia clinic which is ongoing at the UGSC. All clinic attendees receive a general letter saying that we have research projects in which they may be interested. If they express an interest they will be given the participant information sheet and follow up phone calls will be made in the

same manner as with other potential participants. This study is planned to be part of a larger programme of research and recruitment may come directly as well as via a parallel study (Katherine Hooker). Suitable participants recruited into the parallel study will be offered the opportunity to take part in this study and vice versa.

It is expected that people interested in participating in the study will contact the UGSC to declare this interest. Potential participants would be screened via telephone which will include the following:

- Standard screening interview which is administered to all potential research participants at the UGSC with additional questions relating to health and socio-demographic factors as well as International Classification of Sleep Disorders (ICSD-2) diagnostic criteria for insomnia and Non-REM parasomnias.
- Pittsburgh Sleep Quality Index (PSQI)
- Depression Anxiety Stress Scales (DASS)
- Sleep Condition Indicator (SCI)

Further details of the screening measures are included in the proposal for the parallel study (Katherine Hooker). If deemed suitable according to the inclusion/exclusion criteria defined, potential participants will be sent information sheets regarding both studies as well as consent forms. As part of the information provided, they will be informed that participants will be asked to abstain from caffeine, exercise, alcohol and cigarettes for a period of 4 hours prior to the visit. The potential participants will also be informed that this study may highlight cardiac problems or psychopathology and will be informed of the action that will be taken if such a situation should arise. Potential participants will subsequently be given time to consider if they wish to participate before a follow-up phone call is made

approximately one week later. Those who agree at this stage to participate in this particular study will either immediately be given further instructions for attending the UGSC to participate, or will be informed that they will be contacted at a later date with further details.

Inclusion Criteria:

Participants will meet The International Classification of Sleep Disorders, Second Edition (ICSD-2) criteria for either a NREM parasomnia or insomnia, or meet research diagnostic criteria for good sleep. Participants will be aged 18 years and over.

Exclusion Criteria:

Individuals will be excluded if they do not meet criteria for a NREM parasomnia, insomnia or good sleep or if they meet criteria for both a NREM parasomnia and insomnia since participants must only belong to one group. Other exclusion criteria include: other sleep disorders e.g. sleep apnoea or restless leg syndrome (RLS), neurological disorders e.g. narcolepsy or epilepsy, cardiac problems, pregnancy and medications affecting HR. NREM parasomnias are sometimes seen as symptoms of sleep disordered breathing and RSL, and treatment of these disorders has been shown to eliminate the parasomnia. Guilleminault et al. (1996) reports that 28.2% of children aged 8-12 with sleep disordered breathing report sleepwalking. Klackenberg (1987) reports a prevalence rate of 15% for sleepwalking in children aged 5-12. Provini et al. (1999) reported that 34% of 100 individuals with nocturnal frontal lobe epilepsy reported a personal history of parasomnias.

Individuals with DSM based Axis 1 psychiatric disorders will be excluded if their symptoms are severe and/or untreated. Individuals with stable and treated symptoms may be included. Their symptoms will be observed and noted and will be taken account of in the analysis as a

co-variate if appropriate. Ohayon et al. (1999) reported higher rates of mood disorders (30.4% of those with night terrors and 14.6% of sleep walkers) and anxiety disorders (34.2% of those with night terrors and 12.7% of sleep walkers) compared to controls (5.7% and 4.7% respectively).

Measures

Autonomic arousal will be measured via ECG recordings of HR and CVT. UGSC standard operational policy will be followed for electrode attachment and recording. HR is continually under the influence of both sympathetic and parasympathetic activity, each having an antagonistic but complementary influence (Sherwood, L., 2010). Sympathetic activity has the effect of quickening HR while parasympathetic (vagal) tone slows it down. Vagal tone affects HR more quickly (within one heart beat) than sympathetic activity, thus it is vagal tone which is responsible for rapid changes in HR (Little et al., 1999). HR and CVT will be recorded throughout all phases producing continuous data. Average values will be taken from specific, stable periods of each phase, allowing for a period of stabilisation at the beginning of each phase. Prior to the study commencing, training will be provided by the UGSC on autonomic measurement and analysis.

A subjective, self-report measure of stress will also be employed at various times throughout the process. Participants will be asked to rate their level of stress using a visual-analogue scale ranging from 0 - 10. The exact nature of this scale and the times the points at which it will be used will be determined during a pilot of this study.

Research Procedures

On arrival at the UGSC information about the study, the right to withdraw and confidentiality will be discussed. Participants will be informed that the study may highlight cardiac problems or psychopathology and consent will be obtained. An interview will be conducted in which the Mini International Neuropsychiatric Interview (MINI), a standardised measure, will be used to screen for DSM-based Axis 1 disorders.

The study will be carried out at the UGSC at the Southern General Hospital. The room used will be quiet and the light level and temperature will be standardised to ensure calm, pleasant surroundings. Time of day may make a difference to measures of autonomic arousal. Participants will be asked to attend within the time period of e.g. between 10-4pm and a scheme will be put in place to overcome time of day as a confounding factor between groups. It is expected that participants will lie in a prone position (without using postural muscles) on the bed with their head resting on pillows so that a computer screen, which will be positioned a set distance from the participant, can be comfortably viewed. It is thought that one hand will rest on a pillow with a button press and participants will be told to remain as still as possible throughout, other than pushing the response button, since movement will affect the measures. A script will be used to provide instructions and participants will progress through five phases. The exact nature and length of phases will be determined by the pilot study but it is likely that each will continue for five minutes before the subsequent phase commences, taking a total of twenty five minutes. The initial resting baseline phase (RB) will involve participants relaxing whilst looking at a relaxing image on the computer screen. The initial stress phase (S1) will involve participants carrying out a difficult task e.g. a mathematical serial subtraction task using complex numbers. The exact nature of the task will be established out of the pilot study. Stimulus presentation software (SuperLab) will be

used to programme the computer to present the stimuli and participants will respond using the button press with two fingers to provide yes/no or true/false responses. Following phase S1, baseline conditions will be repeated (phase R2) followed by a second stress phase (S2) with unseen stimuli before phase R3 in which baseline conditions will be again repeated. Thus, the five phases will run in the following order: RB, S1, R2, S2, R3. HR and CVT will be measured continuously throughout all phases. Phase R3 can be extended if necessary to allow arousal to return to baseline levels before participants are debriefed and thanked.

Lab Protocol Development and Testing

As part of the developmental stage, a pilot study will be carried out with the help of volunteers in order to inform and validate the experimental procedure. The pilot study will be used to establish whether the stimuli will in fact induce the desired stress response. It may be appropriate to prime such a response by manipulating the experimental instructions at the outset or by altering the stimuli (e.g. frequency of presentation or complexity of the numbers) and this will be considered during the pilot. Some individuals are likely to find the procedure more stressful than others in all three groups and this variation is expected to be normally distributed. Differences in how stressful individuals find the experimental task will be considered during the pilot study and it is hoped that by measuring stress reactivity relative to each individuals' baseline, this difference will not become a confounding variable. The pilot will be conducted in two stages. An initial pilot will be carried out with volunteers without the use of electrodes to record autonomic arousal. Subjective measures of stress will be employed in order to find out how stressful the task is found to be. The task level should be considered difficult and the success rate should not be too high. Autonomic arousal will be measured during the second stage of the pilot with the aim of investigating the pattern of autonomic arousal across phases and how long it takes for participants'

arousal to return to baseline. It will be important to determine, for example, whether acute stress is experienced before habituation as this will inform which portions of continuous data will be used to provide average values for further analysis. The second stage of the pilot will also be used to establish the total time needed to sufficiently test each participant. The aim will be to keep the time as short as possible with no more than two hours (and quite possibly less) estimated for each participant. Following the pilot, the study will be carried out on experimental participants.

Justification of sample size

A prior study in which HR and CVT have been measured in two clinical psychophysiological sleep disorder populations has not been identified. A recent study (Espie et al., unpublished abstract) found statistically significant differences in HR and CVT in individuals with insomnia (n=8) compared to normal sleepers (n=9). A medium-large (d = 0.75) effect size was found for HR and a large effect size was found (d = 1.63) for CVT. This study involved the comparison of one a clinical group with a control. Since this is a preliminary study, both the effect size and sample size must be estimated. The primary research question of whether stress reactivity in reaction to a psychological stressor in a sleep disordered group will significantly differ from stress reactivity in clinical or good sleeper control groups has never before been addressed. This study can therefore hope to provide a definitive answer of how to power future studies. The power calculation is based on the hypothesis that a significant difference in stress reactivity will be found between groups. Stress reactivity will be a measure of the difference between autonomic arousal in response to stress and baseline autonomic arousal. A replication design will be utilised to strengthen the study, involving two stress phases, each followed by a return to baseline conditions. A total sample size of 66 participants (22 per group) was calculated for a one-

way ANOVA using an effect size of 0.4 (f^2), a significance level of 0.05 and standard power of 0.8. It is therefore proposed that 22 participants should be recruited to each of the three groups. If a larger effect size (0.45 or 0.5) was to be used in the calculation, then it would be proposed that a total sample size of 51 or 42 respectively (17 or 14 per group) should be recruited.

Settings & Equipment

- A bedroom at the UGSC, Southern General Hospital.
- Cover sheet for the bed.
- ECG will be recorded using a Lifelines Trackit recorder and disposable wet gel ECG electrodes. Energiser (Ultimate/Ultra) 9V batteries will also be required.
- SuperLab stimulus presentation software.
- A laptop and an appropriate stand to position the screen correctly.

Data Analysis

A continuous ECG output will be recorded and analysed using the NeuroScope^{TM} method (Little et al., 1999), yielding HR (beats per minute; bpm) and the non-invasive index of CVT (linear vagal scale; LVS) on a continuous beat-to-beat bases. Average values will be calculated for particular periods in each phase in which data has stabilised. Data will be exported to Excel and SPSS for analysis. Normative data will be obtained from the UGSC. The demographic data will be analysed to look for similarities between groups with regards gender, age and SES. Any differences between groups will be introduced as a covariate in the analysis. ANOVAs will be used to compare groups for each dependent variable. Dr

Stig Hansen and Christine Salveta will also advise on data analysis (from a technical standpoint).

Health and Safety Issues

Researcher Safety Issues

The study will only be carried out at pre-arranged times at the UGSC when other staff members are present. Sleep Centre staff will be kept informed regarding the participant schedule.

Participant Safety Issues

If necessary, participants will be given extra time to relax at the end of their participation to ensure autonomic arousal has returned to baseline level. Researchers will be trained by the UGSC in autonomic measurement prior to the study commencing and UGSC standard operational policy for electrode attachment, removal and recording will be adhered to.

Ethical Issues

This study employs the use of a mild psychological stressor. Participants will engage in a period of relaxation following the stressor which will allow their level of arousal to subside and return to baseline. Participants will not be left in a stressed state at the end of the study. They will be asked to rate their level of stress at the end of the study and if they indicate feeling stressed, further opportunity for relaxation will be given. There is standard operational policy regarding procedures for attaching and removing electrodes in place at UGCC which will be adhered to. Participants will be warned prior to giving consent both verbally and via an information sheet, that the study may highlight cardiac problems or psychopathology. If evidence of a condition is found, or any indication of an unusual ECG

recording, this will be discussed and participants will be provided with advice, information and referrals as necessary. Details of participants' GPs will be recorded at the outset and informed consent will be sought regarding contacting the GP on the participant's behalf should a serious cardiac or psychiatric disorder be suspected.

Arrangements to give feedback regarding the study will be made if requested. Adequate time and information will be provided to allow participants to make informed consent regarding their involvement. Confidentiality and data protection protocol will be observed. Data will be anonymised data and securely stored. Participant time will be kept to a minimum.

Financial Issues

Photocopying, letters & postage, bed cover, ECG electrodes (3 per person) and 9V batteries (1 per day).

Timetable

The study will be carried out between October 2010 and May 2011.

Practical Applications

The outcome of this study could help inform interventions for individuals with NREM parasomnia.

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