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The feasibility of using brief cognitive behavioural therapy for depression associated with Parkinson's disease

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Award date:
2003

Awarding institution:
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**The Feasibility of Using Brief Cognitive Behavioural
Therapy for Depression Associated
with Parkinson's disease**

by

DR KRISTINA COLE

A thesis submitted to the Department of Clinical Psychology

University of Wales, Bangor

For the degree of

DOCTOR OF CLINICAL PSYCHOLOGY

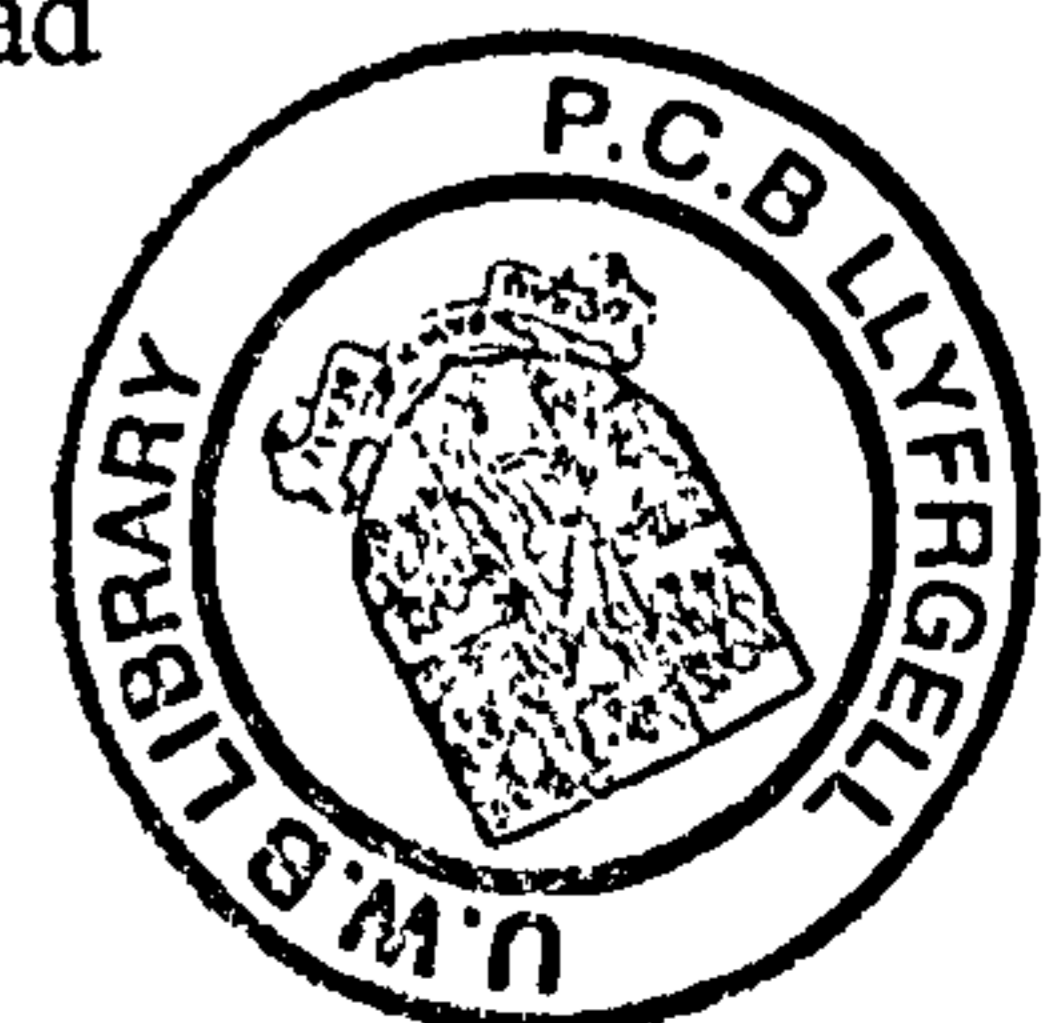
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School of Psychology

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43 College Road

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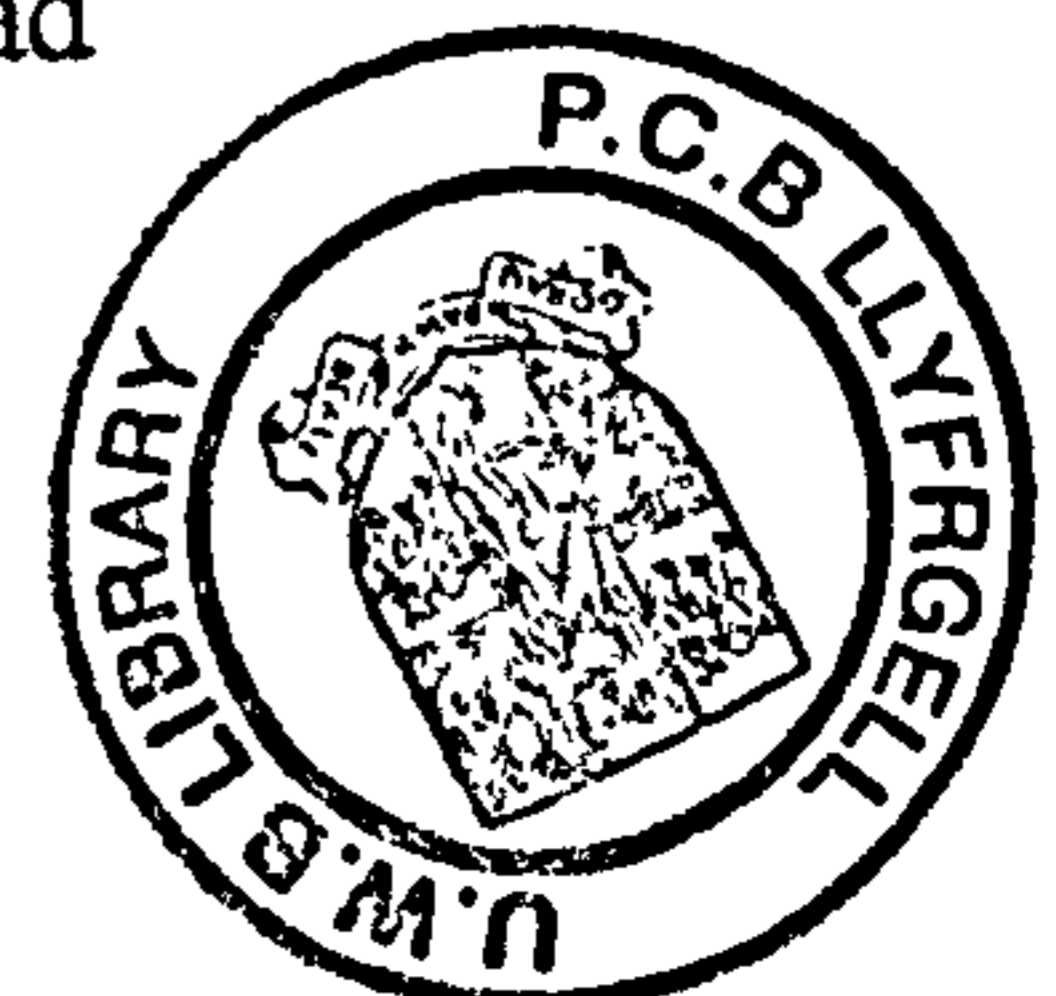
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The Feasibility of Using Brief Cognitive Behavioural Therapy for Depression Associated with Parkinson's disease

People with depression in Parkinson's disease are rarely offered therapeutic assistance. This study set out to investigate the efficacy of an eight-session structured cognitive behavioural intervention in reducing symptoms of depression. Five depressed patients (age range 54 to 82) attending a North Wales movement disorder clinic agreed to participate. Outcome was assessed using mood and quality of life inventories (Beck Depression Inventory; Geriatric Depression Scale; Parkinson's Disease Quality of Life Questionnaire). Four individuals demonstrated a clinically reliable reduction of symptoms according to the GDS scores, with greater improvement reported for the two individuals with more severe pre-therapy levels of depression. The BDI findings suggested most of the improvement related to the cognitive dimensions of guilt, pessimism and failure. The change in mood was not generally accompanied by an enhancement of perceived quality of life, and little variation in the frequency of activities was reported over the course of therapy. The difficulty in challenging rational cognitions and organic factors are highlighted as key issues in the psychological treatment of depression for this population.

ACKNOWLEDGEMENTS

In our curious world of mistaken values it is easy to overlook the incredible achievement of people who live with the adversity of chronic illness. I would like to thank all the people with Parkinson's disease and their carers who made this research possible. I appreciate the assistance of the following individuals based at the Movement Disorder Clinic, Dr Jolyan Meara, Dr Peter Hobson, and Sally Roberts. For offering emotional support when it was most needed I would like to express gratitude to Dr Frances Vaughan my supervisor. Last but not least, to all the gang at the Acquired Brain Injury Unit, thanks for making me laugh!

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RESEARCH PROTOCOL

The Effectiveness of Brief Cognitive Behaviour Therapy in Reducing the Symptoms of Depression in Individuals with Parkinson's disease.

The potential value of addressing this issue

At the present time patients with depression in Parkinson's disease (PD) are not routinely offered psychological therapy in the form of cognitive behaviour therapy (CBT). Evidence that CBT can reduce depressive symptoms would highlight a treatment option for individuals coping with this chronic condition.

Introduction

Depression is the most common neuropsychiatric complaint in PD (Aarsland et al., 1999). Although prevalence estimates vary in accord with the measures used, one local community based study identified depression in 64% of PD patients (Meara, Michelmore, & Hobson, 1999). Studies indicate that depression in PD is often not treated (Cummings & Masterman, 1999). Even when pharmacological approaches have been used they do not seem to be effective for all patients (Cummings, 1992). Untreated depression is likely to lead to poor outcome for the patients in terms of increased disability, morbidity and mortality (Cummings & Masterman, 1999).

Opinions differ regarding the factors thought to contribute to depression in PD. One approach maintains that affective symptoms are linked to the neuropathology of PD (Tandberg, et al., 1996; Mayberg & Solomon, 1995). Another view argues that depression represents a reaction to the changes in lifestyle imposed by physical impairments (Sharag, Jahanshahi, & Quinn, 2001). The question of whether depression in PD is biologically or socially mediated should not preclude attempts to try non-pharmacological treatment strategies.

Cognitive behaviour therapy is a structured, problem orientated approach, which encourages collaboration between therapist and patient. CBT is based on a theory expounded by Aaron T Beck which proposes that during depression people tend to perceive events in an overly negative or distorted manner (Beck, 1995). These appraisals are thought to determine how the individual feels and behaves.

The process of CBT, which commonly includes evaluating alternative solutions to problems and identifying patterns of helpful rather than depressive thinking, suggests that the approach is ideally suited to treating depression linked to medical conditions (Laidlaw, 2001). Literature shows that CBT has been used to facilitate adjustment to a range of health related concerns including pain, chronic fatigue and palliative care (White, 2001; Keelfe, Jacobs & Siddle, 1997; Edelman & Kidman, 1999). Watt & Cappeluz (2000) demonstrated that brief CBT was able to reduce symptoms of depression in patients in the early stages of organic illness. However the efficacy of CBT in treating depression in PD has not been established.

Objectives of the study.

The primary aim of the study is to ascertain whether brief cognitive behaviour therapy can reduce symptoms of depression in patients with PD. Secondly, the study aims to evaluate whether improvement can be maintained a month following cessation of therapy. The hypothesis predicts that there will be a decline in symptoms of depression following brief CBT. The decline will be represented by lower scores on the Beck Depression Inventory and Geriatric Depression Scale in the post therapy and follow up assessments compared to pre treatment scores. The hypothesis predicts that there will be an improvement in the quality of life as measured by the Parkinson's Disease Quality of Life Questionnaire which will be represented as higher scores on items post treatment.

Design

A feasibility study using a repeated measures design will be employed. A sample of up to twenty participants will be asked to complete the Beck Depression Inventory (BDI), the Geriatric Depression Scale (GDS) and the Parkinson's Disease Quality of Life Questionnaire (PDQL) on three occasions, a) pre-therapy, or before the start of the therapy program, b) post-therapy, or after the final therapy session, and, c) follow up, or one month after therapy has finished. Participants may be asked to provide information on a small range of clinically relevant symptoms before during and after the therapeutic intervention (e.g. how low they have been feeling today).

Participants.

Males and females with scores of 5 or more on the GDS, who are currently attending the Parkinson's disease Clinic based at Ysbyty Glan Clwyd will be approached by letter. All patients will have received a diagnosis according to the UK Brain Bank criteria for probable Parkinson's disease. Physical disability levels will vary. Disability will have been assessed by a medical practitioner using the Unified Parkinson's Disease Rating Scale (UPDRS). Some patients may be taking medication for symptoms of Parkinson's disease or depression. Participants who are receiving drug treatment will not be excluded from the trial.

Measures.

The Geriatric Depression Scale (GDS-15, Yesavage et al., 1983). The GDS-15 is a short inventory designed to measure symptoms of depression commonly observed in the older adult population. Research comparing the GDS with clinical diagnoses has established the inventory as a reliable measure of depression in older adults (Lam & Power, 1991). As the Parkinson's disease clinic use the GDS as a routine screening measure, the use of the same inventory would provide clinicians with a recognizable gauge to evaluate the effectiveness of psychological treatment.

The Beck Depression Inventory (BDI-II, Beck & Steer, 1993). The BDI is a 21 item validated self-report inventory measuring symptoms commonly associated with depression. The BDI is a key research tool used to measure outcomes following CBT in late life depression and chronic illness (Laidlaw, 2001). The inclusion of the scale in this study would enable the investigator to examine whether outcomes for CBT in PD compare with outcomes for other chronic illness groups.

Parkinson's Disease Quality of Life Questionnaire (PDQL; Hobson, Holden & Meara, 1999). The PDQL is a 37 item self administered questionnaire containing four subscales: Parkinson symptoms, systemic symptoms, social functioning and emotional functioning. Hobson, Holden and Meara (1999) found the anglicized version of the PDQL was a useful disease-specific measure with good convergent and discriminative validity.

Method

Prospective participants will be sent an approach letter explaining the reason for contact. During a pre research home visit the investigator will outline the aims of the study with the aid of a patient information sheet. The issue of consent will be explained and a second home based appointment will be arranged. No financial reward will be made for participation. The GP will be informed of their patient's participation in the project.

Content and structure of the CBT intervention

Subject to consent, the individual intervention will begin. The program will comprise of eight therapy sessions of approximately one-hour duration. The content of program will be based on the core principles of Cognitive Behaviour Therapy (CBT) as expounded by Beck (1976). The patient will be provided with the booklet 'Coping with Depression When you Have Parkinson's Disease' (Beck, 2000). This short simple booklet describes the main components of cognitive therapy in the context of Parkinson's disease. The structure of the sessions will be guided by the following headings outlined in the booklet;

- How should I change my behaviour
- How should I change my depressive thinking
- How do I solve my problems
- How can I help myself follow my doctor's advice?

The initial session will aim to build a rapport with the participant by providing the opportunity to talk through salient concerns. The plan for subsequent sessions will be discussed. The first two therapy sessions will focus on changing behaviour by encouraging the introduction of appropriate activities. The following two sessions will look at the process of changing depressive thinking. The next session will look at problem solving various situations pertinent for the individual. The final session will review what has been done and consider the future expectations and strategies for coping with depression in Parkinson's disease. A follow up session will be provided one month after the termination of therapy. Following data analysis, the investigator will forward a

short report providing general feedback for the group outcome to each participant. No information on individual participants will be made available.

Data analysis

The outcome data will be checked and scored by a research assistant. The data analysis will be computed using SPSS. Standardised statistical techniques will be used, which, depending on sample size, will include non parametric tests for repeated measures (e.g. Freidmans and Wilcoxon). Information on clinical relevant symptoms may be presented descriptively.

Benefits and hazards.

CBT will teach the participants various techniques to facilitate coping with depression and may result in an improvement in mood. The results will help clarify whether CBT is a viable treatment option for this clinical group. Some participants could assume therapy will 'cure' their depression. However, as the title of the booklet implies, the therapist will emphasis the principle of managing rather than eliminating depressive symptoms. Participants will be informed that they have the right to decline to take part at any stage and withdrawal will not affect the service they receive through the Parkinson's disease Clinic. Participants who are prescribed antidepressant medication during the course of the research will still receive the full psychological intervention.

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Coping with Depression When You Have Parkinson's Disease



For Cognitive Therapy
and Research

Judith S. Beck, Ph.D.

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Revised 2000

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The Geriatric Depression Scale

15 item version

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FDQL

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Daily Mood Rating

Date from To

Keeping a track of your mood can really help to see if there are day to day patterns of feeling better or worse. It can also help to provide a way to get back to the thought which may lie behind the mood changes both those that made you feel low and those that made you feel good.

Please rate (or ask your carer to rate) how good or bad you felt each day on the line below. If you felt good put a pen mark near the start of the line. If you felt "so-so" mark near the middle and if you felt low or depressed mark near the end of the line.

Day one

very happy "so-so" very low

Day two

very happy "so-so" very low

Day three

very happy "so-so" very low

Day four

very happy "so-so" very low

Day five

very happy "so-so" very low

Number of Activities each day

Date from To

Can you write (or ask your carer to write) a list of the things you do on a usual day

.....

.....

.....

Can you or your carer record the number of activities you did during each day in the boxes below (e.g. getting the breakfast ready, tidy the house, going out).

Day one

Day two

Day three

Day four

Day five



**Ymddiriedolaeth GIG Siroedd Conwy a Dinbych
Conwy & Denbighshire NHS Trust**

Tim Gogledd Cymru I Bobl Sydd Wedi Cael Anaf I'r Ymennydd
Ffôn: 01492 807766/807521 Ffacs: 01492 516587
North Wales Team for People with an Acquired Brain Injury
Tel: 01492 807766/807521 Fax: 01492 516587

CONSENT FORM

Title of Project

The effectiveness of Brief Cognitive Behaviour Therapy in reducing the symptoms of depression in individuals with Parkinson's disease.

Name of researcher

Dr Kristina Cole (under the supervision of Dr Frances Vaughan)

Please delete as appropriate

- 1. I confirm that I have read and understand the information sheet for the above study and have had the opportunity to ask questions Yes / No
- 2. I understand that my participation is voluntary and that I am free to withdraw at any time, without giving any reason, without my medical care or legal rights being affected. Yes / No
- 3. I understand my GP will be informed that I am taking part in this study, and I have seen a copy of this letter. Yes / No
- 4. I agree to take part in the above study. Yes / No

<hr/>	<hr/>	<hr/>
Name of Patient	Date	Signature

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Name of person taking consent (if different from the researcher)	Date	Signature

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Name of person taking consent	Date	Signature



**Ymddiriedolaeth GIG Siroedd Conwy a Dinbych
Conwy & Denbighshire NHS Trust**

INFORMATION SHEET

The effectiveness of Brief Cognitive Behaviour Therapy in reducing the symptoms of depression in individuals with Parkinson's disease.

You are being asked to take part in the above study. The study has been reviewed by the North Wales Health Authority Research Ethics Committee, and the University of Wales Ethics Committee, Bangor. Before you decide it is important for you to understand why the research is being done and what it will involve. Please take time to read the following information carefully and discuss it with friends, relatives and your GP if you wish. Ask your psychologist when she calls, if there is anything that is not clear or if you would like more information. Take time to decide whether you would be willing to take part.

What the research is about

Long-term illnesses are sometimes difficult to come to terms with. We are interested in helping people cope with feeling low when they have Parkinson's disease. The aim of the study is to see if a type of therapy which has been shown to help people suffering with various illnesses such as long-term pain, can also help people who get depressed with Parkinson's disease.

Why have I been chosen?

You have been chosen because one of the forms you filled in while attending at the Parkinson's Disease Clinic suggested that there might be times when you feel low. We will be asking 20 people who attend the Parkinson's Clinic to take part in the study.

Do I have to take part?

It is up to you to decide whether or not to take part. If you do decide to take part you will be given this information sheet to keep and be asked to sign a consent form. You are able to withdraw from the therapy at any time without giving a reason. If you do withdraw it will **not** affect the care you are receiving through the Parkinson's disease clinic.



What will happen to me if I take part?

I (Kristina Cole) will visit you at your home for one hour per week for seven weeks, and then once, a month later to see how you have been getting on. To see if the therapy has helped, you will be asked to fill in three questionnaires three times, 1) on the first visit, 2) on the last therapy visit and 3) on the visit a month later. It is OK to allow relatives or friends help you to fill out the forms.

It would also be helpful to know how you have been doing each week. You may be asked to mention a couple of things (e.g. how low or tired you have been or what things Parkinson's disease has stopped you doing) in the two weeks before, during and after therapy.

What type of therapy is it?

You will be given a booklet to keep called

Coping with Depression When You Have Parkinson's disease

The booklet includes the following sections

How do I know if I am depressed?

What can I do?

How should I change my behaviour?

How do I change my depressed thinking?

How do I solve my problems?

The psychologist will work with you to try out various ideas mentioned in the booklet. These ideas have been shown to help people with various illnesses.

Are there any alternative treatments for feeling low?

Sometimes people who have been feeling low will be prescribed antidepressant medication by their GP or by the Doctors working at the Parkinson's Clinic. You can still participate in the research if you are taking any form of medication.

Are there any disadvantages to taking part?

A lot of people think that therapy can take all their problems away. This is not possible when living with a long-term illness. The aim of therapy is to provide people with ways of managing and coping with feeling depressed.

What are the benefits of taking part?

We hope that the therapy will help. However, as this therapy has not been tried with Parkinson Disease, this cannot be guaranteed. The

information we get from the study will help us know what type of treatments help patients with Parkinson's disease.

What happens when the therapy ends?

This therapy is only a short course to help you to cope with your depression. At the end of therapy various options may be discussed with you.

Who will know about the results?

All information will be treated as strictly confidential and will be kept in a locked filing cabinet at all times. If you agree to take part, you will be asked to sign a consent form, which will be kept in your clinical file. We will let your GP know in a letter what is happening, and send a copy of this information sheet. A report of the results for the whole group will be written by the psychologist and sent to you in the post during the summer.

Who is organizing and funding the research?

Dr Kristina Cole is organizing and conducting the research as part of her training for a Doctorate in Clinical Psychology based at the North Wales Clinical Psychology Course, University of Wales, 43 College Road, Bangor, LL57 2DG. (Contact no 01248 386235). No payment will be made for taking part in the study.

What will happen next?

- 1) Read through this information sheet carefully and talk it over with others if you wish.
- 2) I will return at a convenient time. If you have any further questions feel free to ask.
- 3) If you agree to take part, you will be asked to read the consent form and sign it. Arrangements will be made for the home therapy visits.
- 4) If you prefer not to take part, the care you receive through the Parkinson's Clinic will not be affected in any way.

If you would like any further information do not hesitate to contact me

Dr Kristina Cole
Tel.

THANK YOU FOR READING THIS INFORMATION SHEET



**Ymddiriedolaeth GIG Siroedd Conwy a Dinbych
Conwy & Denbighshire NHS Trust**

**The effectiveness of Brief Cognitive Behaviour Therapy in reducing the symptoms
of low mood and depression in individuals with Parkinson's disease.**

Dear Dr

Re

I am writing to inform you that your patient has agreed to participate in the above research study, and a consent form has been signed. This study received ethical approval from the North West Wales Health Authority Research Committee.

The study aims to evaluate whether brief cognitive behaviour therapy (CBT) can help patients with Parkinson's disease who are experiencing symptoms of depression. CBT has been demonstrated to be an effective psychological therapy for individuals coping with a range of medical conditions including palliative care and chronic pain. However it's efficacy with Parkinson's disease has not been established.

Please find enclosed the information sheet passed on to your patient, which outlines the main components of therapy. Participation will not interfere in any way with treatment as usual, received through your practice or the Parkinson's disease clinic. If you would like any more information, or if you have any questions about the study, please do not hesitate to contact me.

Yours sincerely

**Dr Kristina Cole
Trainee Clinical Psychologist**

**Dr Frances Vaughan
Clinical Psychologist.**

Contact telephone number before
After

October 1, 2002 - 01492 531332
October 1, 2002 - 01492 807773



**Ymddiriedolaeth GIG Siroedd Conwy a Dinbych
Conwy & Denbighshire NHS Trust**

**The effectiveness of Brief Cognitive Behaviour Therapy in reducing the symptoms
of low mood and depression in individuals with Parkinson's disease.**

Dear

Staff working at the Parkinson's disease clinic may have mentioned a research study which is aiming to use brief therapy to help people cope with feeling down in Parkinson's Disease. You are being asked if you would like to take part in the study.

I would like to offer you an initial home visit on

The aim of the visit would be to explain a bit about my self, what the therapy involves and to give you the opportunity to ask questions. If you would prefer me not to call, or the above time or date is not convenient please do not hesitate to contact me (Kristina Cole) on the number below. If you decide not to take part in the study the care you receive through the Parkinson's disease clinic will not be affected in any way.

Yours sincerely

Dr Kristina Cole
Trainee Clinical Psychologist

Dr Frances Vaughan
Clinical Psychologist

Contact telephone number before October 1, 2002 - 01492 531332
After October 1, 2002 - 01492 807773

**The Feasibility of Using Brief Cognitive Behaviour Therapy for
Depression Associated with Parkinson's disease: A Literature Review.**

Kristina Cole
University of Wales, Bangor

Frances Vaughan
University of Wales, Bangor

This review forms part of Doctoral thesis by the first author supported by the University of Wales Bangor, School of Psychology, 43 College Road, Gwynedd, LL57 2DG

Running head: Parkinson's disease, depression, cognitive behaviour therapy

**The Feasibility of Using Brief Cognitive Behaviour Therapy for
Depression associated with Parkinson's disease:
A Literature Review.**

Abstract

Medical personnel and researchers have called for more information on how to treat depression related to Parkinson's disease. Cognitive behaviour therapy (CBT) has been identified as the National Health Service treatment of choice for a range of psychological disorders and is increasingly applied to depression associated with chronic medical conditions. The present paper will review the relevant literature on CBT treatment effectiveness and the nature of depression in Parkinson's disease before suggesting how CBT might be adapted to assist this client group.

**The Feasibility of Using Brief Cognitive Behaviour Therapy
for Depression associated with Parkinson's Disease:
A Literature Review.**

The aim of this paper is to assess the feasibility of using cognitive behaviour therapy (CBT) in the treatment of depression in Parkinson's disease (PD) by reviewing the relevant literature. CBT has been successful in reducing symptoms of depression in people with diverse chronic and neurological conditions (Price & Cooper, 1999; Moorey & Greer, 1989; Morley, Eccleston & Williams, 1999), and medical specialists have suggested CBT might also be appropriate to use with depression associated with Parkinson's disease (Meara & Koller, 2000; Playfer & Hindle, 2001). However as there have been few studies endorsing the effectiveness of psychological therapies for PD depression, there is a need to examine research in related fields to see if the CBT treatment option for PD depression is likely to be effective.

There are sound clinical reasons for considering the merits of using CBT for depression associated with PD including; a) depression affects a substantial proportion of people with PD, b) PD depression is often not treated, and c) pharmacological treatment approaches do not always seem to work. Findings supporting each of these statements will be briefly reviewed before presenting an overview of the principle cognitive model of depression and compatible health psychology models. Secondly, the extent to which CBT has been helpful with client groups who share some features with people with PD will be outlined. Finally, literature on the nature of depression in PD will be covered along with the implications for using a cognitive intervention.

Brief Overview of Depression in Parkinson's disease

Parkinson's disease is the second most common neurodegenerative illness (Mastermann & Cummings, 1998). It is an age related disorder, and although some people can develop symptoms in the third decade, the average age of onset is reported to be seventy (Mutch & Lien, 2001). Characteristic symptoms include akinesia (difficulties with movement), rigidity and tremor. These cardinal features would appear to be related to the degeneration of dopaminergic cells within the substantia nigra, one of three brain nuclei comprising the basal ganglia, located in the subcortical region of the brain (Bergaman & Deuschl, 2002). In addition to disorders of movement, people can experience a range of neuropsychiatric complaints, the most common of which is depression (Aarsland et al., 1999).

Prevalence. Providing a reliable estimate of how many people with PD also have depression has proved difficult because studies have employed different assessment methods. However two meta analyses have noted prevalence rates of over 40 per cent for minor depression and 11 per cent for major depression (Cummings, 1992; Miyawaki, Meah, Kormos & Tarsy, 1996). These figures may underestimate the true extent of PD depression as symptoms of low mood such as psychomotor retardation and flat affect can be masked by those of PD, which implies a potential for missed diagnosis. Whatever the current scale of the problem, it would be fair to say that increases in the life expectancy of the general population will result in a rise in PD and associated problems such as depression.

There are data to suggest that people with PD are more likely to experience depressive symptoms than people with other chronic medical conditions. A study of over 200,000 patient records demonstrated that people with PD have a greater risk for developing depression than patients with arthritis or diabetes (Nilsson, Lars, Sorensen, Andersen & Bolwig, 2002). The severity of depression in PD has been found to be equal to or greater than that found in other neurodegenerative conditions (Levy et al., 1998; Gilley, 1990).

Treatment availability. Given the extent and potential consequences of living with depression, it is surprising to find that symptoms of low mood are rarely treated. Studies suggest between 50-93% of people with depression associated with PD do not receive any treatment, with pharmacotherapy being the only option offered to a minority of patients (Richard & Kulan, 1997; Cantello, Riccio, Scarzella, Leotta & Bergamasco, 1984; Lapane, Fernandez & Friedman 1999; Meara, Mitchelmore & Hobson, 1999). There are mixed opinions regarding the use of drugs to treat depression in PD with most reviews highlighting the lack of outcome data and the potential toxic effects of antidepressant medication as reasons to be cautious (Klaasen et al., 1995; Cummings & Masterman, 1999; NINDS, 2001; Burn, 2002). In short, there are substantial disease specific reasons why it would be useful to consider the role of psychological therapies for depression in PD.

Overview of Cognitive Behavioural Therapy

The cognitive model of depression. The most well known form of CBT was informed by a theory proposed by Aaron Beck. In the recent reformulation of the model, depression is characterized as an experience of primal loss associated with sadness,

deactivation, lack of motivation, and withdrawal (Beck, 1996; Clark, Beck & Alford, 1999). Beck maintains that biased information processing constitutes the key for understanding the disorder and that these biases have implications for how people interpret events which, in turn have repercussions for feelings and behaviour. Distorted interpretations were hypothesised to stem from core beliefs held in 'meaning making structures' or schemas, which are formed on the basis of past experience. Evidence for flawed processing is seen in negative automatic thoughts and errors in thinking such as 'over-generalisation', 'mind reading' and 'all-or-nothing thinking'. According to the model, depressed people hold distorted beliefs about themselves, the world around them and are pessimistic regarding the future (Beck, 1967; Wright & Beck, 1983). Although the role of cognitions in mediating depressive illness is fundamental to the cognitive model, Beck acknowledged that biological factors, personality traits and life stressors could contribute to the development of depression (Beck, Rush, Shaw & Emery, 1979).

Components of therapy. CBT for depression is a structured therapy which aims to help people to identify the maladaptive thoughts contributing to emotional discomfort and to replace them with more enabling alternatives (for more detail see, Beck et al. 1979; Padesky & Greenberger, 1995). This objective is achieved through a process of 'collaborative empiricism' which involves the therapist and client working together to explore the implications of various beliefs for the individual. Socratic questioning is the key medium used to guide the client towards identifying, evaluating and, if necessary, modifying thoughts (Padesky, 1993). Questioning can take the form of an inference chain or 'downward arrow', when the therapist asks a series of progressively penetrating questions to uncover maladaptive reasoning. Other features of cognitive therapy include

hypothesis testing of the accuracy of thoughts and evaluating the effect of holding dysfunctional and alternative beliefs on an individual's mood state. The approach also draws upon behavioural techniques such as activity scheduling, graded exposure and experiments to evaluate the authenticity of beliefs.

Mediators of affect. There is no research consensus indisputably identifying the mechanisms of change in CBT (Scott, 2001). Rather there are general assumptions, including the belief that improvement is achieved through the transformation of cognitive structures, the acquisition of new skills, or the consequence of activating more adaptive ways of interpreting experience (Beck, 1996; Gotlib & Hammen, 1992). Similarly, findings are inconsistent regarding which components of cognitive therapy alleviate low mood. Several studies have highlighted behavioural activation (Jacobson, et al. 1996; Malik, Beutler, Alimohamed, Gallagher-Thompson & Thompson, 2003), others have focused on the cognitive elements involved in appraising thoughts (Teasdale & Fennell, 1982), and finally some believe that non-specific elements of CBT such as the quality of the therapeutic alliance play a part in recovery (Garfield, 1998).

The average number and composition of sessions required to facilitate improvement varies over studies. Research findings imply that brief CBT may be as effective as long duration therapy for adults of all ages (Shapiro, Rees, Barkham, & Hardy, 1995; Gallagher-Thompson, Hanley-Peterson & Thompson, 1990), and both group and individual therapy have been shown to be helpful (Chambless & Ollendick, 2001). Even though cognitive therapy has been identified as the treatment of choice for a range of psychological problems (Department of Health, 2001), it does not necessarily follow that it is the best therapeutic option for every client. Factors thought to hinder

progress in CBT are entrenched personality styles, poor motivation, low acceptance of personal responsibility for change, and pessimism regarding therapy outcome (Ruddell & Curwen, 1997; Safran & Segal, 1996).

Other Health Psychology Models

Although Beck's model of psychopathology provides the theoretical foundation to CBT, other cognitive models have specifically addressed how people interpret the experience of illness. These theories offer a health psychology perspective which uphold the case for using a therapeutic intervention aimed at modifying unhelpful cognitions to alleviate depression.

The health belief model. This model was initially conceived by Rosenstock (1966) and highlights the importance of 'perceptions' of health threat in explaining actions. In the main, the approach has been used as a predictive model to explain why people do not engage in preventative behaviour to maintain good health (Sheeran & Abraham, 1996). The original model proposed that if an individual perceives the threat of disease to be credible, following cost- benefits' analysis of consequences, a particular health behaviour would be enacted. Applied to the experience of PD, a link with perceptions and behaviour could be explained in the following way. If an individual perceives a diagnosis of PD to be ominous, s/he may feel that, despite the benefits (e.g. 'at least PD isn't terminal') the costs are dire (e.g. 'there is no cure'), and may therefore choose not to comply fully with medical advice which will have consequences for morbidity.

Transactional model of stress and coping. Appraisal processes again play a crucial role in this theory which attempts to explain reactions to major stressors,

including neurological disease (Roberts, Towell, Golding, & Towell, 2001). Whether an individual becomes distressed is determined by whether an illness is perceived to be harmful, and the beliefs the individual holds about their capacity to cope with it (Lazarus, 1966). Coping is defined as 'cognitive and behavioural efforts to manage psychological distress' (Lazarus, 1993: p.237) and can take a number of forms including avoidance, distancing, positive reappraisal, controlling, seeking support, accepting responsibility, problem solving and confronting (Folkman, Lazarus, Dunkel-Schetter, DeLongis & Gruen, 1986). The coping strategy adopted may depend on the type of demands a particular illness places on the individual. For example, people with PD have reported difficulty adjusting to the symptoms of disease whereas people with chronic fatigue syndrome report a greater struggle with the acceptance of being ill (De Ridder, Schreurs & Bensig, 1998). Evidence suggests that some coping styles may be related to poor adjustment, and potentially to the development of depression (Maies, Leventhal, & De Riddler, 1996).

The self-regulatory model. Unlike the stress and coping theory, Leventhal, Nerenz and Steele's (1984) self-regulatory model was specifically conceptualised to explain adaptation to illness (Buick, 1997). The process of adjustment involves the individual regarding the illness as a problem to be solved. Like other health models, the theory highlights appraisal and coping mechanisms. However the theory differs from other approaches in the categorisation of factors thought to comprise the personal representation of illness. Secondly a dynamic interaction between three stages of processing (representation, coping, appraisal) is proposed. Five components are thought to make up an individual's cognitive representation of a disease, including:

- 1 The identity including the label (e.g. PD) and symptoms (e.g. tremor).
- 2 Ideas about how the disease was caused.
- 3 Belief about the consequences of illness.
- 4 Expectations concerning the duration of illness, or time-line.
- 5 Perceptions of whether the illness can be controlled or cured.

Individual differences in disease perception will occur because past experience and cultural values influence the content of each component of the representation (Leventhal, Diefenbach & Leventhal, 1992). The selection of a coping procedure will depend on the representation the individual has formed about the illness. Finally, the model proposes that people will appraise the effectiveness of their coping strategy and, if it is perceived to be unsuccessful, will select a different approach or change the representation of the illness.

The model has provided a framework to investigate responses to a range of dissimilar medical conditions (see Petrie & Weinman, 1997). Helder et al. (2002) applied the approach to evaluate perceptions, coping strategies and outcomes in Huntington's disease (HD). This neurological condition affects many of the same regions of the brain as PD, and like PD presents with both physical and cognitive decline over a number of years. When compared to the general population, people with HD were more likely to use mental disengagement and venting emotions as coping strategies. The overall findings indicated that negative perceptions/representations of illness and the coping methods of mental and behavioural disengagement were related to reports of poor mental health.

In summary, the cognitive behavioural model provides the rationale to explain the aims and processes of CBT. Health psychology models give direction to the practice of CBT by providing an awareness of the type of issues that people attempt to assimilate

when forming a cognitive interpretation of chronic illness. If aspects of a representation reflect a biased appraisal of the disease, or the cognitively mediated attempts to cope contribute to psychological distress, cognitive behavioural therapy could target these domains.

Outcome literature for CBT

While few would dispute the need for psychological interventions for PD depression, it would be legitimate to argue that CBT should only be considered if the evidence for its efficacy in treating depression is robust. Scott (1996) argues that CBT is “the most extensively researched psychological treatment for non psychotic, unipolar outpatient depressive disorder” (p.1). Roth and Fonagy (1996) conclude that CBT is an empirically validated treatment for a range of psychological problems including depression. Like most reviewers, the authors relied on the ‘gold standard’ meta analyses of randomized control trials (RCTs), followed by the guidelines for ‘well-established’ and ‘probably efficacious’ treatment to determine the effectiveness (see Chambless & Hollon, 1998). CBT studies involving four categories of depressed adults especially relevant to PD will be discussed here; people with medical problems, neurological disorders, older adults, and older adults with comorbid illness. Reported modifications to standard CBT will also be described.

CBT for depression associated with medical conditions. CBT is being applied to an ever-increasing range of medical problems, including illnesses with an enduring, fluctuating or terminal course. The basic components of cognitive therapy are invariably the same, but the issues addressed vary according to the nature of the illness being treated. The effectiveness of CBT for depression associated with chronic pain and

cancer is favourable (Morley, Eccleston & Williams, 1999; Antoni, Lehman, Kibourn, Boyers & Culver, 2001; Edelman & Kidman, 1999). For chronic fatigue syndrome, an illness associated with fluctuating patterns of severity and duration, CBT has been acknowledged as an acceptable treatment provided that the symptoms are not mild or the clients are not too disabled to attend outpatients (Price & Cooper, 2003).

CBT for depression associated with neurological problems. There are few studies looking at CBT for depression in neurological conditions, and only one study which used CBT with a PD client group. Dreisig, Beckmann, Wermuth, Skovlund and Bech (1999) compared outcomes for nine young patients who had received CBT with a treatment as usual control (n = 70). The intervention was not clearly described (e.g. "The written psychotherapeutic strategies are based mainly on cognitive/behavioural psychological theories" p. 219), and did not specifically target depression. Although the between group comparison demonstrated a significant improvement on a range of psychological dimensions for the treatment group, the authors did not comment on within group changes for depression. Given the fact that the majority of participants reported mild symptoms of depression before therapy (90%), it is difficult to determine from this study the extent to which a CBT programme purposely targeting depression in PD would be effective.

Mohr's research group have conducted two controlled trials for depression in multiple sclerosis. In the first study, an eight-week course of CBT administered over the telephone was shown to be more effective than usual-care (Mohr et al., 2000). In the second study, the CBT and the antidepressant medication interventions were superior to supportive therapy (Mohr, Boudewyn, Goodkin, Boston & Epstein, 2001).

Fleminger, Oliver, Williams & Evans (2003) have advocated CBT as a suitable medium for addressing depression following traumatic and acquired brain injury and case study reports seem to support this view (Williams et al., in press; Montgomery, 1995). Similarly preliminary evidence indicates people with inflammation or injury to the brain may benefit from a cognitive intervention (Evans & Williams, 2002). The findings for post-stroke depression are less encouraging with Lincoln and Flannaghan (2003) reporting no difference between CBT and treatment as usual.

Weighed on balance, the outcome literature suggests that CBT is a promising intervention for people with depression associated with medical problems, including some neurologically based disorders, and therefore could also be helpful for PD.

CBT with older adults. A consideration of older adult literature is relevant because the majority of people with PD are in late life. Laidlaw (2001) reports on the results of six meta-analyses for late life depression and reached the general conclusion that the rates of improvement tend to be comparable to those reported with younger adults.

Two studies using brief CBT illustrate the diversity of outcomes possible with older adults. In Rokke, Tomhave and Jocie (2000), ten-session CBT for depression was compared with an educational intervention. Clinically meaningful improvement in mood was demonstrated for both groups (CBT 71%; educational 61%) which was maintained at one year. The second study hinted that the success of CBT with older adults might depend on the severity of depression. Leung and Orrell (1993) evaluated a seven session CBT intervention and looked at the rates of improvement for people with a major depression episode (MDE) and other mood disorders. Ninety-two per cent of the MDE

group did not need any contact with mental health services one year on but 50% of the 'other disorder group' required further support.

CBT for older people with medical and neurological conditions. Despite the fact that an increased association with physical disease has been the only identified factor distinguishing older from younger adult depression (Zeiss & Breckenridge, 1997), there has been little research looking specifically at the consequences of therapy for older adults with medical problems. One exception is Kemp, Corgiate and Gill (1992) who compared CBT for depression in older adults with and without physical illness. The outcome data demonstrated a 40% improvement in mood in both groups, despite no change in levels of functional capacity. Unfortunately the improvements for the disabled group were not maintained over time, a factor which was put down to further deterioration in health.

There is a strong overlap between the illness experiences of people with dementia and PD because both disorders typically occur in older adults and the symptoms are caused by biological decline. Two studies suggest CBT might be helpful in reducing depression when neurological deterioration is taking place. Watt and Cappeluz (2000) reported that CBT combined with reminiscence resulted in a 33% improvement in depressive symptoms for a group in the early stages of organic illness. Scholey and Woods (2003) reported on the outcome and processes of therapy for a case series. On average there was a statistically significant improvement in mood. However the degree of change varied between individuals, with only two of the seven participants demonstrating a clinically meaningful level of improvement.

Taken together the literature on older adults with medical problems suggests CBT can lead to a reduction in depressive symptoms but there are individual differences in treatment responsiveness and it may be difficult to sustain improvement when the individual is coping with ongoing illness and further deterioration.

Modifications to CBT for older adults and people with medical conditions.

As stated previously, the fundamental components of CBT are present in all cognitive intervention packages. Modifications refer to adjustments in the way these core components are delivered. As older adults often benefit to the same degree from CBT as younger adults, specific adaptations to CBT may not be necessary (Gatz et al., 1998). On the other hand it seems reasonable to suggest that changes to style might be required for older adults who are ill or cognitively impaired (Rybarczyk et al., 1992). When clients are in fragile health, Grant and Casey (1995) suggest the therapist may need to supply most of the energy to the collaborative process, slow the pace of delivery and use memory aids. The same authors also propose that CBT may not be feasible when illness becomes so severe that the process of therapy becomes too demanding.

One of the key challenges facing the cognitive model of depression concerns how to address 'realistic' negative cognitions. For medically ill people thoughts about the loss of function and poor prognosis are not always distorted. One approach would be to focus on enabling the patient to develop coping strategies, such as distraction to override the thoughts as they occur (Moorey, 1996). The alternative would be to look for distortions associated with the realistic thoughts using standard cognitive techniques. For example, when beliefs about the consequences of disease appear rational, further exploration may reveal that patients have underestimated their capacity to cope, or hold a misplaced

perception that they are a burden to carers (Rybarczyk et al., 1992). Wright, Thase, Beck and Ludgate (1993) recommend tackling the 'worst case scenario'. Once the individual has confronted the worst eventuality, they may be freed from the intensity of affective distress, which places them in a better position to develop more constructive coping strategies.

The case for using CBT for depression in Parkinson's disease

Given the paucity of evidence for the effectiveness of CBT used with physically ill older adults, it is difficult to know whether CBT could be an effective treatment for depression in people with PD. Various perspectives are possible. Firstly, as PD patients are similar in many respects to adults living with chronic ill health, it would seem fair to speculate that CBT would be as beneficial. Just like anyone else, depressed people with PD may experience symptoms that stem, in part, from distorted appraisals of the difficulties experienced. According to Enright (1997) "there is no psychological or physical problem that cannot potentially be assisted by the cognitive behavioural approach" (p.1812). Analogously, Laidlaw (2001) argues that CBT is a "relevant and accessible therapy precisely because it deals with older people's current concerns, whether that is grief, physical limitations following a stroke or general emotional distress" (p.11), and we could add, the impositions caused by PD.

On the other hand there may be a unique quality to PD depression that could limit the effectiveness of a cognitive approach. Many believe that PD depression, akin to post stroke depression, is not 'reactive' but biologically mediated, a factor which could influence the individual's capacity to engage in and derive benefit from psychological therapy (Mayberg & Solomon, 1995; Gareri, Fazio & De Sarro, 2002). Although

depression in PD is likely to be the consequence of both biological and psychological factors, the only way of establishing whether an organic component will have a bearing on treatment effectiveness would be to run a clinical trial. It is possible that CBT will need to be adapted to accommodate the physiological factors associated with deterioration. In order to appraise the type of modifications that could be required, it would be useful to provide a synopsis of the main biological, cognitive and disability factors potentially associated with PD depression. To set the scene, it would be of value to consider how patients describe their experience of living with PD.

Lived Experience of Parkinson's disease

Findings that people with PD are at greater risk of developing depression following diagnosis and in the advanced stages of disease (Kaiser, Bodey & Bodey, 2000), seems to indicate that demands for adjustment vary over the course of illness. Qualitative studies have provided a real world perspective on what some of these demands might mean on an individual level. Lomas (1999) proposed a transitional model of lived experience in which people with PD move through four stages including pre-diagnosis, diagnosis, initial adjustment and the transition to the chronic phase. In the initial phase patients (n = 9) commented on feelings of sadness when grieving for current and future losses, dealing with PD symptoms, and coming to terms with threats to identity.

Some indication of the intensity of the emotional response and meaning of diagnosis can be gleaned from the following remark made when a patient realised that his symptoms were consistent with PD.

“This predictability was no small part to the entire horror show. One of the things I would be losing here was freedom ... I can't overemphasize what a blow this prospect of being so unfailingly predictable was to my sense of myself as an individual.” (Fox, 2002: 177)

Lomas (1999) concluded that the key task after diagnosis was to find a way of regaining control in order to live with uncertainty and to reduce emotional distress, and if this was not achieved patients would be unlikely to move on. Even if adjustment was successfully negotiated at one stage, it was suggested that deterioration in symptoms could result in a 'resurfacing of dilemmas and emotions which had previously been resolved' (p. 52), at a later stage. On a note of caution, attention was drawn to the fact that patients often described feelings of sadness or sorrow as 'depression', when it was possible that they were experiencing a grief reaction to losses. This observation suggests there may be times when transitive states of sadness are incorrectly diagnosed as depression.

The Nature of Depression in Parkinson's disease

Biological aspects

A range of identified factors implicate the biological disease process in the aetiology and maintenance of depression in PD. Symptoms of anxiety and depression can occur before the onset of motor disturbances leading to the proposition of a common neurological pathway (Murray, 1996). Rates of depression appear to vary between clinical subtypes of PD, with greater depression reported in the akinetic-rigid subtype than in the tremor-dominant type (Jankovic, McDermott & Carter, 1990; Starkstein, et

al., 1998). These subtypes can be distinguished by the location of severe cell loss within the substantia nigra (Kaiser et al, 2000).

With regard to biochemical differences, some post-mortem studies have found lower concentrations of the neurotransmitter serotonin in the cerebrospinal fluid of depressed compared to non-depressed patients with PD (Mayeux, Stern, Sano, Williams & Cote, 1988). Anatomical studies using positron emission tomography (PET) have also found the region of glucose metabolism differs between PD patients with and without depression (Ring et al., 1994).

The 'on-off' phenomenon. Motor fluctuations are a common component of PD affecting 10-50 per cent of patients and have been labelled the 'on-off' phenomena (Menza, Sage, Marshall, Cody & Duvoisin, 1990). Michael J Fox (2002), an actor with PD described the 'off' phase in the following way, "When I am off the disease has complete authority over my physical being. I'm utterly in its possession ... I experience the full panoply of classic Parkinsonian symptoms: rigidity, shuffling, tremors, lack of balance, diminished small motor control, and the insidious cluster of symptoms that makes communication difficult and sometimes impossible" (p.256). Levodopa is the main pharmacological treatment used to control motor instability but, over time, the treatment can become less effective and a proportion of patients will experience unpredictable changes in the mobile 'on' and immobile 'off' states. Some patients will experience mood swings during the 'off' period accompanied by feelings of doom, helplessness and hopelessness (Nissenbaum, Quinn & Brown, 1987; Matson, 2002). Various hypotheses have been offered for mood variations ranging from psychological

reaction to the inability to control illness events to changes in dopamine levels (Erdal, 2001b; Racette et al., 2002).

Cognitive features

Patients with PD can develop dementia, with Biggins et al. (1992) recording an incidence of 19% in a cohort of 87 patients who were followed over a four year period. The risk of dementia increases with age with Mayeux et al. (1990) noting 65% of patients with PD over the age of 85 had dementia. About a third of patients' exhibit subtle cognitive deficits, and a strong link has been established between cognitive impairment and depression (Cubo, Bernard, Leurgans & Raman, 2000). Although basal ganglia dysfunction is the primary cause of PD, the effects of degeneration may be widespread due to neural circuit connections between brain systems, particularly those extending to the prefrontal cortex. This would explain why cognitive problems traditionally associated with the frontal lobe such as deficient problem solving ability, inability to shift attentional set, concrete thought and impaired memory processes, often occur (Dubois & Pillon, 1997; Lawrence & Sahakian, 1996).

Biases in information processing. Within literature evaluating the cognitive model of depression, the Stroop task has been used to investigate biases in attention, one of the earliest stages of information processing. People with depression are more likely to take longer to name emotional words on this task because the content of the words has seized attention (Williams, Mathews & MacLeod, 1996). Findings reported by Serra-Mestres and Ring (2002) suggest non-depressed people with PD show a similar attention processing bias. In an earlier paper the authors argue that PD depression may be triggered

by the neurobiological changes that disturbs brain processing, which in turn increases the vulnerability to reacting negatively to stressors (Serra-Mestres & Ring, 1999).

Content of cognitions. Several studies have reported that people with PD tend to endorse fewer cognitions indicating a negative view of the self, such as guilt and worthlessness on the Beck Depression Inventory (BDI) than people without PD (Gotham, Brown & Marsden, 1986; Huber, Freidenberg, Paulson, Shuttleworth & Christy, 1989). If true, the finding would have implications for the conceptualisation and treatment of depression in PD. Methodological shortcomings have been identified in the above studies (Nilsson et al., 2002). The results reported by Erdal (2001a), who found no ~~difference between the content of cognitions reported by physically healthy depressed~~ adults and people with PD, have limited generalisability because only 4% of the PD sample had major depression. To summarise, there is tentative evidence that PD depression may be associated with fewer negative self-focused cognitions, but more research validation is required.

Disability variables linked with depression

Progressive deterioration. PD is a degenerative condition but the rate and severity of the deterioration for each individual is impossible to predict. Uncertainty over prognosis affects psychological adjustment in chronically ill people (Mullins et al., 2001; Neville, 1998), and Baker (2000) found most people with PD worry about the progression of disease. One study did not find that 'perceived uncertainty in illness' predicted psychological distress in PD, but the authors were careful to point out that the participants were selected from support groups so the results may not be generalisable to all people with PD (Sanders-Dewey, Mullins & Chaney, 2001).

Falling. Approximately two thirds of people with PD fall within a year, and this incidence is two fold higher than that reported for other older adults living in the community (Wood, Bilclough, Bowran & Walker, 2002). Problems with balance and 'freezing' of the lower body are common causes of falling. Findings suggest that people who fall are more likely to be anxious and depressed than people who do not fall (Ashburn, Stack, Pickering & Ward, 2001; Schrag, Jahanshahi & Quinn, 2001).

Observable signs of disability. No quantitative studies have looked specifically at how noticeable physical symptoms, such as tremor, difficulties writing and drooling, impact on the quality of life or emotional well being of people with PD. On the other hand, ~~qualitative data suggests that many people with PD are ashamed to be seen in public.~~ In Nijhof (1995), half of the people interviewed reported significant difficulties coming to terms with visible signs of illness. Schrag et al. (2001) found that reports of feeling stigmatised were linked to the severity of depression which gives substance to the implication that embarrassment over PD symptoms could play a role in psychological distress.

Implications for Cognitive Behaviour Therapy

Presumably all the principles identified in literature covering CBT for chronic illness and older adults would hold for the depressed patient with PD. The therapist may need to consider how to tackle realistic negative thoughts concerning the disease progress. If the adult is older, it may be necessary to think about whether changes to the structure, duration and mode of delivery are required. Whether a psychological intervention can work for a disorder with an assumed intractable organic basis is a controversial issue which requires further comment. In the final section, an argument

will be made supporting the use of CBT with depression compromised by neurodegenerative factors. Also the preceding findings on the nature of PD depression will be used to consider how CBT might be used effectively followed by considering the type of adaptations and themes anticipated arising during therapy. It should be noted that many of the ideas presented are speculative.

Can CBT be effective with an organically mediated disorder?

So far we do not have sufficient evidence to give a conclusive answer to this question. Whilst the biological aspects of PD have been discussed in the context of the implications for drug treatment (NINDS, 2001; Burn, 2002; Cummings, 1992), there has ~~been little information on their significance for psychological therapy.~~ Psychiatric literature has tended to argue that psychological approaches could be beneficial when depression is 'reactive' rather than endogenously based (Lieberman, 1998). The cognitive theory however, maintains that a biological component should not preclude a psychological approach because the model always acknowledged that organic factors might be significant in the disorder (Clark, Beck & Alford, 1999). Even if PD depression is organically determined (as is implied by the possible link between dopamine levels and depression) it should still be possible to use a cognitive intervention effectively.

To use an illustration, if the increased sadness that occurs during motor freezing is caused by neurochemical changes, CBT might still be able to promote more effective coping. The therapist might help the individual to prepare in advance for the type of cognitive distortions likely to happen during the 'off' period and try out more encouraging alternatives. The use of cognitive methods may enable the individual to break the vicious circle of depression exacerbated by the continual stream of

dysfunctional cognitions, irrespective of the initial cause. In this situation, the emphasis would not be on 'curing' low mood but finding a way to manage the reactive responses in order to maintain the most optimistic outlook.

Adaptations to cognitive behaviour therapy

Careful attention needs to be given to how particular symptoms of PD may affect therapy. There is very little information on the impact of subcortical deterioration on therapy responsiveness, but some evidence to implicate cognitive dysfunction with poor treatment outcomes (Jarrett, Eaves, Grannemann & Rush, 1991). Nonetheless as Jones, Miller, Williams and Goldthorp (1997) argue when discussing the application of CBT to ~~people with intellectual disabilities who may be compromised by similar processing~~ problems as people with PD, it should be possible to develop methods to overcome the difficulties posed by cognitive deficits. Crews and Harrison (1995) provide a comprehensive account of the neurological basis of depression and hypothesise adaptations for CBT, which have relevance for the present consideration of PD. For frontal lobe dysfunction, they identify the following deficits that have the potential to impede the effectiveness of cognitive therapy including, impaired capacity for self-talk, literal interpretations of situations and difficulties processing and retaining new information. Rather than discount CBT on account of these deficits, the following recommendations were made; a) to encourage the person to verbalise rational thoughts, b) incorporate frequent discussions of abstract thinking within the therapy session, c) to enlist the support of carers to encourage practice and repeat concepts between sessions, and d) to prepare written material. Stated concisely, the therapist would need to facilitate

the process of guided discovery more conscientiously than would be necessary with a cognitively unimpaired individual.

Themes anticipated to arise in therapy

Former studies suggest illness-focused rather than self-defamatory cognitions commonly feature in the depressive experience of patients with PD (Huber et al., 1989; Gotham et al., 1986). Understandably, most patients worry about the inability to predict prognosis (Backer, 2000). A useful cognitive strategy could be to demonstrate that it is not the uncertainty in itself which is causing distress, but the thoughts connected to uncertainty (White, 2001). If the collaborative discussion reveals that the individual has ~~created a grossly distorted image of future events or has underestimated their perceived~~ capacity to cope, hopeful but realistic thoughts could be encouraged, and the Socratic exploration could build confidence in the individual's capacity to handle changes.

For other anticipated concerns, cognitive strategies have been used to reduce the fear of falling and increase physical activity in the general population (Tennstedt, Lawrence & Kasten, 2001) and should be suitable for PD. Like falling, fear of being seen with signs of disease can lead to a decrease in social activities, an avoidance strategy which could maintain psychological difficulties. Cognitive and behavioural techniques can be utilised to test the reality of predictions of how others will respond to observable markers of disease, and build coping strategies should the 'worst possible scenario' occur (e.g. a person passing a rude comment when PD symptoms cause the individual to shuffle or stumble in public).

Conclusion

A recent review labelled the treatment of PD depression as 'something of a blackhole' with very little evidence-based information to suggest what might work (Burn, 2002: p. 451). Whether CBT can effectively ameliorate symptoms of depression in people with this complex degenerative condition is, at present, an open question. Perhaps the dearth of published research in this area is a testimony to how difficult it has been to use psychological approaches with PD. Also we do not know how much relevance the CBT efficacy literature in the related fields of older adults and chronic illness will have for the PD population. The only established certainty is that people with PD depression are a vulnerable, often neglected group. This would appear to provide a sound clinical basis to attempt to confirm the validity of CBT in this population.

Feasibility studies would not only provide crucial clinically relevant information on how to manage low mood in PD, but also build theoretical understanding in previously neglected areas. The cognitive model presumes that the therapeutic processes involved in evaluating dysfunctional thoughts can be applied universally. People with PD depression not only have to adjust to numerous and ongoing impairments, they have to contend with negative thoughts that often have some basis in reality, and their affect may be determined, in part, by neurodegenerative changes. So far, with the exception of Crews and Harrison (1995), little has been written about the neuropsychology of depression and its implications for cognitive. Even less is known about the consequences for therapy in depression associated with subcortical disease, including PD. If cognitive therapy does not work for patients with PD, one would need to question whether the

model accounts adequately for 'rational cognitions' and the potency of organic moderators of treatment.

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**Brief Cognitive Behavioural Therapy for Depression Associated with
Parkinson's disease: A Single Case Series.**

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Running head: Parkinson's disease, depression, cognitive behaviour therapy

Brief Cognitive Behavioural Therapy for Depression

Associated with Parkinson's disease:

A Single Case Series.

Abstract

The feasibility of a brief home based cognitive behavioural intervention for depression associated with Parkinson's disease was evaluated. Five depressed patients between the ages of 54 and 82 attending a movement disorder clinic agreed to participate. Outcome was assessed using mood and quality of life inventories (Beck Depression Inventory; Geriatric Depression Scale; Parkinson's Disease Quality of Life Questionnaire). Four individuals demonstrated a clinically reliable reduction of symptoms according to the GDS scores, with greater improvement reported for the two individuals with more severe pre-therapy levels of depression. The BDI findings suggested most of the improvement related to the cognitive dimensions of guilt, pessimism and failure. Overall, the change in mood was not accompanied by an enhancement of perceived quality of life, and little variation in the frequency of activities was reported over the course of therapy. Possible explanations for the findings are considered along with implications for theory and clinical practice.

Brief Cognitive Behavioural Therapy for Depression

Associated with Parkinson's disease:

A Single Case Series.

1 Introduction

Few therapeutic approaches have received more research attention in recent decades than cognitive behavioural therapy (CBT). The sphere of health psychology has seen CBT being tried with an ever-increasing range of medical problems including chronic pain, cancer and neurologically based disorders such as multiple sclerosis and brain injury (Morley, Eccleston, & Williams, 1999; Antoni, Lehman, Kibourn, Boyers & Culver, 2001; Mohr, Boudewyn, Goodkin, Boston, & Epstein, 2001; Evans & Williams, 2002). Medical specialists are beginning to consider the role of CBT for depression associated with Parkinson's disease (Meara & Koller, 2000; Playfer & Hindle, 2001), but so far there have been few published studies evaluating its effectiveness.

Parkinson's disease (PD) is a chronic age related neurodegenerative condition, characterised by motor disability, tremor and rigidity. It has been estimated that 1 in 100 of the over 60 population will develop the disorder (Cummings & Masterman, 1999). Depression is a common problem in patients with Parkinson's disease (Aarsland et al., 1999). Estimates of the frequency of PD depression have varied in accord with the assessment methods used, but two meta analyses suggest a rate of 40% (Cummings 1992; Miyawaki, Meah, Kormos, & Tarsy, 1996).

Extensive literature has attributed the high prevalence rates to a range of potential sources. From these identified factors, authors have speculated that PD depression may be organically determined, a psychological reaction to consequences of disease or a

combination of both (Mayberg & Solomon, 1995; Schrag, Jahanshahi & Quinn, 2001; Burn, 2002). Findings potentially supporting the biological perspective include the association between depression and dopamine levels (Racette et al., 2002), subtype of Parkinson's disease (Jankovic, McDermott & Carter, 1990; Starkstein et al., 1998), serotonin levels (Mayeux, Stern, Sano, Williams, & Cote, 1988), degree of cognitive impairment and information processing (Cubo, Bernard, Leurgans & Raman, 2000; Serra-Mestres & Ring, 1999). For the reactive hypothesis, depression has been linked to changes in the quality of life (Karlsen, Larsen, Tandberg & Mealand, 1999), falling (Ashburn, Stack, Pickering & Ward, 2001) uncertainty of prognosis and increased disability (Backer, 2000; Brown, MacCarthy, Gothan, Der & Marsden, 1988), levels of functional activity (Liu et al., 1997), the inability to control motor freezing (Matson, 2002) and shame (Nijhof, 1995).

The complexity of variables associated with PD depression has provided a treatment challenge for medical personnel attempting to help. The literature suggests that between 50-93% of people with depression in PD are not treated (Lapane, Fernandez & Friedman, 1999; Meara, Mitchelmore & Hobson, 1999). Even though a minority of patients are prescribed antidepressant medication, the evidence for its effectiveness remains equivocal (NINDS, 2001). Thus, there would appear to be a clear rationale for trying psychological approaches with this population.

The main theory informing the development of CBT was proposed by Aaron Beck. The model stated that during depression people tend to perceive events in an overly negative or distorted manner (Beck, 1995). These appraisals are thought to determine how the individual feels and behaves. CBT is a structured, problem orientated approach that involves the client and therapist collaborating together to identify patterns of unhelpful thinking and replace them with more reasonable alternatives. Treatment

comprises sharing with patients the cognitive conceptualisation of their problem, behavioural strategies to provide evidence to counteract unhelpful thinking and an exploration of the client's disordered thoughts.

A study by Dreisig, Beckmann, Wermuth, Skovlund and Bech (1999) has highlighted the potential for using CBT with PD. Although therapy did not specifically target depression, individually tailored interventions for nine young PD patients indicated that average scores on a Psychological Profile Questionnaire demonstrated significant improvement for the treatment group compared to the control group. For clients who share similar problems as people with PD, CBT is becoming established as an effective treatment. Laidlaw's (2001) review of six meta-analyses for late life depression concluded that the outcomes of CBT for older people are comparable to those found with younger adults. For depression associated with neurological conditions, CBT has been shown to be more effective than supportive therapy for multiple sclerosis (Mohr et al., 2001), and preliminary findings indicate CBT can help people with depression and dementia (Scholey & Woods, 2003). Cancer patients and people experiencing chronic pain or fatigue appear to fare better when receiving CBT than people who receive treatment as usual (Morley et al., 1999; Greer et al., 1992; Price & Cooper, 2003). These findings suggest CBT should be appropriate for people with depression associated with Parkinson's disease. Furthermore it ought to be possible to look at patterns of unhelpful thinking regardless of whether the content of thoughts are related to organic or psychosocial changes.

Brief psychological interventions are increasing in popularity (Curwen, Palmer, & Ruddell, 2000). In a sample of older adults, the majority of whom had a comorbid chronic illness (82%), ten-session CBT resulted in an improvement in mood which was maintained one year later (Rokke, Tomhave & Jocie, 2000). Leung and Orrell (1993)

evaluated a seven-session CBT and looked at the reduction of depressive symptoms according to DSM diagnostic categories and found that ninety two per cent of people with severe depression did not need to contact mental health services within the following year. Mohr et al. (2000) found eight-session telephone administered CBT was helpful for people with depression associated with multiple sclerosis.

Although these findings suggest that brief CBT holds promise for treating PD depression, it would be worth considering features of the disease with the potential to influence the success of therapy. Parkinson's disease is primarily caused by basal ganglia dysfunction, and the slowness of processing characteristic of this area is likely to impact on the individual's capacity to process information during therapy. Likewise, a proportion of PD patients experience motor fluctuations which have been labeled the 'on-off' phenomena (Menza, Sage, Marshall, Cody & Duvoisin, 1990). As switching off can be accompanied with communication and concentration difficulties, it would seem reasonable to assume that the patient would find it harder to engage in collaborative explorations during these periods (Kaiser, Bodey & Bodey, 2000). Cognitive deficits also feature in Parkinson's disease (Dubois & Pillon, 1997), including those problems traditionally associated with frontal lobe dysfunction such as concrete thinking, and impaired memory. Although suggestions for modifying cognitive therapy to address these problems have been proposed, there is limited information evaluating whether they are effective (Crews & Harrison, 1995).

Depression, if left untreated is likely to lead to poor outcome for the patients in terms of increased disability and morbidity (Cummings & Masterman, 1999). As low mood can reduce functional activity beyond the impairment already caused by motor disability, it would seem reasonable to suggest that treating mental health would contribute to improving the patient's quality of life. In the following study, a structured

self-help booklet will be used as an aid to brief CBT for depression associated with Parkinson's disease. The association between quality of life and depression led us to consider whether any change in depressive symptoms would be accompanied by change in patients' perceived quality of life. A home-based intervention was utilized in order to overcome the practical difficulties patients would face travelling to a clinic.

2. Method

2.1 Design

The effectiveness of a brief form of CBT for depression in Parkinson's disease was evaluated using an A-B design with follow-up. Patients completed pre, post and follow up outcome measures. They also kept pre-treatment baseline measures of activity frequency and mood level for 5 or 10 days prior to intervention. Following baseline, CBT was delivered following the contents of a self-help booklet entitled, 'Coping with Depression when you have Parkinson's disease' (Beck, 2000). Therapy comprised one sixty minute session per week for seven weeks. One month following completion patients were asked to repeat baseline measures.

2.2 Participants.

Four female and three male patients meeting the clinical diagnosis for PD according to the UK Brain Bank criteria for probable Parkinson's disease, were consecutively referred for brief therapy directly from a Movement Disorder Clinic based in a North Wales hospital. Two patients were not included in the clinical trial because it was decided that a different therapeutic intervention would be more appropriate. One male (age 83) was experiencing hallucinations and deluded thought as a side effect of parkinsonian medication. The intervention utilised principles of CBT for psychosis (Kingdom, 1998) and supportive psychotherapy for his carer. A second female patient

(age 79) was experiencing an unpredictable severe form of akinesia during sessions. Supportive psychotherapy was offered until practical assistance for PD symptoms could be obtained. None of the remaining five patients were experiencing symptoms of known dementia and all were taking antiparkinsonian medication. Webster scale scores (1968), measuring the severity of PD indicated that four patients were moderately disabled and one patient (case 3) was in the advanced stage of disease. Scores on the Hoehn and Yahr scale (1967) which categorized patients according to motor ability on a 5-point scale ranged from 2 – 4. A diagnosis of possible depression in PD had been made on the basis of scores of 5 or more on the GDS-15 Geriatric Depression Scale. Two of the five patients were taking antidepressant medication.

2.3. Measures

The Geriatric Depression Scale (GDS-15, Sheikh & Yesavage, 1986).

The GDS-15 is a short inventory requiring a simple 'yes/no' response to fifteen items. The scale was designed to measure symptoms of depression observed in the older adult population and has recently been used as a screening instrument in a Parkinson's disease prevalence study (Meara, Mitchelmore & Hobson, 1999). Research comparing the GDS with other depression scales has established the inventory as a reliable and sensitive measure of depression in older adults with medical conditions (Pomeroy, Clark & Philp, 2001).

The Beck Depression Inventory – II (BDI-II, Beck & Steer, 1993).

The BDI-II contains 21 items, each scored 0 to 3, measuring symptoms commonly associated with depression. The BDI is a key research tool used to measure outcomes following CBT with older adults, and depression associated with medical problems (Laidlaw, 2001; Mohr et al., 2001). It has been established as a reliable and valid measure of depression in PD (Levin, Llabre & Weiner, 1988), and a cut off criteria

for diagnosing depression in the PD population has been proposed (Leentjens, Verhey, Luijckx & Troost, 2000).

Parkinson's Disease Quality of Life Questionnaire (PDQL; De Boer, Wijker, Speelman & De Haies, 1996).

The PDQL is a disease-specific instrument comprising 37 items, scored 1 to 5, looking at PD symptoms, systemic symptoms, emotional and social functioning. The total score gives an indication of the patient's overall perceived quality of life. Hobson, Holden and Meara (1999) found the anglicised version of the PDQL was a useful disease-specific measure with good convergent and discriminative validity.

During baseline, therapy and follow-up, patients monitored mood level using a visual analogue scale adapted from Dick et al. (1995) manualised treatment for older adults. Patients were asked to provide a rating of how low they were feeling on a range from 0 'very happy' to 10 'very low'. They also provided a frequency count of the number of activities engaged in per day. Self-monitoring of mood and activity frequently feature together in depression research (Harmon, Nelson & Hayes, 1980), and commonly occur in CBT and PD studies (Rokke, Tomhave & Jocie, 2000; Kemp, Corgiat & Gill, 1992; Leung & Orrell, 1993; Liu et al., 1997). The use of a simple frequency count of activities along with the visual analogue scale of Dick et al. (1995) have not featured together in published literature on PD but fulfilled the criteria for an unobtrusive repeated measures assessment (Barlow, Hayes & Nelson, 1984), and was believed to be less taxing for people experiencing micrographia.

2.4 Procedure

Following referral, a home visit was arranged to explain the aims and structure of the intervention. Patients who had given informed consent were given a copy of the self-

help booklet to read and asked to complete baseline measures 5 or 10 days prior to treatment. The CBT protocol as guided by Beck (2000) is shown in Table 1. Therapy was delivered in the patient's home by the first author who had appropriate training and experience in CBT. Individual weekly supervision was provided by the second author.

Insert Table 1. here

All patients followed the same structure as outlined in the protocol. The first section outlined the symptoms typical of depression and gave examples of depressed thinking (e.g. 'Life isn't worth living any more'). The behavioural component provided an example of an individual with PD whose mood had improved after behavioural activation and encouraged the patient to plan and attempt activities during the coming week. The cognitive component contained four steps to help the patient identify and evaluate the 'reasonableness' of negative thoughts (e.g. 'I am a burden to everyone'). The therapist worked through the steps with the patient and put together a written summary along with a clinical formulation for the client to refer to as a memory aid. In session six, a difficult situation was problem solved using the steps outlined in the section and issues associated with compliance with medical advice were covered. On the final visit the therapist reviewed the content of previous sessions and discussed how they might apply the principles in the future. During the follow up appointment the patient shared how they had been managing since the end of therapy and options for ongoing mental health problems were addressed.

3. Results

People with Parkinson's disease are a heterogeneous population, and this variation was demonstrated in the diversity of issues raised in each section of the self-

help booklet. Three patients (cases 1, 2 and 4) did not acknowledge particular cognitive identifiers of depression highlighted in the psychoeducation section (e.g. worthlessness and guilt). The recommendation to introduce activities to lift mood was not always straightforward as individuals with mild depression were already attempting to maintain an active life, and those with more severe depression tended to have complex physical problems that hindered the application of behavioural work. The concerns covered during the cognitive restructuring and problem solving components of therapy are shown in Table 2., and a synopsis of the process of intervention per client is presented below. Some personal identifiers have been changed.

Insert Table 2. here

Case 1 was a 51-year-old married male who had been diagnosed with early onset Parkinson's disease two years previously and had no previous mental health history. As his PD symptoms advanced he was forced to give up employment. He admitted that his only concern was the fear of falling. The behavioural evaluation revealed that he was attempting to maintain an active life which included starting new hobbies, and using various strategies to compensate for movement impairment. Freezing was observed as he tried to walk through doorways and symptoms associated with switching off were noted, which led to shorter sessions on three occasions. The psychological component of his fear was evaluated during cognitive restructuring, and an initial worsening of mood occurred (see Figure 1, session 3). The GDS score demonstrated a reduction in depressive symptoms, although BDI and self-ratings of mood suggested ongoing symptomatology, possibly maintained by concerns regarding the outcome of forthcoming major surgery. A request was made for an occupational therapy assessment to evaluate the practical

strategies for managing falls in the home. During the follow up appointment the therapist needed to go over apparently forgotten material previously covered in therapy.

Case 2 was a 72-year-old married female who had been diagnosed with late onset tremor dominant PD two years previously and had a previous mental health history of mild claustrophobia. She acknowledged minimizing her mood symptoms because it 'was rooted in her generation' not to admit to feeling low. Her PD symptoms were relatively minor and the behavioural work indicated that she was keeping up an active social life which involved visiting family, friends and former work colleagues. She had not told many people she had a diagnosis of PD and would sit on her hands in public if the tremor was visible. Symptoms of low mood occurred during the morning when, she reported the internal tremor occurred, and she started to ruminate on what her prognosis would be. Her thoughts were linked to the belief that she would deteriorate in the same manner as her mother-in-law who had died following a dementia related illness. Her symptoms of mild depression as measured by the GDS and self-ratings of mood (see Figure 1) improved over the course of therapy. The follow up appointment showed a return of some symptoms which may have been attributable to death of a friend during the previous week.

Case 3 was an 83-year-old married female who had received a diagnosis of PD six years previously, and had no previous mental health history. Her husband undertook many of the household duties due to the fact that she was incapacitated with many health problems, including fluctuations in PD movement impairment, frequent headaches, poor sleep, urinary infections and back pain. The pervasiveness of her health problems made it difficult to introduce pleasurable activities but it was possible to encourage her to plan replies to her daughter's email messages. During cognitive work this lady was very pessimistic about the outcome of therapy because she believed she was too old and ill to

try anything new. When the therapist shared how other people with PD were managing their thoughts, she became more optimistic and began smiling in the final three therapy sessions. The outcome measures confirmed an improvement in mood had occurred although she was still not symptom free and her own self-reports demonstrated little variation (see figure 1). She did not want to continue with therapy nor did she want to take antidepressant medication because she felt she was feeling much better.

Case 4 was an 80-year-old married male who had received a diagnosis of PD three years previously and had a previous history of health anxiety which he had managed effectively before retirement. He had mild symptoms of tremor dominant PD, but had given up most activities including gardening and social outings. His low mood was linked to concerns about developing a particular health condition, which had been triggered by literature suggesting the condition was a common complication in PD. It was difficult to motivate this client to introduce any pleasurable activities but by the final session he had started to visit an old friend once a week. The irrational content of his thoughts was readily acknowledged, especially given that he had never experienced the problem he was concerned about. Although this patient was very appreciative of the therapy visits, there was little change in his mood levels, quality of life or activity over the course of the intervention. Options for continuing therapy were discussed, and it was decided to focus on strategies suggested by the physiotherapist before continuing with psychological therapy.

Case 5 was an 82-year-old married female who had experienced depression following the suicide of her mother thirty years ago. Depression recurred following the PD diagnosis five years previously. The patient firmly believed her symptoms of depression were closely tied to fluctuations in her PD symptoms and the therapist's observations over the course of the intervention concurred with this view. She was

willing to comply with suggestions for behavioural activation but found it difficult to enact them when PD symptoms were severe. During cognitive work she appreciated the opportunity to discuss thoughts related to becoming a burden to her family. Her mood improved substantially over the course of therapy, as did her perceived quality of life and the number of pleasurable activities engaged in. Other than the fluctuations in PD symptoms and therapeutic intervention, no other extraneous factors could be identified as potential contributors to improvement. As cognitive work revealed that she believed PD was a punishment for not preventing her mother's suicide, it was felt she would find further therapy helpful and she agreed.

Scores on depression inventories and PDQL pre, post-treatment and follow-up for each participant are shown in Table 3. All pre-treatment scores on the GDS fell within the clinical range (recommended cut off point 5) and three individuals had BDI-II scores above the cut off point of 16 recommended to identify PD depression (Leentjens et al., 2000). An improvement in symptoms was shown by the lower scores in all mood measures following treatment, although the degree of reduction was marginal for people with less severe pre-therapy depression (Cases 1, 2, and 4). The changes in BDI-II scores following treatment were no longer evident one month later for Cases 1 and 2. Both of these individuals had experienced significant stressors (e.g. death of friend following terminal illness; preparation for major surgery) in the week prior to the follow-up visit. Apart from case 5, the data suggested that changes in mood did not appear to be linked to an improvement in the client's perceived quality of life.

Insert Table 3. here

In order to determine whether the levels of improvement observed were clinically meaningful, the Reliable Change Index (RCI, Jacobson & Truax, 1991) was calculated for the pre and post intervention difference for the GDS, BDI-II total scores, and BDI-II factors (see Table 4.). The findings suggest a clinically reliable change had taken place for four patients according to the total GDS scores (test re-test reliability 0.85; Yesavage et al. 1983).

Insert Table 4. here

The BDI-II data (test-retest reliability 0.93; Beck, 1996), confirmed a reliable change for the two patients who had more severe pre-therapy depression. For these latter individuals, the scores for BDI factors (Steer, Ball, Ranieri & Beck, 1999) indicated a trend for more change in the cognitive domain, although the results were only significant for case 5, assuming the conservative test-retest reliability score of 0.85. These patients reported less sadness, pessimism, feelings of failure and guilt.

Figure 1. shows the self-reported recordings of mood level and frequency counts of activities throughout the baseline, intervention and follow-up period for each individual. It is worth noting that trends for improvement in mood can be difficult to interpret when factors such as feeling physically unwell have the potential to influence how people respond. Overall there was little variation in the frequency of reported activities throughout the intervention.

In Case 1 there was no obvious decline in self-rated depression during the intervention phase. Rather during the third session there was a noticeable worsening of mood which corresponded to the identification of underlying assumptions. There was a

trend for improvement during the intervention phase for the second client, followed by a small deterioration during the follow up period when the client was grieving the loss of a friend. The trends shown in Case 3 were particularly difficult to interpret given that the improvement in mood supported by the RCI calculations (e.g. less sadness, crying and pessimism) did not correspond with the patient's weekly reports which showed little change. Interestingly, the therapist noted an increased frequency of smiling during the final therapy sessions which suggested that an improvement in mood might have occurred. It is possible that personality factors and ill health during sessions had influenced the style of responding for this client. Consistent with the questionnaire data, there was no obvious pattern of improvement or deterioration for client 4. A clear improvement in mood was apparent at the start of intervention for case 5, which was maintained after therapy was completed. Like Case 1, the Socratic focus on identifying underlying assumptions was however associated with an initial worsening of symptoms.

Insert Figure 1. here

4. Discussion

Overall, the results from this preliminary case series indicate that brief CBT may help to alleviate symptoms of depression in adults, particularly those with greater severity of depression in PD. The rate of improvement reported for the two cases with more severe depression, is consistent with outcomes from CBT group design research assessing adults with similar age and illness circumstances (Mohr et al., 2001; Leung & Orrell, 1993).

The finding that medically ill participants with less severe depressive symptoms did not benefit to the same degree and were not able to sustain the small gains made over time, has also been reported elsewhere (Price & Cooper, 2003; Kemp Corgiate & Gill,

1992). A number of possibilities need to be taken in to account when interpreting this finding. It would be reasonable to claim that a radical shift in mood is less likely for people with mild depression. It is also possible that other factors influenced treatment responsivity including the occurrence of major stressors, comorbid illness or cognitive deficits. Cases 1 and 4 reported symptoms of anxiety, which is consistent with literature suggesting that 67% of people with PD depression have a diagnosis of anxiety (Menza, Robertson-Hoffman & Bonapace, 1993). Cognitive impairment as a potential moderator of treatment was also evident when the therapist needed to remind the first two clients concerning material previously covered.

Since the validity of the outcome measures is a crucial consideration in any study, it is also worth drawing attention to the implications of the disparity between the BDI and GDS-15 in initial diagnostic evaluations and clinical outcomes. The GDS-15 has been shown to have acceptable specificity comparable to other short depression scales for medical patients (Pomeroy et al., 2001). The use of the BDI has been questioned on the basis that somatic items might confound interpretation (Williams & Richardson, 1993). Consequently, based on the findings of Leentjens et al. (2000) a high cut-off of 16 was adopted to limit the chances of false positives in PD. Unfortunately as Leentjens noted, this level of specificity carries the risk of low sensitivity, which means that depression could be missed. Given that the GDS also contains somatic items, one might conclude that the designated cut off has the potential to overrate and the BDI to underrate the presence of depression in PD, which suggests we cannot be sure that the initial assessments of our milder cases were completely accurate. This classification dilemma is not new to PD depression research (Burn, 2002), and illustrates the need to find scales which effectively discriminate between depressive, cognitive and somatic symptoms of

PD. This has been identified as a key research initiative by the National Institute of Neurological Disorders (NINDS, 2001).

Given the hypothesized link between quality of life and depression it was interesting to note that, apart from one patient, there was no obvious connection between improvement in mood and changes in perceived quality of life (Karlsen, et al., 1999). For most individuals, PD symptoms were either unremitting or worsening, which raises the possibility that physical frailty may prove to be more decisive for perceived quality of living in PD patients than poor mental health (De Ridder, Schruers, & Bensing, 1998).

The negligible variation in activity frequency for four individuals was also unexpected. Efforts to increase the number of pleasurable activities were generally ineffective because disease factors often constrained what could be done (e.g. risk of falling in poor weather conditions; switching off). From the perspective of the cognitive model, it is assumed that increasing pleasurable activities will contribute to an improvement in mood by providing evidence to counteract dysfunctional cognitions (Beck, Rush, Shaw & Emery, 1979). Recent and controversial findings have suggested that the behavioural component of CBT can lead to greater change in mood than the attempt to modify thinking (Jacobson & Gortler, 2000). The question raised by the present and similar findings (Kemp, Corgiate & Gill, 1992; Leung & Orrell, 1993) concerns what components of therapy facilitate improvement when the scope for increasing activity is restricted by poor health. Interestingly the two cases who demonstrated the greatest improvement in depression were also the most severely incapacitated by PD symptoms. When their two BDI factors were analysed, the findings indicated that most of the improvement could be attributed to a reduction in cognitive symptoms. These findings suggest the cognitive aspects of therapy might be effective on their own, although further research would be needed to confirm this view.

Some studies (Gotham, Brown & Marsden, 1986; Huber, Freidenberg, Paulson, Shuttleworth & Christy, 1989) but not others (Erdal, 2001) have noted a difference in the type of cognitive symptoms endorsed on the BDI by people with PD depression. In this case series, a disparity was often noted between what people said during therapy (i.e. three patients did not consider themselves worthless, guilty, or punished), and what was reported on the BDI. Nonetheless the observation that people with PD may not acknowledge psychological symptoms has implications for the cognitive model of depression which assumes that depressed people usually attribute their depressogenic experience to a defect within themselves (Beck, 1967; Wright & Beck, 1983). Likewise the concept of a cognitive triad (Beck et al., 1979) which presumes that depressed people also hold distorted views of ongoing experience and the future, does not appear to be a good fit with the PD depression experience. The reporting of negative public responses to observable PD symptoms (e.g. falling, tremor) or concerns about illness prognosis may represent rational appraisals of ongoing circumstances rather than cognitive distortions.

Even though some aspects of the cognitive model do not seem to be fully congruous with PD experience of depression, the therapeutic work undertaken demonstrated that it was possible to utilise cognitive techniques to work with adverse situations (Wright, Thase, Beck & Ludgate, 1993; Moorey, 1996; Scholey & Woods, 2003). Each patient had experienced previous critical incidents (e.g. death of relatives following degenerative illness) that had created negative ways of interpreting current experience (e.g. belief in inability to cope with ongoing health problems), which could be replaced with less distressing alternatives. Whether the focus of reinterpreting thoughts is sufficient to alleviate depression in the PD population will need to be followed up in later studies because the data presented here imply it was not wholly effective for all.

The issue of whether PD depression is biologically mediated was not addressed directly by this study but deserves some comment in light of the opinion that psychological management may be ineffective for endogenous or organically based depression (Lieberman, 1998). Although there were indications that fluctuations in mood corresponded to changes in PD motor symptoms in at least two patients (Cases 1 and 5), it was not possible to determine the direction of the effect. Rather than allowing the biological versus reactive argument to dictate decisions on treatment, we believe the data from this study show that the emphasis on 'coping with' rather than curing depression taken in Beck (2000) can be applicable to all patients with PD depression irrespective of underlying cause.

There were several limitations in the present design. Firstly, we are unsure whether the patients seen were typical of people with PD depression. All participants had not had any contact with PD organizations, because they did not want to know details about prognosis. Also we are aware that some of the problems known to be associated with PD depression, such as dementia, and depression following diagnosis of PD were not represented among those that received the brief intervention (Cubo Bernard, Leurgans & Raman, 2000; Brown, MacCarthy, Gothan, Der & Marsden, 1988).

There was an attempt to control for methodological weaknesses by introducing distinct baseline lengths to vary the timing of the intervention. Following careful deliberation concerning patient needs, it was felt that it would be ethically inappropriate to unduly delay treatment with vulnerable adults. Only one therapist was used to deliver all the interventions. It is not known whether the pattern of results reported here were attributable to individual therapist factors or the CBT intervention (Lambert & Okiishi, 1997). However, given the scarcity of treatment literature for PD depression we think the

evidence for improvement constitutes adequate grounds for following up with a more rigorous research study.

There have been few guidelines highlighting the type of changes to delivery that might be required for people with PD depression. In retrospect, given that the cases presented here were complex, it was felt that therapy of longer duration might have been more appropriate. Psychological issues were often closely tied to disease complications, which highlighted a need for a comprehensive understanding of the disorder, or to work in conjunction with other PD specialists to provide the best management for depression. The observation that some sessions needed to be terminated early as a consequence of switching off highlighted the need for flexibility. Finally it is easy to overlook the impact of cognitive deficits on therapy. Difficulties constructing alternatives to negative thoughts and poor recall of previous sessions may be a consequence of the neurodegenerative process. Strategies such as encouraging the therapist to supply examples of tolerable thoughts, using repetition, frequent summarising, and providing written material may improve treatment effectiveness.

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Table 1. Treatment protocol for CBT following format outlined in Beck (2000)

Session 1:	Education and information on the structure of future sessions. Booklet heading - <i>How do I know if I am depressed</i>
Session 2 – 5:	Behavioural work Booklet heading - <i>How should I change my behaviour</i> Cognitive work Booklet heading - <i>How do I change my depressed thinking</i>
Session 6:	Problems solving and barriers to compliance with medical advice Booklet heading - <i>How do I solve my problems</i> - <i>How can I help myself follow my doctors advice</i>
Session 7:	Review of previous sessions

Table 2. Issues raised during restructuring

	Cognitive restructuring	Problem solving
Patient 1.	Falling in public – embarrassment Consequences of falling at home alone	Preparing for major surgery. Preparing for falls at home.
Patient 2	Unpredictable deterioration Being left alone to cope	How to cope with a friend dying with terminal illness
Patient 3.	Believing one was too old/ill to try anything new or change	How to manage PD symptoms
Patient 4.	Fear that PD symptoms would lead to another disease.	Putting together questions to ask at next PD clinic appointment.
Patient 5.	Unpredictable deterioration Becoming a burden to the family	How to cope with fluctuations in mood. How to ask family about being a burden.

Table 3. Demographic and clinical findings for GDS, BDI-II, and PDQL

Patient (gender/age)	1(M/51)	2(F/72)	3(F/83)	4(M/80)	5(F/82)
Medication for mood	No	Sinemet +	No	No	Mirtazapine
Hoehn and Yahr stage	3	2	4	2/3	3
Previous history of Mental health problems	No	No	No	Yes	Yes
GDS					
Pre	7	5	12	5	13
Post	2	1	7	3	8
Follow up	1	1	6	2	7
BDI-II					
Pre	16	7	20	12	34
Post	12	6	11	10	23
Follow up	15	8	10	8	20
PQLQ (total) *					
Pre	118	158	95	132	86
Post	119	149	91	137	102
Follow up	119	149	94	130	98

* PQLQ higher score indicates a better perceived quality of life

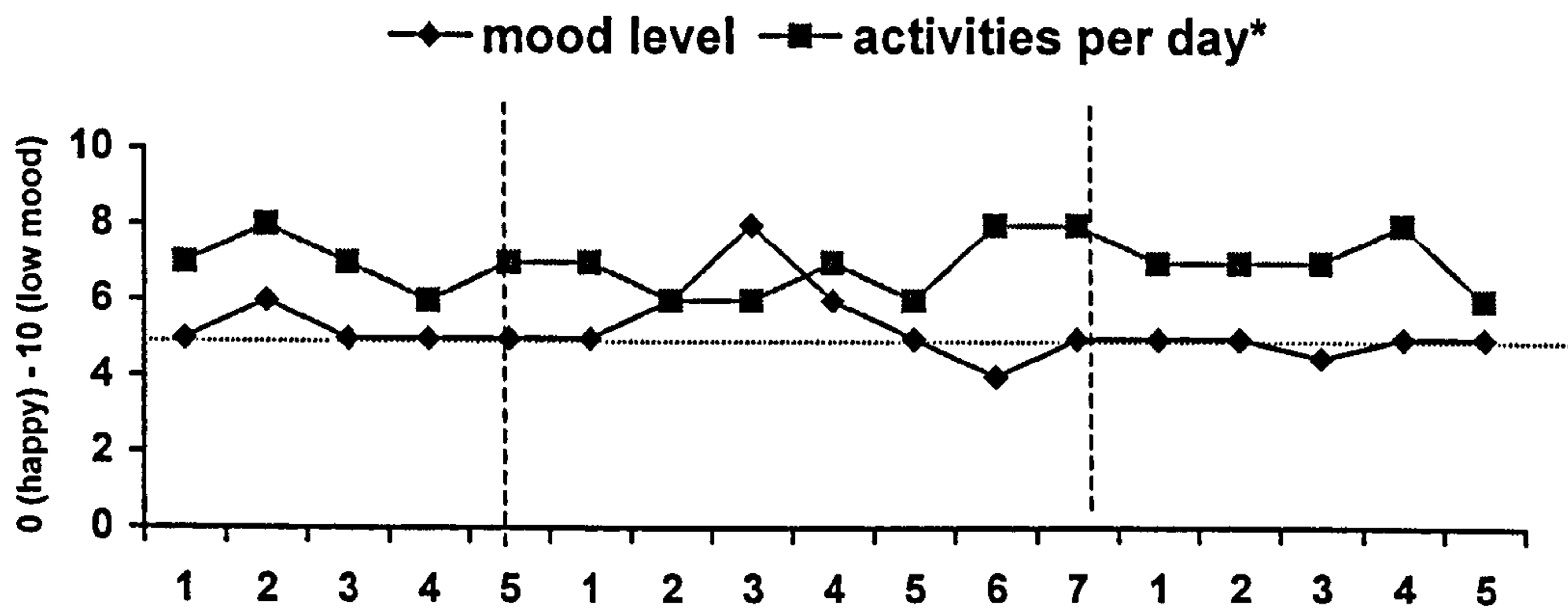
+ Sinemet is an antiparkinsonian drug which was used in this case to alleviate symptoms of low mood

Table 4. Reliable change calculations (RCI) for pre and post GDS and BDI total/factor scores

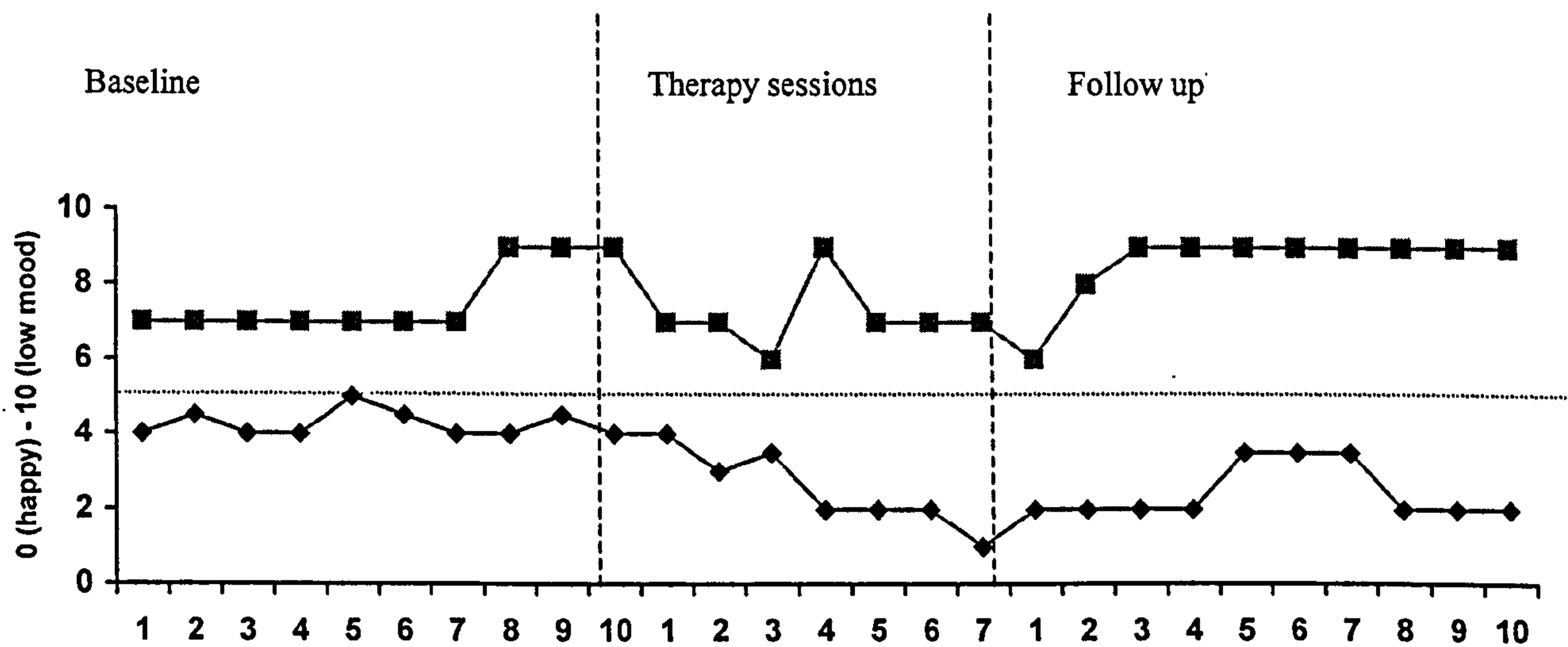
Patient	GDS		BDI total		BDI somatic/affective		BDI cognitive		
	Pre	Post	Pre	Post	Pre	Post	Pre	Post	
1	7	2	16	12	10	9	6	3	1.10
2	5	1	7	6	6	6	1	0	0.36
3	12	7	20	11	14	10	6	1	1.84
4	5	3	12	10	9	9	3	2	0.36
5	13	8	34	23	20	15	14	8	2.21*

* RCI values > 1.96 indicate a reliable change at the 5% significance level

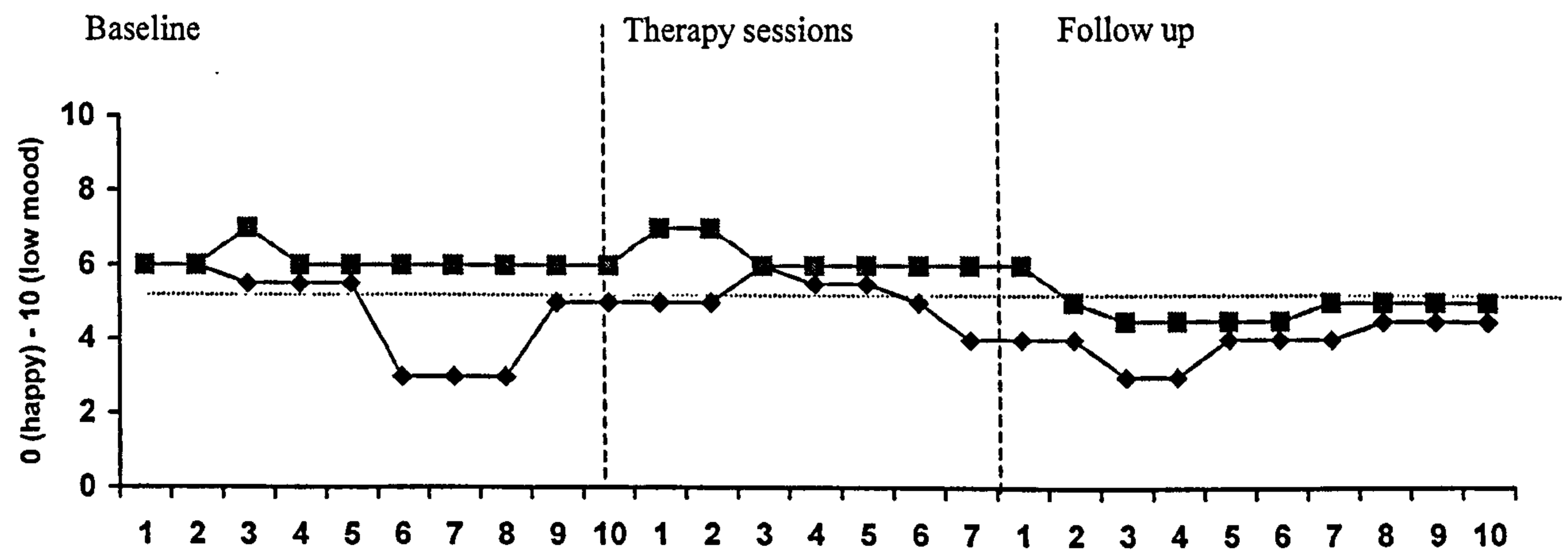
Case one



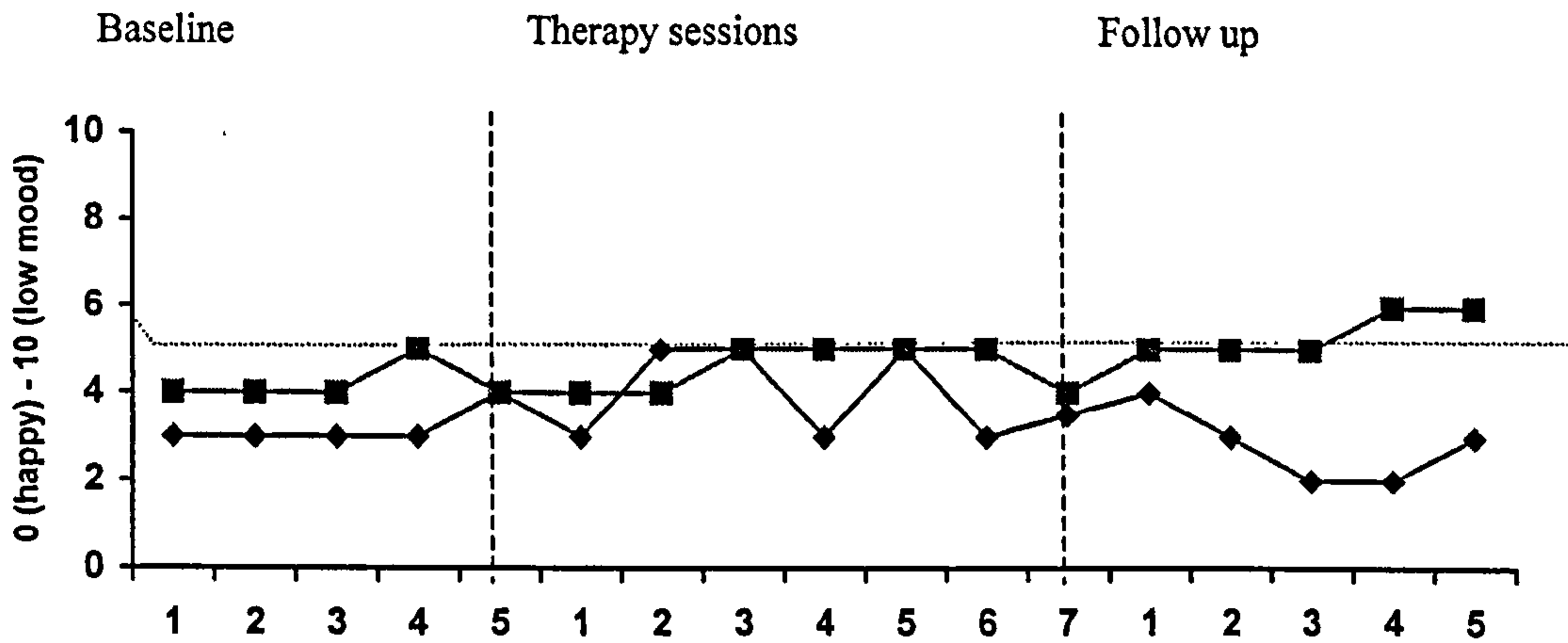
Case two



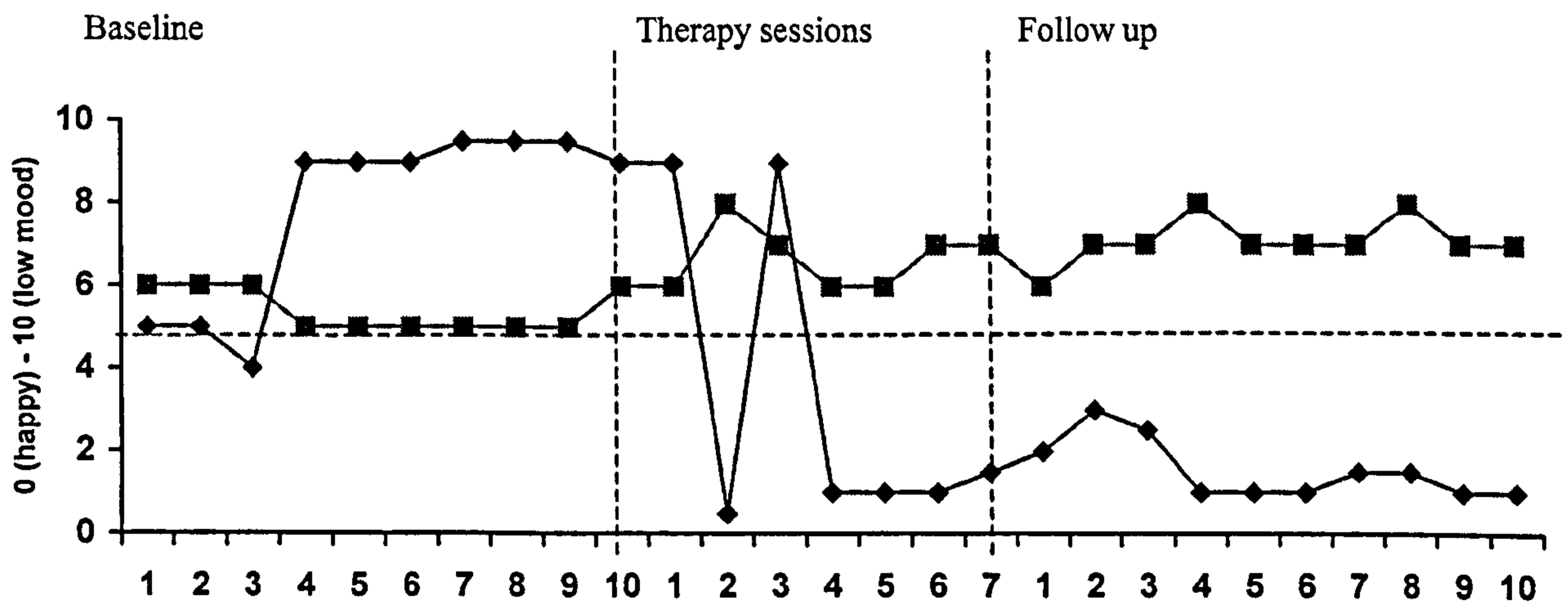
Case three



Case Four



Case Five



* The scale for activities per day represents the actual number of activities reported by the patient

Figure 1. Self ratings of mood and frequency of activities during baseline, treatment and follow-up for each patient

The Feasibility of Using Brief Cognitive Behaviour Therapy for Depression Associated with Parkinson's Disease: Critical Appraisal

Overview

This study aimed to test the hypothesis that brief Cognitive Behaviour Therapy (CBT) would reduce symptoms of depression in people with Parkinson's disease (PD). To borrow a phrase used by Rokke, Tomhave, and Jocie (2000), the results are "positive but guarded" (p. 117). According to the BDI scores, two patients demonstrated a clinically meaningful improvement in mood during the intervention, which apart from neurodegenerative factors, did not appear to be accounted for by other extraneous variables. In the critical review to follow, methodological issues relevant to the interpretation of findings will be discussed before examining the contribution the study has made to theoretical and clinical understanding.

Methodological issues

The implications of change from group design to a single case series.

A repeated measures group study was designed initially to test the prediction that CBT would lead to a reduction in symptoms of depression. Success would have been evaluated by comparing the within subject differences between pre, post and follow up measures. Tests for statistical significance would have been applied to control for the possibility that any differences had not occurred by chance. Although practical difficulties meant that it was not possible to include a between group comparison (e.g. waiting list control), the methodology would have permitted conclusions to be drawn

about CBT effectiveness for an average group of people with PD depression. Just before data collection was to start, it was necessary to change the design because most individuals already identified in patient records with depression had been included in a drug trial which would have compromised their participation in this study. In consequence participants had to be identified prospectively, which made it impossible to complete the study as planned within the time available. Even though only half of the fourteen participants recommended by the power analysis calculation participated in the research (five participants and two were not suitable), it is interesting to note that other researchers have mentioned a comparable inability to collect data as anticipated when faced with similar time constraints (e.g. Myles, 2002, anticipated participants $n = 9$, sample participants $n = 2$).

As an alternative, the investigator used a single case series. In contrast to group studies, the single case approach has more clinical utility because it provides information on whether for an individual with a particular set of illness circumstances, an intervention is more/less likely to facilitate recovery. Similarly Hayes, Barlow, and Nelson-Gray (1999) maintain that single case methods are a useful first step in establishing the efficacy of new treatment programs. If it had been possible for the investigator to select the sample, participants would have been matched for specific factors such as disease type, severity of depression or content of depressed thought (e.g. worry about falling). The intervention started at different points in real time, which controlled for external threats to validity, such as world events. To control for threats to internal validity (Kazdin, 1981), participants were put on a five or ten day baseline in an attempt to ensure that any improvement occurring during the intervention phase could not be attributed to the length

of time that patients had been in the study. From an ethical perspective it would have been inappropriate to impose longer baseline phases, or withhold supportive psychotherapy during pre-intervention visits if the patient was in distress. Also it was considered unlikely to be possible to achieve a stable baseline before intervention if PD depression is connected to fluctuations in the disease process.

In spite of design weakness, suggestions for establishing treatment efficacy were applied (Chambless et al., 1996; Roth & Fonagy, 1996). A 'probably efficacious treatment' can include single case experiments ($n = 3$) provided that the intervention uses treatment manuals, and the characteristics of clients are clearly specified. The study was set up to incorporate the information that would facilitate replication, including a detailed description of, a) patients b) procedure, c) intervention conditions, and d) measures (Hayes et al., 1999). Finally potential moderators of treatment effects were identified, including comorbid anxiety, pessimism and cognitive dysfunction (Kazdin, 2001). Although the results according to the BDI data are not sufficiently strong to establish CBT as a 'probably efficacious' treatment at this stage, it was possible to distinguish selection criteria for a future replication.

A representative sample

The heterogeneity of PD population cannot be overstated (Kaiser, Bodey & Bodey, 2000). Participants varied in disease type, duration and severity of depression. One characteristic that might set this participant group apart from other people with PD relates to their individual decisions not to access information about the disorder or be involved in support groups. A link between avoidant coping and depression has been

established previously (Finset & Andersson, 2000), and it is known that not all people with PD adopt this style (Sanders-Dewey, Mullins, & Chaney, 2001).

Results from two patients were not commented upon in detail in the research paper. One patient who described his affect as 'depression on top of depression' was experiencing deluded thought as a side effect of anti-parkinsonian medication. The intervention, which was based on literature looking at CBT for psychosis (Kingdom, 1998) was difficult to apply because the patient would only engage in therapy during lucid moments. The outcome was positive in the sense that he was visibly more at ease with the symptoms during the final therapy visit. The second patient was in the advanced stages of PD. She was experiencing suicidal ideation but was unable to concentrate on CBT due to the severity of her PD symptoms. It was necessary to attend to the patient's capacity to cope with another hour/day, before arranging respite support. Literature would suggest that these complications do occur in depression related to PD, but clearly require an individually tailored rather than a brief protocol driven intervention.

The validity of materials used

Questionnaire measures. A discussion regarding the validity of GDS and BDI appeared in the paper and therefore will not be repeated here. The fact that the measures were not consistent in screening for depression illustrates the need to identify scales which effectively discriminate between depressive, cognitive and somatic symptoms in PD.

Repeated self-rating measures. Repeated measurement is the hallmark of the single case design. Just as there are problems with interpretation when using questionnaires, so to there are difficulties associated with self-monitoring (see Barlow,

Hayes, & Nelson 1984 for limitations of repeated measurement). In retrospect, the investigator felt that direct observation may have provided a more precise method of measuring change, because cognitive dysfunction and individual factors (e.g. pessimism regarding therapy outcome; Safran & Segal, 1996) appeared to influence the accuracy of some client reports. In case three, the reduction in depressive symptoms demonstrated by the questionnaire data was not matched by a decline of low mood as measured by the patient's self-monitoring (see Figure 1 research paper). At the start of each session the client would preface her mood rating with comments like, 'what can you do to help me when it is the PD that has caused this' and then provide a rating similar to other sessions. As the intervention progressed she began to smile more frequently and it was evident that her mood had lifted. A record of smiling would have been a more reliable indicator of progress, but it would have been impossible to appreciate the relevance of this behaviour at the outset. Alternatively, other methods of gathering data could be considered, such as using carer reports or asking people to provide verbal rather than written records. Although the self-report measures used may present some validity problems, they have a precedent for use in research (Tiplady, Jackson, Maskery & Swift, 1998; Harmon, Nelson & Hayes, 1980; Dick et al., 1995), and the results have given an indication of how to adapt measures in future studies.

The self-help booklet. The use of structured self-help materials for CBT is becoming more common (Keeley, Williams & Shapiro, 2002), and have been advocated as one of the requirements for establishing empirically supported treatments (Chamberless & Ollendick, 2001). The manualised approach is felt to be a way of ensuring therapy is delivered in a uniform and replicable manner, although some findings

suggest therapist variation can be substantial (Malik, Beutler, Alimohamed, Gallagher-Thompson & Thompson, 2003; Najavits & Strupp, 1994; Lambert & Okiishi, 1997).

The use of a treatment protocol is legitimate if the contents of a programme address the issues arising for the targeted group. While Beck (2000) provided a clear articulation of cognitive therapy and clients found it easy to follow, there were indications that the booklet was inadequate in several respects. Two patients did not like to use the word 'depression', a finding which is consistent with literature showing many older adults prefer not to admit to being depressed (Friedhoff, 1994). A more serious limitation relates to the absence of background information on the physical aspects of PD and their potential relationship with depression. The failure to cover the complexity of symptoms experienced on a daily basis by people with PD was seen to be an oversimplification by some and offensive by others.

Therapist adherence to CBT as outlined in the Booklet

Determining whether the therapist adhered to cognitive therapy as outlined in Beck (2000) is important, because it was not possible to enlist an independent assessor to observe delivery. Analogously the therapist believed the use of tape-recording would impose an extra burden on frail adults who already appeared to find the process of reading the information sheet and signing consent forms daunting. Even if adherence to protocol had been evaluated, Jacobson and Gortner's (2000) reported disagreement between recognised master experts in cognitive therapy ratings for therapeutic competency suggests external ratings are not a fool proof method of establishing treatment integrity.

The case overviews outlined in the paper demonstrate adherence to the structure of Beck (2000) but also considerable flexibility in how it was applied. Given that the booklet is designed as self-help material, no provision was made for formulating the problem. As all cases were complex, it was essential to have a comprehensive understanding of how symptoms were uniquely presenting for each individual, before starting the intervention. The delivery of therapy called for a greater degree of sophistication than was implied by the booklet. Participants appeared unable to identify thoughts without the assistance of supplementary Socratic questioning techniques. Also the therapist had to facilitate the process of identifying reasonable alternative thoughts to a greater extent than was anticipated. Additionally, the physical problems associated with psychological difficulties often needed to be addressed. For example, in the 'fear of falling' case, cognitive restructuring was covered in the context of an understanding of how and when motor freezing was likely to occur and the practical strategies that could be applied to overcome the block. The experience of using the booklet has shown that, in practice, rigid adherence to a set format could be invalidating for the client unless the clinician has the scope for adapting the underlying cognitive principles to fit the needs of patients.

Contribution to theoretical and clinical understanding.

The major strength of this study has been its capacity to inform clinicians about how CBT may need to be adapted to address the complex presenting problems of people with PD depression. Findings from general depression research, that not all people benefit from CBT (King, 1998; Shea et al., 1992), show that the mixed results reported in this study are not exclusive to PD. On the other hand, reflecting on the cases where CBT

did not seem to work as well has provided insight into the potential barriers to successful outcomes particular for this client group including (a) the deficiency of Beck's theoretical model when applied to rational thoughts, and, (b) the relevance of biological mediators.

Rational cognitions. Despite refinements to Beck's cognitive model of psychopathology over the years, the key focus on disordered thinking has remained unchanged with the stated aim of therapy being to demonstrate "that a particular belief is wrong or dysfunctional and that another belief is more accurate and adaptive" (Beck, 1996: p.15). When thoughts are both accurate and maladaptive in the sense that a rational appraisal creates emotional discomfort, the application of the cognitive approach becomes more complicated (Moorey, 1996). The process of therapy was able to uncover unhelpful assumptions (e.g. '*if I deteriorate then I will become a burden to my family*'), that could be explored and replaced by more enabling alternatives. Within sessions, clients stated that they found the approach helpful, yet on occasion their symptoms of depression showed little change. This finding might suggest that tackling underlying assumptions may have limited clinical utility when the core fears like falling and deterioration are real. For example, the client who found the evidence that her family did not perceive her to be a burden comforting stated, 'but I still do not want my family to be 'responsible' for me'. Perhaps then, what many depressed people with PD are struggling with is not distress associated with distorted thinking but the discomfort that arises from being in a situation that is understandably difficult to accept.

Health psychology models such as the self-regulatory theory (Leventhal, Mayer, & Merenz, 1980) have highlighted the critical role of cognitive factors in adjustment to chronic illness. In the context of discussing theories of health and illness perception,

Petrie and Weinman (1997) argue that CBT may be an appropriate intervention when the aim of therapy is to “get people to think differently about their illness” (p. 15). While Beck's cognitive theory of depression, focuses primarily on the implications of distorted thinking to inform practice (Beck, 1996; Clark, Beck & Alford, 1999), other clinicians have applied therapeutic strategies arising from the model to alleviate the distress associated with poor adjustment to illness. For example Bates, Burns and Moorey (1989) maintain that encouraging a realistic (e.g. ‘people with PD do fall’) but positive perspective (e.g. ‘here is the evidence that you can cope if you fall’) will enable the medically ill individual to, “accept their disability with a greater sense of inner peace” (p. 273). As this study has shown, when beliefs concerning an illness are plausible it could be invalidating for the client to challenge them. However if the accent of therapy is placed on the implications that the meaning of an illness holds for overall adjustment, it may not matter whether thoughts are distorted or correct, the objective would be to use the tools of cognitive therapy to explore the extent to which the appraisals are damaging for well being. Clearly when the barriers to acceptance are explored from this broader perspective, the content of therapy sessions may cover issues other than the irrationality of cognitions.

Biologically mediated depression. The belief that PD depression is biologically mediated is widely held and was commented upon by Craig White, author of the book ‘Cognitive Behaviour Therapy for Chronic Medical Problems’, as a potential explanation for the present results (White, 2003, personal communication). If the affective experience, as opposed to the cognitive interpretation of that affect, has its roots in the neurochemistry of PD, CBT may not be sufficient on its own to promote full recovery.

There was evidence to indicate that organic variables had adversely affected outcomes (e.g. switching off within sessions; impaired memory), though it was not possible to establish whether these symptoms were specific to PD or a feature of PD depression (Serra-Mestres & Ring, 2002). Until we have more information on the nature of PD depression, we can only surmise that therapists would be advised to acknowledge the potential organic basis of depression with clients.

In the mean time, observing the potential impact of disease processes on therapy advises us to consider what the implications would be for future research of this nature. In the first section of the thesis, adaptations to the mode of delivery were suggested which included, slowing the pace of delivery, providing examples of abstract thinking, and using memory aids (Crews & Harrison, 1995). An awareness of these strategies was worthwhile, but the inclusion of a neuropsychological assessment might have led to more proficiency in delivery by highlighting particular areas of impairment before intervention. Given that the impact of motor fluctuations and associated physiological responses was greater than anticipated, it could be helpful to ask people to provide a detailed record of symptom changes over a typical day, including their responses to medication.

Conclusion

There is an immense gap in the literature guiding service providers in how to manage depression associated with PD. This study has evaluated whether CBT holds promise as a treatment option. The findings infer that CBT may reduce symptoms of depression in certain people with PD, but more empirical validation is required before it is recommended as a routine feature of clinical practice. To carry the research forward there is an urgent need for information on how the neurological aspects of PD (e.g. the

'on-off' phenomena; cognitive difficulties) affect treatment responsivity. Equally we need to establish whether adaptations to overcome the organically mediated impediments actually help, perhaps by comparing a standard CBT delivery to modified delivery. If future studies verify the difficulty in using behavioural strategies with chronically ill patients, it would be important for Beck's theory to establish the relative extent to which non-specific factors and cognitive techniques contribute to successful outcomes. Finally, thinking about actual practice, it is clear that therapy with PD patients presents a considerable challenge. Clinicians considering work in this area require the competence to apply CBT creatively along with the vigilance to be aware of when organically mediated changes may be hindering progress.

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APPENDICES

- I Publication requirements- Notes for contributors**
- II Ethical approval letters**
- III Measures and materials**
- IV Information sheets**
- V Supplementary case material**
- VI Statement of word count**
- VII Statistical information related to the calculation of RCI**

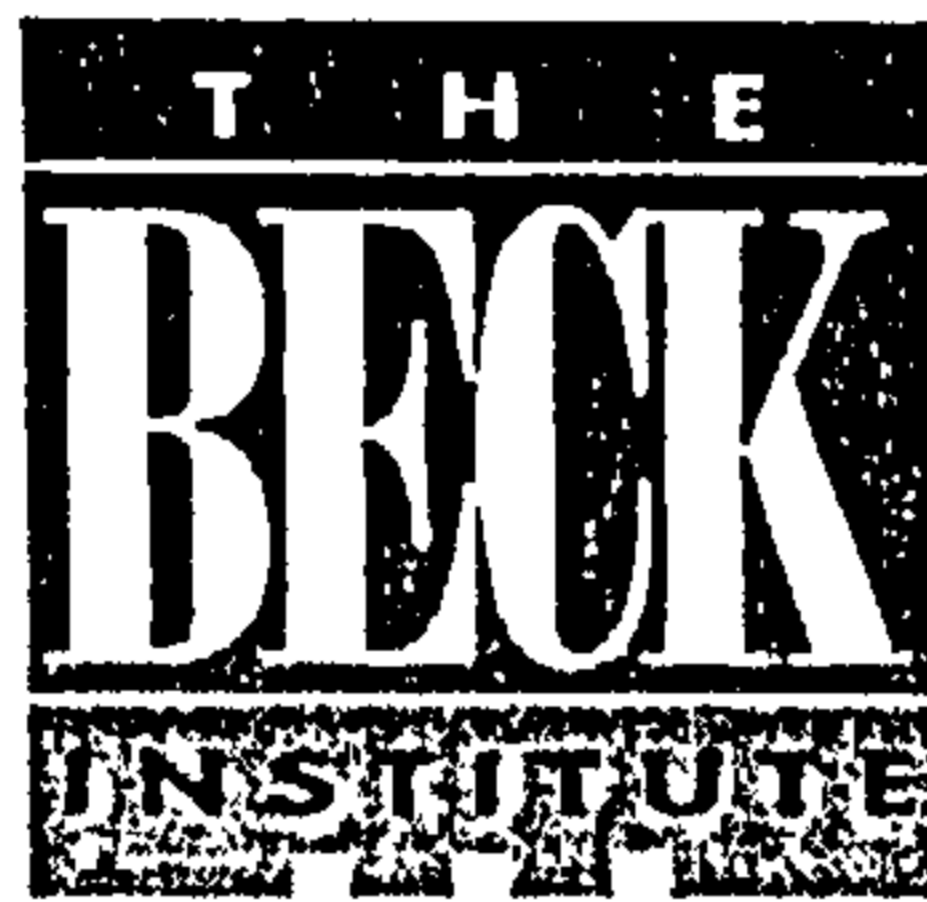
I . Publication requirements- Notes for contributors
(bound with relevant papers)

II Ethical approval letters

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III Materials and measures

Coping with Depression When You Have Parkinson's Disease



For Cognitive Therapy
and Research

Judith S. Beck, Ph.D.

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Revised 2000

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The Geriatric Depression Scale

15 item version

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PDQL

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IV Information sheets



**Ymddiriedolaeth GIG Siroedd Conwy a Dinbych
Conwy & Denbighshire NHS Trust**

INFORMATION SHEET

The effectiveness of Brief Cognitive Behaviour Therapy in reducing the symptoms of depression in individuals with Parkinson's disease.

You are being asked to take part in the above study. The study has been reviewed by the North Wales Health Authority Research Ethics Committee, and the University of Wales Ethics Committee, Bangor. Before you decide it is important for you to understand why the research is being done and what it will involve. Please take time to read the following information carefully and discuss it with friends, relatives and your GP if you wish. Ask your psychologist when she calls, if there is anything that is not clear or if you would like more information. Take time to decide whether you would be willing to take part.

What the research is about

Long-term illnesses are sometimes difficult to come to terms with. We are interested in helping people cope with feeling low when they have Parkinson's disease. The aim of the study is to see if a type of therapy which has been shown to help people suffering with various illnesses such as long-term pain, can also help people who get depressed with Parkinson's disease.

Why have I been chosen?

You have been chosen because one of the forms you filled in while attending at the Parkinson's Disease Clinic suggested that there might be times when you feel low. We will be asking 20 people who attend the Parkinson's Clinic to take part in the study.

Do I have to take part?

It is up to you to decide whether or not to take part. If you do decide to take part you will be given this information sheet to keep and be asked to sign a consent form. You are able to withdraw from the therapy at any time without giving a reason. If you do withdraw it will **not** affect the care you are receiving through the Parkinson's disease clinic.

What will happen to me if I take part?

I (Kristina Cole) will visit you at your home for one hour per week for seven weeks, and then once, a month later to see how you have been getting on. To see if the therapy has helped, you will be asked to fill in three questionnaires three times, 1) on the first visit, 2) on the last therapy visit and 3) on the visit a month later. It is OK to allow relatives or friends help you to fill out the forms.

It would also be helpful to know how you have been doing each week. You may be asked to mention a couple of things (e.g. how low or tired you have been or what things Parkinson's disease has stopped you doing) in the two weeks before, during and after therapy.

What type of therapy is it?

You will be given a booklet to keep called

Coping with Depression When You Have Parkinson's disease

The booklet includes the following sections

How do I know if I am depressed?

What can I do?

How should I change my behaviour?

How do I change my depressed thinking?

How do I solve my problems?

The psychologist will work with you to try out various ideas mentioned in the booklet. These ideas have been shown to help people with various illnesses.

Are there any alternative treatments for feeling low?

Sometimes people who have been feeling low will be prescribed antidepressant medication by their GP or by the Doctors working at the Parkinson's Clinic. You can still participate in the research if you are taking any form of medication.

Are there any disadvantages to taking part?

A lot of people think that therapy can take all their problems away. This is not possible when living with a long-term illness. The aim of therapy is to provide people with ways of managing and coping with feeling depressed.

What are the benefits of taking part?

We hope that the therapy will help. However, as this therapy has not been tried with Parkinson Disease, this cannot be guaranteed. The

information we get from the study will help us know what type of treatments help patients with Parkinson's disease.

What happens when the therapy ends?

This therapy is only a short course to help you to cope with your depression. At the end of therapy various options may be discussed with you.

Who will know about the results?

All information will be treated as strictly confidential and will be kept in a locked filing cabinet at all times. If you agree to take part, you will be asked to sign a consent form, which will be kept in your clinical file. We will let your GP know in a letter what is happening, and send a copy of this information sheet. A report of the results for the whole group will be written by the psychologist and sent to you in the post during the summer.

Who is organizing and funding the research?

Dr Kristina Cole is organizing and conducting the research as part of her training for a Doctorate in Clinical Psychology based at the North Wales Clinical Psychology Course; University of Wales, 43 College Road, Bangor, LL57 2DG. (Contact no 01248 386235). No payment will be made for taking part in the study.

What will happen next?

- 1) Read through this information sheet carefully and talk it over with others if you wish.
- 2) I will return at a convenient time. If you have any further questions feel free to ask.
- 3) If you agree to take part, you will be asked to read the consent form and sign it. Arrangements will be made for the home therapy visits.
- 4) If you prefer not to take part, the care you receive through the Parkinson's Clinic will not be affected in any way.

If you would like any further information do not hesitate to contact me

Dr Kristina Cole
Tel.

THANK YOU FOR READING THIS INFORMATION SHEET



**Ymddiriedolaeth GIG Siroedd Conwy a Dinbych
Conwy & Denbighshire NHS Trust**

Tim Gogledd Cymru | Bobl Sydd Wedi Cael Anaf I'r Ymennydd
Ffôn: 01492 807766/807521 Ffacs: 01492 516587
North Wales Team for People with an Acquired Brain Injury
Tel: 01492 807766/807521 Fax: 01492 516587

CONSENT FORM

Title of Project

The effectiveness of Brief Cognitive Behaviour Therapy in reducing the symptoms of depression in individuals with Parkinson's disease.

Name of researcher

Dr Kristina Cole (under the supervision of Dr Frances Vaughan)

Please delete as appropriate

- 1. I confirm that I have read and understand the information sheet for the above study and have had the opportunity to ask questions Yes / No
- 2. I understand that my participation is voluntary and that I am free to withdraw at any time, without giving any reason, without my medical care or legal rights being affected. Yes / No
- 3. I understand my GP will be informed that I am taking part in this study, and I have seen a copy of this letter. Yes / No
- 4. I agree to take part in the above study. Yes / No

Name of Patient

Date

Signature

Name of person taking consent
(if different from the researcher)

Date

Signature

Name of person taking consent

Date

Signature



**Ymddiriedolaeth GIG Siroedd Conwy a Dinbych
Conwy & Denbighshire NHS Trust**

**The effectiveness of Brief Cognitive Behaviour Therapy in reducing the symptoms
of low mood and depression in individuals with Parkinson's disease.**

Dear Dr

Re

I am writing to inform you that your patient has agreed to participate in the above research study, and a consent form has been signed. This study received ethical approval from the North West Wales Health Authority Research Committee.

The study aims to evaluate whether brief cognitive behaviour therapy (CBT) can help patients with Parkinson's disease who are experiencing symptoms of depression. CBT has been demonstrated to be an effective psychological therapy for individuals coping with a range of medical conditions including palliative care and chronic pain. However its efficacy with Parkinson's disease has not been established.

Please find enclosed the information sheet passed on to your patient, which outlines the main components of therapy. Participation will not interfere in any way with treatment as usual, received through your practice or the Parkinson's disease clinic. If you would like any more information, or if you have any questions about the study, please do not hesitate to contact me.

Yours sincerely

Dr Kristina Cole
Trainee Clinical Psychologist

Dr Frances Vaughan
Clinical Psychologist.

Contact telephone number before
After

October 1, 2002 -	01492 531332
October 1, 2002 -	01492 807773



**Ymddiriedolaeth GIG Siroedd Conwy a Dinbych
Conwy & Denbighshire NHS Trust**

**The effectiveness of Brief Cognitive Behaviour Therapy in reducing the symptoms
of low mood and depression in individuals with Parkinson's disease.**

Dear

Staff working at the Parkinson's disease clinic may have mentioned a research study which is aiming to use brief therapy to help people cope with feeling down in Parkinson's Disease. You are being asked if you would like to take part in the study.

I would like to offer you an initial home visit on

The aim of the visit would be to explain a bit about my self, what the therapy involves and to give you the opportunity to ask questions. If you would prefer me not to call, or the above time or date is not convenient please do not hesitate to contact me (Kristina Cole) on the number below. If you decide not to take part in the study the care you receive through the Parkinson's disease clinic will not be affected in any way.

Yours sincerely

Dr Kristina Cole
Trainee Clinical Psychologist

Dr Frances Vaughan
Clinical Psychologist

Contact telephone number before October 1, 2002 - 01492 531332
After October 1, 2002 - 01492 807773

V Supplementary case material

Summary of CBT sessions per client

Summary of CBT sessions per client

Case One

Session 1 How do I know if I am depressed

Client denied feeling suicidal, worthless, or guilty but stated he was finding it difficult to come to terms with giving up work.

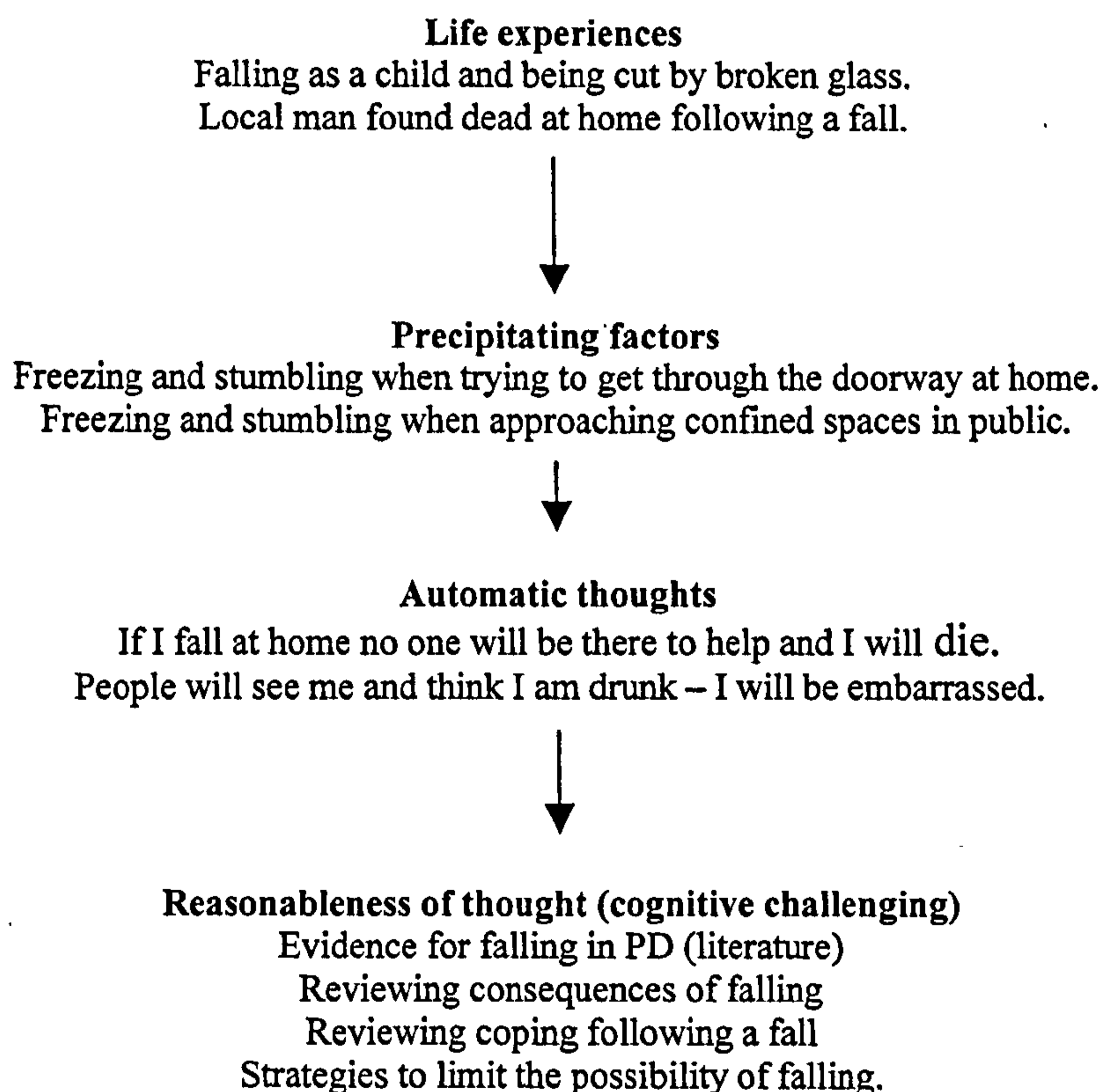
Session 2-3 How should I change my behaviour

Client stated he was already keeping as active as possible to manage feeling low. He attended football matches despite being unsteady on his legs, had taken up a new hobby (collecting for car boot sales) since leaving work, and had taken over household duties such as shopping and cooking.

Session 4-6 How do I change my depressed thinking

The client revealed that fear of falling was the main concern he had with regard to PD symptoms and that it was becoming more difficult to go out in public as his symptoms worsened.

Summary formulation



Session 7 How do I solve my problems

Preparing questions to ask prior to surgery.

Looking into strategies to get through doorways when freezing occurs

Parkinson's symptoms with the potential to influence therapy

Switching off during therapy (e.g. sweating, poor concentration, exacerbation of movement difficulties).

Poor recall of work done during therapy revealed at the follow-up session

Case two

Session 1 How do I know if I am depressed

Client denied feeling suicidal, or guilty and that she was very reluctant to use the word 'depressed', but consented to use the word 'low'. She stated that it is 'ingrained' in the older generation to present as if one is coping well and not to admit to feeling low.

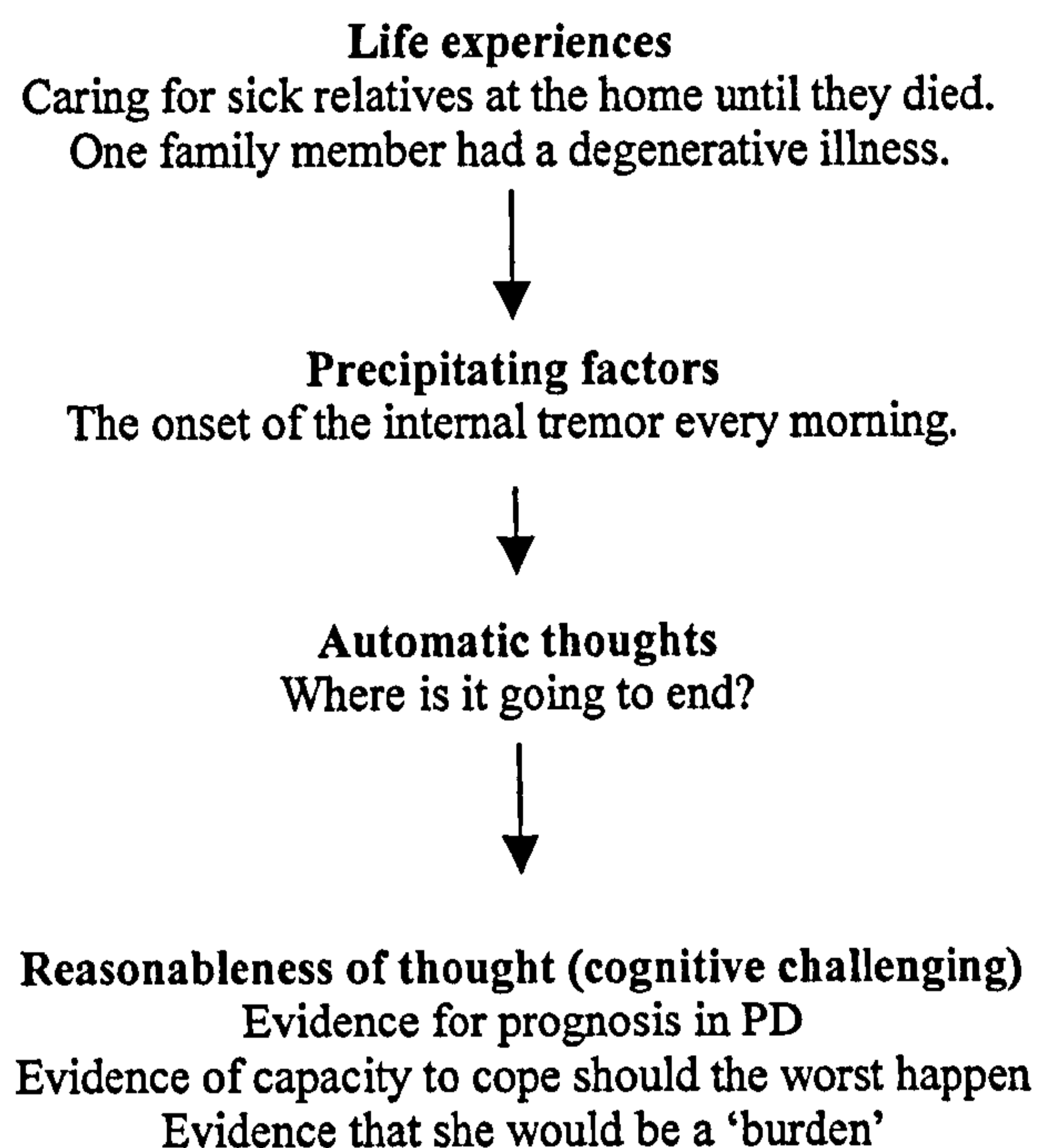
Session 2-3 How should I change my behaviour

Client had a supportive husband and together they kept up an active social life. She was finding it increasingly more difficult to keep up with usual activities as she tired more easily, and was very anxious that people would notice the observable tremor. She was encouraged to include more pleasant activities particularly in the morning when the internal tremor started.

Session 4 – 6 How do I change my depressed thinking

This client was concerned about the prognosis because she had cared for two relatives who had degenerative conditions and did not want her husband to go through the same experience.

Summary formulation



Session 7 How do I solve my problems

Reviewing how to cope when visiting a friend who was dying.

Parkinson's symptoms with the potential to influence therapy

No symptoms appeared to hinder the process of therapy.

Poor recall of work done during therapy revealed at the follow-up session

Case Three

Session 1 How do I know if I am depressed

Client took great offence to the cognitive symptoms mentioned in the booklet stating “what have I got to be guilty about”. She did acknowledge she was depressed and was happy to use the word when describing her symptoms.

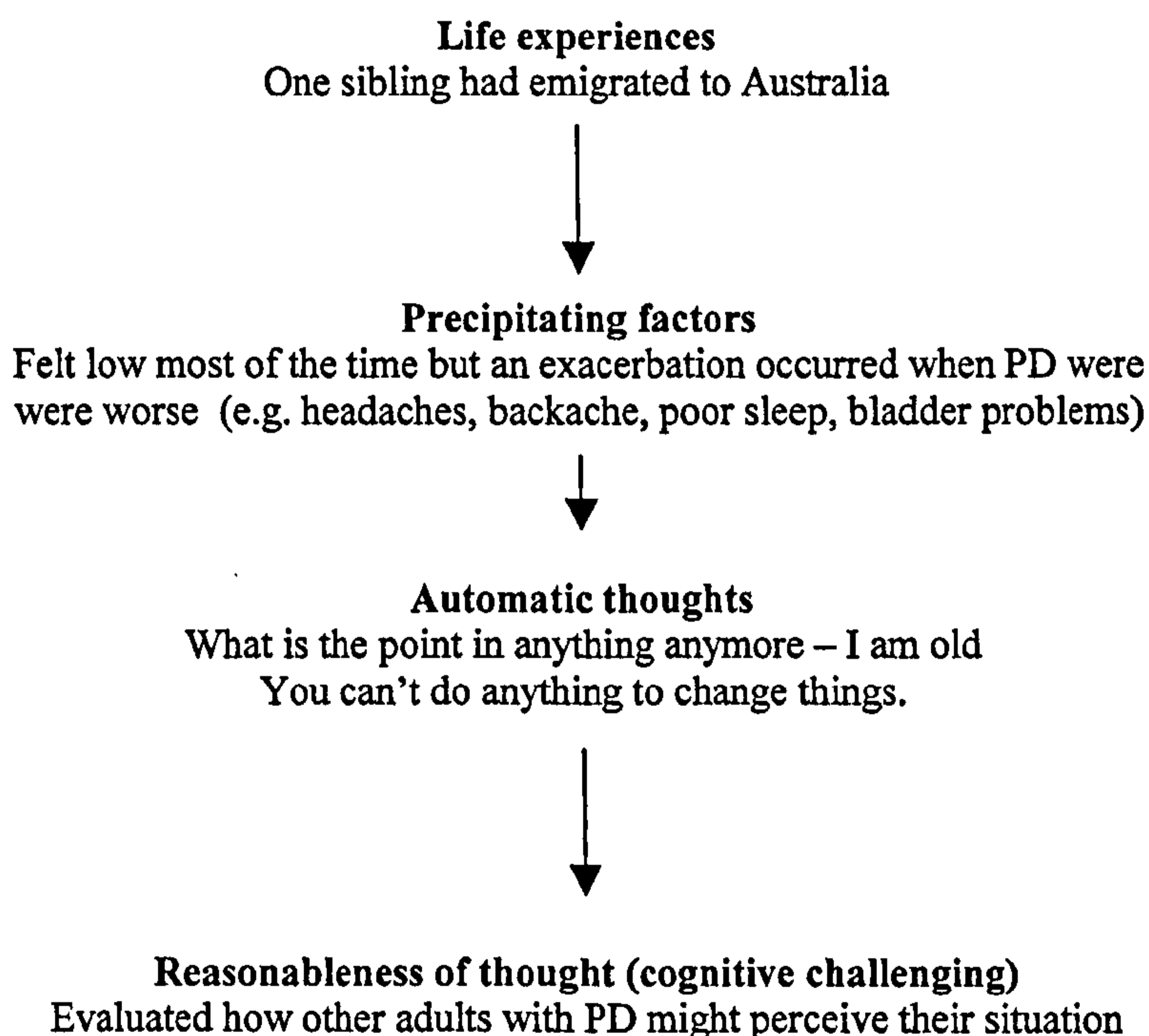
Session 2-3 How should I change my behaviour

Client followed the same routine every day. Her husband brought breakfast in bed and she read the newspaper until dinnertime. After dinner she sat on her settee and did not engage in any activity until teatime. Even though she said the afternoon was the time she felt most low, she was resistant to the idea of introducing pleasurable activities ‘you do not play cards when you are 82’, but finally agreed to plan email messages to her daughter.

Session 4-6 How do I change my depressed thinking

This client felt nothing could be done to change the fact that she was old and had PD and believed that if she did not have PD her mood would be fine. Her mood seemed to lighten (e.g. increase of smiling) as we explored how other people with PD might be managing their mood.

Summary formulation



Session 7 How do I solve my problems

Reviewed methods of keeping in contact with family.

Planned what issues she needed to raise with regard to surgery at the next clinic appointment.

Parkinson's symptoms with the potential to influence therapy

This lady had multiple health problems and experienced frequent headaches following sleep disturbances. Sessions were shortened when her psychical symptoms were severe. Possible executive deficits contributing to difficulty in abstract reasoning.

Case Four

Session 1 How do I know if I am depressed

This client acknowledged he was depressed but did not endorse the cognitive symptoms of worthlessness and guilt. He described his symptoms as a black cloud coming over every day.

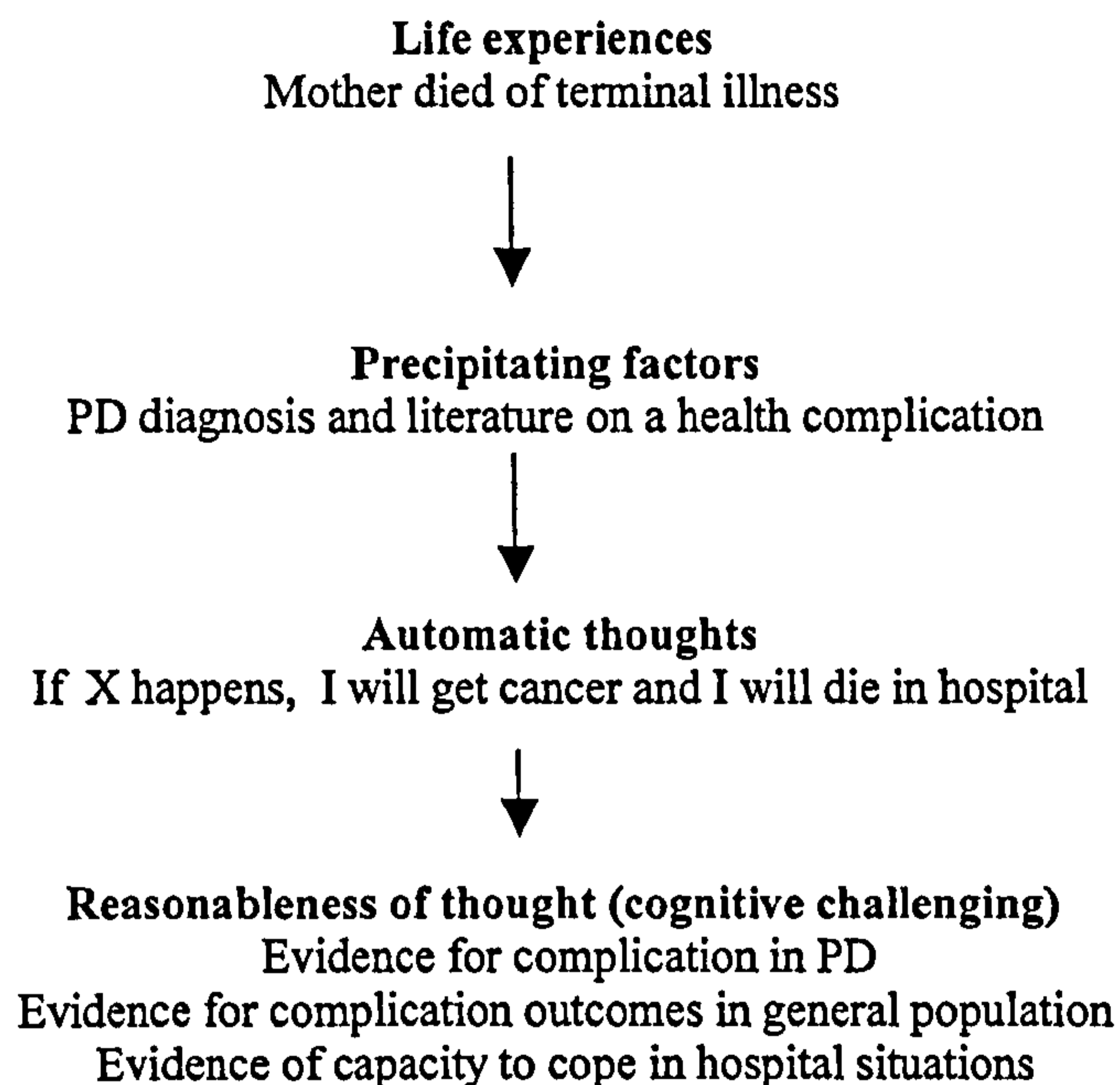
Session 2-3 How should I change my behaviour

This client had become preoccupied with one of the complications of Parkinson's disease and would not engage in any activity because of the problem. He was encouraged to follow his usual routine in spite of his worries, and increase pleasurable activities but he was difficult to motivate.

Session 4-6 How do I change my depressed thinking

He readily acknowledged that his concerns were psychological rather than physical, but he did not seem to be able to process information on alternative thoughts easily.

Summary formulation



Session 7 How do I solve my problems

Planning what questions to raise at the next clinic appointment.
Planning to discuss health issues with long standing friend.

Parkinson's symptoms with the potential to influence therapy

There may have been evidence of slowness of processing information because he appeared to find it difficult to reflect on what was being said.

Case Five

Session 1 How do I know if I am depressed

Client readily acknowledged all symptoms mentioned in the booklet and was happy to use the term depression. She believed mood variations corresponded with the fluctuation of PD symptoms.

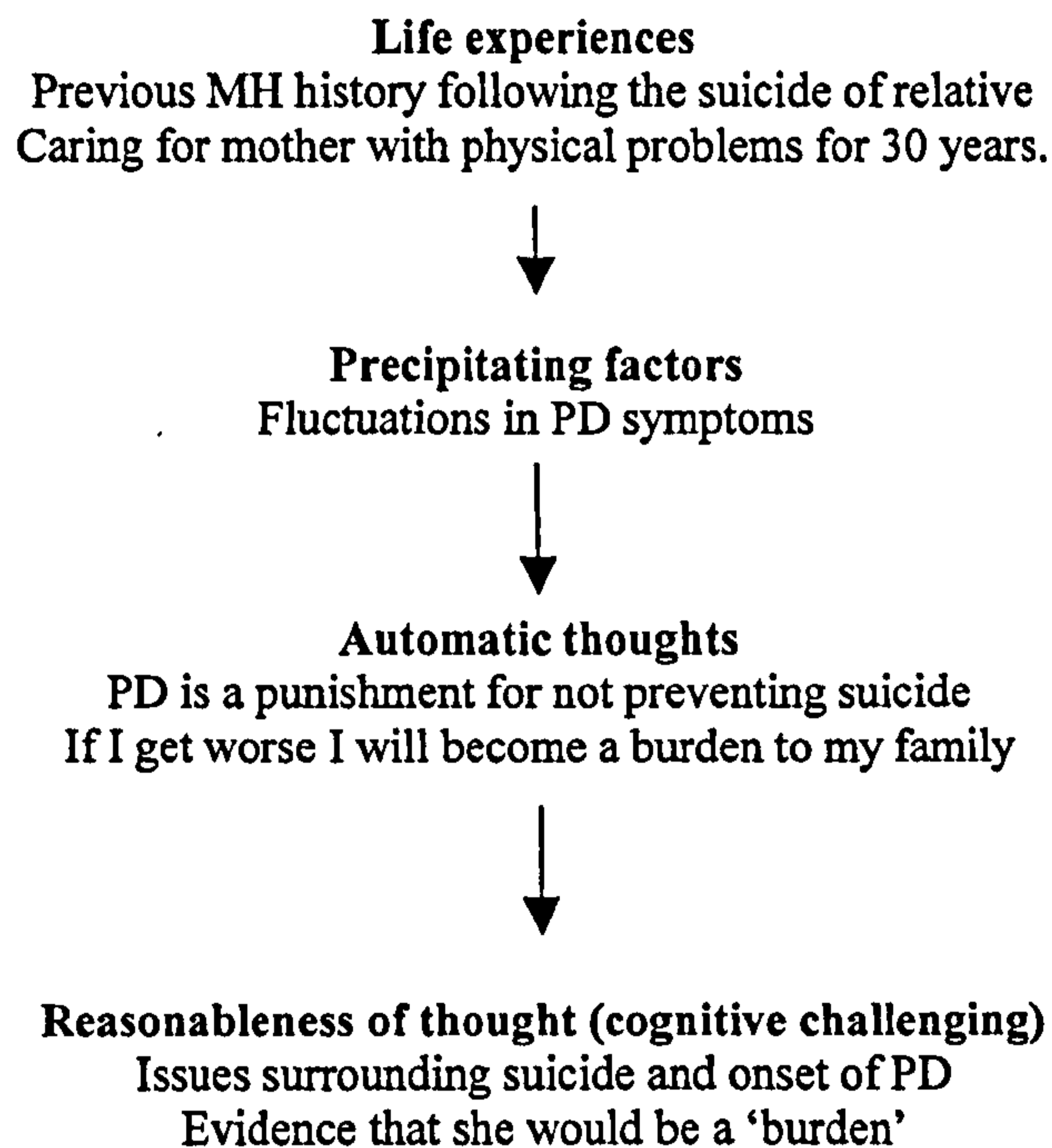
Session 2-3 How should I change my behaviour

Although a previously sociable individual, the depressed mood had resulted in a reluctance to go out of the home. During activity planning, she was encouraged to attend a group for older adults with mental health difficulties for one day a week, and invite the neighbours in for a coffee one afternoon a week.

Session 4-6 How do I change my depressed thinking

Like case 2, having cared for sick relatives, this client was in fear of becoming a 'burden' to her husband and family.

Summary formulation



Session 7 How do I solve my problems

Planning how she could manage low mood on a daily basis. Discuss the advantages and disadvantages of discussion feelings connected to suicide with family members. Arrange continuing psychological support.

Parkinson's symptoms with the potential to influence therapy

Switching off during therapy (e.g. poor concentration, poor recall and exacerbation of movement difficulties).

VI Statement of word count

Word Count

1	Abstract	150
2	Ethics proposal and research protocol (not including text, references or materials)	2250
3	Literature Review (not including references)	6520
4	Research Paper (not including table, figures or references)	5592
5	Critical review (not including references)	2918
	Total	17,430

Appendices

1	Tables, figures and references	
	Ethics proposal	406
	Literature review	3233
	Research paper	2444
	Critical review	878
2	Measures and materials	1505
3	Information sheet, consent, and letters of introduction	1573
4	Supplementary case material	1440
	Total	11479

VII Statistical information

Formula used for the calculation of the Reliable Change Index

$$RC = \frac{X_2 - X_1}{S_{dif}}$$

Figures required for the formula

1 Geriatric Depression Scale

SD = 3.1 Test re-test reliability 0.85 $S_{dif} = 1.69$

2 Beck Depression Inventory – Total Score

SD = 11.74 Test re-test reliability 0.93 $S_{dif} = 4.39$

3 Beck Depression Inventory – Cognitive factor

SD = 4.95 Test re-test reliability 0.85* $S_{dif} = 2.71$

4 Beck Depression Inventory – Affective Somatic Factor

SD = 8.0 Test re-test reliability 0.85* $S_{dif} = 4.36$

* As Steer, Ball, Ranier & Beck, (1999) did not cite test retest reliability scores for separate BDI sub-scales the conservative estimate of 0.85 was used.