

**Initial Insomnia and Paradoxical Intention:
An experimental investigation of putative mechanisms using
subjective and actigraphic measurement of sleep
and Research Portfolio**

**PART ONE
(Part two bound separately)**

Niall Broomfield
August 2001

Submitted in partial fulfilment of the requirements
for the degree of Doctor of Clinical Psychology



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This thesis is dedicated to wee Sam, my new nephew.

Chapter 1: Small Scale Service Evaluation

An investigation of the correspondence between psychological problems diagnosed by GPs and those subsequently targeted for treatment by Clinical Psychologists.

Small Scale service Evaluation submitted in partial fulfilment of the requirements for the degree of Doctor of Clinical Psychology

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Abstract

Objectives

1. To observe the level of correspondence between problems identified by GPs in the referral letter, and primary treatment targets identified by Clinical Psychologists following assessment within an NHS Clinical Psychology Department.
2. To examine whether the pattern of correspondence between GP and Psychologist varies for different psychological problem areas.
3. To highlight problems with the current department referral auditing system.

Design

A retrospective review, and classification, of GP referral letters and Psychologist treatment targets.

Setting

A Department of Clinical Psychology based in Scotland. Sixty five GPs based across fifteen GP practices refer to the department.

Cases

Four hundred and one patients consecutively referred to the Department of Clinical Psychology between April 1 1996 and March 31 1998, and subsequently assessed by one of the qualified Clinical Psychologists. Two hundred and seventy three cases were female (68.08%), one hundred and twenty eight male (31.92%).

Results

Overall agreement between GP referred problem and Psychologist treatment target, coded in terms of EPPIC 'reasons for care' categories was 'fair', and observed for

59.60% of cases. High agreement for eating disorders, moderate agreement for anxiety and depression and low agreement for relationship/social problems was observed. Analysis suggested GP over-referral of anxiety and depression. A number of problems with existing departmental auditing of referrals were also highlighted.

Conclusions

Findings appear to replicate those in the literature regarding over-referral of anxiety and depression. Low levels of agreement for relationship/social problems were not anticipated. The implications of results for future research and service provision are outlined.

Introduction

The quality of care received by patients within the NHS relies, in part, on good communication between General Practitioners (GPs) and clinical specialists (1). Accordingly, an important issue within the primary care setting is the quality of communication between GPs and hospital specialists. Recently, researchers have begun to examine two related aspects of GP-specialist communication: referral content and referral accuracy.

A number of studies have examined the informational content of referrals sent between GPs and Psychiatrists (1,2,3,4). However, recently, the issue of diagnostic agreement between GPs and Psychiatrists has also received attention in the literature (5). GPs provisional diagnoses for 209 referrals to an old age psychiatry service were recorded and compared to diagnoses made by a psychiatrist. Subsequent analysis noted diagnostic correspondence on 81.9% of cases. Notably, GPs misdiagnosed a substantial number of depressive illnesses as anxiety disorders or other functional illnesses. This parallels findings from other research indicating that GPs frequently misinterpret psychiatric symptoms in their patients (6).

A number of related studies in the literature have suggested that GPs often fail to recognise or diagnose mental health problems. For example, in a recent survey (7) detection rates for mental health problems were found to be considerably higher when screening was based on the General Health Questionnaire-28 (33.2 %) and the Schedules for Clinical Assessment in Psychiatry (SCAN) system (31.5%), than when screening involved direct GP observation (14.1%). Similar results were reported in a study (8) where trained medical students presented to GPs suffering from generalised anxiety disorder. Analysis indicated that 25 of 42 GPs studied made no diagnosis at all, or diagnosed only physical problems. Other studies have demonstrated that GPs may have difficulties recognising and diagnosing depression (9).

Despite receiving such attention in the psychiatric literature, the notion of GP referral accuracy has been largely ignored in the clinical psychology literature. This is somewhat surprising, particularly given the fact that GPs are the most frequent referral pathway to adult mental health psychology services (10). However, data from two studies are of some relevance.

In one, a Clinical Psychologist sat in on 366 GP consultations, rating each for psychological relevance (11). Researchers then compared these data with ratings of psychological relevance made by the GP for the same cases. Results indicated low levels of agreement between GP and Psychologist as to which cases required psychological input.

In a second recent study (12), 115 GP referral letters to a Clinical Psychologist were examined for referral problem and compared to diagnoses based on ICD-10 criterion made by the clinician for the same cases. Results indicated agreement between GP and Psychologist in 56.52% of cases surveyed. Nearly half of all cases referred by the GP for anxiety were diagnosed differently by the psychologist, most frequently depression. In addition, two thirds of cases diagnosed by the psychologist with relationship problems were initially referred with a different problem. However, these data were generated from an audit of one Clinical Psychologist, and therefore reflect the views of a small number of GPs and one Psychologist. A broader survey of GP-Psychologist correspondence would clearly be useful (12).

In addition, it is important to note that the above study examined GP referrals and compared these to a Psychologists diagnoses. Whilst Psychologists in primary care may diagnose specific disorders, current models of clinical psychology delivery emphasise formulation and the development of treatment targets as processes central in the clinical management of cases (13). Thus, observation of the correspondence between GP referrals and Psychologists treatment targets would provide a more direct

comparison of what patients are referred for and how they are subsequently managed.

Currently, details of all new GPs referrals to the NHS Department of Clinical Psychology under study are placed on an audit sheet (see Appendix 1.2). Included in this information are treatment targets and type of disorder, both recorded by the Psychologist following completion of the intervention. Discussions between the auditor and clinicians in the department concluded that the recording system with regard to treatment target was comprehensive and reliable, although some concerns were raised with respect to the type of disorder section. It was agreed the present audit would provide a useful forum for reviewing methods for recording disorder type. Importantly though, by examining correspondence between GP referral and treatment target, the present data were entirely unaffected by any limitations of the type of disorder recording system.

The present study therefore aimed to survey the correspondence between problems referred by the GP and problems subsequently targeted for treatment following assessment by the Psychologist. In doing so, it was anticipated that it would both complement, and extend, findings in the literature with respect to communication between General Practitioners and clinical specialists.

Method

Design

The present study was designed to provide a review of the concordance between referred problem and primary treatment target identified by the Psychologist following assessment. All patients referred by GPs to the Department of Clinical Psychology between April 1 1996- March 31 1998 were retrospectively included in the study.

In order to achieve a working degree of uniformity, problems referred (GP) and

problems targeted for treatment (Psychologist) were each coded according to Effective Purchasing and Providing in the Community (EPPIC; 14) 'reason for care' categories (see Appendix 1.3). These categories encompass the most common psychological problems, and thereby provide a reasonable working definition of psychological dysfunction. Two additional categories derived from the EPPIC 'contributory factors' categories were also included in the adopted coding system: abuse/trauma and psychiatric illness. EPPIC contributory factors were developed to complement 'reasons for care' categories by clarifying precisely what psychological interventions are targeted at (14).

In order to ensure reliability of coding, the system of allocation to EPPIC categories was subjected to a test of inter-rater reliability. A random sample of 20% (n = 80) of all problems referred and problems targeted were allocated to the same set of modified EPPIC categories as those utilised by the auditor. A Trainee Clinical Psychologist completed this procedure in order to control for any bias due to differential level of experience.

Obtained data indicated a high degree of inter-rater agreement overall (89.38%), as is evident in Table 1.1.

Insert Table 1.1 here

Kappa Coefficient analysis confirmed a high degree of inter-rater reliability, indicating this was 'excellent' (15) for both problems referred ($K = 0.73$) and problems targeted ($K = 0.98$).

Procedure

Data collection consisted of two phases. First, the referral letters of all cases were examined, and a primary reason for referral identified, and coded using the

modified EPPIC categories. Direct reference to a particular problem (e.g. he is now at the stage he is clinically depressed.), but also an accurate behavioural description of symptoms (e.g. she is low in mood, weepy, her sleep pattern is disturbed, she is negative in her thinking and she shows reduction in appetite and concentration.), led to final categorisation. When more than one presenting problem was described, the problem which most closely reflected the GPs treatment priority was encoded as primary. An unstated category was relied upon for those letters where no problem was apparent. This occurred on only 11 (0.03%) of all cases.

Secondly, the primary target problem for each case, identified following assessment by the Psychologist was coded according to the modified EPPIC categories. Obtained data were then subjected to descriptive and inferential analysis.

Discussion sessions were also held with the departmental psychologists to highlight and clarify any potential problems with the departmental audit sheet structure and content.

Results

1. Overall Correspondence Levels

Analysis indicated there was agreement between problem referred and problem targeted on 239 of 401 cases, a correspondence level of 59.60%. This equates to agreement between GP and Psychologist on three fifths of cases. Obtained data were subjected to a test of inter-rater reliability using Kappa Coefficients. This demonstrated agreement levels were fair ($K = 0.46$; cf. 15).

Insert Table 1.2 here

2. The Distribution of Correspondence levels across EPPIC categories

Table 1.2 delineates the relationship between problem referred and problem

targeted. The shaded area denotes correspondence. Percentage agreement levels for each problem area were also computed and are displayed in Figure 1.1. The equation and workings used to calculate these data is displayed in Appendix 1.4. Note that the frequency of cases in each category was not equal.

Highest agreement was seen for eating disorders (68.42%) and for abuse/trauma (60.00%). Moderate agreement was seen for anxiety disorders (54.08%), with one third of referred cases treated for a different problem. Agreement was lower for depression (42.45%), with half of depression referrals treated differently. Agreement was very low for relationship/social problems (21.33%), with half of cases referred for relationship/social difficulties treated differently. Moreover, two thirds of cases treated for relationship/social difficulties were referred with a different problem.

There was no agreement for Personality Disorder, Psychiatric Illness and Addictions, although the frequency of cases allocated to these categories was low (< 5).

Insert Figure 1.1 here

3. Referral Letter Content

During examination of referral letters, themes were recorded and categorised. A very small number of these were relevant to the issue of discrepancies between GPs and Psychologists. In seven instances, GPs alluded to the notion that patients had "underlying problems which they are not able to discuss". In a further two, GPs referred for one problem but noted that input in other areas might be more relevant: "I wonder whether this patient requires input in a different area".

A number of other unrelated themes emerged. There were sixteen instances of the use of "urgent/soon". In twenty seven letters, the GP noted the patient would

"benefit from psychological counselling", whilst in eight cases, the GP noted that "the patient feels they would benefit from psychological input". In a further twenty letters, GPs wrote that the patient was "keen/motivated to make changes".

Discussion

The present study aimed to extend previous findings regarding psychological problems identified and referred by GPs, and those subsequently targeted for treatment by Clinical Psychologists. This was achieved by examining the rate of concordance between primary referred problem (GP) and primary treatment target (Clinical Psychologist) across 401 consecutive GP referrals to a Clinical Psychology Department. A secondary aim was to highlight any shortcomings with departmental recording of new referrals. Both these aims were broadly achieved.

Overall agreement was fair (15) and observed for 59.60% of cases. Analysis across problem categories suggested high agreement for eating disorders, moderate agreement for anxiety and depression, and low agreement for relationship/social problems. In addition, a number of problems with departmental audit sheet procedures (type of disorder, outcome scale, opt-in details) were usefully detected.

Overall agreement levels were lower than that seen in the psychiatric literature (5), although this may be because the present study examined referrals from GPs to Psychologists, rather than from GPs to Psychiatrists. GPs and Psychiatrists share an initially generic medical training and may rely on similar approaches (e.g. ICD-10 or DSM-IV) to conceptualise and categorise mental health problems. Moreover, the present study examined agreement between referral and treatment targets rather than referral and diagnosis. Psychologists may agree with a diagnosis of a particular problem (e.g. depression), but choose to initially target a specific aspect of the problem (e.g. sleep problem).

A number of other factors may have contributed to observed disagreement levels. As noted, GPs may diagnose and refer for depression which, whilst central to a Psychologists formulation, may not in itself become a key target for treatment. In addition, GPs see patients for short consultation times, commonly 6 minutes (16), and must decide on psychological problems based on limited information. Moreover, given such time limits, patients may themselves be unwilling, or unable, to disclose details regarding their personal problems during GP consultations. GPs and Clinical Psychologists experience different training backgrounds and may utilise different assessment approaches. GPs are guided by categorical classification systems whereas Psychologists more often rely on a hypothesis driven, semi-structured interview procedure aimed at developing a case formulation. Again, this may result in the identification of treatment targets quite different to an initial diagnosis. Finally, referral to a specialist service, in this case clinical psychology, necessitates a greater level of detail reported by the patient. In sum, therefore, some disagreement between GP referral and Psychologists treatment targets is quite likely, as previous research has suggested (11,12).

Despite some of the above factors, agreement for certain problem areas was high e.g. eating disorders. All but one of the cases referred for an eating disorder subsequently received treatment for this following assessment. This is perhaps not surprising. Physical and psychological symptoms of eating disorders are specific and tangible (e.g. weight loss, fear of fatness, distorted body image). Such symptoms are therefore more easily observed. A relatively high level of agreement was also seen for the category of abuse/trauma. This may reflect GPs awareness of their patients history and personal background as part of the doctor patient relationship.

Analysis indicated moderate agreement for anxiety, with one third of anxiety referrals subsequently treated for a different problem, most frequently depression or

relationship/social difficulties. This might be consistent with GP over-referral of anxiety (5,6,12), and may be the result of patients reporting secondary anxiety symptoms caused by an underlying primary problem. The present data appear to suggest this may be occurring with respect to relationship difficulties, PTSD, anger and addictions (see Table 1.2). Similarly, approximately half of depression referrals were treated differently following psychological assessment. This also appears consistent with previous findings (e.g. 6,9), and again may be the result of GPs labelling effect (depression) rather than cause (e.g. relationship difficulties).

Finally, analysis indicated that approximately two thirds of cases treated for relationship/social difficulties were initially referred with a different problem. This may relate to different training backgrounds, with GPs more likely to diagnose a medical problem, rather than underlying relationship difficulties. It may also reflect the difficulties of disentangling whether a relationship difficulty is causing a patients symptoms, or vice versa. It is also possible that whilst a patient may have met GP criterion for depression, formulation led the Psychologists to treat underlying dysfunctional relationships hypothesised as exacerbating the problem. This may reflect the orientation of a particular Psychologist e.g. a psychodynamic focus on relationships. Further research is required to clarify these issues.

Completion of the present study demonstrated a clear need to improve departmental recording of patient diagnoses. The system utilised at the time of audit presented clinicians with a limited choice of only six disorder types, not representative of DSM-IV categories. Discussions with departmental psychologists confirmed this, and as a result, the "type of disorder" list has recently been extended to cover DSM-IV categories. This may facilitate more accurate recording of patient diagnoses, and allow for more accurate comparisons of levels of correspondence between referred problem, psychologist diagnosis and psychologist treatment target. Discussions also

highlighted problems with the audit outcome scale (1-5 of improvement) and opt-in information. Changes to recording procedures have been suggested by the auditor, and are to be implemented in the near future.

Further studies examining concordance between GP and Psychologist could also be conducted in a larger department or across a number of departments. This would overcome the limitations of assessing the views of a small group of GPs and Psychologists, as seen in the present study. Further studies may also benefit from examining concordance between treatment targets and secondary/tertiary diagnoses. Many cases currently referred are complex and comprise more than one problem. Therefore, by examining only the primary referred problem, the present data may omit valuable information. In addition, the present study did not examine the influence of time spent on the waiting list, on subsequent agreement levels between GP and Psychologist. Greater agreement might have been observed at a short waiting time, although analysis of this was beyond the scope of the present study. Finally, in the present study, agreement levels across individual GPs were not calculated. Thus, perhaps one or two outliers in the data have distorted findings. Future research could attempt to control for the influence of such extreme data points.

Despite such limitations, the present study appears to replicate findings in the literature regarding the differential treatment by Psychologists of some cases initially referred by GPs for anxiety and depression (5,6,9,12). In addition, by examining correspondence between referral and treatment target, it arguably offers a more direct examination of correspondence between what patients are referred with and how they are subsequently managed. It also outlined some improvements with respect to departmental recording of patient type of disorder, although as noted, these changes did not influence the treatment target data set in any way. Overall agreement levels were encouraging, although low agreement on certain categories (e.g.

relationship/social problems) probably indicates the need for some changes in referral practices at a local level. For instance, it might be useful to offer more detailed information to GPs relevant to the broad domain of problem areas Psychologists work in. As a result of the present study, these proposed changes are currently under review, and the effects of these changes could be monitored using future departmental audit.

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Table 1.1 Inter-rater reliability concordance levels for each type of data allocated to EPPIC Categories

Type of Data	% Concordance between raters
Problem referred (GP)	80.00 %
Problem targeted (Psychologist)	98.75%
Mean concordance	89.38%

Number of cases referred (GP)	Number of cases targeted (Psychologist)																Referred Total	
	A	O	P	D	R	C	An	E	B	Ab	Ph	S	Pd	Pi	Ad	Sd		Ot
Anxiety (A)	106		6	17	16	1	2	1	3	1	1	1			2			157
OCD (O)	2	3																5
PTSD (P)	1		7													1		9
Depression/Mood Disorders (D)	17		1	45	13	3	3		2	1	1	1		1	1		1	89
Relationship/social problems (R)		1	1	4	16	2		1	2	2	2	1			1		1	32
Develop/Cognitive Disorder (C)					1	7	1											9
Anger (An)	2				4		5			1								12
Eating Disorder (E)	3				1			13	1									18
Behavioural/Movement Disorder (B)					1				2									3
Abuse/Trauma (Ab)	2			4	3					24						1		34
Problems related to physical health	1			1	1						5							8
Sexual Disorder (S)	1											5						6
Personality Disorder (Pd)	1			1									0					2
Psychiatric Illness (Pi)				1										0				1
Addictions (Ad)															0			0
Sleep Disorder (Sd)			1													1		2
Other/ Unstated (Ot)	9			2	3												0	0
Targeted Total	145	4	16	75	59	13	11	14	6	30	8	9	1	1	4	3	2	401

Table 1. 2 Relationship between problems referred and problems targeted.

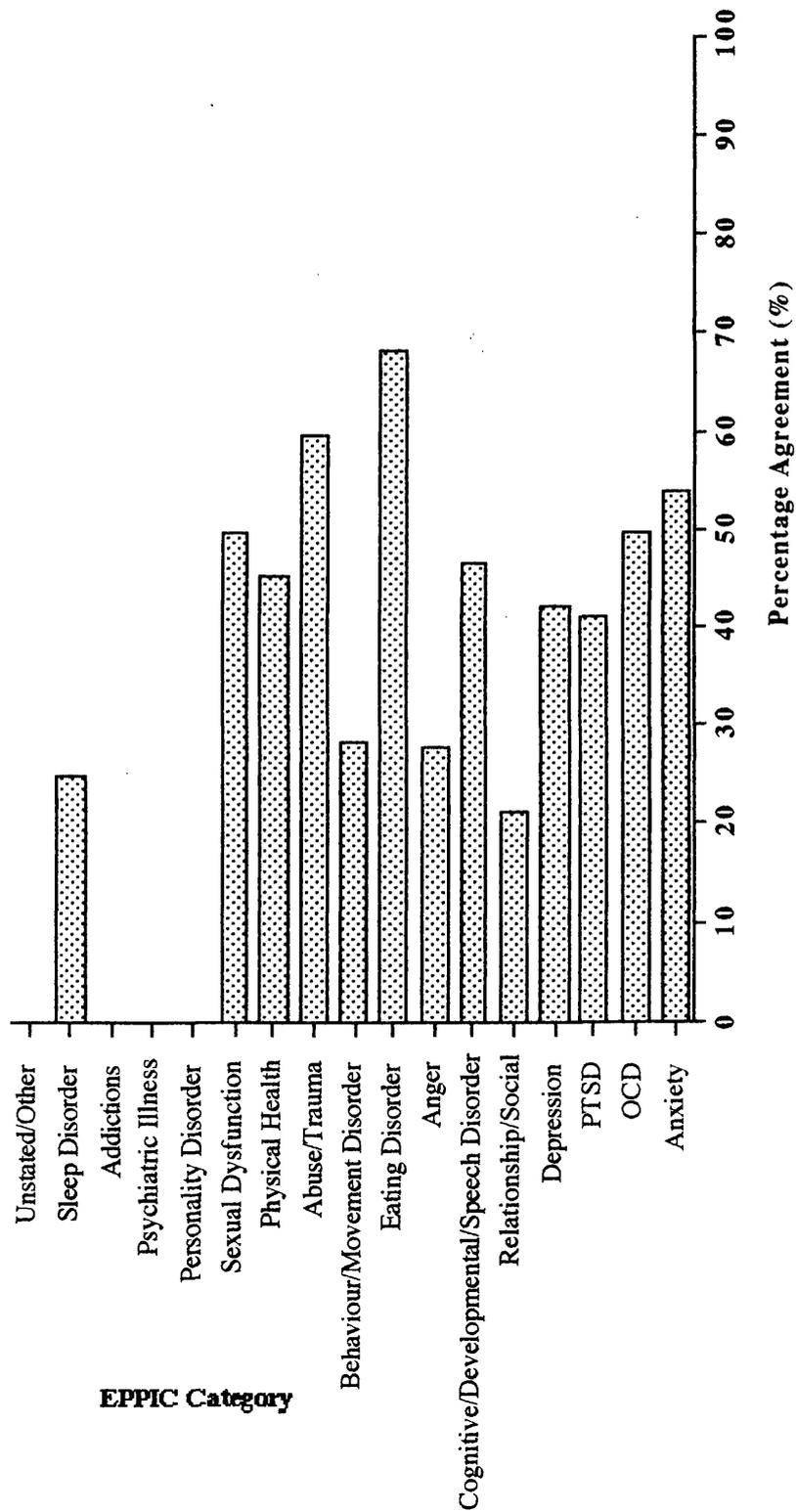


Figure 1.1 Percentage agreement between GP and Clinical Psychologist as a function of EPPIC category

Chapter 2: Literature Review

Initial Insomnia and Paradoxical Intention: A Review of the Literature.

Literature Review submitted in partial fulfilment of the requirements
for the degree of Doctor of Clinical Psychology

Prepared in accordance with requirements for submission to
Behaviour Research & Therapy
(Appendix 3.1)

RUNNING HEAD: Insomnia and paradoxical intention

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Abstract

A review of the paradoxical intention literature with respect to initial insomnia is presented. Initially, evidence for the role of pre-sleep cognitions in initial insomnia is reviewed. Cognitive approaches to insomnia management are then outlined, and paradoxical intention is defined. The performance anxiety model of paradoxical intention is delineated, and the need to clarify this model, empirically, is emphasised. Findings demonstrating the efficacy of paradoxical intention are described, although evidence for some response variability, and the lack of studies employing objective sleep measures, is noted. The need for further research clarifying putative mechanisms and response variability, using both objective and subjective sleep measures, is therefore stressed, and the clinical implications of this research discussed.

Key words: Initial Insomnia, Paradoxical Intention, Performance Anxiety

Introduction

Chronic insomnia is a widespread disorder affecting 9 -12% of the general population (Bixler, Kales, Soldatos, Kales & Healy, 1979; Gallup Organisation, 1991; Mellinger, Balter & Uhlenhuth, 1985), which is characterised by dissatisfaction with sleep duration, efficiency and/or quality (American Sleep Disorders Association, 1997). It is associated with both daytime fatigue, impaired performance, poor physical health and reduced quality of life, and is also a known risk factor for major depressive disorder (Breslau, Roth, Rosenthal & Andreski, 1996; Buysse, Reynolds, Kupfer, Thorpy, Bixler, Manfredi et al, 1994; Ford & Kamerow, 1989; Roth & Ancoli-Israel, 1999). Insomnia is a heterogeneous condition which encompasses a variety of forms. For example, sufferers may experience difficulty initiating sleep, difficulty remaining asleep, or they may awaken too early. The present review focuses on initial or sleep-onset insomnia i.e. difficulty initiating sleep.

Despite its prevalence, the precise mechanisms underlying initial insomnia remain only partially understood (Wicklow & Espie, 2000). This is problematic, particularly given the contraindication of long term pharmacological approaches managing the condition (NIMH, 1984). However, recent research suggests that pre-sleep cognitive activity may mediate the development and maintenance of initial insomnia (e.g. Espie & Wicklow, 2001; Harvey, 2000 a,b). The present paper will review this work, and then examine findings regarding paradoxical intention, a cognitive technique used to treat the condition (e.g. Ascher & Turner, 1979). Suggestions for future research exploring paradoxical intention will also be made.

Initial insomnia and pre-sleep cognitive activity

Recently, evidence has converged supporting a cognitive arousal model of initial insomnia aetiology (Espie & Wicklow, 2001). This proposes that, rather than

physiological hyperarousal (cf. Monroe, 1967), pre-sleep cognitions may be central in the development and maintenance of the disorder. Data supporting this hypothesis has been generated from a number of sources.

Several reports indicate that insomniacs consistently attribute poor sleep to cognitive factors. For example, in their survey of 296 self defined initial insomniacs, Lichstein & Rosenthal (1980) found that cognitive arousal was blamed 10 times more frequently than physiological arousal as the central determinant of sleep difficulty. 55% of insomniacs attributed their disturbed sleep to cognitive arousal, whereas only 5% attributed it to somatic arousal. Consistent with this, both Espie, Brooks & Lindsay (1989) and Harvey (2000a) noted that cognitive items in the Sleep Disturbance Questionnaire (SDQ; e.g. 'My mind keeps turning things over') were most frequently endorsed by initial insomniacs in accounting for their sleeping difficulties.

There is also evidence of a correlation between pre-sleep cognitive activity and sleep-onset latency (SOL). Nicassio, Mendlowitz, Fussel & Petras (1985) found that, relative to somatic Pre Sleep Arousal Scale items (e.g. 'heart racing'), cognitive items (e.g. 'can't shut off thoughts') were highly correlated with length of SOL. Similar data have also been reported by Van Egeren, Haynes, Franzen & Hamilton (1983). However, not all correlational studies support this proposal. Sanavio (1988) observed only a low correlation (0.09) between pre-sleep cognitions and self-report SOL. It therefore remains possible that excessive pre-sleep cognitive activity is merely an epiphenomenon of night-time wakefulness (Freedman & Sattler, 1982; Morin, 1993).

Several studies examining the content of insomniacs pre-sleep thoughts using questionnaire measures also suggest sleep onset difficulties may be linked to cognition. Coyle & Watts (1991) factor analysed 92 'sleep disturbed' participants responses to both the SDQ, and a second questionnaire assessing sleep-onset difficulties. Two of their resulting factors specifically related to cognition: sleep attitudes (reflecting concerns

about not sleeping), and mental anxiety (reflecting elevated general cognitive activity). Using a similar methodology, Watts, Coyle & East (1994) explored pre-sleep cognitions in 38 insomniacs and 41 non-insomniacs, separated into worrier and non-worrier groupings. Factor analysis revealed 6 thought categories linked to initial insomnia: mental activity and rehearsal, thoughts about sleep, family and long time concerns, somatic preoccupation, work and recent concerns, and positive concerns and plans. Further analysis indicated insomnia was particularly associated with thoughts about sleep. Finally, Harvey (2000a) has shown that, relative to good sleepers (N = 30), insomniacs (N = 30) pre-sleep cognitions are more intrusive, and focused particularly with not sleeping, and with recent concerns. Other data also show that initial insomniacs employ strategies of thought suppression and reappraisal to control these negative pre-sleep cognitions (Harvey, 2001), suggesting metacognitive beliefs about the significance of sleep-related intrusions may play a part in insomnia maintenance (Wells, 2001).

Other researchers have directly sampled the content of insomniacs pre-sleep cognitions. Van Egeren et al (1983) recorded the pre-sleep cognitions of 34 undergraduates with initial insomnia who spent five nights in a sleep laboratory. Regression analysis indicated that cognitions during the pre-sleep phase, particularly thoughts about not falling asleep, were significantly associated with subjective, although not objective (EEG), SOL. Kuisk, Bertelson & Walsh (1989) sampled participant pre-sleep cognitions by interviewing normal sleepers, objective insomniacs and subjective insomniacs (each N = 8) at 4 minute intervals during the pre-sleep phase. Their analysis indicated that both subjective and objective insomniac groups experienced more negative pre-sleep thoughts, relative to good sleepers.

Finally, Wicklow & Espie (2000) used voice activated tape recorders to sample the pre-sleep cognitions of 21 initial insomniacs. Content analysis of recordings revealed eight categories of pre-sleep intrusion: general problem solving, sleep and its

consequences, reflection on quality of thoughts, arousal status, external noise, autonomic experiences, procedural factors and rising from bed. Wrist actigraphy was utilised as an objective measure of SOL, and correlational analysis of sleep data with intrusion categories demonstrated that the anticipated consequences of poor sleep, and general problem solving, were the strongest predictors of objective SOL.

Overall, the above studies clearly demonstrate an association between pre-sleep cognitive activity and sleep-onset difficulties. Data indicate that initial insomniacs pre-sleep cognitions are typically intrusive, negative in affect, and relate to both recent problems, and the sleep process itself (cf. Borkovec, 1982).

It is important to note though that these findings are correlational, and therefore do not directly link pre-sleep cognitive activity with initial insomnia. However, direct evidence for this association has been provided by six studies which manipulated pre-sleep cognitions using experimental methods. Four of these studies utilised good sleepers as participants. Gross & Borkovec (1982) demonstrated that increasing the likelihood of pre-sleep cognitions experimentally increases SOL amongst good sleepers. They informed one group of participants they were required to give a speech pertaining to a given topic ('speech plus topic') following an afternoon nap. 'Sleep only' controls were simply required to go to sleep. Data analysis based on both objective (EEG criteria) and self report measures indicated that, relative to controls, 'speech plus topic' participants displayed significantly longer objective SOL and shorter sleep duration (EEG). Similar data have also been reported by Hall, Buysse, Reynolds, Kupfer & Baum (1996). In their study, good sleepers told they were to deliver a 15 minute speech following a nights sleep displayed more stress-related intrusive thoughts, and significantly longer objective SOL, relative to controls. Similarly, Haynes, Adams & Franzen (1981) demonstrated that difficult mental arithmetic tasks completed during the pre-sleep period significantly increased objective (EEG) and subjective SOL amongst 11 good sleepers. Taken

together, these findings offer more direct evidence that elevated pre-sleep cognition leads to delayed sleep-onset (cf. Espie & Wicklow, 2001).

Ansfield, Wegner & Bowser (1996) have also reported data which support this. They allocated 83 good sleepers to one of four experimental conditions aimed at testing Wegner's theory of ironic mental control (Wegner, 1994). Wegner's model suggests that under certain conditions, the desire to control a particular mental state (e.g. sleep, mood) can yield the ironic opposite of what is desired. Consistent with the model, Ansfield et al's data indicated that good sleepers instructed to sleep quickly whilst listening to marching music took the longest to fall asleep. They concluded that sleep urgency under conditions of high cognitive load may result in prolonged sleep-onset latency. Besides implicating the central role of pre-sleep thoughts in prolonging sleep-onset, these data also suggest that treatments which encourage insomniacs to minimise sleep effort and urgency (e.g. paradoxical intention; Ascher & Turner, 1979) under conditions of high cognitive load (e.g. sleep anxiety) may prove highly effective in reducing SOL.

Two further studies in the sleep literature experimentally manipulated the pre-sleep cognitions of poor sleepers. Harvey (2000b) explored the effects of suppressing pre-sleep cognitive activity on SOL. A cohort of 30 insomniacs and 30 good sleepers were instructed to suppress the thought most likely to dominate their pre-sleep thinking (suppress condition) or to think about anything (non-suppression condition). Results indicated that 'suppress' participants showed longer subjective SOL and poorer sleep quality, regardless of whether they were insomniac or not. These data further implicate the role of cognition in initial insomnia, and suggest thought suppression, whilst used to turn off pre-sleep thinking, can have an opposite 'ironic' effect, fuelling cognitive activity and preventing sleep-onset (cf. Wegner, 1994). Finally, Haynes et al (1981) demonstrated in their study outlined above, that exposure to stressful mental arithmetic tasks decreased the objective and subjective SOL of 11 poor sleepers. These data may

also be consistent with a cognitive arousal model of initial insomnia. They suggest that the stressful arithmetic tasks had a distracting effect on cognition, disrupting the usual pre-sleep thoughts of the poor sleepers, thereby hastening sleep-onset.

Taken together therefore, the above data indicate that experimental manipulation of pre-sleep cognitions can directly impact sleep-onset. Accordingly, they extend the correlational data outlined above, and offer the strongest and most direct support for a cognitive arousal model of initial insomnia (cf. Espie & Wicklow, 2001).

The relevance of metacognitive models of emotional disorder to initial insomnia

Metacognitive models of the anxiety disorders suggest that beliefs, processes and strategies which appraise, monitor or control cognition may be central in the aetiology of emotional disorder (Wells, 1997; 2001). As outlined above, considerable evidence indicates that insomniacs show marked levels of sleep-related intrusions. Given this, and the recent finding that insomniacs employ metacognitive thought control strategies to minimise sleep-related intrusions (Harvey, 2000b), it seems likely that metacognitive models of emotional disorder (see e.g. Wells, 1997; 2001) may offer a relevant theoretical framework for the study of insomnia. Specifically, the model assumes that anxiety incubates intrusions (Wells & Papageorgiou, 1995), and that intrusions act to trigger negative metacognitive beliefs regarding their meaning and significance (Wells, 1997; 2001). Thus, cognitive treatments such as paradoxical intention, which target sleep anxiety and lower sleep effort (Ascher & Turner, 1979), may not only reduce the frequency of sleep-related intrusions, but also lower activation of negative metacognitive beliefs (e.g. "Thinking about sleep makes my sleep worse"; "Thinking about sleep means I'm a poor sleeper"), which may characterise poor sleepers (cf. Harvey, 2000b). In turn, by reducing activation of negative sleep-related metacognitive beliefs, paradoxical intention may reduce poor sleepers conviction in their declarative beliefs regarding sleep

("I'm a poor sleeper"), and thus alter their tendency to overestimate sleep-latency (Borkovec, 1982). There is growing experimental evidence of the links between cognitive process, intrusions and metacognition in the anxiety literature (Wells, 2001), and in accord with this, future insomnia research programmes should take account of the influence of cognitive insomnia treatments on both pre-sleep intrusions and related metacognitive belief structures. There remains, at present, a lack of clinical and research data linking metacognitive constructs to disturbed sleep, although its conceptual relevance to insomnia is clear.

A cognitive arousal model of initial insomnia

As is evident, there is now considerable evidence suggesting that pre-sleep cognitive activity mediates sleep-onset difficulties. Consequently, recent formulations of initial insomnia aetiology increasingly emphasise the role of cognitive variables in the development and maintenance of the condition.

For instance, in his multi-component model of insomnia aetiology, Espie (1991) highlights the central role of cognitive arousal in promoting and maintaining the condition. Similarly, Morin (1993) argues that a range of cognitive variables, including excessive cognitive activation, worry over sleep loss, effort to sleep, and beliefs that insomnia is uncontrollable and unpredictable, together act to bring on and maintain cognitive hyperarousal and insomnia. Both Harvey (2000 a,b) and Lundh (1998) have also suggested that pre-sleep cognitive activity, dysfunctional beliefs, and anxiety and worry about insufficient sleep are central in mediating initial insomnia. Indeed, research has shown that when insomniacs and good sleepers are awoken from Stage 2 sleep, insomniacs report being drowsy but thinking, whereas good sleepers report being asleep (Borkovec, Lane & VanOot, 1981).

Consistent with these accounts, Espie & Wicklow (2001) have recently argued

that due to cognitive overarousal, insomniacs are not 'mentally well prepared' for bed. Their model predicts that good sleep should occur if individuals can achieve pre-sleep cognitive dearousal, comprising four desired states. First, a state of minimal information processing, where acute mental activity is attenuated; second, a state of minimal cognitive drive, where consideration of important matters and problem solving is minimised; third, a state of minimal effort, where effort to sleep and sleep urgency are attenuated, and fourth, a state of minimal affective load, where concern and anxiety relating to sleep, and the feared consequences of sleep loss, are minimised.

Cognitive treatments for initial insomnia

Based on these models, there has recently been a marked increase in the use of cognitive approaches in the clinical management of initial insomnia (Lundh, 1998; Morin, 1993). Thought stopping and articulatory suppression are cognitive treatments which focus on blocking pre-sleep intrusive thinking (Levey, Aldaz, Watts & Coyle, 1991), whilst cognitive control attempts to prevent pre-sleep intrusions by scheduling time earlier in the day for worry (Espie & Lindsay, 1987). Paradoxical intention is thought to obviate performance anxiety and catastrophisation about sleep loss by requiring that patients attempt to remain awake (Espie & Lindsay, 1985). Finally, cognitive restructuring targets the content of sleep related cognitions, aiming to alter faulty expectations, attributions, beliefs and appraisals about insomnia (Morin, 1993).

Most of these cognitive techniques tend to be subsumed within a multi-faceted cognitive-behavioural therapy (CBT) package (cf. Morin, 1993). Preliminary CBT outcome data are highly promising, indicating that primary insomniacs treated with CBT show significant improvements on a range of sleep measures, relative to those receiving placebo treatments (Edinger, Radke, Wohlgemuth, Marsh & Quillan, 1997; Morin, Collechi, Stone, Sood & Brink, 1995). There is also evidence that items in the

Dysfunctional Beliefs about Sleep Scale (DBAS-10) are sensitive to change following CBT treatments (Espie, Inglis, Harvey & Tessier, 2000). Unfortunately, the adoption of such integrative approaches has meant that few studies in the sleep literature provide any direct evidence of the utility of individual cognitive components subsumed within CBT treatment packages (Espie, 1991, Edinger & Wohlgemuth, 1999). However, one exception to this has been the clinical application of paradoxical intention.

Paradoxical Intention: A definition

The technique of paradoxical intention was originally developed by Frankl (1955) within the context of logotherapy. It has been widely utilised in the clinical treatment of a range of problems where performance anxiety has developed, including initial insomnia, urinary retention and orgasmic dysfunction (Ascher, 1979; Seltzer, 1986; Strong, 1984). The technique instructs patients to "intend" behaviour which is apparently in opposition to their goal. With respect to initial insomnia, paradoxical intention requires that insomniacs give up trying to sleep, and attempt instead to remain awake for as long as possible (Espie & Lindsay, 1985). Paradoxically, therefore, the likelihood of staying awake is reduced by encouragement to do so.

Paradoxical Intention: A performance anxiety model

Paradoxical intention is thought to operate by eliminating voluntary efforts to sleep, therefore minimising performance anxiety, an aroused state incompatible with sleep (Ascher & Turner, 1979). Insomniacs thus may inadvertently maintain their problem by trying to control the sleep process. Since sleep is a member of a class of behaviours which cannot be placed under full voluntary control, efforts to control sleep-onset fail, resulting in performance anxiety and, therefore, continued sleep disruption. Paradoxical intention appears to help by directing insomniacs to give up voluntary efforts to sleep

which maintain their anxiety, thereby reducing arousal levels and promoting sleep (Ascher & Turner, 1979). Consistent with this model, good sleepers report they do nothing to fall asleep; good sleep is thus associated with minimal effort- an absence of performance effort, rather than performance success (Espie & Wicklow, 2001).

In terms of cognitive mechanisms of action, paradoxical intention acts as a de-catastrophising technique (Espie & Wicklow, 2001). The treatment requires insomniacs give up sleep effort and attempt instead to remain awake. It therefore decatastrophises anxious thoughts about staying awake initially by focusing on and enhancing these (habituation), and subsequently by appraising them via rationalisation and experience. As a result, cognitive arousal levels are reduced, and sleep is promoted.

Recently, authors have framed the performance anxiety model of paradoxical intention in terms of Wegner's theory of ironic control (Wegner, 1994), with performance anxiety thought to constitute Wegner's 'cognitive load'. Consistent with this, a number of findings demonstrate that urgent attempts to sleep under circumstances of high cognitive load result in ironic wakefulness (e.g. Ansfield, Wegner & Bowser, 1996; Gross & Borkovec, 1982).

However, at present, the performance anxiety model of paradoxical intention remains largely untested. There is some evidence that paradoxical intention is more effective when used to treat individuals high in performance anxiety (Ascher & Schotte, 1999), although these data come from a study of social phobics. Only one insomnia study has measured performance anxiety pre and post paradoxical intention in order to test Ascher & Turner's model directly. Fogle & Dyall (1983) allocated 22 chronic insomniacs to a 'give up trying' to sleep condition framed as a paradoxical sleep improvement method, or to a 'try giving up' treatment presented as a way of improving night-time comfort. A further 11 insomniacs received a control treatment (self monitoring). Results indicated that relative to controls, performance anxiety reduced

significantly amongst both treatments groups. However, the authors failed to collect SOL data, nor did they measure effort to sleep. Moreover, analyses indicated all participants, including controls, improved on a subjective sleep efficiency measure. Thus, no firm conclusions regarding Ascher & Turner's performance anxiety model may be drawn. The theoretical link between paradoxical intention, reduced effort to sleep, and reduced performance anxiety in initial insomnia, therefore requires clarification (Espie & Lindsay, 1985).

It should also be noted that there is a marked lack of paradoxical intention research utilising objective sleep measures (Ott, Levine & Ascher, 1983). This is problematic, particularly given that insomniacs' self-report sleep data can be unreliable (Carskadon, Dement, Mitler, Guilleminault, Zarcone & Spiegel, 1976). Objective and subjective sleep measures may also reflect differing response systems (Wicklow & Espie, 2000).

A reliable and minimally intrusive method of measuring sleep data objectively is the actigraph- a small wrist attachment containing internal motion sensors which record the wearers movements. There is increasing recognition that movement is a good predictor of wakefulness, whilst lack of movement is a good predictor of sleep (American Sleep Disorders Association, 1995). Moreover, actigraphic measures have been found to correlate highly ($r = 0.80 - 0.95$) with polysomnographic (PSG) data for global measures of sleep duration and total wake time (e.g. Sadeh, Hauri, Kripke & Lavie, 1995; Mullaney, Kripke & Messin, 1980), although lower rates of agreement have also been reported (Blood, Sack, Percy & Pen, 1995; Hauri & Wisbey, 1992). An examination of paradoxical intention and insomnia using actigraphy would therefore be desirable.

Paradoxical Intention: Evidence of efficacy

The application of paradoxical intention to initial insomnia has received some

research attention. A number of clinical case studies have demonstrated the utility of the procedure in reducing insomniacs SOL (Ascher, 1975; Ascher & Efran, 1978; Espie & Lindsay, 1985; Relinger, Borstein & Mungus, 1978; Relinger & Bornstein, 1979).

Following the earliest case reports, a series of experimental studies were conducted comparing alternative methods of paradoxical intention delivery. Two approaches have dominated the literature. Reframing is based on traditional methods for delivering paradoxical directives, and involves instructing patients to stay awake in order to collect data about their thoughts. Patients are told this information will be used to develop an individualised therapeutic program (e.g. Relinger et al, 1978). In contrast, a second approach involves providing the patient with a straightforward description of paradoxical intention, followed by an outline of how the technique may help reduce SOL (e.g. Ascher & Turner, 1979). A series of studies suggest that, in experimental settings, where participants are randomly allocated to condition, the latter method of delivery is consistently superior in alleviating sleep-onset insomnia (Ascher & Turner, 1980; Ott et al, 1983).

A number of randomised-controlled trials examining the efficacy of paradoxical intention have also been reported. Findings demonstrate the superiority of paradoxical intention over control conditions in reducing SOL (Ascher & Turner, 1979, 1980), and its equivalence to other active treatments (stimulus control, progressive relaxation) in ameliorating initial insomnia (Espie, Lindsay, Brooks Hood & Turvey, 1989; Ladocuer & Gros-Loius, 1986; Turner & Ascher, 1979). Indeed, paradoxical intention is now regarded as a "probably efficacious" insomnia treatment according to American Psychological Association criteria (Chesson, Anderson, Littner, Davila, & Hartse, 1999; Morin, Hauri, Espie, Spielman, Buysse & Bootzin, 1999).

Paradoxical Intention: Evidence of response variability

However, despite Morin et al's (1999) recent conclusions, other research findings have been less positive. For instance, data reported by Lacks, Berteson, Gans & Kunkel (1983) and Turner & Ascher (1982) failed to demonstrate any significant differences between paradoxical intention and control conditions. Other authors have highlighted considerable variability in therapeutic response following paradoxical intention, with some insomniacs actually worsening as a result of its application (Espie et al, 1989; Lacks et al, 1983).

Espie & Lindsay (1985) presented a series of six case studies of insomniacs treated as part of a larger study using paradoxical intention. Of these, three responded promptly to paradoxical intention, maintaining improvements at 3 month follow up. However, three individuals showed increased SOL, two of whom had to discontinue paradoxical treatment due to the extent of their sleep latency exacerbation. Interestingly, subsequent successful treatment using progressive relaxation suggested they were not merely poor treatment responders.

The reasons for such response variability remain unclear (Morin, 1993). However, in attempting to account for this, Espie & Lindsay (1985) have suggested that some patients may follow paradoxical instructions ("stay awake") and/or counterdemand instructions ("do not expect to see any improvement for 2 weeks of the study") literally. Counterdemand instructions are routinely included in paradoxical intention studies to control for demand characteristics and patient outcome expectations. Certainly, future research is needed to examine the extent to which certain patients may worsen as a result of adhering to such paradoxical instructions literally. Moreover, there is also a need to explore the effect of including and excluding counterdemand instructions on therapeutic response within a paradoxical intention paradigm.

Espie & Lindsay (1985) also suggest that paradoxical intention may only be suitable for individuals high in effort to sleep and performance anxiety (cf. Ascher &

Turner, 1979). As noted, it is assumed that paradoxical intention operates to reduce SOL by alleviating effort to sleep and obviating performance anxiety, and as outlined above, there is tentative evidence for this proposal from the insomnia (Fogle & Dyall, 1983) and clinical treatment literature's (Ascher & Schotte, 1999). Thus, non-responders may be unsuited to paradoxical intention due to low levels of performance anxiety and effort to sleep. An initial empirical examination of changes in effort to sleep and performance anxiety amongst insomniacs exposed to paradoxical intention would resolve this, and clarify the performance anxiety model of paradoxical intention. Subsequent research could then allocate individuals to insomnia treatment programmes including or excluding paradoxical intention, based on pre-morbid effort to sleep and performance anxiety measures. It has also been suggested that paradoxical intention may not be suitable for individuals who show severe pre-sleep cognitive activity (Espie & Lindsay, 1985). Research is therefore required to clarify this issue. Moreover, future studies should also examine whether response variability relates to the severity of sleep disturbance (Espie & Lindsay, 1985).

Paradoxical intention is an empirically-supported psychological treatment for insomnia (Morin et al, 1999). However, for a significant minority of patients it may result, at least initially, in exacerbated sleep disturbance (Espie & Lindsay, 1985; Lacks et al, 1983). There is therefore a clear need for experimental research using objective sleep measures (Ott et al, 1983) in order to clarify both the theoretical rationale, and underlying mechanisms, of paradoxical intention (Ascher & Turner, 1979). In turn, such work should determine the utility of paradoxical intention as a general treatment approach (Espie & Lindsay, 1985).

Summary and conclusions

Recently, evidence has converged supporting a cognitive arousal model of initial

insomnia (Espie & Wicklow, 2001). Accordingly, in recent years, cognitive treatments for the condition have become increasingly popular. One widely studied cognitive technique in treating initial insomnia is paradoxical intention - a treatment approach thought to operate by eliminating effort to sleep, and therefore reducing sleep related performance anxiety (Ascher & Turner, 1979). However, this proposal remains largely untested. Moreover, some response variability following paradoxical intention has been reported, the reasons for which remain largely unclear (Espie & Lindsay, 1985).

Research is therefore required to clarify the performance anxiety model of paradoxical intention, and to explore further whether some patients exposed to the treatment follow instructions to stay awake literally (Espie & Lindsay, 1985). There is also a need to clarify the association between severity of both sleep-onset insomnia, pre-sleep cognitive activity and therapeutic response. Finally, an examination of paradoxical intention and insomnia using objective actigraphic methods is required. Together, such research should prove useful in clarifying the utility of paradoxical intention as a general treatment approach for initial insomnia.

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Chapter 3: Proposal for Major Research Project

Initial insomnia and Paradoxical Intention: An experimental investigation of putative mechanisms using subjective and actigraphic measurement of sleep.

Major Research Proposal submitted in partial fulfilment of the requirements for the degree of Doctor of Clinical Psychology

Prepared in accordance with D.Clin.Psy. course guidelines

(Appendix 2.1)

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1.2 Title

Initial insomnia and paradoxical intention: an experimental investigation using actigraphic measurement of sleep.

1.3 Summary

In recent years, cognitive treatments for initial insomnia have become increasingly popular (Espie & Wicklow, 2001). One widely studied cognitive technique for initial insomnia is paradoxical intention. Paradoxical intention is thought to alleviate insomnia by eliminating effort to sleep, and therefore reducing sleep related performance anxiety (Ascher & Turner, 1979a). However, this proposal remains largely untested (Fogle & Dyall, 1983). Moreover, whilst outcome studies from clinical case reports and experimental studies suggest the technique can be very useful (Ascher & Turner, 1979a), some response variability has also been reported. The reasons for this response variability remain largely unclear, but may relate to literal interpretation of counterdemand instructions (Espie & Lindsay, 1985).

The present study will therefore examine experimentally, using objective measures of sleep, the extent to which paradoxical intention can alleviate sleep difficulties in a cohort of initial insomniacs. In addition, by recording levels of sleep related performance anxiety pre and post intervention, the study will also explore the performance anxiety model of paradoxical intention.

A cohort of 38 chronic initial insomniacs will be randomly assigned to one of two

conditions (paradoxical intention ; control). A range of objective and subjective measures of sleep, effort to sleep, and performance anxiety will be recorded at baseline, intervention and post-intervention phases. Subsequent analyses of variance will examine the effects of paradoxical intention on participants sleep patterns and performance anxiety levels. Results will be discussed in terms of the performance anxiety model of paradoxical intention (Ascher & Turner, 1979a), and Wegner's model of ironic cognitive control (Wegner, 1994).

Obtained data will have important implications for the clinical application of paradoxical intention, both in terms of its utility as a general treatment approach, and its predicted suitability particularly for individuals high in performance anxiety and effort to sleep (Espie & Wicklow, 2001).

1.4 Introduction

Recently, evidence has converged that pre-sleep cognitive activity may be an important determinant of initial insomnia aetiology (Espie, 1991). Accordingly, current models of insomnia posit the role of cognitive factors in the development and maintenance of the condition (Espie & Wicklow, 1999; Morin, 1993). Furthermore, insomnia treatment programmes now routinely emphasise cognitive techniques in the treatment of the condition. However, these are almost always incorporated in a CBT "package", with few studies directly using cognitive techniques alone (Espie, 1991).

One exception has been the clinical application of paradoxical intention. Originally labelled by Frankl (1955), the technique instructs clients to "intend" behaviour which is apparently in opposition to their goal. With respect to sleep difficulties, paradoxical intention requires that insomniacs give up trying to sleep, and attempt, instead, to remain awake for as long as possible (Ott, Levine & Ascher, 1983). Paradoxically, therefore, the likelihood of staying awake is reduced by encouragement to

do so.

In terms of mechanisms of action, paradoxical intention is thought to operate by eliminating voluntary efforts to sleep, therefore minimising performance anxiety, an aroused state incompatible with sleep (Ascher & Turner, 1979a). Insomniacs thus may inadvertently maintain their problem by trying to control the sleep process. Since sleep is a member of a class of behaviours which cannot be placed under full voluntary control, efforts to control sleep-onset fail, resulting in performance anxiety and, therefore, continued sleep disruption. Paradoxical intention appears to help by directing insomniacs to give up voluntary efforts to sleep which maintain their anxiety, thereby reducing arousal levels and promoting sleep (Ascher & Turner, 1979). Consistent with this model, good sleepers report they do nothing to fall asleep; good sleep is thus associated with minimal effort- an absence of performance effort, rather than performance success (Espie & Wicklow, 2001). More recently, certain authors have framed this performance anxiety model in terms of Wegner's theory of ironic control, with performance anxiety thought to constitute Wegner's 'cognitive load' (Ansfield, Wegner & Bowser, 1996; Ascher & Schotte, 1999).

However, at present, the performance anxiety model of paradoxical intention remains largely untested. There is some evidence that paradoxical intention is more effective when used to treat individuals high in performance anxiety (Ascher & Schotte, 1999), although these data come from a study of social phobics. Only one insomnia study has recorded performance anxiety data pre and post paradoxical intervention. Fogle & Dyall (1983) noted that performance anxiety reduced significantly amongst insomniacs following paradoxical but not control instructions. However, no data for sleep-onset latency (SOL) were collected, and since both paradox and control participants improved on a subjective sleep efficiency measure, no firm conclusions regarding the performance anxiety model of paradox can be drawn. Clearly therefore, the theoretical link between paradoxical intention, reduced effort to sleep, and reduced performance

anxiety merits clarification. It has also been suggested that paradoxical intention may only be suited to individuals high in performance anxiety and effort to sleep (Espie & Lindsay, 1985). This issue also merits experimental clarification, particularly given its relevance in determining the utility of paradoxical intention as a general treatment approach.

Moreover, there is a lack of research in insomnia and paradoxical intention utilising objective measures of sleep (Ott et al, 1983). This is problematic, particularly given that self report sleep data may have limited reliability and validity (Carskadon, Dement, Mitler, Guilleminault, Zarcone & Spiegel, 1976). A closer examination of paradoxical intention and insomnia using objective sleep data would therefore be desirable.

Evidence regarding the efficacy of paradoxical intention is somewhat mixed. Findings from a number of clinical case studies indicate its superiority over control conditions in alleviating insomnia (Ascher, 1975; Ascher & Efran, 1978; Relinger & Borstein, 1979). Moreover, experimental studies of paradoxical intention, where patients are randomly allocated to treatment conditions, indicate its superiority over placebo control (Ascher & Turner, 1979a), and suggest it matches the efficacy of other active treatments (stimulus control, relaxation) in alleviating insomnia (Espie, Lindsay, Brooks, Hood & Turvey, 1989; Turner & Ascher, 1979). However, other studies have revealed some response variability, with certain insomniacs worsening following its application (Espie & Lindsay, 1985).

Counterdemand instructions, where participants are told not to expect immediate sleep improvements (Steinmark & Borkovec, 1974), have been used in a number of outcome studies comparing paradoxical intention with other insomnia treatments (e.g. Turner & Ascher, 1979; Espie et al, 1989). Such instructions are included to eliminate demand characteristics and placebo effects. It has been suggested that response variability following paradoxical intention may be due to participants interpreting these

instructions literally, although this is unclear (Espie & Lindsay, 1985). An experimental examination is therefore necessary to clarify to what extent non-responding may relate to misinterpretation of counterdemand instructions. In turn, this will further ascertain to what extent paradoxical intention may be suitable as a general treatment approach for initial insomnia.

1.5 Aims and hypotheses

Primary aims:

1. To examine, experimentally, the utility of paradoxical intention in reducing objective (wrist actigraphy) and subjective (sleep diary) SOL and sleep efficiency in chronic initial insomniacs.
2. To examine the effect of paradoxical intention on self-reported performance anxiety and effort to sleep, thus exploring, empirically, the performance anxiety model of insomnia and paradox.
3. To examine differential therapeutic response due to the presence/absence of counterdemand instructions.

Hypotheses are as follows:

1. Insomniacs allocated to the paradoxical intention condition will, relative to controls, show a reduction in objective and subjective SOL, and an increase in objective and subjective sleep efficiency.
2. Insomniacs allocated to paradoxical intention will, relative to controls, show a reduction in sleep related performance anxiety and effort to sleep.
3. Insomniacs who do not receive counterdemand instructions may respond significantly better to paradoxical intention in terms of objective and subjective SOL and sleep efficiency.

1.6 Plan of investigation

1.6.1 Participants

Following previous research, a cohort of 38 chronic initial insomniacs, each randomly allocated to one of two conditions (Paradoxical Intention; Control), will be run. Power calculations across the three primary outcome variables (Objective SOL, Subjective SOL and Sleep related performance anxiety) were run using the UCLA power calculator. Mean and standard deviation data were drawn from Ott, Levine & Ascher (1983) and Fogle & Dyall (1983), two recent similar studies. Significance level was set at $p < 0.05$, power = 0.8. Calculations indicated the need for between 15 - 20 participants in each group (n = 15 for Objective SOL; n= 19 for Subjective SOL; n = 14 for Performance Anxiety).

Participant inclusion and exclusion criteria will be as standard for identification of sleep-onset insomniacs (cf. Wicklow & Espie, 2000) i.e. age between 16 and 65 years, clinically significant sleep problems cf. ICSD (American Sleep Disorder Association, 1997) = > 30 mins. to fall asleep at least 4 nights per week, and scores in excess of 5 on the Pittsburgh Sleep Quality Index (Buysse, Reynolds, Monk et al, 1989; see Appendix 2.2). Exclusion will be based on concurrent treatment, complicating medical conditions, psychopathological disorders, etc.

Possible participants will be identified using email, posters in the university and local hospitals, and via the university staff newsletter. These methods have been utilised successfully by previous researchers in this department.

Participation will require attendance at an initial screening interview, an instructions interview and a feedback interview. Each participant will take 3 weeks to run, and all participants will receive both the paradoxical intention treatment (whether during the study or after), and general sleep hygiene education at the end of the study.

1.6.2 Measures:

Participants will complete several intake questionnaires prior to participation (e.g. Pittsburgh Sleep Quality Index- Buysse et al, 1989- see Appendix 2.2; Sleep History Questionnaire- Morin, 1993- see Appendix 2.3). A measure of depression (Beck Depression Inventory; Beck, Steer & Brown, 1996) will be utilised to exclude participants (> 20 points), whilst a measure of anxiety (Penn State Worry Questionnaire; Meyer, Miller, Metzger & Borkovec, 1990) will also be completed pre-baseline. Participants will, following allocation to experimental condition, complete a credibility rating sheet to assess their perception of the credibility of instructions given. Self-report data regarding participant adherence to the experimental instructions, and paradoxical intention participants understanding of the treatment given, will also be collected at the end of the study.

Objective sleep data will be collected using actigraphic methods (cf. Wicklow & Espie, 2000). The actigraph is a small wrist attachment containing internal motion sensors which record the wearers movements. There is recognition that movement is a good predictor of wakefulness, whilst lack of movement is a good predictor of sleep (American Sleep Disorders Association, 1995). Wrist actigraphic recordings will be made using the "Actiwatch" (Model AW2 Registered Cambridge Neurotechnology Ltd).

Two sleep variables will be recorded:

Sleep-onset latency (minutes)

Sleep efficiency (%)

Subjective sleep data will be recorded via sleep diaries (Espie, 1991), completed each morning during the study. Measures will include:

Estimated sleep-onset latency

Estimated sleep efficiency (%)

Restedness rating upon awakening (6 point Likert scale)

Sleep related performance anxiety will be assessed at Baseline, Weeks One and Two via the scale developed and utilised by Fogle & Dyll (1983). Some internal consistency data have been reported for the scale (see Fogle & Dyll , 1983, p. 26). A second scale may also be developed and piloted by the authors prior to commencement of the study, and then additionally employed to measure sleep anxiety.

1.6.3 Design and procedure

Following Ott et al (1983), the experimental design will integrate one Between participants factor (Instructional Set: Paradoxical Intention; Control) and one Within participants factor (Time: baseline; Treatment Week One; Treatment Week Two). A series of repeated measures ANOVAs will note effects of manipulation of these independent variables on a range of dependent measures of sleep (objective and subjective), performance anxiety, and effort to sleep.

Sleep data will be analysed in terms of weekly averages (cf. Ott et al, 1983). Thus, baseline data (all sleep measures, performance anxiety and effort to sleep) will be collected for a week and averaged. Two 'treatment' weeks of data will then be collated and meaned across 'Treatment' Week One and 'Treatment' Week Two. Instructional sets will be delivered on a piece of paper to participants following their baseline week at a meeting with the researcher. Participants will be invited to ask any questions regarding what is required of them. These will be answered in as standardised a fashion as is possible.

Half of the paradoxical intention participants will receive counterdemand and positive demand instructions as standard ("Do not expect to see any improvement during Week 1 of the experiment, but by the beginning of Week 2 you will begin to see dramatic improvements"; cf. Steinmark & Borkovec, 1974), whilst half will only receive

positive demand characteristics ("The above instructions should have an immediate effect on your sleep"). This will clarify whether the non-responding reported in the literature may relate to participants misinterpreting counterdemand instructions as a literal demand to show no improvement.

1.6.4 Settings and equipment

All equipment will be obtained within the Department of Psychological Medicine at Glasgow University. This includes actigraph equipment, questionnaires, self report measures and all necessary computing resources.

1.6.5 Data analysis

Data analysis will be conducted using SuperAnova and SPSS. Measures of sleep (subjective and objective) and performance anxiety will be scored and analysed using both descriptive and inferential (ANOVA) methods. Any necessary data transformations will be completed prior to inferential analyses.

1.7 Practical Applications

The study has important implications for the clinical application of paradoxical intention to initial insomniacs. It is clear from the literature that not all individuals benefit from the intervention. Demonstration, experimentally, of the mechanisms of action of paradoxical intention i.e. reduction of performance anxiety and effort to sleep, will have implications for identifying which individuals (e.g. those high in performance anxiety and sleep effort pre-intervention) are most likely to benefit from the procedure in future clinical settings (cf. Espie & Lindsay, 1985). In turn, this will determine the utility of paradoxical intention as a general treatment approach for chronic initial insomnia.

1.8 Timescales

Data collection will commence immediately following ethical approval (05/2000) and should require 6-10 months. Data analysis and scoring will be completed by 03/2001 and write up completed by 06/2001.

1.9 Ethical Approval

Forms will be submitted to the Greater Glasgow Primary Care Trust Ethics Committee for the May 11th meeting (Submission deadline 27th April). The proposal will also be sent to the relevant University Faculties for approval to contact potential participants thereafter.

Trust ethical approval was granted in August 2000- see Appendix 2.4. Approval was also granted from the various University Faculties concerned prior to students being contacted by email for participation.

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Wicklow, A. & Espie, C.A. (2000). Intrusive thoughts and their relationship to actigraphic measurement of sleep: Towards a cognitive model of insomnia. *Behaviour Research and Therapy, 38*, 679-693.

Chapter 4: Major Research Project Paper

Initial insomnia and Paradoxical Intention: An experimental investigation of putative mechanisms using subjective and actigraphic measurement of sleep.

Major Research Project Paper submitted in partial fulfilment of the requirements for the degree of Doctor of Clinical Psychology

Prepared in accordance with requirements for submission to Behaviour Research & Therapy
(Appendix 3.1)

RUNNING HEAD: Insomnia and Paradoxical Intention

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Abstract

Although Paradoxical Intention (PI) is now regarded as an empirically-supported treatment for initial insomnia, few PI studies have measured outcome using objective sleep measurement. Moreover, mechanisms underlying PI remain unclear. The present experiment therefore examined the effect of PI on both objective (wrist actigraphy) and subjective (self-report) measures of sleep. Sleep performance anxiety and sleep effort data were also collected. Following a seven night baseline, thirty-four initial insomniacs were randomly allocated to fourteen nights of PI, or to a control (no PI) condition. Analysis of actigraphic sleep data indicated no significant differences between PI participants and controls in terms of objective SOL. However, PI resulted in a clinically significant reduction in self-reported SOL and sleep anxiety. These results may relate to recent metacognitive models of anxiety. Consistent with the performance anxiety model of PI, there was a significant reduction in sleep effort as well as sleep anxiety following PI. Correlational analysis also revealed a significant association between reduced sleep effort and reduced subjective SOL, which supports recent models of ironic cognitive control. Together, results help determine putative mechanisms underlying PI, and have important implications for the clinical application of PI. The need for further research investigating sleep effort, sleep anxiety and PI using objective and subjective sleep measurement is emphasised.

Key words: Initial Insomnia, Paradoxical Intention, Performance Anxiety, Actigraphy

Introduction

Recently, evidence has converged suggesting that pre-sleep cognitive activity may be an important determinant of initial insomnia (cf. Espie & Wicklow, 2001). For instance, research has shown that insomniacs attribute their poor sleep to cognitive factors (Espie, Brooks & Lindsay, 1989a; Harvey, 2000a; Lichstein & Rosenthal, 1980), and typically experience pre-sleep thoughts which are intrusive, negative in affect, and related to both recent problems, and the sleep process itself (Coyle & Watts, 1991; Harvey, 2000a; Wicklow & Espie, 2000). Moreover, correlational studies show a strong association between pre-sleep cognitive activity and sleep-onset latency (SOL; Nicassio, Mendlowitz, Fussel & Petras, 1985; Van Egeren, Haynes, Franzen & Hamilton, 1983), whilst experimental studies indicate that direct manipulation of pre-sleep cognitive activity can delay SOL amongst both good (Ansfield, Wegner & Bowser, 1996; Gross & Borkovec, 1982; Hall, Buysse, Reynolds, Kupfer & Baum, 1996; Harvey, 2000a) and poor sleepers (Harvey, 2000a).

Accordingly, recent formulations now emphasise the role of cognitive variables in insomnia aetiology (Espie & Wicklow, 2001; Lundh, 1998; Morin, 1993), and as a result, cognitive techniques have become increasingly popular in the management of the condition (e.g. cognitive restructuring- Morin, 1993; cognitive control- Espie & Lindsay, 1987; articulatory suppression- Levey, Aldaz, Watts & Coyle, 1991; paradoxical intention- Espie & Lindsay, 1985). However, the majority of these techniques tend to be subsumed within a multifaceted cognitive-behavioural therapy (CBT) package (e.g. Espie, Inglis, Tessier & Harvey, 2001; Edinger, Radtke, Wohlge-muth, Marsh & Quillan, 1997; Morin, Colecchi, Stone, Sood, & Brink, 1995). Such integrative approaches have meant few studies provide direct evidence of the utility of individual cognitive components subsumed within CBT treatment packages (Espie, 1991, Edinger & Wohlge-muth, 1999).

One cognitive technique that has been studied in isolation is Paradoxical Intention (PI). PI instructs patients to "intend" behaviour which is apparently in opposition to their goal (Ascher, 1979; Seltzer, 1986; Strong, 1984). With respect to initial insomnia, it requires that insomniacs give up trying to sleep, and attempt instead to remain awake (Espie & Lindsay, 1985). Paradoxically, therefore, the likelihood of staying awake is reduced by encouragement to do so.

Both clinical case reports (e.g. Ascher, 1975; Ascher & Efran, 1978; Espie & Lindsay, 1985) and group studies (e.g. Ascher & Turner, 1979, 1980; Espie, Lindsay, Brooks, Hood & Turvey, 1989; Ladocuer & Gros-Loius, 1986) demonstrate the efficacy of PI in reducing SOL, and PI is now regarded as a "probably efficacious" insomnia treatment according to American Psychological Association criteria (Chesson, Anderson, Littner, Davila, & Hartse, 1999; Morin, Hauri, Espie, Spielman, Buysse & Bootzin, 1999).

However, very few studies have examined the effects of PI using objective sleep measures (Ott, Levine & Ascher, 1983). Insomniacs' self-report sleep data can be unreliable (Carskadon, Dement, Mitler, Guilleminault, Zarcone & Spiegel, 1976), and objective and subjective sleep measures may reflect differing response systems (Wicklow & Espie, 2000). An examination of PI using objective sleep measurement would therefore be desirable.

A reliable and minimally intrusive objective sleep measure is the actigraph- a small wrist attachment which records the wearer's movements. There is recognition that movement is a good predictor of wakefulness, whilst lack of movement is a good predictor of sleep (American Sleep Disorders Association, 1995; Mullaney, Kripke & Messin, 1980). Moreover, actigraphic measures correlate highly with polysomnographic (PSG) data for sleep duration and total wake time (e.g. Sadeh, Hauri, Kripke & Lavie, 1995; Mullaney, Kripke & Messin, 1980). Therefore, wrist actigraphy provided a valid, minimally intrusive measure of objective sleep in the context of the present study.

The precise mechanisms underlying PI also remain unclear. PI is thought to operate by eliminating voluntary sleep effort, therefore minimising performance anxiety, an aroused state incompatible with sleep (Ascher & Turner, 1979). However, this performance anxiety model remains untested (Espie & Lindsay, 1985). Whilst one study noted that performance anxiety declined following PI (Fogle & Dyall, 1983), the researchers failed to collect SOL or sleep effort data. Moreover, participants not receiving PI also improved on a subjective sleep efficiency measure. Thus, the link between improved sleep, PI, sleep effort and sleep anxiety remains unclear. The present study aimed to clarify this performance anxiety model of PI.

Recently, PI has also been framed in terms of ironic cognitive control (Wegner, 1994), with sleep performance anxiety thought to constitute Wegner's *cognitive load* (Ansfield, Wegner & Bowser, 1996). In Wegner's model, two cognitive processes involved in mental control are identified: a conscious *intentional operating process* which searches for cognitions yielding a desired mental state, and an automatic *ironic monitoring process* which searches for opposing cognitions. Wegner suggests that under high cognitive load, the intentional operating process is undermined, and the ironic monitor predominates. Applying this to sleep, urgent attempts to fall asleep under high cognitive load should result in ironic wakefulness, and there is already some experimental evidence for this (Ansfield et al, 1996). By measuring sleep effort and sleep anxiety data, the present study aimed to clarify a complementary prediction- that attempts to stay awake under high cognitive load (sleep performance anxiety) result in an opposite, ironic mental state i.e. faster sleep onset (cf. Wegner, 1994.)

Metacognitive models of emotional disorder (Wells, 1997; 2001) may also prove relevant in considering the impact of PI on initial insomnia, particularly given the hypothesised central role of pre-sleep intrusions in the condition (e.g. Harvey, 2000a). Such models posit the existence of negative meta-cognitive beliefs which are triggered by, and concern the meaning and significance of, intrusive thoughts. Since

it has already been demonstrated that anxiety incubates intrusive thoughts (Wells & Papageorgiou, 1995), PI, by reducing sleep anxiety, may in turn lower activation of negative metacognitive beliefs, thereby leading to declarative belief change, and a reduction in overestimated self-report SOL frequently seen in insomnia (Borkovec, 1982).

Although PI is now regarded as an empirically-supported insomnia treatment (Morin et al, 1999), certain researchers have failed to report significant treatment effects (Lacks, Berteson, Gans & Kunkel, 1983; Turner & Ascher, 1982), whilst others have reported some insomniacs worsening as a result of the approach (Espie et al, 1989b; Lacks et al, 1983). Counterdemand instructions ("do not expect to see any improvement for 2 weeks of the study") have been included in some PI studies to control for demand characteristics and patient outcome expectations (cf. Steinmark & Borkovec, 1974). It has been suggested non-responding following PI may relate to participants interpreting these and/or paradoxical instructions ("stay awake for as long as possible") too literally (Espie & Lindsay, 1985). In order to clarify this, only half of participants allocated to PI in the present experiment received counterdemand instructions, whilst at the end of the study, questioning explored how PI participants employed the technique. Clarification of these non-response issues may in turn help determine the suitability of PI as a general treatment approach for insomnia.

In sum, the present study offers an experimental examination of the efficacy of brief (2 week) PI, using both objective (actigraphy) and subjective (self-report diary) measurements of sleep. Furthermore, by recording both effort to sleep and sleep performance anxiety data, putative mechanisms underlying PI were explored. Factors thought to account for non-responding following PI were also examined.

Primary hypotheses were that insomniacs allocated to PI would, relative to controls, show a reduction in objective and subjective SOL (cf. Ascher & Turner, 1979, 1980), and an increase in objective and subjective sleep efficiency. In addition, insomniacs allocated to PI would, relative to controls, show a reduction in both sleep

performance anxiety and sleep effort (cf. Ascher & Turner, 1979). Finally, insomniacs not receiving counterdemand instructions would respond significantly more to PI in terms of objective and subjective sleep parameters (Espie & Lindsay, 1985).

Method

Participants

Participants were recruited using the University email system and via notices placed locally. Ethical approval was granted by both the Health Trust and University. Prior to participation, potential participants completed questionnaires assessing sleep (Pittsburgh Sleep Quality Index [PSQI; see Appendix 2.2]- Buysse, Reynolds, Monk, Berman & Kupfer, 1989; Sleep History Questionnaire- Morin, 1993, see Appendix 2.3), anxiety (Spielberger Trait Anxiety Inventory- STAI; Spielberger, Gorsuch & Lushene, 1970), worry (Penn State Worry Questionnaire- PSWQ; Myer, Miller, Metzger & Borkovec, 1990) and depression (Beck Depression Inventory- BDI; Beck, Steer & Brown, 1996).

Respondents were included if they were between 16 and 65 years, complained of clinically significant problems falling asleep according to the International Classification of Sleep Disorders (American Sleep Disorder Association, 1997; i.e. SOL greater than 30 min at least 4 nights per week, with or without disruption to other sleep variables), and scored in excess of 5 on the PSQI (Buysse et al, 1989). Participants were excluded if they experienced intermittent awakenings without sleep-onset difficulties, were receiving treatment for sleeping difficulties, or were suffering any medical or psychopathological disorder impacting on sleep.

Forty-six participants were finally recruited all meeting criteria. 34 completed the study (73.91%). A further seven failed to attend the initial meeting, three withdrew during baseline, whilst two were excluded due to unreliable diary and scale

completion. Mean age of completing participants was 25.18 years, with an average sleep disturbance of 6.35 years.

Measures

Participants completed a daily sleep diary (Espie, 1991) upon rising, recording time to bed, rise time, time to fall asleep, and total sleep time (see Appendix 3.2). This data provided measures of subjective SOL, and sleep efficiency- a percentage of total sleep time divided by total time in bed widely employed in sleep research (Morin, 1993). Daily effort to sleep and restedness data were also recorded on a seven point scale (anchor points 0 "not at all ", 6 "very much"; see Appendix 3.2).

Wrist actigraphic recording using the 'Actiwatch' (Model AW2; Cambridge Neurotechnology Ltd) provided objective SOL and sleep efficiency data. Participants wore the actigraph continuously on their non-dominant hand except during wet activities. An event marker on the actigraph was depressed at lights out, and upon rising. Epoch length was set at 1 min (cf. Wicklow & Espie, 2000), with 'sleep' or 'wakefulness' determined by the program's algorithm.

Two self-report scales were utilised to measure sleep-related performance anxiety. First, in order to extend previous findings, the Sleep Anxiety Scale (SAS; Fogle & Dyall, 1983; see Appendix 3.3) was employed. Some internal consistency data for this scale have been reported (see Fogle & Dyall , 1983, p. 26).

Participants also completed a specially developed scale, the Sleep Performance Anxiety Questionnaire (SPAQ; Broomfield & Espie, 2001; see Appendix 3.4). This comprised seven components of dysfunctional sleep monitoring (sleep effort, sleep control, sleep avoidance, bedtime worry, performance failure, anticipatory anxiety, and daytime worry). Prior to the study, the SPAQ was administered to four good and four poor sleepers. Data suggested the scale readily distinguished these groups (good sleepers mean score = 7.75, SD = 0.96; poor

sleepers mean score = 15.50, SD = 3.69).

Following training in paradoxical intention, PI participants completed a credibility rating sheet (see Appendix 3.5). Control subjects did not complete a credibility rating sheet. At the end of the study, they were also asked how they employed the procedure ("What did you do when you went to bed?"), and any effects it had ("What effects, if any, did the treatment have?"). At the end of the study, all participants completed a compliance rating sheet (see Appendix 3.6).

Design

The study employed a One Between (Condition: Paradoxical Intention Therapy [PI], Control) and One Within participants factor (Time: Baseline, Week One, Week Two) design. Participants were randomly assigned to experimental condition by the experimenter using a predetermined list of numbers.

Procedure

Following a telephone interview, participants were sent screening questionnaires, an information sheet, and an informed consent slip. These were completed and returned by post. Participants then met with the experimenter (meeting 1), were instructed to complete their sleep diary upon rising, for 21 days, and were supplied with a wrist actigraph. Participants were then told the next seven days was a baseline measurement week.

At meeting 2, participants completed the two sleep anxiety scales (Baseline), and were issued with further copies to be completed after seven days (Week One). Participants were then randomly allocated to an experimental condition (PI with counterdemand instructions; PI with no counterdemand instructions; Control).

PI participants were introduced to a diagram of the performance anxiety model of PI (cf. Ascher & Turner, 1979; see Appendix 3.7). The role of sleep effort

and performance anxiety in maintaining initial insomnia was highlighted, and the rationale of PI was explained. PI participants were then given an instruction sheet (cf. Ott et al, 1983), asking them to apply PI for fourteen nights. Those allocated to the PI counterdemand condition were told not expect to see sleep improvement for the first week of the study, but to improve thereafter (see Appendix 3.8). PI participants not receiving counterdemand instructions were told to expect immediate improvement in their sleep (see Appendix 3.9). Control participants received instructions to continue wearing the actigraph and completing their sleep diary (see Appendix 3.10).

At meeting 3, all participants returned their diaries and actigraph, and completed two final sleep anxiety scales (Week Two), and a compliance rating sheet. PI participants were asked about their use of the technique, and its general effects. Control participants received PI training. All participants were debriefed and thanked, and issued with the "Good Sleep Guide" (National Medical Advisory Committee, 1993; see Appendix 3.11). No further treatment was offered.

Insert Table 4.1 here

Results

Participant Characteristics

Demographic summary data are presented in Table 4.1. Chi Square analysis indicated participants allocated to the two conditions were equitable in terms of gender ($\chi^2 = 0.11$, $df = 1$, NS). Independent t-tests also demonstrated participants did not differ on age ($t(32) = 0.63$, $p > 0.1$, NS), duration of sleep problem ($t(32) = -0.48$, $p > 0.1$, NS), trait anxiety ($t(32) = 0.01$, $p > 0.1$, NS), worry ($t(32) = 0.09$, $p > 0.1$, NS), depression ($t(32) = -0.75$, $p > 0.1$, NS) or sleep quality ($t(32) = 0.63$, $p > 0.1$, NS).

Outcome Variables

Unless otherwise indicated, analyses relied on a Two (Condition: Paradoxical Intention vs. Control) by Three (Time: Baseline vs. Week One vs. Week Two) ANOVA design based on weekly means. Prior to ANOVA, all data were examined for kurtosis and skewness and fell within acceptable limits. Data also showed homogenous variance, following Hartley's Fmax test (Winer, 1971). Alpha level was set at .05, two-tailed, throughout. Means and standard deviations for primary outcome variables are presented in Tables 4.2, and 4.3.

Insert Table 4.2 here

Sleep

Objective Sleep (Actigraphy)

Two participants' actigraph data were lost due to faulty equipment. Objective sleep analyses are therefore based on 16 participants per condition.

Analyses of variance for objective SOL failed to demonstrate significant effects of Time ($F[2,60] = 0.48, p > 0.1, NS$) or Condition ($F[1,30] = 0.06, p > 0.1, NS$). The interaction of Time x Condition was also non-significant ($F[2,60] = 0.13, p > 0.1, NS$). Objective SOL across conditions remained at approximately 25 - 30 minutes, with no significant differences between PI and control participants (see Figure 4.1 and Table 4.2).

Analysis of objective sleep efficiency data failed to reveal significant effects of Time ($F[2,60] = 0.53, p > 0.1, NS$) or Condition ($F[1,30] = 0.67, p > 0.1, NS$). The interaction of Time x Condition was also non-significant ($F[2,60] = 0.19, p > 0.1, NS$). Objective efficiency across conditions remained at approximately 80%, with no significant differences between PI and control participants (see Figure 4.2 and Table 4.2)

Insert Figure 4.1 here

Subjective Sleep (Diary)

Analysis revealed a main effect of Time ($F[2,64] = 6.72, p = 0.002$), and an interaction of Time x Condition ($F[2,64] = 9.16, p = 0.0001$), displayed graphically in Figure 4.3. In order to clarify this interaction, Bonferroni corrected simple main effects analyses for Condition were completed across the Time variable (critical p value = 0.017; Keppel, 1993). Results indicated PI participants and controls did not differ significantly at Baseline ($F[1,33] = 1.15, p > 0.1, NS$), and there was only a trend approaching significance for lower SOL amongst PI participants at Week One ($F[1,33] = 3.36, p = 0.076$) and Week Two ($F[1,33] = 3.34, p = 0.077$). Inspection of mean subjective SOL data indicated allocation to PI resulted in a 41.85% reduction in SOL, compared to a 1.21% increase amongst controls (see Table 4.2). Subjective SOL treatment effect size was "moderate" ($d = 0.61$; cf. Cohen, 1988).

Analysis of subjective sleep efficiency data revealed a main effect of Time ($F[2,64] = 6.65, p = 0.024$), although the Time x Condition interaction failed to reach significance ($F[2,64] = 0.73, p > 0.1, NS$). Analyses for morning restedness data also demonstrated a main effect of Time ($F[2,64] = 6.97, p = 0.018$), although the Time x Condition interaction was non-significant ($F[2,64] = 1.55, p > 0.1, NS$).

Insert Figure 4.2 here

The Relationship between Objective and Subjective Sleep Onset Latency

As inspection of Table 4.2 and Figures 4.1 and 4.2 reveals, objective SOL at baseline was 30 minutes for PI participants, and 27 minutes for controls. At Week Two, these scores were unchanged for both PI participants (mean = 24 minutes) and controls (mean = 25 minutes). No significant objective SOL change following PI was therefore observed.

Subjective SOL scores at baseline were much higher than objective SOL scores amongst both PI participants (mean = 66 minutes), and controls (mean = 55 minutes). This suggests participants *overestimated* SOL relative to actigraphic

assessment as an objective criteria. Analyses indicated allocation to PI led to a significant reduction in subjective SOL, reducing the discrepancy between subjective (mean = 38 minutes) and objective (mean = 24 minutes) SOL scores by Week Two. Control subjects showed no significant subjective SOL change, and thus no reduction in discrepancy by Week Two (mean subjective SOL = 55 minutes, mean objective SOL = 25 minutes). Together, these data suggest, in contrast to hypotheses, allocation to PI did not impact on objectively assessed SOL, but did significantly reduce participants tendency to overestimate subjective SOL.

Pearson correlation coefficients were computed across all participants to examine the association between objective and subjective SOL, and objective and subjective sleep efficiency. This revealed correlations were 'low' for SOL ($r = 0.25$), and 'low' for sleep efficiency ($r = 0.17$).

Insert Table 4.3 here

Sleep Performance Anxiety

Separate ANOVAs were run for the two performance anxiety scales employed, based on participants total scale scores at Baseline, Week One and Week Two.

Sleep Anxiety Scale (SAS; Fogle & Dyall , 1983)

Analysis of variance revealed an effect of Time ($F[2,64] = 6.69, p = 0.002$), and an interaction of Time x Condition ($F[2,64] = 9.84, p = 0.0001$). As is evident in Figure 4.4, simple main effects demonstrated no differences across Condition at Baseline ($F[1,33] = 0.09, p > 0.1, NS$). However, at Week Two, PI participants displayed significantly lower sleep anxiety than controls ($F[1,33] = 8.26, p = 0.007$; critical p value = 0.017), and there was a near significant trend for lower sleep anxiety at Week One ($F[1,33] = 5.41, p = 0.026$; critical p value = 0.017).

In order to examine the internal consistency of obtained scale data, baseline

item scores across all participants were subjected to appraisal using Cronbach's alpha. This revealed an overall alpha coefficient of 0.86, with the range of alpha values, if item deleted, of 0.83 - 0.87. The mean corrected item-total correlation was 0.62.

Insert Figure 4.3 here

Sleep Performance Anxiety Questionnaire (SPAQ; Broomfield & Espie, 2001)

Analysis revealed a main effect of Time ($F[2,64] = 15.68, p = 0.0001$), and a Time x Condition interaction ($F[2,64] = 15.48, p = 0.0001$), displayed in Figure 4.5. As is evident, simple main effects revealed no differences across Condition at Baseline ($F[1,33] = 0.30, p > 0.1, NS$), but significantly lower scores for PI participants, relative to controls, at Week Two ($F[1,33] = 9.89, p = 0.004$; critical p value = 0.017), and a near significant trend for lower sleep anxiety amongst PI participants at Week One ($F[1,33] = 5.66, p = 0.023$; critical p value = 0.017).

Cronbach's alpha analysis of baseline item scores revealed an overall alpha coefficient of 0.70, with the range of alpha values, if item deleted, of 0.63 - 0.73. The mean corrected item total correlation was 0.42.

Finally, pearson correlation coefficients were computed across participants baseline scores on the SAS and SPAQ. This indicated correlation was 'moderate' ($r = 0.62, p < 0.01$), representing 38.5% shared variance.

Insert Figure 4.4 here

The Relationship between Objective and Subjective Sleep Onset Latency and Sleep Performance Anxiety

As is evident in Figures 4.1, 4.3, 4.4. and 4.5, PI significantly reduced sleep performance anxiety. Moreover, allocation to PI significantly reduced participants subjective SOL, but did not impact on objectively assessed SOL. These data highlight a possible relationship between sleep anxiety and self-report SOL, whereby

PI lowered participants sleep anxiety levels, in turn reducing their overestimations of time to sleep onset, without actually impacting on objective SOL.

Sleep Effort

Analysis revealed a main effect of Time ($F[2,64] = 9.05, p = 0.0001$), and a Time x Condition interaction ($F[2,64] = 6.37, p = 0.003$). As is evident in Figure 4.6, simple main effects indicated PI participants showed significantly lower sleep effort at Week One ($F[1,33] = 7.72, p = 0.009$; critical p value = 0.017) and near significant lower effort at Week Two ($F[1,33] = 5.55, p = 0.025$; critical p value = 0.017). No differences were observed at Baseline ($F[1,33] = 0.01, p > 0.1, NS$).

Pearson correlational analyses indicated baseline sleep anxiety was significantly associated with sleep effort (SAS; $r = 0.56, p = 0.001$), (SPAQ; $r = 0.58, p = 0.0001$).

Insert Figure 4.5 here

The Association between Sleep Effort, Sleep Performance Anxiety and Subjective Sleep Onset Latency

To explore whether reduced sleep effort or sleep anxiety best predicted reduced subjective SOL, change scores for each variable (mean week 1 - mean week 3) were computed and subjected to Pearson correlational analyses. Effort change scores were unsuitable for parametric analysis and therefore, Spearman's correlations are reported. Results indicated SOL change was significantly associated with sleep anxiety change (SAS: $r = 0.36, p = 0.035$; SPAQ: $r = 0.43, p = 0.01$), but appeared more strongly associated with effort change ($r = 0.56, p = 0.001$).

Given that sleep effort and sleep anxiety were correlated, partial correlations were computed to reveal the individual effect of effort change, and sleep anxiety change (both scales), on subjective SOL change. This revealed effort change significantly correlated with SOL change when sleep anxiety was partialled out (SAS:

$r_p = 0.42, p = 0.016$; SPAQ: $r_p = 0.39, p = 0.026$). In contrast, when effort change was partialled out, neither measure of sleep anxiety was significantly associated with SOL change (SAS: $r_p = 0.08, p > 0.1, NS$; SPAQ: $r_p = 0.2, p > 0.1, NS$). This suggests SOL reduction was most strongly associated with sleep effort reduction.

Insert Figure 4.6

Counterdemand Instructions

A series of Two (Instructions: Counterdemand vs. No Counterdemand) by Three (Time: Baseline vs. Week One vs. Week Two) ANOVAs on objective and subjective SOL and sleep efficiency data revealed no significant effects.

PI: Treatment Credibility, Application, and Reported Effects

PI participants reported a mean rating = 4.70 (SD = 1.10) that "the treatment approach seemed sensible", mean = 4.11 (SD = 0.76) that they were "confident it would work" , and mean = 4.64 (SD = 0.86) that they would be "confident in recommending it to a friend with insomnia" (all scales 0-6). Note that since controls did not receive any formal intervention, no credibility rating data were collected for this group.

All 17 PI participants reported they lay awake with their eyes open when using PI. None reported using active methods (reading, television) to stay awake.

Twelve participants (70.6%) reported PI helped them get to sleep quicker. None reported PI worsened their sleep. Three (17.6%) indicated it made no difference to their sleep.

Compliance with Experimental Instructions

Participants reported correctly following experimental instructions (diary

completion, actiwatch use) on mean = 19.17 nights. Mean compliance rating (scale 0-6) was 5.03 (SD = 0.79). Areas of non-adherence included forgetting to press the actiwatch, or to replace the actiwatch after wet activities. These events were relatively unusual.

Discussion

A first key aim of the study was to examine the effect of PI on objective and subjective sleep-onset latency (SOL). Objective SOL differences across condition failed to reach statistical significance, with PI participants mean objective SOL remaining largely constant relative to baseline. However, PI produced a subjective SOL reduction of 41.85% after two weeks, lowering the objective-subjective SOL discrepancy (higher subjective SOL) seen at baseline. The subjective SOL treatment effect size was "moderate" (cf. Cohen, 1988), with more than two thirds of PI participants reporting they fell asleep quicker by the end of the experiment, and none indicating it worsened their sleep. Clinically significant interventions are defined as those which have a genuine impact on client functioning (Kadzin, 1999), and these data suggest clinically significant subjective SOL change following PI was observed. Subjective SOL findings therefore appear consistent with previous demonstrations of the efficacy of PI (e.g. Ascher & Turner, 1979; Espie et al, 1989), although since only PI participants completed a treatment credibility rating, the unbalanced demand characteristic between groups may have contributed to this effect.

It is of some importance, however, that in contrast to previous findings (Ott et al, 1983), and contrary to our predictions, allocation to PI acted only to lower subjective SOL, and did not appear to impact on objective SOL. One possible interpretation of this data however is that participants may have overestimated their sleep-latency, and that PI acted to ameliorate this overestimation, without impacting objective sleep. Research findings have shown that insomniacs do frequently overestimate sleep-latency (Borkovec, 1982). Moreover, inspection of Table 4.2

reveals a clear discrepancy between estimated (subjective) and objective SOL across both groups at baseline, with much higher estimated subjective relative to objective sleep latency. Interestingly, this objective-subjective SOL discrepancy remained amongst control participants at Week 2, but reduced considerably following PI. Given this, and the lack of objective SOL change across groups, the notion that PI acted to reduce SOL overestimation seems credible.

One possible explanation for how PI may have reduced sleep-latency overestimation draws on metacognitive models of anxiety disorder (Wells, 1997, 2001). Metacognitive models of anxiety suggest that beliefs, processes and strategies which appraise, monitor or control cognition may be central in the aetiology of emotional disorder (Wells, 1997; 2001). Their relevance to sleep seems particularly likely, given the key role of pre-sleep intrusions in insomnia, and recent evidence that insomniacs employ metacognitive thought control strategies to minimise sleep-related intrusions (Harvey, 2000b).

A metacognitive interpretation of the sleep findings relates to a third variable recorded during the study - namely sleep anxiety. Research indicates that anxiety and worry can incubate intrusive thoughts (Wells & Papageorgiou, 1995), and suggests that intrusions activate negative meta-cognitive beliefs concerning their meaning and significance (see Wells, 1997; 2001). Our analysis indicated that PI significantly lowered sleep anxiety. Invoking a metacognitive analysis, by reducing sleep anxiety, PI may have reduced the frequency of sleep-related intrusions, in turn lowering activation of negative sleep-related metacognitive beliefs (e.g. "Thinking about sleep makes my sleep worse"; "Thinking about sleep means I'm a poor sleeper"). This lowered activation will have minimised participants catastrophic thoughts and perceptions about sleep difficulty, reduced conviction in declarative beliefs about sleep ("I'm a poor sleeper"), and shifted their tendency to overestimate sleep latency, in the absence of objective sleep improvement (Wells, 2001). In other words, PI reduced excessive anxiety about sleep, in turn altering beliefs about sleep via

metacognitive processes, thereby reducing SOL overestimation. The possible effect of lowered sleep anxiety on negative interpretive biases (Williams, Watts, Macleod & Mathews, 1997) may also have reduced PI participants tendency to overestimate SOL.

Due to the lack of measurement of these variables, this interpretation is speculative, although it appears able to account for the reduction in sleep anxiety and subjective SOL seen in the absence of objective SOL change. Certainly, the data add to growing evidence linking intrusions and metacognitive beliefs (Wells, 2001), and suggest insomnia research should take account of metacognitive concepts. Findings also highlight the clear similarities between paradoxical directives and anxiety-based treatments aimed at metacognitive and behavioural change (e.g. graded exposure, exposure plus response prevention).

A competing explanation of the sleep data relates to actigraphic measurement error. It is entirely possible that objective and subjective sleep-latency changes occurred following PI, but objective changes were not detected actigraphically. Although actigraphic measures of sleep duration and total wake time correlate highly with PSG data (Mullaney et al, 1980; Sadeh et al, 1995), lower agreement rates for discrete variables such as SOL have been reported (Blood et al, 1997; Hauri & Wisbey, 1992). This is because actigraphy measures body movements which only indirectly reflect sleep (American Sleep Disorders Association, 1995). Quiet wakefulness in the pre-sleep phase may therefore have been coded actigraphically as 'sleep' by the program's algorithm, resulting in the low objective SOL scores observed (see Table 4.2). These scores are consistent with previous insomnia studies employing actigraphy (Harvey, 2000b; Wicklow & Espie, 2000), and may have precluded the likelihood of significant change due to a floor effect. The lack of other objective sleep data does however limit this explanation, although the use of such measures (e.g. PSG, Behavioural Response Monitoring [BRM]- see Blood et al, 1997) alongside actigraphy in future PI research may resolve this. Certainly, the low

correlation between objective and subjective SOL data seen here, and elsewhere (e.g. Wicklow & Espie, 2000) suggests these measures reflect differing response systems (cf. Wicklow & Espie, 2000), and strengthens the case for recording both in a full assessment of sleep.

Certain methodological aspects of the present study may also have reduced the likelihood of observing objective SOL change. The present experiment integrated a short 'treatment' phase (2 weeks). Whilst objective SOL reduction following brief PI has been reported (Ott et al, 1983), a study of longer duration may have demonstrated objective SOL effects here. There is also evidence PI delivered by trainee clinicians is less likely to result in reduced sleep latency and this may have impacted on objective SOL findings (Turner & Ascher, 1982). In addition, only thirty-four participants completed the study, four less than were necessary for adequate power (see Chapter 2). Inclusion of additional participants may have increased the likelihood of objective SOL effects. It is certainly likely the lack of significant effects observed with respect to counterdemand instructions relates, at least in part, to inadequate power. Reliance on a somewhat robust statistical model to avoid Type I errors may also have contributed to the sleep data observed.

Importantly, it seems unlikely the sleep findings were confounded by poor participant compliance. Participants showed good adherence to the experimental protocol, and importantly, all PI participants reported correct employment of paradoxical instructions. In addition, in contrast to previous suggestions (Espie & Lindsay, 1985), there was no evidence of literal interpretation of paradoxical instructions.

The present study also aimed to examine, empirically, the sleep performance anxiety model of PI (Ascher & Turner, 1979). As noted, PI participants showed a significant reduction in sleep anxiety on both the SAS and SPAQ, which is consistent with a previous report (Fogle & Dyall, 1983). In addition, PI participants displayed a significant reduction in sleep effort. This indicates they correctly followed PI

instructions, and suggests successful operationalisation of the sleep effort construct in this study.

Moreover, alongside the sleep anxiety data, the observation of significant sleep effort reduction confirms the performance anxiety model of PI (Ascher & Turner, 1979). Ascher & Turner suggest PI ameliorates initial insomnia by eliminating voluntary efforts to sleep, thereby minimising performance anxiety, an aroused state incompatible with sleep (Ascher & Turner, 1979). Results here are entirely consistent with this model.

By confirming, empirically, Ascher & Turner's performance anxiety model, obtained data have clear clinical implications. If PI does operate by reducing sleep anxiety and sleep effort, the approach may be best suited to insomniacs high in these variables at baseline assessment (Espie & Lindsay, 1985). There is already evidence of a link between elevated performance (recursive) anxiety and response to PI in the clinical literature (Ascher & Schotte, 1999), and given this, and our finding, research should now examine whether insomniacs high in sleep anxiety and sleep effort respond better, and more quickly, to interventions integrating PI. This would help determine whether PI should be utilised as a general treatment approach, or may be best suited to insomniacs with high pre-morbid levels of these variables. The operationalisation of both constructs here suggests their routine clinical assessment is entirely feasible.

The development of the SPAQ (Broomfield & Espie, 2001) represents an important step in improving the operationalisation of sleep anxiety. The scale was employed because we felt the SAS (Fogle & Dyllal, 1983) fails to measure crucial aspects of the construct. The SPAQ comprises seven components of dysfunctional sleep monitoring, with items addressing sleep effort, performance failure, sleep avoidance and the desire to control sleep, alongside more general measures of anxiety/worry about sleeplessness seen in the SAS. Analysis suggested the scale shows reasonable internal consistency, and appears to readily distinguish good and

poor sleepers. Moreover, PI resulted in significant reductions in SPAQ scores. Clearly, research is necessary to clarify the reliability and validity of the scale, and this is planned. However, future research must employ scales like the SPAQ to assess more accurately the influence of PI on sleep anxiety.

The sleep anxiety and effort data are also consistent with Wegner's model of ironic cognitive control (Wegner, 1994). Wegner suggests that under conditions of high cognitive load (e.g. anxiety, stress), the action of the intentional operating system is undermined, and the ironic monitor predominates. If sleep anxiety constitutes a cognitive load (cf. Ansfield et al, 1996), it follows that sleep anxious insomniacs using PI should fall asleep quicker, as their ironic monitor searches for cognitions opposing the desired mental state of wakefulness. The present findings appear to confirm this, with analysis revealing a strong association between reduced sleep effort and reduced subjective SOL. This suggests, for insomniacs high in sleep anxiety, sleep-onset was promoted when attempts were made to stay awake. In other words, under high cognitive load, PI resulted in the ironic monitoring process detecting restful cognitions, thereby promoting faster sleep-onset (cf. Ansfield et al, 1996; Wegner, 1994).

It should finally be noted that our analysis did not reveal any significant subjective or objective sleep efficiency differences following PI. Previous findings regarding PI and sleep efficiency are ambiguous though (Fogle & Dyall, 1983), and there is little evidence to suggest PI increases total sleep time, a closely related variable (see e.g. Ascher & Turner, 1979; Espie et al, 1989). Thus, it still remains unclear whether PI influences sleep efficiency. The approach instructs clients to give up effort to sleep at the point of sleep-onset (Ascher & Turner, 1980). Therefore, PI may primarily impact SOL.

In summary, following PI, there was evidence of subjective SOL and sleep anxiety reduction, but no evidence of objective SOL reduction. One explanation of this data is that by lowering sleep anxiety, PI reduced activation of negative

metacognitive beliefs, thus minimising SOL overestimation (cf. Wells, 2001).

Alternatively, the lack of objective SOL change may relate to the insensitivity of actigraphy, although this seems less likely. The demonstration of significant sleep effort reduction amongst PI participants, alongside the sleep anxiety data, certainly offers empirical clarification of the performance anxiety model of PI (Ascher & Turner, 1979), and these results are also consistent with Wegner's model of ironic cognitive control (Wegner, 1994). Research is now required to explore further the link between these underlying putative mechanisms and response to PI, and to extend progress made here in operationalising these constructs. This will have important implications for the future clinical application of PI in managing chronic initial insomnia.

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Condition	Mean Age (years)	Females : Males Ratio	Mean Duration of Initial Insomnia (years)	STAI-Trait Anxiety	PSWQ Worry	BDI Depression	PSOL Sleep Quality
Paradoxical Intention (N = 17)	26.00	9:8	5.93	37.35	40.58	7.94	11.18
Control (N = 17)	24.35	10:7	6.77	37.29	40.11	9.88	10.59

Table 4.1 Demographic and questionnaire (mean scores) data for subjects allocated to the paradoxical intention and control conditions.

Condition	Objective Sleep Data				Subjective Sleep Data							
	SOL (mins)		Efficiency (%)		SOL (mins)		Efficiency (%)					
	Baseline	Week 1	Week 2	Baseline	Week 1	Week 2	Baseline	Week 1	Week 2			
Paradoxical Intention												
M	29.92	27.23	24.47	80.87	81.84	80.65	65.74	41.76	38.24	80.87	86.10	85.79
SD	17.16	15.06	17.20	6.67	7.56	7.57	33.97	19.62	25.28	13.49	11.85	11.07
Control												
M	26.62	25.19	25.03	78.93	79.80	80.64	54.67	58.82	55.33	80.04	82.46	82.82
SD	21.83	16.37	16.49	7.93	7.34	8.95	25.67	32.98	29.16	11.85	10.32	12.40

Table 4.2 Means and standard deviations for Objective and Subjective Sleep Latency and Sleep Efficiency at Baseline, Week One and Week Two.

		Sleep Anxiety Data		Sleep Effort Data						
		SAS (Fogle & Dyall, 1983)	SPAQ (Broomfield & Espie, 2001)							
Condition		Baseline	Week 1	Week 2	Baseline	Week 1	Week 2			
Paradoxical Intention	M	16.18	13.59	12.29	15.41	12.53	11.41	2.33	1.11	1.10
	SD	4.05	3.78	4.03	2.87	3.89	3.61	1.37	1.20	1.30
Control	M	15.76	16.35	16.05	14.88	15.18	14.76	2.28	2.21	2.14
	SD	3.83	3.12	3.60	2.71	2.43	2.51	1.44	1.12	1.28

Table 4.3 Means and standard deviations for Sleep Performance Anxiety and Sleep Effort data at Baseline, Week One and Week Two.

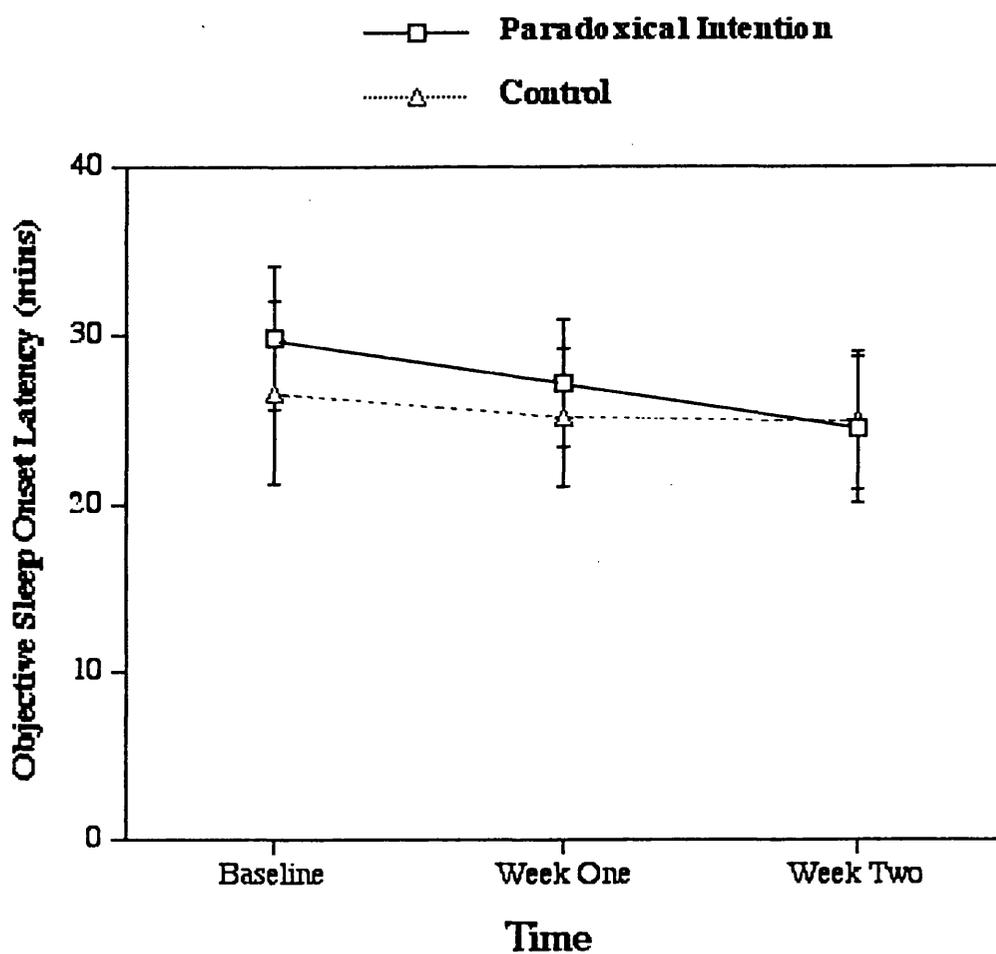


Figure 4.1 Mean and standard error objective sleep onset latency for paradoxical intention and control subjects, as a function of Time.

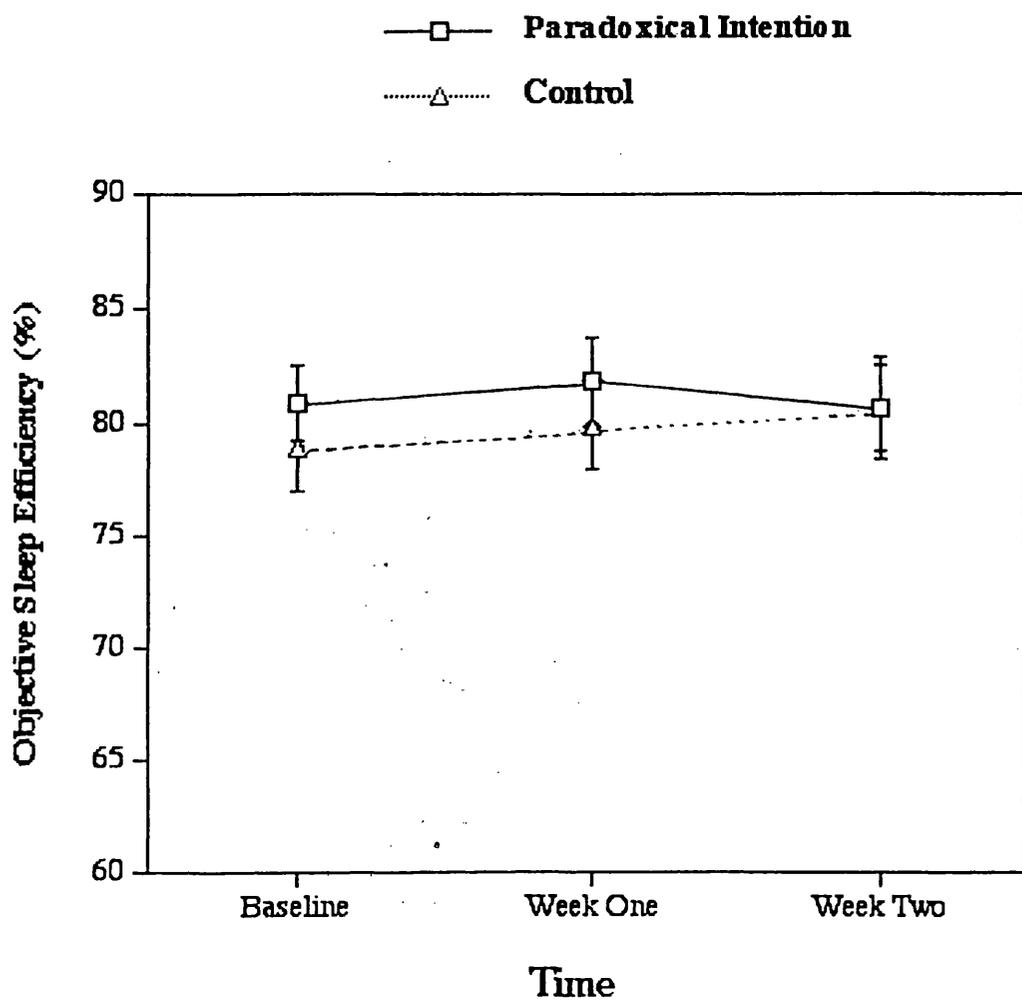


Figure 4.2 Mean and standard error objective sleep efficiency for paradoxical intention and control subjects, as a function of Time.

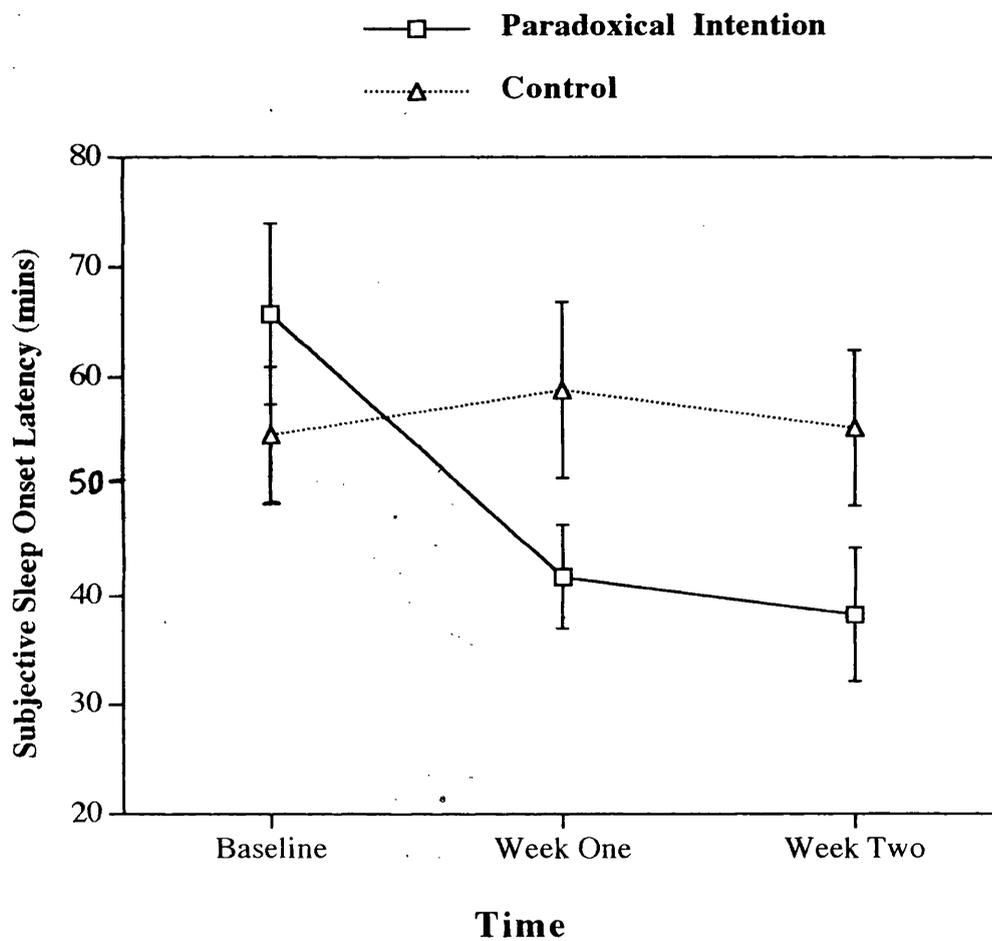


Figure 4.3 Mean and standard error subjective sleep onset latency for paradoxical intention and control subjects as a function of Time.

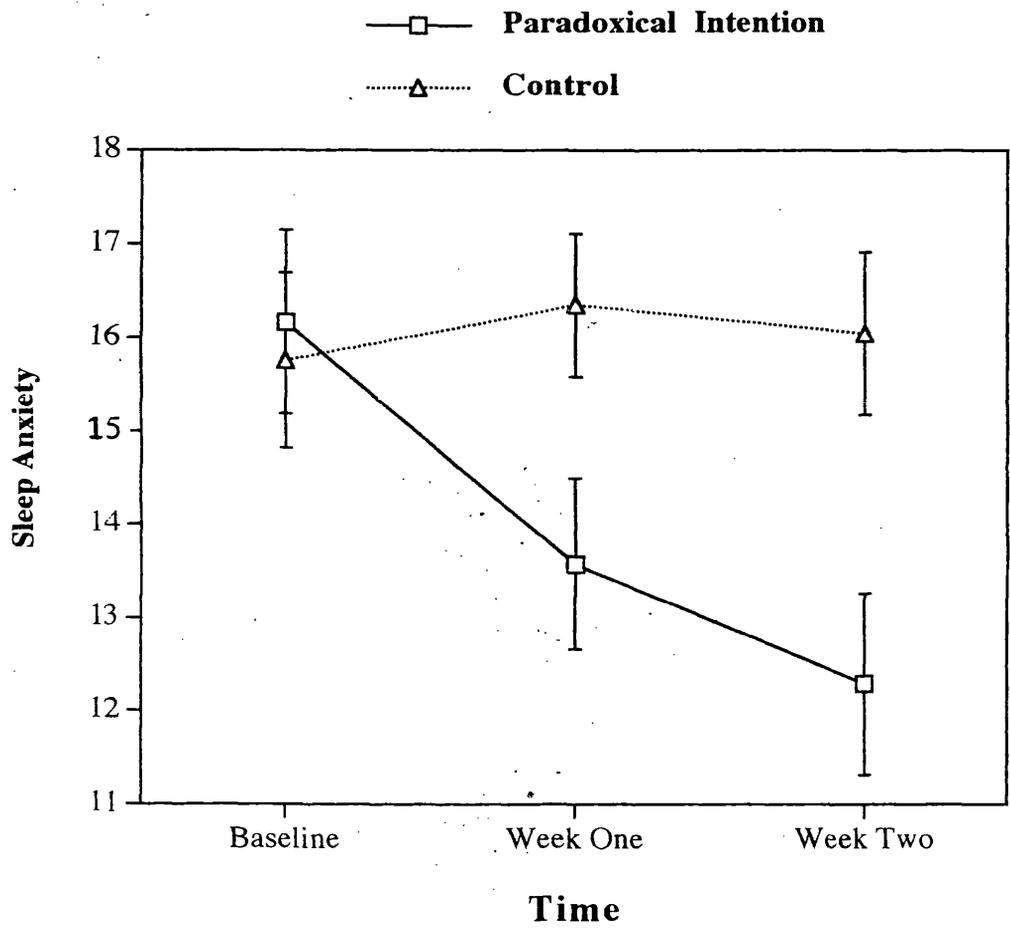


Figure 4.4 Mean and standard error sleep anxiety scores on the SAS for paradoxical intention and control subjects, as a function of Time.

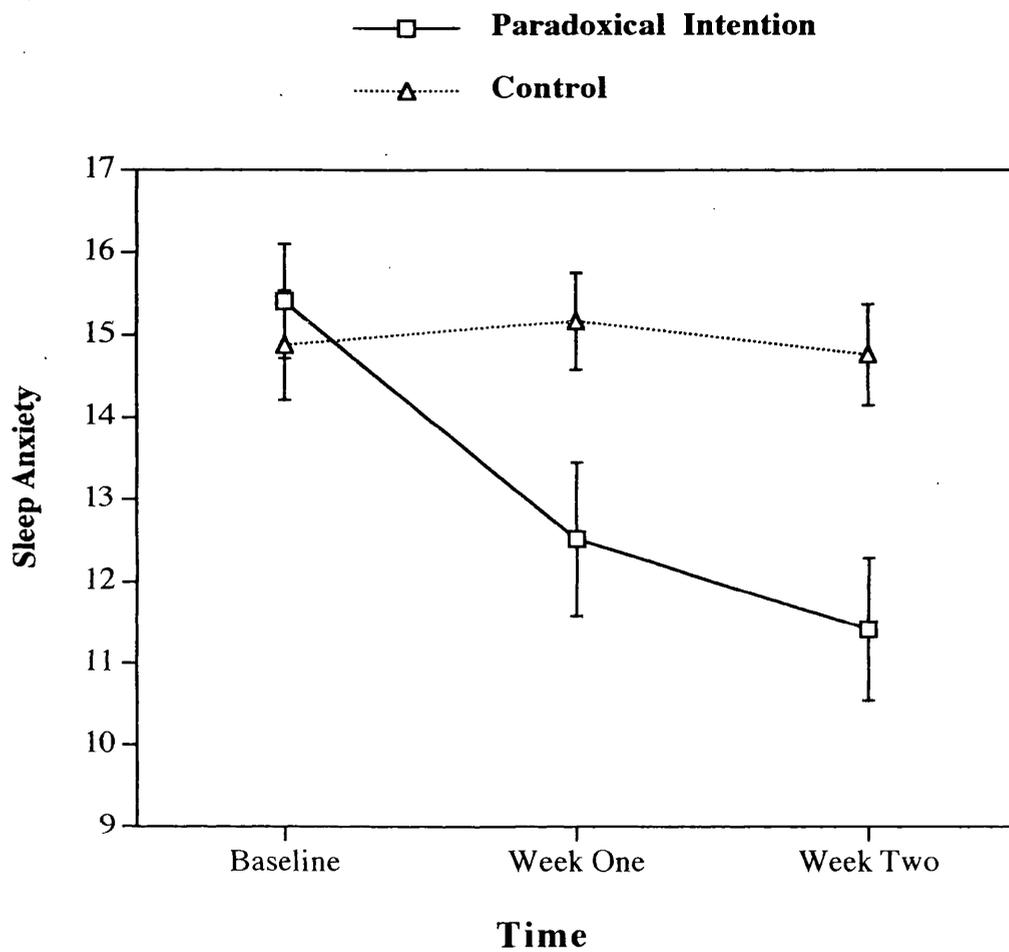


Figure 4.5 . Mean and standard error sleep anxiety scores on the SPAQ for paradoxical intention and control subjects, as a function of Time.

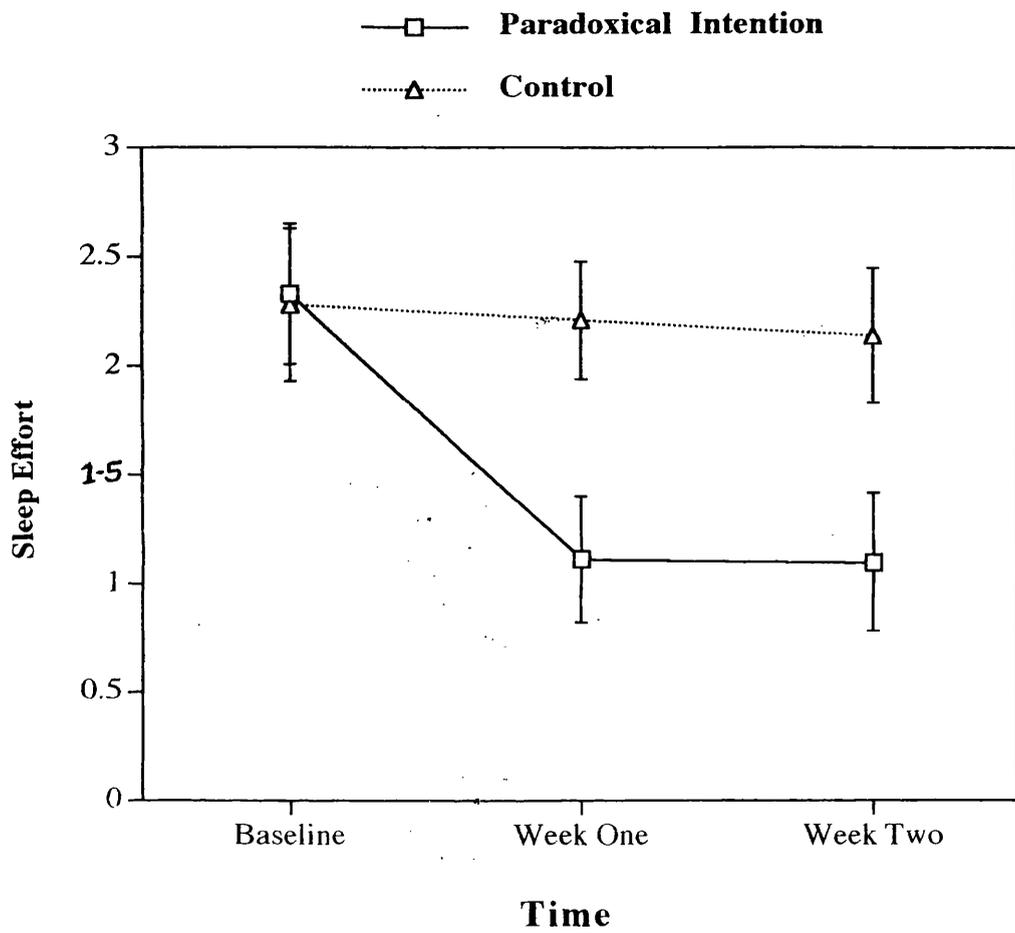


Figure 4.6 Mean and standard error sleep effort ratings for paradoxical intention and control subjects, as a function of Time.

Chapter 5: Clinical Case Research Study

A single case experimental investigation of the utility of structured relapse prevention treatment in a problem drinker with co-morbid panic disorder.

Clinical Case Research Study submitted in partial fulfilment of the requirements
for the degree of Doctor of Clinical Psychology

Prepared in accordance with requirement for submission
to Behavioural and Cognitive Psychotherapy
(Appendix 4.1)

Running Head: Problem drinking, SRP and panic disorder

Address for correspondence:

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Abstract

This study tested the effectiveness of Structured Relapse Prevention (SRP) in the treatment of alcohol dependence and comorbid panic disorder. A single case experimental design was employed. Consistent with previous research, daily self-report and questionnaire data demonstrated SRP significantly reduced alcohol intake, increased client self-efficacy, and promoted abstinence. The association between problem drinking and comorbid anxiety disorder was also examined. Problem drinking may promote anxiety disorder, and consistent with this, panic attack frequency and daily anxiety rating data indicated that, following abstinence, criteria for panic disorder were not met. However, residual avoidance and safety behaviours (carrying alcohol to prevent panic attack) remained, which were subsequently treated using cognitive-behavioural methods. In particular, graded exposure involving the dropping of carrying alcohol achieved some change in associated beliefs, although this was non-significant. Together, findings underline the complex association between alcohol dependence and anxiety disorder, and indicate the need for further single-case and group research in this area.

Keywords: Alcohol Dependence, Relapse Prevention, Self-Efficacy, Controlled Drinking, Comorbid Panic Disorder, Safety Behaviours,

Chapter 6 Appendices

Appendix 1: Small Scale Service Evaluation

Notes for Contributors

Papers, articles and other contributions should be sent to the Editor, *Health Bulletin*, Scottish Executive Health Department, Room IE05, St Andrew's House, Edinburgh EH1 3DE. They must be submitted exclusively for *Health Bulletin*. Acceptance is on the understanding that editorial revision may be necessary. All papers are reviewed by the Editor and by peer review, referees being drawn from a panel of appropriate professionals. No correspondence can be entered into in relation to articles found to be unsuitable and returned to authors.

Potential contributions can be submitted in two ways. Material submitted for publication must be typewritten on one side of the paper only, in double spacing and with adequate margins, and each page should be numbered. The top typed copy should be submitted, with four other copies. We are willing to receive one copy typewritten in the above format and accompanied by a disk (Microsoft Word version 98, Excel for tables and figures). All papers should be prefaced by a structured Abstract, of about 250 words in length. It should normally contain six clearly headed sections entitled Objective, Design, Setting, Subjects, Results and Conclusion. The name, appointment and place of work of the authors should be supplied on a separate title page. This same page should include the full postal address of one author, to whom correspondence and reprints will be directed. There should be adequate references to any relevant previous work on the subject; these references should appear at the end of the material on a separate page or pages, using the Vancouver style, which in the case of papers in journals includes:

- Surname and initials of author(s)
- Title of paper
- Full name of journal
- Year published
- Volume number
- Opening and closing page numbers

Reference to books should similarly include author's name and initials, full title, edition (if necessary), place of publication, publisher's name, year and, if required, volume number, chapter number or page number.

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CLINICAL PSYCHOLOGY SERVICE
AUDIT OF NEW REFERRALS**

Psychology No. <input style="width: 100%;" type="text"/>	Opt in: YES / NO	Age: 15 or under <input type="checkbox"/>	C
		16 - 64 <input type="checkbox"/>	A
		65 or over <input type="checkbox"/>	E
Date of Birth <input style="width: 100%;" type="text"/>	Sex <input style="width: 100%;" type="text"/>	Neuro <input type="checkbox"/>	N
		Physical Health <input type="checkbox"/>	PH

GP Name <input style="width: 100%;" type="text"/>	Practice Code <input style="width: 100%;" type="text"/>	
Date Referral Received <input style="width: 100%;" type="text"/>	Date of First Appointment Offered <input style="width: 100%;" type="text"/>	
Date of Last Appointment Offered <input style="width: 100%;" type="text"/>	Tick if Medico-legal report <input type="checkbox"/>	
No. of Times Attended <input style="width: 100%;" type="text"/>	No. of UTA's <input style="width: 100%;" type="text"/>	No. of DNA's <input style="width: 100%;" type="text"/>

Location For First Appointment

1. Joint Hospital <input type="checkbox"/>	7. Victoria Infirmary <input type="checkbox"/>
2. Clyde Unit, Joint Hospital <input type="checkbox"/>	8. Out-patient Clinic, VOLDGH <input type="checkbox"/>
3. Geriatric Day Unit, Joint Hospital <input type="checkbox"/>	9. Ward, VOLDGH <input type="checkbox"/>
4. Cottage Hospital <input type="checkbox"/>	10. Christie Ward <input type="checkbox"/>
5. Dumbarton Health Centre <input type="checkbox"/>	11. Home Visit <input type="checkbox"/>
6. Bank St <input type="checkbox"/>	12. Other : _____ <input type="checkbox"/>

Referred By (Profession/Agency)

1. Clinical Psychologist <input type="checkbox"/>	8. Medical Consultant <input type="checkbox"/>
2. Educational Psychologist <input type="checkbox"/>	9. Voluntary Agency <input type="checkbox"/>
3. Psychiatrist (name) _____ <input type="checkbox"/>	10. Community Team <input type="checkbox"/>
4. GP <input type="checkbox"/>	11. Speech Therapist <input type="checkbox"/>
5. Social Worker <input type="checkbox"/>	12. HMS Neptune <input type="checkbox"/>
6. Health Visitor <input type="checkbox"/>	13. Other <input type="checkbox"/>
7. Occupational Therapist <input type="checkbox"/>	

Psychologist

1. Daryl Foot <input type="checkbox"/>	<input type="checkbox"/>
2. Elizabeth Irvine <input type="checkbox"/>	<input type="checkbox"/>
3. Ishbel Dumughn <input type="checkbox"/>	<input type="checkbox"/>

Trainee/Assistant

1. Trainee <input type="checkbox"/>	<input type="checkbox"/>
2. Assistant <input type="checkbox"/>	<input type="checkbox"/>

Other Agencies Involved

Choose up to 4 categories

1. CPN <input type="checkbox"/>	<input type="checkbox"/>
2. Psychiatry <input type="checkbox"/>	<input type="checkbox"/>
3. Social Work <input type="checkbox"/>	<input type="checkbox"/>
4. OT <input type="checkbox"/>	<input type="checkbox"/>
5. Counsellor <input type="checkbox"/>	<input type="checkbox"/>
6. Voluntary Sector <input type="checkbox"/>	<input type="checkbox"/>
7. Other: _____ <input type="checkbox"/>	<input type="checkbox"/>

Appendix 1.3

EPPIC reason for care categories

Anxiety and Phobias
Post Traumatic Stress Disorder
Anger
Depression/Mood Disorder
Obsessional Disorder
Eating Disorder
Sleep Disorder
Personality Disorder
Behavioural/Movement Disorder
Addictions
Cognitive/Development/Speech Disorder
Problems related to Physical Illness
Sexual Disorder
Elimination
Relationship/Social Problems

Additional categories taken from EPPIC contributory factors

Abuse/Trauma
Psychiatric Illness

Appendix 1.4

The proportion of agreement for each EPPIC category was calculated by dividing the number of instances that the GP and the Psychologist agreed (Total no. of agreements), by the sum of that figure and the number of instances when the GP and Psychologist did not agree (Total no. of Disagreements). For reference see e.g., (15). The total number of disagreements per category constituted all occasions when the GP referred for the category in question but the Psychologist treated for a differing problem, and those occasions when the Psychologist treated for a category in question but the GP referred for a different problem. These data can be read off in *Table 2*. Proportions for each category were then expressed in percentage form.

% Agreement per

category =

Total no. of Agreements

Total no. of Agreements + Total no. of Disagreements x 100/1%

Anxiety	= 106/196 x 100% = 54.08%
OCD	= 3/6 x 100% = 50.00%
PTSD	= 7/17 x 100% = 41.18%
Depression	= 45/106 x 100% = 42.25%
Relationship/Social	= 16/75 x 100% = 21.33%
Cognitive/Development/Speech Disorder	= 7/15 x 100% = 46.67%
Anger	= 5/18 x 100% = 27.78%
Eating Disorder	= 13/19 x 100% = 68.42%
Behaviour/Movement Disorder	= 2/7 x 100% = 28.57%
Abuse/Trauma	= 24/40 x 100% = 60.00%
Physical health	= 5/11 x 100% = 45.45%
Sexual Dysfunction	= 5/10 x 100% = 50.00%
Personality Disorder	= 0/3 x 100% = 0.00%
Psychiatric Illness	= 0/2 x 100% = 0.00%
Addictions	= 0/4 x 100% = 0.00%
Sleep Disorder	= 1/4 x 100% = 25.00%
Unstated/other	= 0/16 x 100% = 0.00%

Appendix 2: Major Research Proposal

Appendix 2.1

Major Project Research proposal guidelines from D.Clin.Psy Course Handbook.

The research proposal should be laid out according to the format described below. This format is based upon the application for a mini-project grant in Health Services Research (SOHHD- Chief Scientist Office). Trainees may find that forms provided by ethical committees are substantially similar to this and this may be an acceptable alternative format.

1.1 Applicants

Names and addresses including the names of co-workers and supervisor(s) if known.

1.2 Title

No more than 15 words.

1.3 Summary

Of less than 300 words, including a reference to where the study will be carried out.

1.4 Introduction

Of less than 600 words summarising previous work in the field, drawing attention to gaps in present knowledge and stating how the project will add to knowledge and understanding.

1.5 Aims and hypotheses

These should wherever possible be stated as a list of questions to be answered to which answers will be sought.

1.6 Plan of investigation

Consisting of a statement of the practical details of how it is proposed to obtain answers to the questions posed.

1.6.1 Subjects

A brief statement of inclusion and exclusion criteria and anticipated number of participants.

1.6.2 Measures:

A brief explanation of interviews/observations/rating scales etc. to be employed, including references where appropriate

1.6.3 Design and procedure

A brief explanation of the overall experimental design with reference to comparisons to be made, control populations, timing of measurements, etc. A summary chart may be helpful to explain the research process.

1.6.4 Settings and equipment

A statement on the location(s) to be used and resources or equipment which will be employed (if any).

1.6.5 Data analysis

A brief explanation of how data will be collected, stored, and analysed.

1.7 Practical Applications

The applicants should state the practical use to which the research findings could be put.

1.8 Timescales

The proposed starting date and duration of the project.

1.9 Ethical Approval

Stating whether this is necessary and, if so, whether it has been obtained.

Appendix 2.2

Pittsburgh Sleep Quality Index

Name _____ Date _____ ID No _____

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 of the questions.

1. During the past month, when have you usually gone to bed at night?

USUAL BED TIME _____

2. During the past month, how long (in minutes) has it taken you to fall asleep each night?

NUMBER OF MINUTES _____

3. During the past month, when have you usually got up in the morning?

USUAL GETTING UP TIME _____

4. During the past month, how many hours actual sleep did you get at night (this may be different than the number of hours you spend in bed).

HOURS OF SLEEP PER NIGHT _____

For each of the remaining questions, check the one best response. Please answer all the questions.

5. During the past month, how often have you had trouble sleeping because you

(a) Cannot get to sleep within 30 minutes

Not during the past month _____	Less than once a week _____	Once or twice a week _____	Three or more times a week _____
------------------------------------	--------------------------------	-------------------------------	-------------------------------------

(b) Wake up in the middle of the night or early morning

Not during the past month _____	Less than once a week _____	Once or twice a week _____	Three or more times a week _____
------------------------------------	--------------------------------	-------------------------------	-------------------------------------

(c) Have to get up to use the bathroom

Not during the past month _____	Less than once a week _____	Once or twice a week _____	Three or more times a week _____
------------------------------------	--------------------------------	-------------------------------	-------------------------------------

(d) Cannot breathe comfortably

Not during the past month _____	Less than once a week _____	Once or twice a week _____	Three or more times a week _____
------------------------------------	--------------------------------	-------------------------------	-------------------------------------

(e) Cough or snore loudly

Not during the past month _____	Less than once a week _____	Once or twice a week _____	Three or more times a week _____
------------------------------------	--------------------------------	-------------------------------	-------------------------------------

(f) Feel too cold

Not during the past month _____	Less than once a week _____	Once or twice a week _____	Three or more times a week _____
------------------------------------	--------------------------------	-------------------------------	-------------------------------------

(g) Feel too hot
 Not during the past month _____ Less than once a week _____ Once or twice a week _____ Three or more times a week _____

(h) Had bad dreams
 Not during the past month _____ Less than once a week _____ Once or twice a week _____ Three or more times a week _____

(I) Have pain
 Not during the past month _____ Less than once a week _____ Once or twice a week _____ Three or more times a week _____

(j) Other reason(s), please describe _____

6. During the past month, how would you rate your sleep quality overall?

Very good _____
 Fairly good _____
 Fairly bad _____
 Very bad _____

7. During the past month, how often have you taken medicine (prescribed or “over the counter”) to help you sleep?

Not during the past month _____ Less than once a week _____ Once or twice a week _____ Three or more times a week _____

8. During the past month, how often have you had trouble staying awake while, driving, eatings meals, or engaging in social activity?

Not during the past month _____ Less than once a week _____ Once or twice a week _____ Three or more times a week _____

9. During the past month, how much of a problem has it been for you to keep up enough enthusiasm to get things done?

Not a problem at all _____
 Only slight problem _____
 Somewhat of a problem _____
 A very big problem _____

10. Do you have a bed partner or roommate?

No bed partner or roommate _____
 Partner/roommate in other room _____
 Partner in same room, but not same bed _____
 Partner in same bed _____

Appendix 2.3

The Sleep History Questionnaire

1. Nature of Sleep-Wake problem

Do you have a problem with falling asleep?	No	Mild	Moderate	Severe
Do you have a problem with staying asleep?	No	Mild	Moderate	Severe
Do you have a problem with waking up too early in the morning?	No	Mild	Moderate	Severe
Do you have a problem with staying awake during the day?	No	Mild	Moderate	Severe

2. Current Sleep-Wake Schedule

What is your usual bedtime on weekdays ? _____ o'clock
 At what time do you last awaken in the morning? _____ o'clock
 What is your usual rising time on weekdays? _____ o'clock
 Do you have the same sleep-wake schedule on weekends? YES NO
 How often do you take naps (including unintentional naps)? _____ days/week
 Do you ever fall asleep at inappropriate times/places? YES NO
 How many nights/week do you have a problem with falling asleep/staying awake? _____ nights

On a typical night (past month), how long does it take you to fall asleep after you go to bed & turn the lights off? _____ hours _____ min
 On a typical night (past month) how many times do you wake up during the middle of the night? _____ times

What wakes you up at night? Pain Child Noise Spontaneous

On a typical night, how long do you spend awake in the middle of the night (total no. of min/hrs for all awakenings)? _____ hours _____ min
 How many hours of sleep per night do you usually get ? _____ hours _____ min

3. Sleeping Aids

In the past 4 weeks have you used sleeping pills? YES NO
 Which drugs?
 Which dosage?
 How many nights/week?
 If no, have you ever?
 When did you first use sleep medication?
 When did you last use sleep medication?

In the past 4 weeks have you used alcohol as a sleep aid? YES NO
 If no, have you ever? YES NO

4. History of Sleep Problems (onset, course, duration)

How long have you been suffering from insomnia? _____ years _____ months

Were there any stressful life events related to it's onset

(e.g. death of a loved one, divorce, retirement, medical or emotional problems etc)?

Gradual or sudden onset?

What has been the course of your insomnia problem since it's onset (e.g. persistent, episodic, seasonal etc)?

5. Bedroom Environment

Are you sleeping with a bed partner?	YES	NO
Do you have a TV, radio, phone in your bedroom?	YES	NO
Is there a desk with paperwork to be done in the room?	YES	NO
Do you read in bed before bedtime?	YES	NO

What is your room temperature at night? Hot Warm Cool Cold

6. Lifestyle

How many times per week do you exercise?	_____ times per week
Do you sometimes exercise prior to bedtime?	YES NO
How many caffeinated drinks do you drink per day?	_____ per day
How many cigarettes per day do you smoke?	_____ per day
How many units of alcohol per day do you drink?	_____ per day

7. General

What is your pre-bedtime routine like?

What do you do when you can't fall asleep, or return to sleep?

Is your sleep better/worse/same when you go away from home?

Is your sleep better/worse/same on weekends?

What types of factors exacerbate your sleep problem (e.g. stress at work, travel plan etc)?

What types of factors improve your sleep?

How concerned are you about sleep/insomnia?

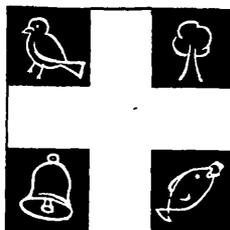
What impact does insomnia have on your life (e.g. mood, alertness, performance)?

How do you cope with these daytime sequelae?

Have you received treatment in the past other than sleeping aids?

If you would like to add any further information that you think would be relevant, then please do so in the space provided below.

Thank-you.



**GREATER GLASGOW
PRIMARY CARE
NHSTRUST**

Ref: AmcM/0016

3 August, 2000

Dr N Broomfield
Academic Centre
Gartnavel Royal Hospital
1055 Gt Western Road
Glasgow
G12 0XH

Dear Dr Broomfield

PROJECT: *Insomnia and paradoxical intention - an experimental investigation using actigraphic measurement of sleep*

Many thanks for sending the required amendments for the above named submission to the Research Ethics Committee. I am pleased to be able to tell you that the Committee has no objections from an ethical point of view, to this project proceeding and ethical approval is formally granted.

Before your project commences you will also require to obtain management approval via the Research & Development Directorate, Gartnavel Royal Hospital.

I would also like to take this opportunity to remind you that you should notify the Committee if there are any changes, or untoward developments, connected with the study – the Committee would then require to further reconsider your application for approval. The Committee expect to receive a brief regular update every 6 months, and then a brief final report on your project when the study reaches its conclusion. (Failure to keep the Committee abreast of the status of the project can eventually lead to ethical approval being withdrawn).

May I wish you every success with your study.

Yours sincerely

A W McMAHON
Administrator – Research Ethics Committee

Appendix 3: Major Research Project

*Information for Contributors**Submission of Papers*

Authors are requested to submit their original manuscript and figures with two copies. Manuscripts for the regular section should be sent to Dr S. Rachman, Department of Psychology, University of British Columbia, Vancouver, British Columbia, Canada, V6T 1Z4. Manuscripts for the *Behavioral Assessment* Section should be sent to Dr S. Taylor, Department of Psychiatry, 2255 Westbrook mall, Vancouver, British Columbia, Canada, V6T 2A1.

Submission of a paper implies that it has not been published previously, that it is not under consideration for publication elsewhere, and that if accepted it will not be published elsewhere in the same form, in English or in any other language, without the written consent of the publisher.

Manuscript Preparation

General: Manuscripts must be typewritten, double-spaced with wide margins on one side of white paper. Good quality printouts with a font size of 12 or 10 pt are required. The corresponding author should be identified (include a Fax number and E-mail address). Full postal addresses must be given for all co-authors. Authors should consult a recent issue of the journal for style if possible. An electronic copy of the paper should accompany the final version. The Editors reserve the right to adjust style to certain standards of uniformity. Authors should retain a copy of their manuscript since we cannot accept responsibility for damage or loss of papers. Original manuscripts are discarded one month after publication unless the Publisher is asked to return original material after use.

Abstracts: A summary, not exceeding 200 words, should be submitted on a separate sheet in duplicate. The summary will appear at the beginning of the article.

Keywords: Authors should include up to six keywords with their article. Keywords should be selected from the APA list of index descriptors, unless otherwise agreed with the Editor.

Text: Follow this order when typing manuscripts: Title, Authors, Affiliations, Abstract, Keywords, Main text, Acknowledgements, Appendix, References, Vitae, Figure Captions and then Tables. Do not import the Figures or Tables into your text. The corresponding author should be identified with an asterisk and footnote. All other footnotes (except for table footnotes) should be identified with superscript Arabic numbers.

References: All publications cited in the text should be present in a list of references following the text of the manuscript. In the text refer to the author's name (without initials) and year of publication, e.g. "Since Peterson (1993) has shown that . . ." or "This is in agreement with results obtained later (Kramer, 1994)". For 2-6 authors, all authors are to be listed at first citation, with "&" separating the last two authors. For more than six authors, use the first six authors followed by et al. In subsequent citations for three or more authors use author et al. in the text. The list of references should be arranged alphabetically by authors' names. The manuscript should be carefully checked to ensure that the spelling of authors names and dates are exactly the same in the text as in the reference list.

References should be prepared carefully using the *Publication Manual of the American Psychological Association* for style as follows:

Birbaumer, N., Gerber, D., Miltner, W., Lutzenberger, W., & Kluck, M. (1984). Start with biofeedback and continue with behavior therapy in migraine. *Proceedings of the 15th Annual Meeting of Biofeedback Society of America* (pp. 33-36) Albuquerque.

Gray, J.A. (1976). The behavioral inhibition system: a possible substratum for anxiety. In M. P. Feldman & A. Broadhurst. *Theoretical and experimental bases of the behaviour therapies* (pp. 3-41). London: Wiley.

Taber, I.I., McCormick, R.A., Russo, A.M., Adkins, B.J., & Ramirez, L.F. (1987). Follow-up of pathological gamblers after treatment. *American Journal of Psychiatry*, 144, 757-761.

Illustrations: All illustrations should be provided in camera-ready form, suitable for reproduction (which may include reduction) without retouching. Photographs, charts and diagrams are all to be referred to as "Figure(s)" and should be numbered consecutively in the order to which they are referred. They should accompany the manuscript, but should not be included within the text. All illustrations should be clearly marked on the back with the figure number and the author's name. All figures are to have a caption. Captions should be supplied on a separate sheet.

Line drawings: Good quality printouts on white paper produced in black ink are required. All lettering, graph lines and points on graphs should be sufficiently large and bold to permit reproduction when the diagram has been reduced to a size suitable for inclusion in the journal. Dye-line prints or photocopies are not suitable for reproduction. Do not use any type of shading on computer-generated illustrations.

Appendix 3.3

Sleep Anxiety Scale

Please complete the following questions regarding your sleep for a typical night *in the last week*. Indicate your answer by **circling one of the options** on the scale outlined.

1. For me, having difficulty falling asleep initially at night is

<i>a major worry</i>	<i>a minor worry</i>	<i>not a worry</i>
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2. For me, having difficulty getting back to sleep after waking at night is

<i>a major worry</i>	<i>a minor worry</i>	<i>not a worry</i>
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3. For me, waking up too early in the morning is

<i>a major worry</i>	<i>a minor worry</i>	<i>not a worry</i>
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4. For me, not feeling rested the next day is

<i>a major worry</i>	<i>a minor worry</i>	<i>not a worry</i>
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Now, please complete the following questions regarding your worries about not sleeping. Again, please indicate your answer by **circling one of the options** on the scale outlined.

1. When I go to bed, my sleeplessness worries me

<i>very much</i>	<i>some</i>	<i>not at all</i>
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2. When I wake up at night, my sleeplessness worries me

<i>very much</i>	<i>some</i>	<i>not at all</i>
------------------	-------------	-------------------

3. When I wake up early in the morning, my sleeplessness worries me

<i>very much</i>	<i>some</i>	<i>not at all</i>
------------------	-------------	-------------------

4. When I think about it during the day, my sleeplessness worries me

<i>very much</i>	<i>some</i>	<i>not at all</i>
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Appendix 3.4

Sleep Performance Anxiety Questionnaire

The following seven statements relate to your night-time sleep pattern *in the past week*. Please indicate by circling one response how true each statement is for you.

1. I put too much effort into sleeping at night when it should come naturally

Very much To some extent Not at all

2. I feel I should be able to control my sleep at night

Very much To some extent Not at all

3. I put off going to bed at night for fear of not being able to sleep

Very much To some extent Not at all

4. I worry about not sleeping if I am in bed at night and cannot sleep

Very much To some extent Not at all

5. I am no good at sleeping at night

Very much To some extent Not at all

6. I get anxious about sleeping before I go to bed at night

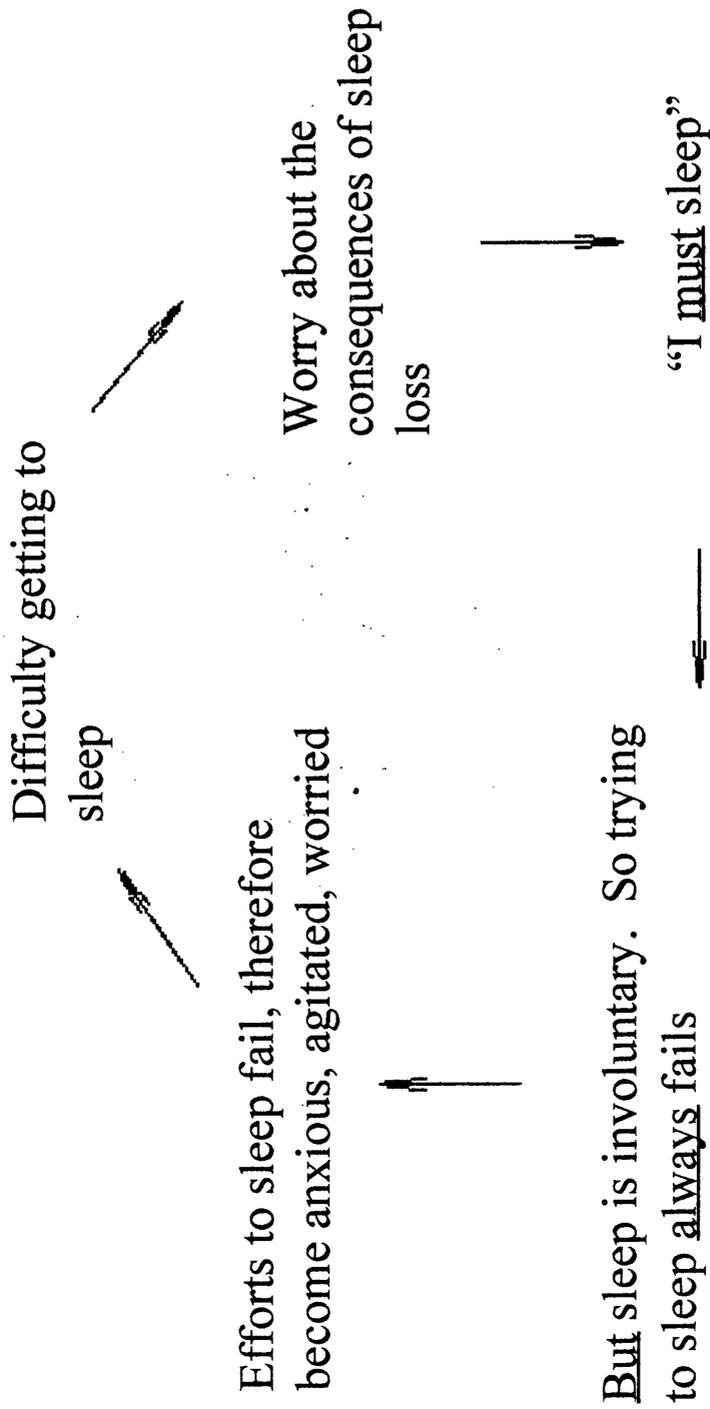
Very much To some extent Not at all

7. I worry about the long term consequences of not sleeping at night

Very much To some extent Not at all

Thank-you.

Appendix 3.9. Diagram outlining rationale of Paradoxical Intention (cf. Ascher & Turner, 1979)



Appendix 3.8

Instructions

Often, people who find sleeping difficult are so eager to fall asleep that they try too hard. Unfortunately, sleep is a natural process which happens involuntarily. So you cannot make yourself fall asleep- it must happen naturally. And since sleep is involuntary, trying too hard to get to sleep will always fail. This can cause worry about not being able to sleep, which in turn makes trying to sleep even more difficult.

So, the more you try to control sleep, the less you are likely to get to sleep. This then makes you more worried about not sleeping, and in turn your sleep problem gets even worse. So by giving up trying to sleep, your sleep pattern should improve.

Therefore, in this experiment, we want you to give up trying to fall asleep when you go to bed. Instead, we want you to try to stay awake. Paradoxically, staying awake should help you get to sleep more quickly because it stops you worrying, and it stops you trying. So, for the next 14 nights, give up trying to sleep and instead, try to stay awake. Here's how to do it.

1. As you go to bed tonight, lie down in a comfortable position and put the lights out.
2. In the darkened room, try to stay awake with your eyes open. Each time they feel like closing tell yourself "it would be good to keep them open for another little while".
3. Congratulate yourself on your success at remaining awake. Remind yourself that staying awake will help you get to sleep quicker.
4. If at any stage you feel worried or irritable at not sleeping remind yourself "the plan is to remain awake so I'm doing fine."
5. Try to stay awake as long as you can.
6. Do not, however, use active methods to stay awake such as reading or physical movement. The idea is to resist sleep-onset gently but persistently.

Remember, the above instructions take time to have an effect on your sleep. Patience and perseverance are essential. Do not expect to see any improvement during Week 1 of the experiment, but by the beginning of Week 2 you will begin to see dramatic improvements, and you will be relaxing yourself to sleep.

Please continue to wear your actigraph wristwatch and complete your sleep dairies for the next 14 nights. Good luck.

Appendix 3.9

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5. Try to stay awake as long as you can.
6. Do not, however, use active methods to stay awake such as reading or physical movement. The idea is to resist sleep-onset gently but persistently.

Remember, with patience and perseverance, the above instructions should have an immediate effect on your sleep. You will begin to see immediate and dramatic improvements as soon as you start to follow these instructions, and begin to relax yourself to sleep.

Please continue to wear your actigraph wristwatch and complete your sleep dairies for the next 14 nights. Good luck.

Appendix 3.10

Instructions

In order for us to understand sleep problems in more detail, we want you to continue to wear your actigraph wristwatch and complete your sleep dairies.

Thank you.

Appendix 3.11

THE GOOD SLEEP GUIDE

During the evening

1. Put the day to rest. Think it through. Tie up "loose ends" in your mind and plan ahead. A notebook may help.
2. Take some light exercise early in the evening. Generally try to keep yourself fit.
3. Wind down during the course of the evening. Do not do anything that is mentally demanding within 90 minutes of bedtime.
4. Do not sleep or doze in the armchair. Keep your sleep for bedtime.
5. Do not drink too much coffee or tea and only have a light snack for supper. Do not drink alcohol to aid your sleep- it usually upsets sleep.
6. Make sure your bed and bedroom are comfortable- not too cold and not too warm.

At Bedtime

1. Go to bed when you are "sleepy tired" and not before.
2. Do not read or watch TV in bed. Keep these activities for another room.
3. Set the alarm for the same time every day- 7 days a week, or at least until your sleep pattern settles down.
4. Put the light out when you get into bed.
5. Let yourself relax and tell yourself that "sleep will come when it's ready". Enjoy relaxing even if you don't at first fall asleep.
6. Do not try to fall asleep. Sleep is not something you can switch on deliberately but if you try to switch it on you can switch it off!

If you have problems getting to sleep

1. Remember that sleep problems are quite common and they are not as damaging as you might think. Try not to get upset or frustrated.
2. If you are awake in bed for more than 20 minutes, get up and go into another room.
3. Do something relaxing for a while and don't worry about tomorrow. People usually cope quite well even after a sleepless night.
4. Go back to bed when you feel "sleepy tired".
5. Remember the tips from the section above and use them again each time you waken up.
6. A good sleep pattern may take a number of weeks to establish. Be confident that you will achieve this in the end by working through the "GOOD SLEEP GUIDE".

