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An investigation into negative appraisals and dysfunctional coping strategies associated with posttraumatic stress disorder symptoms following myocardial infarction

Claire Copland

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Adult Mental Health Essay

Discuss the role of cognitive behaviour therapy in the management of a psychotic disorder.

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Year 1
**Introduction**

People with psychotic disorders present with a variety of severe and complex problems. These can include delusions, hallucinations, thought disorder and perceptual disturbances, which are in many cases accompanied by emotional disturbance and social disability (Fowler, Garety & Kuipers, 1995).

Following Kraeplin (1919), the medical model of psychotic disorders has prevailed. Thus, for people experiencing psychosis the usual first line treatment is antipsychotic medication. This will provide some benefit in the majority of cases. However, despite medication, between twenty-five and fifty percent of people diagnosed with schizophrenia will continue to experience hallucinations and delusions (Fowler et al., 1995). A similar percentage will suffer from anxiety and depression (Garety, Kuipers, Fowler, Chamberlain & Dunn, 1994). These distressing and disabling symptoms have significant cost implications in terms of loss of individual potential, family burden and service use. For example, the treatment of schizophrenia consumes about three percent of the total health service spending in the UK and most of these costs are for people who respond poorly to, or who are intolerant of medication (Lewis, 1998). The development of treatments that may further improve outcome, is therefore an important consideration in planning services for people with psychosis.

Behavioural and psychosocial interventions, such as family therapy, social skills training and training in illness management, have been proven successful in reducing relapse and improving social functioning (Kingdon, Turkington & John, 1994). However, in general, these approaches have been criticised for neglecting important aspects of client's experiences (Fowler et al., 1995).

Therefore, there is a need to develop therapies that offer a more comprehensive approach to the understanding and management of psychotic disorders.

**Theory behind the application of Cognitive Behaviour Therapy in Psychosis**

Cognitive behaviour therapy (CBT) is an effective, well-established, treatment for a range of 'neurotic' disorders (Hawton, Salkovskis, Kirk & Clark, 1989). However,
CBT has been slow to be applied to psychosis, despite a number of studies dating from the 1950’s suggesting that cognitive-behavioural strategies may be effective (Slade & Haddock, 1996). This is no doubt in part due to the number and severity of problems associated with the disorder but also reflects the fact that the concept of schizophrenia has precluded the use of reasoning based approaches (Chadwick, Birchwood & Trower, 1996).

Traditionally it has been assumed that psychotic ‘syndromes’ are caused by an underlying biological pathology and as such are discontinuous from normal experience and somehow qualitatively different from other psychiatric disorders (Fowler et al., 1995). This assumption has important clinical implications as it implies that psychotic symptoms will not respond well to psychological intervention. Thus while beliefs in depression are considered open to change and directly targeted in therapy, delusions have been described as “beliefs which are not weakened by counter argument or direct refutation” (Chadwick et al., 1996, pg. xiv). Similarly, the interpretation and personal meaning of hallucinations and delusions have largely been ignored (Chadwick & Birchwood, 1994). However, of the up most importance in the theory and treatment of other psychiatric disorders is an individual’s interpretation of events. For example in the cognitive model of panic, it is the (mis)interpretation of bodily sensations and not the occurrence of sensations per se that is thought to cause panic (Clark, 1989).

Cognitive theorists have instead focussed on individual symptoms of psychosis (e.g. delusions and hallucinations) and not syndromes. This approach has revealed evidence to suggest that it may be more appropriate to regard psychotic symptoms as more severe forms of experiences that may occur in the general population (Fowler et al., 1995). For example, hallucinations have been reported by individuals who have been sexually abused or bereaved and can occur when people are hypnotised, or suffer sensory deprivation or severe stress (Chadwick et al., 1996; Fowler et al., 1995). In addition, examination of the thinking and reasoning styles associated with hallucinations and delusions have identified ‘normal’ thinking process that are biased or distorted. For example, Bentall (1996) suggests that hallucinators tend to misattribute internal events (e.g. thoughts) to external sources and that this process is
influenced by the individual’s beliefs and expectations about what kinds of events are likely to occur. Research also suggests that similar to neurotic disorders it is the beliefs that people have about events that is problematic (e.g. Chadwick & Birchwood, 1994). Thus delusions and hallucinations may be conceptualised in terms of antecedents (A), beliefs (B) and consequences (C) (Chadwick et al., 1996). For example in hallucinations an individual hears a voice (A), which they interpret in a certain way e.g. as threatening (B) and this impacts on the way that they feel or behave (C).

This recognition of the similarities between normal experience and psychosis has led to interest in how CBT techniques used to tackle distorted thinking and poor coping in neurotic disorders may be successfully applied to psychosis and the problems associated with it.

**Cognitive Behavioural Techniques in Psychosis**

A number of different CBT techniques for psychosis have been developed. For example, belief modification, reality testing and rational responding have been used to reduce the impact of positive symptoms. Collectively these techniques aim to identify the beliefs underlying delusions and hallucinations. The person is then helped to question the evidence for these beliefs and to generate alternative responses (Haddock, Morrison, Hopkins, Lewis & Tarrier, 1998). Similar techniques may be used to address the dysfunctional assumptions that a person holds about themselves and others (Fowler et al., 1995). Another approach is coping strategy enhancement. It aims to build on the existing strategies used by individuals so that they are better able to cope with and control their reactions to positive symptoms (Tarrier, Beckett, Harwood, Baker, Yusupoff & Ugarteburu, 1993).

There has also been some interest in applying CBT techniques within a normalising framework that aims to reduce the stigma and anxiety associated with the experience of psychotic symptoms (Turkington & Kingdon, 1996). A central aspect of this intervention is to offer information that helps people to decatastrophise psychosis. For example, the person is told that psychotic symptoms may be experienced by other people, that it is continuous with normal experience and therefore not
necessarily dangerous (Fowler et al., 1995).

As well as employing differing techniques, there is also variation in how CBT is used to manage psychosis. Some authors have taken a specific approach to treatment and focused on one particular symptom. Others have developed more comprehensive treatment approach aimed at tackling positive symptoms, emotional disturbance, relapse and social disability.

However, there are a number of features that are common to all cognitive behaviour therapies for psychosis. Emphasis is placed on detailed assessment in order to tailor interventions to the specific needs of clients. Collaboration is also important and may help to relieve the sense of alienation and loneliness felt by many clients (Chadwick et al., 1996). In addition, all techniques engage clients in discussion about the nature and meaning of their experiences (Fowler et al., 1995).

**Research on CBT in Psychosis**

*Reduction of Drug Resistant Positive symptoms*

The majority of studies of CBT for psychosis have targeted people who continue to experience psychotic symptoms despite taking neuroleptic medication. The effectiveness of these therapies has largely been evaluated via case studies or small case series but there have been a small number of controlled trials.

Tarrier et al. (1993) compared the effectiveness of two CBT approaches - Coping Strategy Enhancement (CSE) and Problem Solving (PS). Results at six-month follow-up indicated that there was a decrease in the number and severity of symptoms for those in the PS and CSE group although changes were mainly restricted to delusions and not hallucinations. There were no changes for those on the waiting list control group. There was some evidence that CSE was superior to PS but this may have been because of pre-treatment differences in the groups.

Haddock, Slade, Bentall, Reid and Faragher (1998) compared focusing therapy to distraction techniques in the management of auditory hallucinations. Twenty-five participants were randomly allocated to either the focusing or distraction group. At
the end of treatment, there was a non-significant trend for participants in CBT groups to experience reductions in the amount of time spent hallucinating and the distress and disruption caused by the hallucinations. When group data was combined there was a significant reduction in time spent hallucinating and disruption, but this improvement was not maintained at two-year follow-up. The only significant difference between groups was that at follow-up focusers showed a significantly greater belief that their voices were their own thoughts.

Garety, Kuipers, Fowler, Chamberlain and Dunn, (1994) evaluated CBT compared to a waiting list control group. CBT was aimed at reducing positive symptomatology, increasing understanding of psychotic disorders and reducing dysfunctional emotions. In relation to positive symptoms, the results showed that the CBT group improved significantly in comparison to controls on delusion conviction. Small reductions were noted in the distress, interference, preoccupation and action associated with delusions. There were no changes reported for hallucinations.

Kuipers, Garety, Fowler, Dunn, Bebbington, Freeman and Hadley (1997) compared CBT and standard care to a control group receiving standard care only. Standard care consisted of case management and medication and CBT was similar to that used by Garety et al. (1994). After nine months, there was a significant difference in overall symptomatology between groups. Fifty percent of the CBT group achieved reliable clinical improvement compared with thirty-one percent in the control group. This improvement was mainly due to changes in delusions and hallucinations with a trend for reduction in delusional conviction, delusional distress and the frequency of hallucinations for the CBT group. These gains were maintained and even improved upon at 18 months after start. Sixty-five percent of the CBT group demonstrated a reduction in overall symptomatology compared to seventeen percent for the control group (Kuipers, Fowler, Garety, Chisholm, Freeman, Dunn, Bebbington & Healy 1998).

Taken together these studies suggest that there is good evidence that CBT has an impact on reducing the severity of delusions, in particular the degree of conviction with which delusions are held. There is less convincing evidence in the treatment of
hallucinations, with studies varying in the success. This observation is supported by a critical analysis carried out by Bouchard, Vallieres, Roy and Maziade (1996) of fifteen studies published between 1973 and 1994 on CBT for chronic psychotic symptoms in schizophrenia. It was found that in general CBT led to a decrease on the measures specific to hallucinations and delusions, but that hallucinations seemed to be more resistant to treatment.

**Acute Stage Psychosis**

The success of reducing drug resistant positive symptoms has prompted interest in how similar techniques may be used to tackle positive symptoms in the acute stage of a psychotic disorder. This may be important for reasons outlined by Drury, Birchwood, Cochrane and Macmillian (1996a). Firstly, studies have demonstrated that in schizophrenia, symptoms that are present after an acute episode persist and indeed worsen after each acute episode. Secondly, long periods of untreated psychosis are associated with increased relapse risk and treatment resistance. Therefore, a shortening of an acute episode may be beneficial and has implication in terms of health service costs. In addition, cognitive flexibility has been demonstrated to be important predictor of change in delusions (Garety et al., 1997). Thus, it makes sense to target CBT in the acute stages of psychosis when delusions and hallucinations may be less entrenched (Haddock et al., 1998).

So far, only one study in this area has been carried out. Drury et al (1996a) investigated the efficacy of CBT compared to a control group. CBT consisted of individual sessions, group and family work aimed at modifying delusion beliefs, enhancing coping strategies for positive symptoms and fostering more adaptive attitudes towards psychosis (i.e. perceiving it as a manageable, meaningful and containable experience). The control group received social and leisure activities, which were similar in terms of the time spent with therapists to CBT. After twelve weeks, both groups demonstrated reductions in positive symptoms, but the CBT group experienced significantly fewer symptoms and rate of improvement was faster. For delusional beliefs, CBT produced significant reduction in belief conviction when compared to the control group, but there was no significant difference in preoccupation. Differences between the groups were maintained at nine months.
follow-up with five percent of the CBT group reporting moderate or severe residual symptoms compared to fifty-six percent of the control group. CBT was also found to produce changes in other features of psychosis that were not directly targeted including, depression, insight and psychotic thinking. Recovery was also time greatly reduced (Drury, Birchwood, Cochrane & Macmillian 1996b)

This study provides evidence that it is possible and effective to use CBT techniques with people who are experiencing acute psychosis. However further research is needed to determine if these findings can be replicated.

**Depression**

Estimates of the prevalence of depression in psychosis range from twenty-two to seventy-five percent (Birchwood & Iqbal, 1998) and may be predictive of relapse and suicide (Fowler et al., 1995). However, as with psychotic disorders in general, depression in psychosis has not been widely addressed within the cognitive framework (Rooke & Birchwood, 1998). Recent research suggests that depression in psychosis may develop as a reaction to the appraisals that individuals make about their symptoms and the experience of chronic mental illness (Birchwood & Iqbal, 1998).

Depression has been linked to the presence of hallucinations. Specifically it seems to be related to the beliefs held by the individual about the power and purpose or intent of their voice. Birchwood and Chadwick (1997) found that sixty-eight percent of people who believed their voices to be malevolent were depressed compared to thirty-five percent who regarded their voices as benevolent. Believing a voice to be very powerful was also associated with higher levels of depression. It is hypothesised that it is the feelings of powerlessness caused by these threatening and powerful voices that leads to depression (Birchwood & Iqbal, 1998).

It is argued that depression in psychosis may also be understood as a reaction to the changes in lifestyle and functioning that are associated with psychosis (Birchwood & Iqbal, 1998). In particular, it is the way that an individual interprets these changes, which is important. Rooke and Chadwick (1998) suggest the onset of psychosis can
lead to a loss of valued roles or goals, humiliation in the face of social stereotypes and entrapment by symptomatology. This may lead to thoughts such as “I am powerless to control my illness”, “I am incapable of little as a result of my illness” and “I cannot talk to people about my illness”. Appraisals of this type were found to distinguish between depressed and non-depressed psychotic patients (Rooke & Chadwick, 1998).

This research suggests that CBT is ideally suited to the management of depression in psychosis. Techniques aimed at modifying people’s beliefs about the power and authority of voices, teaching to control or cope with positive symptoms and identifying and challenging appraisals of psychosis may go some way to easing depression (Birchwood & Iqbal, 1998). In addition, the ‘non specific’ elements of cognitive behaviour therapy, for example the normalising rationale and the emphasis on collaboration, may address some of the feelings of powerlessness and sigma implicated in the development of depression.

However, the success of CBT in achieving changes in depression related to psychosis is mixed. Tarrier et al. (1993) and Haddock et al. (1998) found no changes in depression scores. This may be explained by the fact these studies did not attempt to target individuals’ appraisals of the meaning of their illness. In studies where this was targeted, Kuipers et al. (1997) found no changes in depression scores while Garety et al. (1994) and Drury et al. (1996b) were able to demonstrate an effect of CBT on depression.

**Relapse Prevention**

The issue of relapse is central to the management of psychotic disorders as each relapse is associated with increased probability of future relapse, residual symptoms and social disability (Birchwood, 1996). Long-term neuroleptic medication reduces the risk of relapse, but even then between forty and sixty percent may still relapse over a five-year period (Fowler et al., 1995). Relapse is also an issue of great concern to sufferers themselves with fear of relapse being one of the most common worries of people with psychosis (Fowler et al., 1995).
A central aspect of CBT for all psychiatric disorders is that strategies are taught that will not only alleviate current symptoms but also provide the individual with ways of coping with or preventing the reoccurrence of symptoms in the future. Formal response prevention strategies may also be included in therapy. For example, the collaborative relationship between therapist and client can be used to provide education regarding the benefits and disadvantages of medication. The person can then make an informed decision about how medication can benefit and techniques such as motivational interviewing may be used to increase compliance (Haddock et al., 1998).

Teaching people about the early warning signs of relapse may also be beneficial. Research has demonstrated that individuals and families are able to recognise the signals that suggest relapse is about to occur (Birchwood, Smith, MacMillian, Hogg, Parsad, Harvey & Benny, 1989). These include anxiety, poor attention, withdrawal and low level psychotic thinking (e.g. ideas of references and misinterpretations) which may be detectable between one and four weeks before full blown relapse (Birchwood, 1996). If people are able to mobilise intervention at this stage, e.g. increased medication, support and counselling, a psychotic episode may be curtailed or even prevented (Birchwood, 1996).

There is limited data available at present on how effective CBT approaches are in preventing relapse. Several studies do indicate that the gains made during treatment are maintained in the short term (Tarrier et al., 1993, Drury et al., 1996b), but this may not be the case in the long term (Haddock et al., 1998). However, these studies did not include relapse prevention strategies in the treatment package. Kuipers et al (1997) did include strategies to address the problem of relapse. Although relapse rates are not directly reported for this study, there were differences between the control group and the CBT group in relation to the number of days spent on an inpatient psychiatric ward (Kuipers et al., 1998).

Another area that has yet to be systematically evaluated is the role that CBT plays in supplementing existing relapse prevention strategies. Birchwood (1996) proposes a cognitive model of relapse whereby the attributions that an individual makes about
the exacerbation of symptoms (e.g. "I am relapsing and there is nothing I can do about it") helps to accelerate the relapse process. Thus, CBT may be used as part of an early intervention strategy, to identify faulty cognitions and then help the individual to modify them. In addition, research has shown that high levels of expressed emotion (EE) in families of people with psychosis may contribute to relapse. Behavioural interventions aimed at modifying the levels of EE in families have been demonstrated to lower relapse rates (Kingdon et al., 1994). However, a cognitive approach suggests that certain families behave in ways that an individual may interpret as controlling, critical or rejecting. Thus, these interpretations should be targeted, as well as the behaviour of families (Chadwick et al., 1996).

**Social Disability**

Surveys suggest that up to sixty percent of people with schizophrenia will show signs of social disability, including the inability to work, poor interpersonal relationships and the inability to carry out daily living skills (Fowler et al., 1995). It is therefore an important aspect of the problems experienced by people with psychosis and CBT may provide a structured approach to the management of social disability (Fowler et al., 1995).

However, the impact of CBT on social disability has been disappointing, with none of the controlled studies demonstrating benefits in this area. It is possible that a longer time is needed for changes in positive symptoms to translate into changes of a broader social nature, or that more explicit strategies need to be included to improve social functioning (Garety et al 1994). Although, it may be that the role of CBT may lie in helping people to a stage where they are less depressed and preoccupied with positive symptoms. They would then be more able to access rehabilitation services (Garety et al., 1994) or take part in behavioural interventions such as social skills training.

**Limitations of Research**

As well as small numbers of studies, any discussion of the role of CBT in psychosis is necessarily limited by methodological problems with the research in this area. Difficulties include small sample sizes (e.g. Haddock et al., 1998) which are often
eroded further by drop out (Tarrier et al., 1993). The strict selection criteria of the studies e.g. specifying compliance with medication (Drury et al., 1996a) may also limit the application of studies to the general population of people with psychosis. The diversity of measurement tools used to assess outcome makes it difficult to get a clear picture of the outcome results. Assessments may also be biased when not carried out by blind raters (e.g. Kuipers et al., 1997).

It is also difficult to assess whether changes between groups are the result of CBT or due to other differences between groups. For example the effects of medication may not be controlled for adequately and participants may receive differing levels of medication (e.g. Drury et al., 1996; Kuipers et al., 1997). In addition, control groups may not be comparable. For example, in the Drury et al. study (1996a and b) the activity carried out by the control group could actually overstimulate participants and increase stress and prolong recovery (Johnston, 1996).

Assessing meaningful change in people with psychosis may be particularly problematic. Even if a clinically noticeable change is achieved this may not place people back in the normal range of functioning. On the other hand, small changes in symptoms might make an important difference to an individual's ability to cope with problems or cope in the future (Kuipers et al., 1997). In addition measuring change is complex. In relation to delusions, change may involve an individual becoming less certain that the delusion is true, the content may be modified, the delusion may become less distressing, or the person may be less prone to act on it (Chadwick et al., 1996). These changes may also be difficult to interpret, as cognitive therapy requires people to attend to and consider beliefs. Thus, a person may become more preoccupied with delusion and hallucination during and soon after therapy (Chadwick et al., 1996).

**Theoretical and Practical Limitations of CBT in Psychosis**

Several theoretical and practical factors may also place limitations on the role of CBT in the management of psychotic disorders.

Although cognitive models of psychosis are able to offer an understanding of how
symptoms are maintained and how these processes may relate to treatment, they fail to explain how such symptoms may be caused (Chadwick et al., 1996). This is important because it begs the question, what remains when a symptom disappears? Chadwick et al (1996) suggest that symptoms may be the product of an underlying psychological vulnerability. At present, this theory is in its early stages but it emphasises that careful attention needs to be paid to the function that symptoms may serve for people with a diagnosis of psychosis. For example, Haddock, Sellwood, Tarrier and Yusupoff (1994) describe a case where a patient lived in poor social circumstances and had little social support apart from that provided by hospital staff. A diagnosis of schizophrenia therefore helped this person to cope by maintaining links with mental health services.

A major challenge to the role of CBT in psychosis is the difficulty of engaging clients in therapy. This is demonstrated by high drop out rates e.g. fifty percent in the Tarrier et al. (1993) study. The problem of engagement and drop out is not uncommon in the treatment of psychiatric disorders and has frequently been reported for psychosocial interventions for people with schizophrenia (Tarrier et al., 1993). Therefore, it is to be expected that any intervention with highly disturbed patients may be problematic. However, Chadwick et al. (1996) outline several features of CBT that may make engagement more difficult. These include failure of therapist empathy (e.g. the difficulty of relating to the unusual experiences reported by people with psychosis may make it difficult for therapists to empathise with clients); therapist beliefs (e.g. beliefs about the impact of psychological therapy) and framing delusions as beliefs not facts (e.g. clients may take this as an indication that they are not believed by the therapist). In addition, some clients may be unwilling to discuss their experiences with a therapist. This may be because clients believe that revealing the extent of their delusions and hallucination may result in increased medication or hospitalisation (Chadwick et al., 1996), or because they find the presence of voices reassuring and therefore reluctant to lose them (Chadwick & Birchwood, 1994).

A related problem is that of cognitive deficits. There is good evidence that cognitive deficits may be found in people suffering from psychosis and these include problems with verbal learning, memory functions, attention and concentration and the ability to
understand complex information (Fowler et al., 1995). It has been argued that including an explanation of the possible contribution of cognitive deficits to psychotic symptoms may offer framework to help individual understand their experiences and that recognition of cognitive deficits helps the therapist to tailor their approach to a level which is manageable for the client (Fowler et al., 1995). In some cases, it may be beneficial to combine CBT with cognitive-rehabilitation techniques that attempt to directly modify cognitive deficits (Bentall, Haddock & Slade, 1994). However, individuals with severe cognitive deficits will probably be unable to engage in CBT.

With the current emphasis on value for money in the National Health Service, the question of cost effectiveness will no doubt play a part in determining the role of CBT in psychosis. Kuipers et al. (1998) demonstrated that although CBT was more costly than standard care, these costs were offset by a reduction in overall symptomatology and subsequently in service use. However these results were achieved by intensive treatment (e.g. the mean number of sessions was eighteen and a half with a maximum of fifty) with clients who in addition to CBT received the ‘best’ current routine treatment. Therapy was also carried out by highly experienced clinical psychologists. It is therefore unclear how cost effective CBT may be in routine settings. In addition research suggests that for those people who are willing and able to engage in CBT approximately fifty percent of participants with chronic psychotic symptoms will improve (Kuipers et al., 1997; Tarrier et al., 1993). This is comparable to the response rate for the new generation of neuroleptic medication which includes clozapine (Lewis, 1998). There have as yet not been any studies that compare CBT to clozapine. However, any advantages of medication over CBT would have to take into consideration the problems of possible non-compliance and the physical side effects of medication.

Conclusion
The cognitive behavioural approach has made a valuable contribution to our understanding of psychotic symptoms. It has highlighted the importance of the meaning that people attach to their experiences of psychosis and demonstrated that normal psychological processes contribute to positive symptoms, depression and
relapse. Thus, engaging clients in discussion about symptoms may be beneficial and reassuring rather than useless or harmful.

However, the role of CBT in psychosis is yet to be clearly defined. A relatively limited amount of research has been carried out so far. It may be that the 'therapeutic enthusiasm' (Fowler et al., 1995) for CBT in psychosis has overlooked the benefits actually achieved to date. Studies seem to indicate that CBT may be effective in producing changes in positive symptomatology, while its impact in other areas is less certain. Although, research in acute psychosis suggests that CBT may be more successful if used in the early stages of the disorder.

More research is therefore needed to further develop CBT techniques for psychosis. In addition, the aspects of CBT which may be most useful, which clients may benefit and the optimum length of therapy need to be more thoroughly investigated. Follow-up studies over longer periods are also required to assess whether gains made during therapy are maintained.

At present, it seems that CBT may play a role in complimenting and even improving upon existing interventions such as family therapies, early intervention and medication. CBT is a generally acceptable approach and clients reported high levels of satisfaction and no demonstrable negative consequences (Kuipers et al., 1997). Therefore, it may also be an important technique for people who have previously refused other kinds of treatment.

The challenge for future research is to discover to what extent the role suggested for CBT by theory may actually apply in practice.
References


What is an autistic spectrum disorder (ASD) and what aetiological models have contributed to our understanding of autism? To what extent have these models guided intervention practices?

June 1999

Year 1
Introduction

“They’re withdrawn….they can’t communicate….they’re very musical….they’re very good at maths….they’re very clever….they’re mentally handicapped” (Arrons & Gittens 1992, pg. 5) are some of the wide ranging ideas expressed by the public about autism. Indeed, throughout history, there have been folktales and legends about children and adults with communications problems and unusual social and repetitive behaviours together with ‘islets’ of preserved ability (Wing, 1996).

Such patterns of behaviour have been noted by clinicians since the nineteenth century, for example Itard’s (1801) description of the ‘Wild boy of Aveuron’ (cited in Trevarthen, Aitken, Papoudi & Robarts, 1996). However, it was not until Kanner’s (1943) systematic description that the syndrome of autism was recognised. The features of what Kanner named ‘early infantile autism’ included a lack of emotional contact with other people, desire for sameness in routines, excellent rote memory, abnormalities in speech and a fascination with handling objects (Wing, 1996). Kanner later suggested that it was the first two of these features that were key to defining the syndrome of autism (Wing, 1996).

The diagnostic criteria for autism have been refined over the years, but today many children and adults with autistic disorder still conform to the basic picture described by Kanner over fifty years ago (Happé, 1994). However, there has been a growing awareness that there are many people with autistic like behaviours who do not fit the criteria for typical autism. This has led to the introduction of the concept of ‘autistic spectrum disorders’, which will be discussed in the first section of this essay.

In the second section of the essay, aetiological models of autism are discussed. Although a vast number of theories have been proposed to account for autistic behaviour, many have contributed little to our understanding of autism. Indeed some, for example psychogenic theories, which claim that inadequate parenting is the cause of autism, have been positively damaging (Happé, 1994). Others models, for example those developed from behavioural theories, have been helpful in developing specific interventions but have focussed on only some aspects of autism and ignored other important factors (Frith, 1991). At present cognitive models of autism provide
the most complete explanation of the cause of autism (Frith, 1991) and it is these models which this essay will focus on.

Finally, interventions for autism will be discussed and the extent to which cognitive models have contributed to clinical practice will be investigated.

**Autistic Spectrum Disorders**

The development of the concept of autistic spectrum disorders begins with research into the core features of autism. Although Kanner described the basic nature of autism, his observations were based on a small and highly selective sample of children. Consequently his description of autism included some features, which were later shown to be secondary or unrelated to autism (Happe, 1994). For example, Kanner assumed that autistic children came from middle class families (Volkmar & Lord, 1998) and that they were all of normal intelligence (Arrons & Gittens, 1992). Therefore, it was necessary to more precisely define diagnostic criteria for autism.

Significant progress in determining diagnostic criteria for typical autism was made in a series of empirical studies carried out at the Maudsley Hospital in the 1960's. This research demonstrated that typical autism could be reliably discriminated from other psychiatric disorders occurring in childhood such as psychosis (Rutter, 1999). The three areas of behaviour which were significantly more frequent in autistic children were (1) a failure to develop social relationships (2) language impairments and (3) ritualistic and compulsive behaviours. These features, together with an onset before thirty months, were taken as the defining characteristics of autism (Rutter, 1999). It was this description that shaped the first official definition of typical autism in DSM-III (American Psychiatric Association, 1980), (Volkmar & Lord, 1998).

Research into nature and aetiology of typical autism has also changed the way in which the disorder is thought about. For example, follow-up studies that showed the link between autism and epilepsy, neurological impairment and learning disabilities suggested that autism may be a biologically based disorder of development (Lord & Rutter, 1994). Subsequently, interest was focused on the skills which children with autism did not develop. This highlighted that children with autism not only had
impairments in social and communication skills, but also showed a lack in the development of imagination (Wing, 1996). For example, autistic children show a marked absence of pretend play when compared to normal children of the same age (Happé, 1994). This so-called 'triad' of impairments in socialisation, communication, and imagination has been demonstrated to be central to autism. Its presence reliably discriminates between autistic and non-autistic individuals (Wing & Gould, 1979). The idea of the triad of impairments has been incorporated in the subsequent classification systems for typical autism, which is now termed autistic disorder (DSM, IV, American Psychiatric Association, 1994).

However, just as there was beginning to be a consensus as to the diagnosis and nature of autistic disorder, there was an increasing awareness of how variable the presentation of autism could be. This was highlighted in an epidemiological study of one hundred and thirty-two children who had learning disabilities and / or social impairment, verbal and non-verbal language impairments and stereotyped activities (Wing & Gould, 1979). The study demonstrated that the triad of social, communication and imagination impairments not only marked out those children already diagnosed with typical autism but also applied to a wider sample of children. For example, in the sample there were not only children who were withdrawn, as described by Kanner, but also children who had “active but odd” social behaviour. Similarly, the communication impairments observed ranged from muteness to fluent but inappropriate speech (Happé & Frith, 1996).

Recognition of the variable picture of autism renewed interest in the work of Hans Asperger who had described a pattern of behaviour, which he referred to as ‘autistic psychopathology’ (now called Asperger's syndrome) (Happé, 1994). Despite publishing his research around the same time as Kanner, Asperger’s description did not receive attention in America and the U.K. and was largely ignored until reviewed by Wing (1981). The syndrome described by Asperger showed many similarities to that described by Kanner including social isolation, impaired communication, lack of flexibility in play and isolated special interests (Wing, 1991). However, Asperger also noted features in his children that were different from Kanner’s group (Wing, 1991). Firstly, Asperger reported his subjects used fluent, albeit odd language in
contrast to sparse use of words observed by Kanner. Secondly, Asperger commented on poor motor co-ordination, while Kanner was struck by the dexterity that his group of children displayed. Lastly, Asperger’s group of children seemed to have a better capacity for learning (Happe, 1994).

As with autistic disorder, the diagnostic criteria for Asperger’s syndrome has undergone a number of refinements over the years. At present DSM IV (American Psychiatric Association, 1994) criteria for Asperger’s syndrome requires the presence of the triad of impairments together with repetitive patterns of behaviours and interests as with autistic disorder. The significant difference between the two disorders is that in Asperger’s syndrome there is no clinically significant delay in language development and intelligence is in the normal range.

There has been much debate by researchers and clinicians as to whether Asperger’s syndrome is qualitatively or quantitatively different from autistic disorder. Some authors have argued that Asperger’s syndrome and autistic disorder are entirely different syndromes. For example Van Krevelen (1971: cited in Happe, 1994) suggested that autistic disorder was the result of a psychotic process, whereas Asperger’s syndrome was a personality trait. However, other researchers point to the relationship between the two disorders. For example, individuals diagnosed with autistic disorder in childhood have been observed to develop Asperger’s syndrome in later life (Wing, 1991). There also seems to be a high incidence of autistic disorder and Asperger’s syndrome occurring in the same family (Happe, 1994). This suggests that Asperger’s syndrome is on a continuum with autism and may be viewed as a milder form of autistic disorder (Wing, 1991).

Adopting Asperger’s syndrome as a diagnostic category helped to describe some people who did not fit the criteria for autistic disorder. However, there still remained the problem of how to classify syndromes such as those described by Rett (1966) and Heller (1930) which could be associated with autistic features (both cited in Volkmar & Lord, 1998). In addition, there were a number of children identified in the Wing and Gould (1979) study who did not fit the criteria for either autistic disorder or Asperger’s syndrome (Wing, 1991).
Wing (1991) suggests that the best way to explain this wide variation of autistic behaviour is to employ the concept of an 'autistic spectrum.' Disorders on the autistic spectrum share the triad of impairments but differ from autistic disorder in the severity of impairments, presentation across time and/or may have a number of additional defining features (Lord & Risi, 1998). The spectrum of autistic disorders is recognised in DSM IV (American Psychiatric Association, 1994) under the term 'pervasive developmental disorders.' This category includes autistic disorder, Asperger's syndrome, Rett's syndrome and Childhood Disintegrative disorder. The term 'Pervasive Developmental Disorder not otherwise specified' is also included to encompass cases with features of autism that do not meet any of the diagnostic features for the other defined disorders (Lord & Risi, 1999).

Although there is still controversy as to which disorders should be considered as part of the autistic spectrum (Happe, 1994), the term does appear to have some clinical value. It has enabled clinicians to recognise children and adults who do not met the criteria for autistic disorder and provide these people and their families services and support which they may not previously have received (Lord & Risi, 1999). In addition, stipulating the features necessary for a disorder to be part of the autistic spectrum has defined what needs to be explained in causal models of autism.

Aetiological Models of Autism
A minimum requirement for aetiological models of autism is to explain the co-occurrence of the triad of deficits (Happe, 1994). However, this is not as easy as it may appear. Each of these areas is made up of a great variety of different behaviours with specific patterns of deficits and preserved abilities. For example, in the area of socialisation, autistic children sometimes show attachment behaviours that are no different from those of non-autistic children. However, autistic children seem to be impaired in the ability to recognise emotions (Happe, 1994).

In addition, causal models should be able to integrate research findings from a number of different fields of study (Frith & Happe, 1995). There is now a wealth of evidence which demonstrates basic and persistent cognitive deficits in autism such as
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impairments in planning, attention control and the ability to sequence information (Bailey, Philips & Rutter, 1996). There is also data to suggest the role of biological factors in autism. The high incidence of learning disabilities and epilepsy in people with autism is suggestive of some kind of brain damage (Happé, 1994). In addition, the higher rate of autism in males, the high concordance rate between identical twins and the association of autism with disorders such as Fragile X and tuberous sclerosis suggests a genetic component (Fisher, Van Dyke, Sears, Matson, Lin Dyken, & McBrien, 1999).

Cognitive models of autism have addressed these issues by considering aetiology in terms of levels of explanation. Here the relationship between behavioural features and biological and cognitive factors are explored (Happé, 1994). Morton and Frith (1994: cited in Happé, 1994) have proposed a number of ways to describe this relationship in developmental disorders where there may be either single or multiple deficits at each level. The systematic co-occurrence of the triad of impairments in autism suggested that a single cognitive deficit may be responsible for these behaviours (Frith, 1996). Therefore, a causal model for autism maybe similar to that shown in Figure 1.

Figure 1. Morton and Frith's proposed model of autism (from Happé, 1994)

Research into the nature of this single cognitive deficit has produced three main cognitive theories of autism.
Theory of Mind

A number of theories have sought to explain autistic behaviour as due to a lack of social insight (Happe & Frith, 1996). The best researched of these is ‘theory of mind’.

‘Theory of mind’ is a term that has been used in developmental psychology. It refers to an individual’s ability to (a) appreciate one’s own and other people’s mental states and (b) to understand the link between mental states and action (Baron-Cohen & Swettenham, 1997). An individual’s ability to use theory of mind is investigated using ‘false belief test’ such as the Sally–Ann test (Frith, 1996). In such a test, theory of mind is demonstrated by understanding and predicting the behaviour of the character based on that character’s beliefs and knowledge and not what is known by the observer (Happe, 1994).

Experiments using a variety of different false belief tests have demonstrated that from around four years of age normal children are able to pass such tests (Happe, 1994). Children with autism, on the other hand, fail these tests and thus demonstrate a lack of ‘mentalising’ – the ability to think about thoughts (Frith, 1996). Different experimental paradigms have highlighted other problems autistic children have because they are unable to mentalise. These studies show that autistic children are poor at understanding how objects differ from thoughts about objects, understanding functions of the mind, making appearance-reality distinctions and understanding metaphor (Baron-Cohen & Swettenham, 1997).

The theory of mind account is able to neatly explain why autistic people have impairments in social, communicative and imagination because mentalising is a prerequisite for all these skills (Happe, 1994). Theory of mind can also explain why people with autism are impaired in only certain aspects of this triad of behaviours. Not all social interactions require an understanding of other people’s mental states and it is only in situations which require theory of mind that people with autism will be at a disadvantage (Happe, 1994). For example, Atwood, Frith and Hermlin (1988: cited in Happe, 1994) showed that autistic children are able to use gestures to
communicate ideas about behaviour e.g. 'come here', but do not use gestures to signal mental state e.g. consolation.

One challenge to the theory of mind account is that between fifteen and sixty percent of people with autism will pass theory of mind tests (Happe, 1994). One explanation of this is that their performance is not based on theory of mind and that they are able to somehow compute solutions (Happe, 1994). However, some people who pass such tasks are also able to apply theory of mind skills in everyday situations (Happe & Frith, 1996). Proponents of theory of mind counter this argument with evidence from research which demonstrates that no child with autism who has been tested possesses normal theory of mind skills at the appropriate age (Baron-Cohen & Swettenham, 1997). Thus people with autism may eventually acquire theory of mind skills, but it is the delay in its development which causes the triad of impairments (Happe, 1994).

Despite the success of theory of mind in explaining the triad of impairments in autism, there are a number of limitations to this model. It does not address the reasons for the varying severity of symptoms and cannot account for why learning disability is prevalent in autism (Frith, 1996). In addition, theory of mind focuses on explaining the nature of deficits in the areas of imagination, social interaction, and communication. However, lack of mentalising cannot explain the non-triad features of autism that can include a restricted repertoire of interests or preoccupation with parts of objects (Frith & Happe, 1995).

In answer to these criticisms, two other cognitive theories have been developed to tackle the issues neglected in the theory of mind account.

**Executive function deficits**

In attempting to explain the presence of restricted, repetitive and stereotyped patterns of behaviour in autism, researchers have drawn parallels between autism and the behaviour of patients who have acquired frontal lobe injuries (Frith, 1996). Such patients are said to have deficits in executive function. Executive function refers to a range of higher cognitive processes such as the ability to disengage from context,
inhibition of inappropriate responses, planning, and organisation (Happe & Frith, 1996). There is some evidence that individuals with autism do display problems characteristic of executive function deficits. Ozonoff, Rogers and Pennington, (1991) found that subjects with autism were all impaired on two typical tests of executive function – the Tower of Hanoi and the Wisconsin Card Sort Test. Individuals with autism have also been demonstrated to have problems in planning, organisation, and switching set (Happe & Frith, 1996). Thus, an executive function deficit may explain stereotyped behaviours and interests seen in autism.

It has also been suggested that executive function deficit may underlie some of the social deficits seen in autism. Prior and Ozonoff (1998) suggest certain executive function skills, such as flexibility, may be a prerequisite for social behaviours. However, it is clear that executive function deficits cannot completely account for the triad of impairments in autism (Bailey et al., 1996).

A limitation to the executive function deficit theory of autism is that acquired frontal lobe injury patients do not show other behaviours which are typical of autism (Frith, 1996). One possible explanation of this is that brain damage from birth and damage in later life give rise to a different clinical picture (Frith, 1996). However, executive function impairments have also been found children with other psychiatric disorders, such as attention deficit hyperactivity disorder and conduct disorder (Happe & Frith, 1996). A further limitation is that suggesting a global impairment in executive function makes it difficult for the theory to account for preserved skills (Happe & Frith, 1996).

Weak Central Coherence Theory
A third cognitive theory proposes that autism might be explained by a difficulty in integrating different levels of information (Happe, 1994). Normal information processing seems to involve the ability to extract the overall meaning of a situation by drawing together information from different sources including context (Frith & Happé, 1995). This is called ‘central coherence’. An example of this is the way that ambiguous words e.g. pear – pair, are appropriately recognised when used in context (Frith & Happé, 1995).
If weak central coherence is a feature of autism then it is predicted that autistic subjects would be good at tasks that require attention to detail, but poor on tasks that require more global processing. Support for this theory has been demonstrated by the fact that autistic children are superior to controls on tasks where processing of parts over wholes is useful, such as the Embedded Figures Test and the Block Design subscale of WAIS –R (Frith & Happé, 1995). In contrast, in tests involving the use of overall context and meaning, e.g. processing faces, autistic subjects do less well (Frith and Happé, 1995).

The weak central coherence theory is able to explain why autistic people pay excessive attention to detail (Frith, 1996) and the presence of islets of ability (Baron-Cohen & Swettenham, 1997). However, like the theory of executive function deficit, weak central coherence has little to say about the triad of autistic behaviours.

**The relationship between the three theories**

Each of the three theories reviewed here are able to account for differing types of behaviour observed in people with autism. This suggests a model of autism that specifies a single cognitive deficit may not be the best causal explanation of the disorder. A model which incorporates multiple cognitive deficits might be a more realistic view and may help to explain the variation of behaviours in autism (Frith, 1996).

Indeed, research suggests that the three cognitive theories are not mutually exclusive. For example, because people with autism show evidence of weak central coherence, regardless of level of ability on theory of mind tests, central coherence cannot replace theory of mind in explaining autism (Frith & Happé, 1995). Thus, weak central coherence and theory of mind impairment may exist independently in autism (Baron –Cohen & Swettenham, 1997). A possible way to explain the presence of both these deficits has been proposed by Frith & Happé (1995). They suggest that the ability to mentalise may be caused by a single area of damage in the brain, while lack of central coherence is a cognitive style that may be subject to both environmental and genetic influences.
There is also likely to be an overlap between executive function deficits, impairment in theory of mind and weak central coherence (Baron-Cohen & Swettenham, 1997). For example, Bailey et al., (1996) suggest that executive function problems may impair the development of theory of mind.

Cognitive models of autism are clearly at an early stage, but they have made a significant contribution to our understanding of autism. Firstly, the development of cognitive models has produced careful investigation of the patterns of deficits and preserved abilities in autism and has led to a better understanding of core behaviour patterns (Happé & Frith, 1996). Secondly, the theories provide a framework to link biology, cognition and behaviour and have already started to guide the investigations into the specific biological basis of autism (Frith, 1991). Thirdly, in the process of constructing cognitive models, a number of tasks have been devised to test the validity of the theories. Use of these tests may help to clarify the boundaries between autism and other disorders e.g. semantic pragmatic disorder (Bailey et al., 1996).

Research into cognitive models of autism, however, is not only of academic importance. Improvements in clinical practice are also reliant on understanding the causal psychological processes in the disorder (Rutter, 1999). So what practical implications do cognitive models have for people with autism and to what extent have these models guided intervention?

**Intervention**

People with autism frequently present with not only the triad of impairments and obsessive behaviours but also less specific emotional and behaviour problems. These may include overactivity, restricted diets and sleep problems (Howlin & Rutter, 1987). In addition there is a high rate of learning disabilities in people with autism and associated challenging behaviour (Howlin, 1998).

Although most people improve in functioning with age (Howlin, 1998), almost all children with autism will continue to require care and support throughout their adult
lives (Rogers, 1998). In response to the obvious need for effective interventions, a range of therapies have been developed over the years, for example, Holding therapy, music therapy and auditory integration. Although these therapies claim to be miracle 'cures' for autism, few of them are supported by empirical data (Howlin, 1998). In addition, these therapies target only one area of impairment in autism. As autism is associated with a range of both primary and secondary problems, it is likely that effective therapies would have to involve intervention in a number of areas (Lord & Rutter, 1994).

Rutter (1985) suggests there the four important aspects of interventions for people with autism. These are (1) fostering social and communicative development, (2) enhancing learning and problem solving, (3) decreasing behaviours that interfere with learning and access to opportunities for normal experiences and (4) helping families cope with autism.

Several behavioural intervention packages have been developed which attempt to address several, if not all of these areas. For example, the Home-Based Teaching Programme (Howlin & Rutter, 1987). This is an outreach service for young autistic children and their families. The programme employs a range of behavioural techniques together with counselling sessions for families. Evaluation of this programme showed that in comparison to controls, autistic children showed a reduction in maladaptive behaviour, an improvement in play and social behaviours and the use of communicative language, which were maintained at eighteen month follow-up (Howlin & Rutter 1987). Another well validated intervention is TEAACH developed by Scholper and Reicher (1979) which focuses on the development of appropriate communication skills and personal autonomy to maximise the quality of life of people with autism (Trevarthen et al., 1996).

At first glance, it would appear that cognitive theories have had very little influence on such behaviourally oriented programmes. However, cognitive theories of autism have contributed to these interventions in a number of ways.

Firstly, it could be argued that cognitive theories of autism have led the way for the
application of education and behaviour programmes. Research demonstrating the presence of cognitive deficits in autism turned the tide against psychoanalytic theories. It suggested that the focus of intervention should be on educational and behavioural programs to help children better cope with a basic cognitive deficit (Rutter, 1999). Secondly, cognitive models that emphasise the developmental nature of autism have shaped behavioural programmes to take into the developmental level (Howlin & Rutter, 1987). Thirdly, cognitive models of autism have allowed clinicians, parents and teachers to more thoroughly understand the behaviour of people with autism. For example, the deficit in theory of mind account of autism suggests that people with autism are unable to think about thoughts. From this perspective it is no wonder that the world seems terrifying and unpredictable to a person with autism and that they try to withdraw from it (Happe, 1994).

As far as contributing to specific interventions for autism it is only the deficits in theory of mind model that has been investigated thus far. As the theory suggests that the inability to mind read is at the heart of the triad of autistic behaviour, there has been interest in teaching autistic children to pass theory of mind tests and thus acquire theory of mind. Several methods, for example repetition, explanation and correction, prompting and modelling of correct responses, have been used to aid children pass theory of mind tests (McGregor, Whiten & Blackburn, 1998). Although children were able to learn to pass the test used in the teaching sessions, none of the approaches resulted in generalisation of theory of mind to other tests or situations (McGregor et al. 1998). Given the difficulty of teaching autistic children to acquire a conceptual understanding of theory of mind, Swettenham, Baron-Cohen, Gomez and Walsh (1996) aimed to teach children an alternative route to predicting the behaviour of others. These authors developed a teaching strategy based on the finding that autistic children are able to understand physical representations and suggested to them that seeing was like taking a picture. Results showed that autistic children could use this method to pass a variety, but not all of theory of mind tests.

Although studies so far have only demonstrated limited success, training packages which increase autistic people’s ability to use theory of mind, may be an important addition to intervention programmes (Howlin, 1998)
Theory of mind has also contributed in the area of early detection of autism. Research into the developmental origin of theory of mind suggests that joint attention skills such as pointing gestures and gaze monitoring are absent in very young children with autism (Baron-Cohen & Swettenham, 1997). It is suggested that this may result from a failure to appreciate other people’s point of view. Thus, joint attention deficit may be a precursor to the development of theory of mind (Baron-Cohen & Swettenham, 1997). This idea may have value in detecting autism in children at an earlier age and has been used in the development of the Checklist for Autism in Toddlers (Baron-Cohen & Swettenham, 1997). This is an important contribution as early detection of autism impacts on the effectiveness of interventions in two ways (Trevarthen et al., 1996). Firstly, one of the predictors of good outcome is the level of language ability at thirty months. Secondly, early intervention can minimise secondary behaviour difficulties that can arise because of obsessions and rituals.

Conclusion
Kanner’s (1943) now classic description of autism brought the syndrome to the attention of clinicians and was the starting point for over fifty years of research into the definition, aetiology and treatment of the disorder. Indeed autism is now one of the most discussed syndromes in the literature on developmental disorders (Wolf-Shein, 1994). This intense interest has had a significant impact on the concept of autism. It is no longer thought of as psychogenically caused disorder and there is a wealth of evidence to suggest that biological and cognitive factors play a role in causing the behaviour patterns seen in autism. In addition, behaviour therapies have contributed a great deal to reducing the impact that autism has on individuals and families.

However, autism remains a difficult disorder to understand. There is still confusion surrounding the definition of what is and what is not autism. This difficulty is reflected in the changing criteria for autism and related disorders in classification systems (Wing, 1996). Adopting the concept of a spectrum of autistic disorders, which have the triad of impairments in socialisation, communication and imagination...
as their defining features, has gone some way to clarifying diagnostic issues. Although controversy remains about the boundaries between autistic spectrum disorder and other disorders.

Cognitive theories have contributed significantly to our understanding of autism and in the future may be able to inform judgements about autistic spectrum disorders and their biological basis. At present though, the current limitations of these theories means that they are unable provide a complete explanation of the behavioural impairments seen in autism. More research is needed to refine and extend the explanatory power of the theories, in particular theories of weak central coherence and executive function deficit. In addition, the relationship between the cognitive deficits proposed by the three major theories needs clarification. In particular, if these deficits are all present in autism, what combination is necessary and sufficient for autism to develop (Baron-Cohen, 1997)?

Although cognitive theories have influenced intervention practices at a general level, it is only theory of mind that has made any direct contribution to intervention. The current limitations of the theories is one explanation of why this gap between theory and practice exists. In addition specific cognitive theories have only recently been developed and as Rutter (1999) points out it often takes many years before research findings are translated into interventions.

It remains to be seen whether the early promise shown by cognitive theories of autism in enhancing our understanding of and interventions for autism is borne out in the future.
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Children, Adolescents and Families Essay

What theories have been advanced to explain childhood phobias? Consider their strengths and limitations and the evidence base that would support their use in clinical practice.

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Year 2
Introduction

Fear is a normal reaction to a situation that poses a threat to a person's wellbeing, safety or security (Carr, 1999). Fear reactions can be described in terms of three response systems – cognitive, physiological and behavioural (Lang, 1968: cited in King, Ollendick & Murphy, 1997). In children, cognitive responses to fearful situations include thoughts of being scared and self-deprecatory thoughts. Physiological responses are likely to be increased heart rate and changes in breathing, while behavioural responses include rigid posture, thumb sucking, and avoidance (King, et al., 1997).

During the course of normal development, children experience many fears (King, Eleonara & Ollendick, 1998). These fears tend to follow developmental 'trends' with infants fearing for example, loud noises and unfamiliar people, while in early childhood, fear of small animals and the dark is typically experienced (Silverman & Rabian, 1996). Many of these fears can be seen as adaptive responses to everyday situations and reflect the development of cognitive and representation capacities (King & Ollendick, 1997). Moreover, in the majority of cases these fears are usually short lived and do not cause significant problems for the child or their family (King, Eleonora & Ollendick, 1998).

For some children, however, fear reactions can persist and become maladaptive. The diagnostic classification system DSM IV (American Psychiatric Association, 1994) recognises such fears as 'phobias'. The main features that distinguish a phobia from a normal childhood fear are: (a) a marked and persistent (more than six months) fear that is excessive and cued by the presence or anticipation of an object or situation; (b) exposure to the phobic stimulus almost invariably provokes an immediate anxiety response; (c) the person recognises that the fear is excessive, although this may not be the case for all children; (d) the phobic situation is avoided or else is endured with intense anxiety or distress; and (e) the avoidance or distress in the feared situation interferes significantly with the person's normal routine. DSM IV (American Psychiatric Association, 1994) also makes a distinction between social and specific phobias. Social phobia describes a fear of situations where the person is exposed to evaluation by others, while in specific phobia, excessive fear is experienced in non-
evaluative situations e.g. at the sight of blood.

Epidemiological studies have shown that the estimates of the prevalence of specific phobias in childhood range from two percent to nine percent with an average of five percent across studies (King, Eleonara & Ollendick, 1998). The presence of other types of anxiety disorder, including social phobia, is around two percent (King, Eleonara & Ollendick, 1998). Thus, phobias are a frequent problem in childhood.

It is hoped that by understanding the determinants of childhood phobias, assessment and treatment of such problems will be improved (King, Eleonara & Ollendick, 1998). However, explaining childhood phobias has proved to be a perplexing issue for researchers (King, Eleonara & Ollendick, 1998). This has resulted in multiple theories being advanced. To review them all would be beyond the scope of this essay and therefore only the most notable contributions in the areas of psychoanalytic, behavioural, nonassociative, biological and cognitive theories will be discussed. Family systems theories will not be considered separately as there is no integrated theory (Carr, 1999). Instead, family factors will be addressed when they contribute to the theories discussed.

**Psychoanalytic Theories**

Perhaps the most appropriate starting point for a discussion on childhood phobias is with Freud (1909) because his case study of 'Little Hans' was one of the first systematic accounts of phobia development (Davidson & Neale, 1990). Freud argued that due to the Oedipus complex, a stage of psychosexual development that all children pass through, Little Hans wished to sleep with his mother and kill his father. However, the anxiety caused by these unacceptable fantasies was repressed and instead manifested as a phobia of horses – animals that symbolically represented Little Hans' father (Menzies & Clarke, 1995). Based on information from this case study, Freud proposed that all phobias stemmed from unconscious, forbidden thoughts and impulses (Herbert, 1994). In an attempt to keep these unacceptable thoughts and impulses from entering consciousness, various defence mechanisms are employed to repress them. In phobias, the prime defence mechanism is displacement (Carr, 1999). In displacement, anxiety provoked by the unacceptable impulse is
transferred onto another more socially acceptable object that symbolises the taboo, original object or situation (Carr, 1999). The object or situation onto which the anxiety is displaced then becomes the phobic object. Subsequent avoidance of the phobic object 'protects' the person from having to confront repressed impulses (Herbert, 1994).

There is some anecdotal evidence to suggest the clinical utility of Freud's idea of displacement. For example, Carr (1999) points out that often children who are worried about one thing say that they are worried about something else. However, there is no evidence that displacement is associated with unconscious psychosexual conflicts (Carr, 1999) and for the most part Freud's theory of phobia development has been heavily criticised.

Firstly, the evidence cited to support the model is obtained from uncontrolled clinical case reports. Therefore, the information presented is the analyst's interpretation of events and may contain inferences that are not supported by the available evidence (Menzies & Clarke, 1995). For example, Wolpe and Rachman (1960 cited in Menzies & Clarke, 1995) re-examined Freud's description of the Little Hans case. They concluded that there was no evidence to suggest that Little Hans wanted to sleep with his mother or that he hated his father. Indeed, Wolpe and Rachman (1960 cited in Menzies & Clarke, 1995) point out that some of Freud's inferences were later proved to be unfounded by information from Little Hans himself. Secondly, the concepts proposed in the theory e.g. unconscious psychosexual conflicts, do not lend themselves to experimental manipulation (Bateman & Holmes, 1995). It is therefore impossible to develop hypotheses that can be tested and to generate an empirical evidence base that would support the theory. Lastly, Freud's account of phobias fails to take account of environmental influences. For example, in the case of Little Hans, Rachman and Wolpe (1960; cited in Menzies & Clarke, 1995) argue that phobia development may be more simply explained with reference to a traumatic event involving horses that Little Hans witnessed.

Notwithstanding the limitations of psychoanalytic theory, psychoanalytic psychotherapy is widely used in the treatment of anxious and phobic children (Roth
Psychoanalytic theory suggests that therapy should concentrate on uncovering the unconscious processes associated with the development of the phobia. Therefore, the aim of therapy is to interpret the defence, the repressed impulse and the associated anxiety (Carr, 1999). However, few studies evaluate the effectiveness of psychoanalytic psychotherapy (Roth & Fonagy, 1996). Those that do exist suggest that the approach may be successful in treating childhood phobias. For example, Target and Fonagy (1994) reviewed treatment information on three hundred and fifty-two children seen at the Anna Freud Centre in London for various emotional disorders including specific phobias. Therapy was conducted for one to five sessions a week over an average of two years. Results showed that of the forty-eight children with specific phobias, eighty-seven percent showed significant improvement by the end of therapy. However, there are various methodological problems with this study, which limit the conclusions that can be drawn. These include, the non-random assignment of patients, lack of control group, reliance on chart based data and the possibility of bias in the results because of dropout (Target & Fonagy, 1994). Hence, it is difficult to determine whether improvement is due to resolution of unconscious conflicts, non-specific therapeutic factors arising from intensive and long-term therapy or natural remission of the phobia.

Given the lack of empirical support for the psychoanalytic model and limited evaluation of therapy derived from it, one must conclude that the status of the theory is controversial (Herbert, 1994).

**Behavioural Theories**

Dissatisfaction with the psychoanalytic model has paved the way for interest in behavioural explanations of phobias (Menzies & Clarke, 1995). Indeed, there are many behaviourally based accounts of phobia development, for example: incubation theory (Eysenck, 1979: cited in Carr, 1999) and operant conditioning (Davidson & Neale, 1990). However, arguably the most influential behavioural theory of phobia development has been Rachman’s (1977) three-pathway model (Silverman & Rabian, 1994). This model suggests that phobias may be acquired through the process of classical conditioning, vicarious conditioning or transmission of negative information.
Classical conditioning theory has featured in explanations of specific and social phobias for many years and has undergone various refinements e.g. latent inhibition and unconditioned stimulus inflation (King, Eleonara & Ollendick, 1998). However, classical conditioning has traditionally been described as involving the pairing of a neutral stimulus with a frightening incident. The previously neutral stimulus then becomes associated with negative experiences and itself becomes a source of fear (King, Eleonara & Ollendick, 1998). The individual then learns that fear can be reduced by avoiding the phobic stimulus. In this way avoidance becomes part of the phobic response (Merckelbach, de Jong, Muris, & van de Hout, 1996). The likelihood of fear developing via classical conditioning is increased by factors such as confinement and exposure to high-intensity fear (King, Eleonara & Ollendick, 1998). The power of classical conditioning in acquiring a phobia has been demonstrated in numerous studies across different settings, for example laboratory experiments with humans and animals and in real life situations (Rachman, 1977).

However, Rachman (1977) argues that limitations inherent in the classical conditioning model means that it cannot provide a complete explanation for the development of all phobias. Classical conditioning fails to account for occasions when phobias fail to develop after a traumatic event and not all phobias appear to start after a traumatic event (King Eleonara & Ollendick, 1998). In addition, classical conditioning assumes that all stimuli have an equal chance of provoking fear. However, research demonstrates that some fears are far more common than others (Rachman, 1977). Finally, Rachman (1977) points out that indirect learning, such as vicarious conditioning and transmission of negative information, can also play a role in phobia acquisition.

Vicarious conditioning involves the learning of a fear response through repeated observations of fear and maladaptive coping in others. For example, a child may develop a fear of the dark through observing fearful nighttime behaviour of siblings (King, Eleonara & Ollendick, 1998). This assumption is supported by studies in both animals and humans (Merckelbach de Jong, Muris, & van de Hout, 1996). In exposure to negative information, it is argued that children learn to become fearful
because of the information provided by peers and family. For example, a child may learn to fear visiting the dentist after hearing frightening stories and jokes about dentists (King, Eleonara & Ollendick, 1998).

By incorporating classical conditioning, vicarious conditioning, and transmission of negative information into one model, Rachman's three-pathway model is an attempt to unify what is known about the influence of learning processes in phobia acquisition. A number of studies have attempted to evaluate Rachman's three-pathway model as an explanation of phobia acquisition in childhood. For example, King, Clowe-Hollins and Ollendick (1997) evaluated Rachman's theory with a sample of thirty dog phobic children by asking parents about the influential factors in the onset of their children's fear. In all but four cases, parents were able to attribute their children's phobia to modelling, classical conditioning, or instruction. Support for the Rachman's theory was also found by Ollendick and King (1991: cited in King, Eleonara & Ollendick, 1998) in a sample of one thousand and ninety-two children with common fears. These researchers also demonstrated that frequently more than one type of learning was required to contribute to the development of the phobia. This finding suggests that the pathways may be interactive (King, Eleonara & Ollendick, 1998). When this is the case, the phobia may be associated with higher levels of fear (Merckelbach, Muris & Schouten, 1996).

The strength of Rachman's model is that it appears to account for various aspects of childhood phobias (King, Eleonara & Ollendick, 1998) including variations in phobia severity and both the sudden and gradual development of fear (Rachman, 1997). In addition, Rachman (1977) suggests that by acknowledging the idea that people are genetically pre-programmed to condition to certain fears, the three-pathway model can also explain the non-random distribution of types of phobias. The three-pathway model can also be used to guide the selection of treatment strategies. Since phobias are acquired by direct and indirect learning process, the use of deconditioning procedures such as systematic desensitisation and modelling therapies should be effective.

Systematic desensitisation has a long history of use with phobic children (Ollendick
& King, 1998). In this procedure fear is counterconditioned by gradually presenting a hierarchy of phobic stimuli (either in vivo or imaginally) to the child while he or she is relaxed (Ollendick & King, 1998). In reviewing four randomised controlled trials of imaginal desensitisation for children with phobias, Ollendick and King (1998) conclude that it is more effective than no treatment and various alternative treatments offered e.g. relaxation only and exposure without relaxation. However, there is some evidence to suggest that in vivo desensitisation appears to be superior to imaginal desensitisation for younger children aged five to ten (Ollendick & King, 1998). Thus, systematic desensitisation is a reasonably effective procedure for the treatment of phobias in children. It is also well received by parents and children (King & Ollendick, 1997). This is obviously an important consideration, and one which has prevented flooding, another classical conditioning based procedure being widely used or evaluated (King & Ollendick, 1997).

A variant of systematic desensitisation that has been used with children is emotive imagery. As with systematic desensitisation, emotive imagery involves the use of a fear hierarchy. However, an exciting story that generates positive feelings in the child is used instead of relaxation to inhibit fear (King, Molloy, Heyne, Murphy & Ollendick, 1998). It is argued that emotive imagery may be a more useful procedure with younger children who find the relaxation component of systematic desensitisation too demanding (King, Molloy, Heyne, Murphy & Ollendick, 1998). Cornwall, Spence and Schotte (1996: cited in King, Molloy, Heyne, Murphy & Ollendick, 1998) conducted a randomised controlled trial of emotive imagery for twenty-four children with darkness phobia. After six weekly sessions, children in the emotive imagery group showed significant reductions in darkness fears and significant improvements in a darkness toleration test compared to waiting list controls. These improvements were maintained at three-month follow-up. However, this study is the only controlled trial of emotive imagery and further research is needed before it can be concluded that it is an effective therapy.

Modelling procedures aim to modify fear responses by exposing children to situations were they can observe someone demonstrating non-fearful behaviour in an anxiety-provoking situation (Ollendick & King, 1998). A number of case and
analogue studies have demonstrated that various forms of modelling (e.g. filmed, live and participant) are more effective than no treatment and exposure only (Ollendick & King, 1997). Participant modelling is also more effective than imaginal desensitisation (Ollendick & King, 1998). Research comparing the different types of modelling has shown that participant modelling seems to be superior, especially when parents are involved in the intervention (Ollendick & King, 1997). Indeed parental involvement could be viewed as critical if the phobias developed as a result of observing parents reacting fearfully.

The outcome studies reviewed above are limited by various methodological problems such as small sample size and frequent use of non-clinical samples (King, Eleonara & Ollendick, 1998). Nevertheless, the evidence suggests that therapies derived from classical and vicarious conditioning are relatively effective for children with phobias. However, Herbert (1994) states that behaviour therapy is an empirical rather than a rational treatment that works regardless of the origin of the problem. For example, although systematic desensitisation appears to work, uncertainty exists as to precisely why it is successful (Herbert, 1994). Does it decondition the response, as the theory predicts, or is the effect achieved by other means? In addition, it has been argued that emotive imagery, supposedly based on classical conditioning theory is not really a behavioural intervention (King, Molloy, Heyne, Murphy & Ollendick, 1998). As there is a heavy emphasis in emotive imagery on affecting the beliefs about a feared object and changing a child’s perceptions of threat and danger, it could be argued that the intervention is primarily cognitive (King, Molloy, Heyne, Murphy & Ollendick, 1998). Therefore, the efficacy of behaviour therapy does not necessarily support behavioural theory (Herbert, 1994).

Perhaps a more fundamental limitation of the three-pathway model is that it cannot account for the development of all childhood phobias. For example Menzies and Clarke (1993: cited in King, Eleonara & Ollendick, 1998) asked parents about the origin of water phobia in a sample of fifty children. The majority of parents believed that their child’s phobia had existed since their first contact with water and neither direct nor indirect learning processes were involved. In addition, studies that claim to support the three-pathway model have a number of methodological problems
leading to the suggestion that they overestimate the influence of learning in childhood phobias. For example, the Phobic Origins Questionnaire has been used in several of studies evaluating Rachman’s model. Merckelbach, Muris and Schouten, (1996) point out that items in the questionnaire are restricted to conditioning, modelling and negative information experiences. This means that other non-learning factors that may play in the development of childhood phobias are not addressed.

**Nonassociative Model**

The nonassociative account of phobia development takes its lead from the above evidence (e.g. Menzies & Clarke, 1993: cited in King, Eleonara & Ollendick, 1998) that not all phobias result from direct or indirect learning. Nonassociative theory draws on the work of Charles Darwin who proposed that through a process of natural selection, humans have developed fears concerning various survival-related situations (Menzies & Clarke, 1995). In line with this reasoning, Menzies & Clarke (1995) argue that fears in humans emerge spontaneously at appropriate developmental stages. Young children, for example, may be pre-programmed to develop a fear of separation from caregivers, as in the evolutionary past separation would have increased the likelihood of exposure to danger (Menzies & Clarke, 1995). Thus, as many fears are innate, individuals will show fear on their first encounter with evolutionary significant stimuli (Menzies & Clarke, 1995).

According to Menzies and Clarke (1995), developmentally appropriate fears develop into phobias for a number of reasons. The first reason is that individuals fail to habituate to the fear provoked by the stimulus. This may be because the person is a poor habituators to fear or because the individual does not have safe exposure opportunities. Support for this type of phobia development is said to be gained from studies in which parents report that their children had always been afraid of various phobic stimuli. The second route to phobia development is that during periods of stress, a process of ‘dishabituation’ occurs and previously conquered fears resurface. To support this claim, Menzies and Clarke (1995) cite evidence from a study that showed that sixteen percent of height phobic students claimed that their fear developed during a period of severe stress or anxiety.
The nonassociative model therefore provides an explanation for childhood phobias that develop in the absence of direct or indirect learning. It is also able to account for the fact that phobias and fears relating to survival-relevant stimuli are more common than others (Merckelbach, de Jong, Muris, & van de Hout, 1996). However, the nonassociative account does have a number of limitations. Firstly, the theory is based on assumptions about important survival behaviours that existed in the distant past but there is no way to test these assumptions (Menzies & Clarke, 1995). Secondly, the nonassociative account cannot explain phobias of modern phenomenon such as medical or dental procedures – both of which are common fears in childhood (King, Eleonara & Ollendick, 1998). Thirdly, the reasons for phobia development are not adequately dealt with (Merckelbach et al., 1996). Why are there variations between individuals in their ability habituate to stimuli? Similarly, the idea that stress provokes the return of developmental fears suggests that many more people should suffer from phobias than epidemiological studies indicate. Lastly, the nonassociative explanation of phobias may be of theoretical interest. However, Menzies and Clarke (1995) do not elaborate on how nonassociative theory may be used in clinical practice.

**Biological Theories**

Family and twin studies are often cited as evidence to suggest the role of biological factors in phobias. For example, Fyer, Mannuzza, Gallops, Martin, Aaronson, Gormon, Liebowitz and Klein (1990: cited in Silverman & Rabian, 1994) found that relatives of people with specific phobias are more likely to have a specific phobia compared to relatives of non-phobics. Moreover, phobias shared by relatives appear to be type specific e.g.; members of the same family are all dog phobics (Graham & Gaffan, 1997). A similar pattern of phobia heritability is found for social phobia (Beidel & Randall, 1994). Of course, these findings may not only reflect shared genes, but also other shared experiences in the family such as vicarious conditioning (Graham & Gaffan, 1997). One method to disentangle the contributions of environmental and genetic factors is to study the occurrence of phobia in identical and fraternal twins. Torgersen (cited in Silverman & Rabian, 1994) found that in relation to the strength and nature of phobias, identical twins are more similar than fraternal twins.
It is uncertain as to the precise biological factors and the mechanisms by which phobias are genetically transmitted (Carr, 1999). However, certain neurotransmitters have been implicated in the development of phobias (Carr, 1999). It is argued that in phobic individuals, neurones that normally produce gamma-aminobutyric acid (GABA), are dysfunctional. When functioning normally, GABA is released when arousal reaches a certain level and has the effect of decreasing anxiety. Therefore, when GABA function is disrupted, the anxiety reduction mechanism cannot work effectively (Carr, 1999). The main support for this model has been gained from trials of benzodiazepines - drugs which bind with GABA receptors in the brain (Bernstien, 1994). Benzodiazepines have been demonstrated to reduce anxiety in adults (Bernstein, 1994). However, there are few controlled trials of this drug with children. Where controlled trials have been carried out, these have mainly been with children with generalised anxiety rather than simple or social phobias. In these trials medication was only marginally better than a placebo (Bernstein, 1994).

In addition to being of questionable benefit, benzodiazepines have a number of well documented side effects in children such as behavioural disinhibition, sedation and tremor (Bernstein, 1994). Lengthy treatment with benzodiazepines has also been demonstrated to lead to drug tolerance and physiological and psychological dependence in adults. However, the risks are unknown in children (Bernstien, 1994). It is therefore recommended that the length of benzodiazpine treatment should be short and only in conjunction with psychological interventions (Bernstein, 1994). Thus, medication may be most useful to reduce severe anxiety so that a behavioural programme can be initiated (Bernstein, 1994).

Biological theories may be able to explain the genetic link in social and specific phobias and suggest how children may be vulnerable to phobias. However, theories do not adequately explain the mechanisms by which phobias develop and are maintained.

**Cognitive Theories**

Theories explaining childhood phobias discussed thus far have ignored the thoughts
and feelings of individuals. However, cognitive models assert that individuals do not respond directly to their environment, but that emotion and behaviour is mediated by cognitive factors (Kendall & Gosch, 1994). Beck (1976) argues that emotional disorders are maintained by distortions in thinking that reflect underlying beliefs about the world and the self that are formed through early experiences. For example, family interactions may shape a person's thoughts, attributions, and expectations (Kendall & Gosch, 1994).

In anxiety disorders, beliefs concerning personal danger and threat are thought to play a central role (Thorpe & Salkovskis, 1995). In social phobia, beliefs are related to threat of incompetence and failure in social situations (Wells, 1997). This leads social phobics to interpret social encounters as signs of danger and view them in a negative way. Clark and Stopa (cited in Clark, 1997) investigated this hypothesis in a sample of adult social phobics. Results showed that social phobics were more likely to endorse catastrophic interpretations (e.g. "I am a boring person") of mildly negative social events than other anxious people or non-anxious controls. In relation to specific phobias, studies have demonstrated the importance of beliefs about expectations of harm and helplessness in specific phobias. For example, in a sample of twenty-five phobic adults, Thorpe and Salkovskis (1995) found that negative beliefs concerning harm and coping e.g. "I would go mad", "I would not stay calm", were related to phobic fear and avoidance. In addition, it was demonstrated these beliefs were specific to the phobic object.

Other cognitive factors such as faulty information processing have also been implicated in the maintenance of phobias. In specific phobias individuals have been shown to demonstrate a selective attention bias for phobic related stimuli, while social phobics selectively attend to and monitor their own behaviour (Eysenck, 1997). According to the cognitive model, these biases may affect the way that events are interpreted and therefore, serve to maintain negative beliefs (Wells, 1997).

Cognitive models of phobias have a number of strengths. Firstly, theories are supported by a range of empirical evidence such as that described above. Secondly, cognitive models are able to explain both the development and maintenance of
phobias. Lastly, for social phobia at least there is a large body of evidence to suggest that cognitive behaviour therapy (CBT) which aims to modify distorted cognitions is effective for adult populations (Roth & Fonagy, 1996). However, cognitive models of phobias have been developed using research on adult populations. The question therefore arises as to how well cognitive models explain childhood phobias.

Unfortunately, there is a limited amount of research examining the presence of maladaptive cognitions in phobic children (King & Ollendick, 1997). This may reflect the problems in assessing children’s cognitions. For example, the difficulties in determining the age at which children able to reliably report their cognitions (Ollendick, King & Murphy, 1997). However, there is some evidence that anxious children do have more negative automatic thoughts than non-anxious children (Kendall & Gosch, 1994) and that these cognitions concern the possibility of threat or danger. Under test conditions, phobic children report more negative self-evaluations and fewer positive evaluations (Ollendick & King, 1998). In non-clinical samples of children, research has also demonstrated that in situations such as taking tests, diving and visiting the dentist, anxious children have more negative automatic thoughts such as “I am going to mess up” or “I am going to get hurt” (Kendall & Gosch, 1994).

The presence of maladaptive cognitions in children suggests that CBT may be an effective intervention. CBT for children with phobias is similar to that used for adults in that it aims to tackle distorted cognitions. The main difference being that the techniques used are modified to take into account children’s ages and cognitive-verbal skills (King & Ollendick, 1997). Generally, CBT for childhood phobias involves a package of treatment strategies including various behavioural techniques, cognitive restructuring, and problem solving (Kendall & Gosch, 1994). In reviewing CBT for phobic children, Ollendick and King (1998) conclude that it is “probably efficacious” with the few studies that have been carried out demonstrating positive results. For example, Graziano and Monney (1980: cited in Ollendick & King, 1998) compared a cognitive intervention (verbal self-instruction) to a waiting list control group in a sample of thirty-three children with severe nighttime fears. After treatment, the verbal self-instruction group demonstrated significantly less fear than
the waiting list control group. These gains were maintained at six-month, one year, and three-year follow-up.

CBT for children with phobias may be enhanced when families are involved in the intervention. For example Barrett, Dadds and Rapee (1996: cited in Ollendick & King, 1998) compared CBT, CBT and family therapy, and no treatment, in a sample seventy-nine children with anxiety disorders, including social phobia. In this study, the family therapy component involved helping parents to become more aware of their own anxiety responses in addition to training them to reinforce their children’s use of CBT strategies. Results showed that both CBT and the combination of CBT and family therapy were more effective than no treatment, but that CBT plus family therapy was superior to CBT alone.

It can be seen that the cognitive model of phobias losses some of its explanatory power when it is applied to children. This is mainly because of lack of research into the role of cognitions in phobic children. The positive results of CBT for children does lend support to the theory but here too, further research is needed to establish the efficacy of CBT for phobic children, especially with younger children. In particular, to what extent are cognitions expressed and if they can be modified in children under five (King & Ollendick, 1997).

Conclusions
Psychoanalytic, behavioural, nonassociative, biological, and cognitive explanations of childhood phobias have been discussed in this essay. With the possible exception of the psychoanalytic model, the theories reviewed have all outlined important influences on the development and maintenance of childhood phobias. Each theory has strengths in explaining different aspects of phobia development. Nonassociative accounts provide an explanation for the development of fears in the absence of learning while Rachman’s three-pathway model outlines the impact of learning on phobia acquisition. Biological theory explains why certain children may be vulnerable to developing phobias and cognitive models provide an account of how beliefs and expectations about stimuli effect phobia development and maintenance.
However, each model is limited in its ability to comprehensively explain childhood phobias. These limitations not only stem from the theoretical aspects of the models such as poor definition of process involved in phobia development, but also the in research base that is said to support the models. For example, with cognitive models there is little research investigating the presence of maladaptive cognitions in children. Assessing the evidence base that would support the use of theories in clinical practice also poses problems. Limitations of therapy outcome research include small sample sizes and the use of non-clinical samples of children (Silverman & Rabian, 1994) and a lack of comparisons of different types of intervention. In addition, Roth and Fonagy (1996) note that a major difficulty with evaluating psychological interventions for childhood disorders is the lack of randomised controlled trials. Therapies for childhood phobias are no exception.

Despite these limitations, it must be concluded that childhood phobias are probably best explained by an interaction of the processes described in behavioural, nonassociative, biological, and cognitive models. Family factors too may play an important role in shaping the types of learning experiences, attitudes, and beliefs to which children are exposed.

Recognising the complex nature of childhood phobias has a number of implications for assessment and treatment. A comprehensive assessment would have to evaluate the relative influences of biological vulnerability, cognitive, learning and family factors for individual children (King, Ollendick & Murphy, 1997). Treatment strategies can then be tailored to address the pertinent factors for each child in the development and maintenance of the phobia.
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Older Adults Essay

Discuss the difficulties in distinguishing depression and early stage dementia and evaluate ways in which these difficulties might be addressed.

June 2000

Year 2
Introduction

‘Dementia’ is a generic term used to describe a syndrome of symptoms including acquired cognitive impairment, behaviour change and loss of social functioning (Feldman & O’Brien, 1999). Dementia may be caused by more than seventy disorders (Kaszniak & Christenson, 1994), but the most common dementias are the result of progressive cerebral degeneration (Walsh & Darby, 1999). These so-called ‘primary’ dementias, for example Alzheimer’s disease, Multi-infarct dementia, and Lewy body dementia are at present irreversible. Nonetheless, accurate and early diagnosis is extremely important. Newly developed drugs may slow the rate of deterioration in Alzheimer’s disease if used in the early stages of the disorder (Wilcock & Skoog, 1999). In addition, timely provision of information about the nature of the dementia can help sufferers and their families to come to terms with the condition and plan future support (Jobst, Suarez & Miller, 1999).

Unfortunately, the assessment and diagnosis of dementia in older adults is beset with problems. Older adults have high incidence of health problems that can impair cognitive function (Yesavage, 1993). Dementia caused by medical conditions, commonly called ‘secondary’ dementia, may also be more prevalent in older adults (Yesavage, 1993). Sensory impairments such as hearing and eyesight problems can give the false impression of cognitive decline and older adults tend to be prescribed multiple medications that can also effect cognitive function (Miller & Morris, 1993). In addition, when physical causes are ruled out, clinicians have to distinguish between cognitive changes caused by early stage dementia and those associated with normal ageing (O’Connor, 1994).

However, of all the difficulties associated with the diagnosis of dementia in older adults, distinguishing depression from early stage primary dementia may be particularly problematic (Teri & Wagner, 1992). Indeed, Lezak has described it as “probably the knottiest problem of differential diagnosis” (1995, p327: cited in O’Carroll, Conway, Ryman & Prentice, 1997). Consequently, misdiagnosis can be a frequent occurrence, with studies suggesting that up to thirty-one percent of people who are diagnosed with primary dementia are actually depressed (Kaszniak & Christenson, 1994).
This essay will first discuss why difficulties arise in distinguishing depression from early stage dementia. Each type of primary dementia has separate diagnostic criteria (American Psychiatric Association, 1994) and varying issues relating to differential diagnosis (Rosenstein, 1998). Thus, for clarity the focus of the discussion will be on Alzheimer’s disease, as it is the most common type of dementia in older adults (Teri & Wagner, 1992).

After considering the difficulties in distinguishing depression from early stage Alzheimer’s disease, this essay will go on to evaluate how these difficulties may be addressed. Numerous techniques have been suggested to distinguish early stage Alzheimer’s disease from depression. These range from analysis of clinical features (Emery & Oxman, 1992) to tests for olfactory function (Solomon, Petrie, Hart & Brackin, 1998) and genetic markers (Jobst et al., 1999). However, many of the techniques proposed are experimental and not widely used or too costly to be used routinely. Therefore, the techniques discussed will be limited to the evaluation of clinical features, neuropsychological assessment, and neuroimaging and neurophysiological tests as these are more commonly used (Pryse-Philips & Wahlund, 1999).

**Why difficulties arise in distinguishing early stage dementia from depression**

Standard diagnostic criteria for depression and Alzheimer’s disease, such as DSM IV (American Psychiatric Association, 1994), give the impression that the two disorders are very different (Teri & Wagner, 1992). Alzheimer’s disease is described as a disorder involving the development of multiple cognitive deficits that cause significant and progressive impairment in social or occupational functioning. Memory impairment is the cardinal symptom of Alzheimer’s disease, but one or more additional cognitive disturbances, including aphasia, apraxia, agnosia and a decline in executive functioning, must also be present (American Psychiatric Association, 1994). On the other hand, depression is defined primarily in terms of depressed mood and loss of pleasure or interest in activities. Changes in weight, sleep problems, psychomotor changes, fatigue, inability to concentrate, feelings of worthlessness or guilt and recurrent thoughts of death are also prominent features of
major depression (American Psychiatric Association, 1994). Given the apparent marked differences between depression and Alzheimer’s disease, why is it difficult to distinguishing between these two disorders?

As Alzheimer’s disease progresses it can be distinguished relatively easily from depression as the cognitive impairments are severe (Crowe & Hoogenraad, 2000). However, in the early stages, individuals with Alzheimer’s disease present with a range of more subtle of cognitive and behavioural symptoms (Feldman & O’Brien, 1999). It is at this stage of the disorder that differentiating between Alzheimer’s disease and depression can become difficult because the clinical picture of the two disorders are more alike (Crowe & Hoogenraad, 2000).

Typical depression in older adults is often associated with poor performance on standardised tests cognitive function (Ballard & Eastwood, 1999). This type of cognitive impairment is thought to be due to a lack of interest, poor concentration or a lack of motivation (Ballard & Eastwood, 1999) and may be easily distinguishable from early Alzheimer’s disease (McNeil, 1999). However, for approximately twenty percent of older adults, depression may be accompanied by more severe cognitive impairments (McNeil, 1999). Various labels have been given to depression associated with significant cognitive deficits including ‘psuedodementia’, ‘dementia syndrome of depression,’ ‘depressive dementia’ or ‘depression related cognitive dysfunction’ (Kaszniak & Christenson, 1994).

As these terms suggest the cognitive impairment seen in depression is often severe enough to mimic dementia. Indeed studies have shown that the cognitive deficits found in depressive dementia seem consistent with global impairment in brain functions (Elliott, 1998). For example, people with depressive dementia have been demonstrated to have deficits in memory and executive function on a range of neuropsychological tests when compared to normal controls (McNeil, 1999; Elliott, 1998). Moreover, when directly compared on various neuropsychological tests, there is often little difference between the performance of people with depressive dementia and those with early stage Alzheimer’s disease (Christensen, Griffiths, MacKinnon & Jacomb, 1997).
In addition to cognitive impairment, a number of behavioural features are also commonly observed in both depression and early Alzheimer’s disease depression. These include, agitation, psychomotor changes, disrupted sleep–wake cycle and personality changes (Kaszniak & Christenson, 1994). Researchers point to the fact that in Alzheimer’s disease, other symptoms crucial to the diagnosis of depression e.g. low mood and feelings of hopelessness may not be present (Myers, 1998). However, Kaszniak & Christenson, (1994), suggest that older adults with depression may also be less likely to report low mood and focus more on the somatic symptoms of depression that are similar to the behavioural symptoms seen in Alzheimer’s disease. Thus, the presence of depression-like symptoms in Alzheimer’s disease, or ‘psuedodepression’ (Myers, 1998) can significantly confuse differential diagnosis. This may lead to people being classified as suffering from depressive dementia, rather than the early stages of Alzheimer’s disease.

Besides the symptomatic overlap between early stage dementia and depression, an additional source of difficulty in differential diagnosis is the growing recognition that many individuals have a dual diagnosis of Alzheimer’s disease and depression (Teri & Wagner, 1992). It is estimated that up to twenty percent of people with Alzheimer’s disease will have concurrent major depression and this may be more prevalent in mild dementia (Ballard & Eastwood, 1999).

At present, it is unclear whether it is cerebral degeneration or a reaction to being diagnosed with an untreatable problem that causes depression in Alzheimer’s disease (Emery & Oxman, 1992). Nonetheless, the fact that dementia and depression can coexist makes the diagnostic process even more complicated. In many such cases, depression manifests as the more prominent problem (desRosiers, Hughes & Berrios, 1995). Hence, an individual may be diagnosed with depressive dementia, rather than both early stage dementia and depression.

A further complication in distinguishing depressive dementia and Alzheimer’s disease is the suggestion that the two disorders may be related (Teri & Wagner, 1992). Studies suggest that in some cases, depressive dementia may lead to the
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devlopment of Alzheimer's disease. Alexopolous, Myers, Mattis and Kakuma (1993; cited in McNeil, 1999) found that individuals with depressive dementia were five times more likely to develop dementia over a period of three years than those with typical depression. Overall, follow-up studies suggest that between nine and ninety-one percent of people diagnosed with depressive dementia will go on to develop primary dementia (McNeil, 1999). Thus, as well as distinguishing early stage Alzheimer's disease from depression, clinicians must also be able to differentiate cases of depressive dementia that may lead to the development of 'true' dementia (McNeil, 1999).

From the above discussion, it can be seen that depression, early stage Alzheimer's disease, and early stage Alzheimer's disease with concurrent depression often appear identical in presentation. Indeed, it has been suggested that accurate diagnosis can only be made on a post hoc basis. That is, if cognitive impairment is caused by depression, this will resolve after treatment for depression (McNeil, 1999). However, the outcome of treatment for depression may not provide a definitive diagnosis. Kaszniak & Christenson (1994) suggest that when people with a dual diagnosis of Alzheimer's disease and depression are treated for depression, many show improvements in mood, functional abilities and cognition. Thus, when Alzheimer's disease is in the early stages, these improvements in functioning may appear to indicate that the person was suffering depressive dementia. In addition, treatment with antidepressant medication can have undesirable effects in certain cases. Kaszniak and Christenson (1994) point out that treatment of non-existent depression in an individual with primary dementia with certain antidepressant drugs (e.g. imipramine) can exacerbate memory impairments

Thus, clinicians and researchers have attempted to identify techniques whereby depression and early Alzheimer's disease can be distinguished from each other without the need to rely on the outcome of treatment.

Techniques used to distinguish dementia from depression

Analysis of clinical features

Various researchers have pointed out that although symptoms of early stage
Alzheimer's disease and depression appear similar, subtle variations do exist. Thus, analysis of the clinical features of depressive dementia and Alzheimer's disease has been proposed as a potentially useful method to differentiate between the two disorders (Kaszniak & Christenson, 1994). To this end, differences in background history and clinical presentation have been investigated.

Firstly, it has been suggested that if the individual’s description of the onset of the presenting problem is explored, Alzheimer’s disease is found to progress slowly and insidiously. Consequently, it may take some time before the person or their family seeks medical help (Emery & Oxman, 1992). On the other hand, in depressive dementia, symptom onset appears to be more rapid and individuals seek help more quickly (Emery & Oxman, 1992). Secondly, a previous history of depression is thought to be more common in individuals with depressive dementia than those with Alzheimer’s disease (Emery & Oxman, 1992). Thirdly, individuals with depressive dementia more commonly report feelings of helplessness and worthlessness than people with early stage Alzheimer’s disease (Emery & Oxman, 1992). Lastly, people with depressive dementia will more often answer ‘I don’t know’ to questions where as people with Alzheimer’s disease will tend to confabulate (Emery & Oxman, 1992). Similarly, people with Alzheimer’s disease will consistently have deficits in functioning, while people with depressive dementia may fluctuate in their abilities (Emery & Oxman, 1992).

The utility of using clinical features to distinguish between Alzheimer’s disease and depression has received some empirical support. In a prospective study, Yousef, Ryan, Lambert, Pitt and Kellet (1998) developed a checklist of forty-four features noted by other authors to be possible differences between depressive dementia and primary dementia. This checklist included items pertaining to: (1) history, such as onset of problem and previous psychiatric problems; (2) clinical data such as, presence of depressed mood and fatigue; (3) insight and concern regarding problems and; (4) quality performance for example presence of ‘don’t know answers’. This checklist was administered along with four psychometric assessments to one hundred and twenty-eight people who had been referred for a differential diagnosis of depression or dementia. Final diagnosis was made twelve to fourteen months later.
Sixty-three people received a diagnosis of depression and forty-four people received a diagnosis of dementia (type unspecified). The remaining twenty-one patients either had died by follow-up or received a dual diagnosis of depression and dementia and were excluded from the analysis. Results showed that checklist scores were able to distinguish reliably between dementia and depression. Additional analyses identified a subset of eighteen questions that the authors suggest may form a questionnaire sensitive enough to detect cases of depressive dementia.

However, there are a number of factors limiting the use of clinical features to differentiate early stage dementia and depression. Firstly, the technique relies on clinicians making subjective evaluations of an individual’s presenting symptoms. Thus, there may be discrepancies between clinicians in how symptoms are classified. Secondly, Ballard and Eastwood (1999) warn that many individuals may not fit the typical patterns cited in the literature. For example ‘don’t know’ responses to questions have been found as often in dementia as in depression (Crowe & Hoogenraad, 2000). Lastly, if a person has a dual diagnosis of dementia and depression, there may well be a previous history of depression (Crowe & Hoogenraad, 2000). Therefore, most authors agree that while the analysis of clinical features may assist in differentiating between depression and early Alzheimer’s disease, they cannot be used to make a definitive diagnosis (Kaszniak & Christenson, 1994).

**Neuropsychological assessment**

Neuropsychological tests are often considered to be the most sensitive way of detecting the presence and nature of cognitive deficits (Crowe & Hoogenraad, 2000). Indeed they have been proven to be useful in differentiating between various forms of dementia and distinguishing early dementia from cognitive changes associated with normal ageing (Butters, Salmon & Butters, 1994). Thus, researchers have used neuropsychological tests to identify differences between the cognitive impairment found in early stage Alzheimer’s disease and depression.

As memory impairment is the cardinal symptom of Alzheimer’s disease there is a wealth of data concerning the types of memory impairment seen in this disorder.
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(Butters, Salmon & Butters, 1994). There is also converging evidence to suggest that assessment of memory impairment may provide the best way to differentiate between mild Alzheimer’s disease and depression (desRosiers et al., 1995). In a meta-analytic review of memory tests, Lachner and Engel (1994) showed that people with dementia (mixed aetiology) and depression could be differentiated by their performance on tasks involving the delayed recall of previously presented information. This conclusion is supported by the results of more recent studies that focus on Alzheimer’s disease. For example, Coen, Kirby, Swanwick, Maguire, Walsh, Coakley, O’Neill and Lawlor (1997) used the delayed word recall test with an additional delayed recognition task to compare twenty-six participants with mild Alzheimer’s disease and twenty participants with depression. The results demonstrated that both the delayed recall and recognition tasks were highly accurate in distinguishing between participants with mild Alzheimer’s disease and those with depression.

In a more sophisticated study, desRosiers et al. (1995) compared twenty-four people with mild Alzheimer’s disease (twelve with concurrent depression and twelve without), twelve people with major depression and twelve healthy controls. All participants were matched for age, sex, education levels, and estimated pre-morbid intellectual level. The participants were also followed up for a minimum of two years so that correct diagnosis was ensured. Each participant completed the following tests; Wechsler’s Logical Memory, Wechsler’s Visual Reproduction, Wechsler’s Paired Associate learning, Kendrick’s Cognitive Tests for the Elderly and Warrington’s Recognition Memory for Faces. Results showed most of the tests differentiated between those with Alzheimer’s disease (both with and without depression) and those with depression. However, on both statistical and case by case analysis, two tests involving retention of information over short period of time were the most sensitive. These were Wechsler’s Logical Memory and the Object Learning Test from the Kendrick’s Cognitive Tests for the Elderly.

Similarly, McNeil (1999) compared thirteen people with depressive dementia with fourteen people with Alzheimer’s disease and concurrent depression on a range of neuropsychological tests. The assessment covered the areas of mental status,
attention, memory, abstraction / problem solving, language, visuospatial and motor abilities. Results of this study showed that only short-term memory tests were able to distinguish between the two groups.

However, Christensen et al. (1997) suggest that there has been an overemphasis on the use of memory tests to distinguish mild Alzheimer’s disease from depression. In a meta-analytic review, Christensen et al., (1997) conclude that memory tests such as, Wechsler’s Logical memory, Wechsler’s Visual reproduction and the Kendrick battery are not useful in distinguishing between Alzheimer’s disease and depression. Similarly, O’Carroll, Conway, Ryman & Prentice (1997) compared the performance of fifty people with Alzheimer’s disease and fifty people with depression on the delayed word recall and recognition task. The sample of participants differed from the Coen et al. (1997) study in that: (1) the Alzheimer’s disease group were more cognitively impaired and; (2) the depressed participants were more severely depressed. O’Carroll et al. (1997) found that the mean scores for delayed word recognition and recall were significantly higher for the depressed group than the Alzheimer’s group. However, there was considerable overlap in performance when individual scores were inspected and the authors conclude that the delayed word recall test does not distinguish satisfactorily between people with mild Alzheimer’s disease and those with severe depression.

Given the inconclusive results of using memory tests to differentiate between depression and dementia, researchers have explored other types of neuropsychological assessments. For example, Hermann, Kidron, Shulamn, Kaplan, Binns, Leach and Freedman (1998) compared forty-two people with Alzheimer’s disease, thirty-three with depression and thirty age-matched controls on a clock drawing task. The task consisted of: (1) drawing a clock showing a certain time on blank paper; (2) adding numbers and time onto a pre-drawn circle; (3) setting the time on a pre-drawn clock face; (4) clock copying and; (5) clock reading. Results showed that people with Alzheimer’s disease scored significantly worse than people with depression and controls on all clock tasks. Similarly, Christensen et al., (1997) found that tests that assess visuoconstructional skills for example the Block Design subtest of the Wechsler Adult Intelligence Scale – Revised could distinguish between
Alzheimer's disease and depression.

However, there is often an overlap between the performance of people with Alzheimer's disease and depression when single tests are used. It has therefore been suggested that analysis of patterns of performance across a number of tests may provide a better way to distinguish between early stage Alzheimer's disease and depression (Crowe & Hoogenraad, 2000; Lachner & Engel, 1994). Crowe and Hoogenraad (2000) point out that there is evidence to suggest that depressive dementia may have a pattern of cognitive deficits similar to those seen in subcortical dementias e.g. Parkinson's and Huntington's disease. As Alzheimer's disease is a cortical dementia, the difference in the patterns of deficits between depression and Alzheimer's disease may offer a way of distinguishing the two disorders. Crowe and Hoogenraad (2000) compared fifteen people with depressive dementia, fifteen with Alzheimer's disease, and fifteen normal controls on measures of memory, language, visuospatial function and praxis. It was hypothesised that, similar to subcortical dementia, the depressed group would show normal performance on tests of praxis and language but show deficits on measures of memory and abstraction. This pattern would be different for the Alzheimer's group. Results confirmed the hypotheses and Crowe and Hoogenraad (2000) suggest that this supports the use of a cortical / subcortical distinction between Alzheimer's disease and depression. However further studies, particularly ones with prospective designs are needed (Crowe & Hoogenraad, 2000).

Major difficulties exist when attempting to evaluate neuropsychological assessment as a way in which dementia and depression may be distinguished. Differences in design and methodological make comparison of results difficult. Firstly, studies frequently use different types of neuropsychological assessments to measure similar aspects of cognitive functioning. Secondly, there are often differences between studies in the level of cognitive impairment of both Alzheimer's disease and depression groups. Thirdly, Christensen et al., (1997) point out that the cognitive deficits seen in depression vary with age, treatment setting, whether the person has had ECT, and the severity of depression. However, across studies there are considerable differences in the characteristics of the participants with depression.
Lastly, the studies all appear to use different methods of assigning participants to experimental groups. Thus, the people labelled with 'mild dementia' may vary considerably for people given the same label in a different study. Related to this point is that few studies make use of prospective or follow-up designs. This seems especially crucial. Given the difficulties described in distinguishing depression from early stage Alzheimer's disease, lack of prospective studies or follow-up of participants, means that participants may be wrongly diagnosed. This would significantly confound the findings of a study.

Given these limitations, firm conclusions regarding the use of neuropsychological tests in distinguishing between early stage Alzheimer's disease and depression cannot be made. While there appears to be some evidence supporting the use of memory tests to distinguish between depression and Alzheimer's disease with and without depression, further research is needed to address in inconsistencies in the findings across studies. Additional studies are also needed to support the findings regarding the use visuoconstructional tasks and detection of cortical versus subcortical patterns of cognitive deficits.

Even if neuropsychological tests can be shown to distinguish between early Alzheimer's disease and depression, there may be several practical issues that will limit their use with older adults. Bucks and Loewenstein (1999) point out that neuropsychological tests are often inappropriate for older adults by virtue of the size and content of test materials. In addition, there is often a lack of normative data for 'old' older adults (Bucks & Loewenstein, 1999). A further problem arises with the introduction of new versions of neuropsychological tests. The Weschler Adult Intelligence Scale-III and Wechsler Memory Scale-III have recently been published and as yet there is no data available comparing how people with Alzheimer's disease and people with depression perform on these test batteries.

**Neuroimaging Techniques**

As Alzheimer's disease is an 'organic' disorder and depression is an 'functional' or psychiatric disorder (O'Connor, 1994), brain imaging methods may assist in differential diagnosis. There are two basic kinds of neuroimaging methods. Firstly,
techniques that assess the structure of the brain, for example computerised
topography (CT) and magnetic resonance imaging (MRI). Secondly, techniques
which measure how the brain functions, for example positron emission tomography
(PET) and single photon emission scanning (SPECT) (Emery & Oxman, 1992).

Few studies have directly compared CT or MRI scans for people with Alzheimer’s
disease and people with depression. Those that do exist have found similarities on
both CT and MRI scans for both disorders (O’Brien, Desmond, Ames, Schweitzer,
Tuckwell & Tress, 1994). However, O’Brien et al., (1994) suggest that these results
may have been found because the techniques did not target areas in the brain thought
to be most effected by Alzheimer’s disease such as the temporal lobes and the
hippocampus. O’Brien et al., (1994) assessed the usefulness of visual ratings of MRI
scans of temporal lobe structures including the hippocampus, in differentiating
between forty-three patients with Alzheimer’s disease and thirty-two patients with
depression. Results showed that temporal lobe MRI scans were shown to be able to
differentiate between Alzheimer’s disease and depressive dementia. However, the
people in the Alzheimer’s disease group differed from the depressive dementia group
in terms of severity of cognitive impairment and this may have confounded the
results. In order to control for this difference between groups, a subgroup of
participants from both groups were matched for degree of cognitive impairment.
Hippocampal MRI could still distinguish between these patients with eighty-nine per
cent sensitivity

Use of MRI scans in differentiating between Alzheimer’s disease and depression is
also supported in a study by Pantel, Scroder, Essig, Popp, Dech, Knopp, Schas,
Eyenbach, Backenstaff and Friedlinger (1997). MRI scans assessing brain and
cerebral spinal fluid volume were compared for nineteen people with depression,
twenty-seven with people with Alzheimer’s disease, and thirteen healthy controls.
Results showed that volumetric MRI scans of the frontal and temporal lobes and the
hippocampus differentiated between depression and Alzheimer’s disease. Similarly
found that CT scans of the medial temporal lobe may be significantly different in
dementia and depression.
There is less evidence available at present to support the use of functional imaging techniques such as PET and SPECT scans. The use of PET and SPECT scans with people with Alzheimer's disease show brain abnormalities such as, reduced activity in the posterior parietotemporal areas of the brain (Ebmeier, Prentice, Ryman, Halloran, Rimmington, Best & Goodwin, 1997). In depression, functional imaging techniques have shown reduced brain function in anterior areas of the brain (Ebmeier et al., 1997, Elliot, 1998). Thus, PET and SPECT scans may be potentially useful diagnostic techniques. However, there is a lack of studies directly comparing PET and SPECT scans in both Alzheimer's disease and depression.

Given the small number of studies directly comparing Alzheimer's disease and depression it is difficult to evaluate the utility of using neuroimaging to distinguish between early stage Alzheimer's disease and depression (O'Brien, 1994). The studies that do exist use small numbers of participants and employ slightly different scanning techniques making it difficult to draw conclusions (Pantel et al., 1997). In addition, the comments made regarding participant characteristics and selection in the section on neuropsychological testing, also apply to studies investigating neuroimaging.

Further research and follow-up studies are needed in this area (Pantel et al., 1997). However, even if techniques such as MRI, PET and SPECT are demonstrated to differentiate between depression and Alzheimer's disease, their use may be limited because they are time consuming to perform and may not be widely available (Jobst et al., 1999).

**Neurophysiological techniques**

As with neuroimaging techniques, electoencepalography (EEG) allows researchers to assess brain function. EEG's detect brain electrical activity and studies show that patterns of activity generally slow in dementia (Ihl & Brinkmeyer, 1999). In addition, the main area of brain activity shifts from occipital to frontal areas of the brain (Ihl & Brinkmeyer, 1999). Such patterns of activity have been shown to be correlated with the severity of dementia and discriminate between older adults with
dementia and those without (Ihl & Brinkmeyer, 1999). Consequently, it has been proposed that EEG may be able to differentiate between dementia and depression.

Various studies have shown that inspection of EEG patterns do have some utility in distinguishing between primary dementia and depression, but there are limitations in its use (Emery & Oxman, 1992). For example, Pozzi, Golimstock, Petracchi, Garcia and Starkstien (1995) compared EEG patterns for fourteen people with Alzheimer’s disease, thirteen people with depression, seventeen people with both depression and Alzheimer’s disease and ten age controls. Results showed that different EEG patterns could distinguish between all four groups of participants. However, Pozzi et al. (1995) point out that inspection of individual cases showed some overlap between EEG patterns in mild Alzheimer’s disease and depression. In addition, although the Alzheimer’s group was classified as mildly cognitively impaired, the depressed group did not have similar levels of cognitive impairment. The authors conclude that EEG patterns may be more useful to distinguish between depressed and non-depressed people with Alzheimer’s disease, rather than differentiating between those with depression and Alzheimer’s disease.

In a more recent study, Ihl & Brinkmeyer (1999) investigated the power of an EEG technique known as EEG segmentation to differentiate between dementia and depression. Twenty-one normal adults (ten older adults and eleven younger adults), twenty depressed individuals (twelve older adults and ten younger adults), and twelve older adults with Alzheimer’s disease were compared in the study. The study generated a number of interesting findings. Most relevant to this discussion, was that EEG patterns in depression were significantly different from those found in participants with mild Alzheimer’s disease. However, when individual results were reviewed there was an overlap in the EEG patterns for people with Alzheimer’s disease and those with depression. In addition, like other studies, the results may have been confounded by differences in the severity of cognitive impairment between the groups.

The results of EEG studies suffer from similar limitations to the research on the use of neuroimaging. These include small sample sizes, few studies and inconclusive
evidence as to how useful the techniques maybe in differentiating between the Alzheimer’s disease and depression. However, one advantage that EEG has over neuroimaging techniques is its low cost (Jobst et al., 1999). Therefore, if future work substantiates the utility of EEG, it may be significant addition to the diagnostic process (Jobst et al., 1999).

Conclusion
The difficulties associated with distinguishing between early stage Alzheimer’s disease and depression in older adults have been written about extensively in the literature (Emery & Oxman, 1992). A number of factors contributing to this difficulty have been highlighted. Firstly, the similarities between the cognitive and behavioural symptoms observed in early Alzheimer’s disease and those found in depression. Secondly, the co-morbidity of Alzheimer’s disease and depression and lastly, the possibility that depressive dementia may be prodromal to Alzheimer’s disease (Kaszniak & Christenson, 1994).

In order to address these difficulties in distinguishing between early stage Alzheimer’s and depression, clinicians and researchers have suggested various assessment techniques. The evaluation of clinical features, neuropsychological assessment, neuroimaging and neurophysiological techniques have been discussed in this essay.

The evidence to support the use of the evaluation of clinical features to make a differential diagnosis between early Alzheimer’s disease and depression is limited. Neuropsychological assessment, neuroimaging and neurophysiological techniques on the other hand have shown more promise in distinguishing between depression and Alzheimer’s disease, with and without depression. However, conclusions are limited by the lack of research into the use of neuroimaging and neurophysiological techniques. While, in the area of neuropsychological assessment, there are numerous studies but the results are often conflicting. In addition, the studies reviewed evaluating the use of neuropsychological assessment, neuroimaging and neurophysiological techniques have numerous methodological difficulties. These include small sample sizes, lack of prospective or follow-up studies and differences
between studies in how people are allocated to diagnostic groups. Further research is needed on all three techniques to address the conflicting results and the methodological problems of current studies.

An additional focus for future research should be whether neuropsychological assessment, neuroimaging or neurophysiological techniques are able to predict which people with depressive dementia will go on to develop Alzheimer’s disease (desRosiers et al., 1995).

Given the difficulties associated with the techniques described, differential diagnosis between Alzheimer’s disease and depression should not be based solely on information from any one technique. It is important to gather information from various sources. For example, O’Brien et al., (1994) suggest that a combination of MRI scan data and neuropsychological assessment may significantly increase the accuracy of diagnosis. The authors point out that in their study, the individuals that could not be accurately identified by MRI scan were not the same individuals who were misclassified by neuropsychological assessment. Thus, various researchers suggest that the differential diagnosis of dementia and depression in older adults should involve multidisciplinary assessment to decrease the possibility of misdiagnosis (Kaszniak & Christenson, 1994; Pryse-Philips & Wahlund, 1999).
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Discuss the concept of Chronic Fatigue Syndrome. How have psychological theories contributed to our understanding of Chronic Fatigue Syndrome?

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Year 3
Introduction

'Fatigue' can be described as a feeling of weariness, lack of energy, and exhaustion that worsens with exertion (Sharpe, 1997). It is an experience familiar to us all and community surveys suggest that fatigue may be reported by up to half of the general population at any one time (Afari, Schmaling, Herrell, Hartman, Goldberg & Buchwald, 2000). Fatigue is also one of the most common symptoms reported by people seeking treatment in primary care settings (Afari et al., 2000: Sharpe, 1997). At one end of the spectrum, feelings of fatigue may be transient and associated with factors such as stress, overwork or lack of sleep (Sharpe, 1997). At the other end of the spectrum, fatigue can be persistent, impair occupational and social functioning, and lead some people to become bedridden (Wessely, Hotopf & Sharpe, 1998). Of course, severe and disabling fatigue may be a symptom of almost any medical condition (Sharpe, 1997). However, in many cases, severe fatigue remains unexplained after extensive medical investigations (Sharpe, 1997).

Severe fatigue in the absence of an identifiable organic cause has been reported in the psychological and medical literature for at least a hundred years (Wessely et al., 1998). Various terms have been used to describe the condition including 'neurasthenia' and 'myalgic encephalomyelitis' (ME), 'postviral fatigue syndrome' and 'chronic Epstein Barr virus infection' (Sharpe, 1997: Moss-Morris, 1997). Such terms imply an organic cause for the symptoms of severe fatigue and their use has sparked considerable controversy (Moss-Morris, 1997). Consequently, the term 'chronic fatigue syndrome' (CFS) has been proposed (Holmes, Kaplan, Gantz, Komaroff et al., 1988) as it does not suggest a particular aetiology for the symptoms.

Despite general acceptance in the United Kingdom of the term 'chronic fatigue syndrome' (Joint Working Group of the Royal College of Physicians, Psychiatrists and General Practitioners, 1996), other aspects of the condition remain the subject of intense and at times acrimonious debate (Sharpe, 1997). There are several definitions of CFS (Moss-Morris, 1997) and although CFS has been the topic of numerous research studies, the cause and treatment of the syndrome remains uncertain (Sharpe, 1997). Indeed, CFS has been dubbed the 'clinical conundrum' (Kyle & deShazo, 1992; cited in Sharpe, 1997).
This essay will first discuss the controversy surrounding the diagnosis and aetiology of CFS. In the second part of the essay, psychodynamic, systemic and cognitive behavioural theories of CFS will be reviewed and their contribution to our understanding of chronic fatigue will be discussed. Leventhal's self-regulatory model of illness representations (Leventhal, Nerenz & Steele, 1994) will not be discussed in detail as the concepts referred to in this model have been incorporated into cognitive behavioural models of CFS (Moss-Morris, 1997). In addition, the essay will focus on CFS in adults, although reference will be made to research on CFS in children and adolescents where relevant.

The concept of Chronic Fatigue Syndrome

The controversy surrounding the concept of CFS stems in part from the difficulties in defining the nature of the condition. As there are no clear biological markers for CFS, diagnosis is based on the presence of a cluster of symptoms (Moss-Morris, 1997). CFS was initially defined by Holmes et al. (1988), however, these criteria were not universally accepted and several other definitions of CFS have been proposed (e.g. Sharpe, Archard, Banatvala et al., 1991: Lloyd, Wakefield, Boughton & Dwyer, 1988). The use of differing definitions of CFS has implications for both research and clinical practice, for example it is difficult to compare the findings of research studies when different diagnostic criteria for CFS have been used.

In order to standardise CFS definition, Fukada, Straus, Hickie, Sharpe, Dobbins & Komaroff (1994) published criteria agreed by an international panel of researchers. Fukada et al. (1994) propose that in CFS, fatigue: (1) must have a definite onset; (2) be present for at least 6 months; (3) results in substantial reduction in previous levels of occupational, educational, social and personal activities; (4) is not alleviated by rest and (5) is not the result ongoing exertion. In addition, at least four other key symptoms must be present such as muscle and joint pain, headaches, unrefreshing sleep, sore throat, tender lymph nodes, subjective memory impairment and fatigue following exertion lasting more than twenty-four hours. Exclusion criteria for a diagnosis of CFS include: (1) the presence of a medical condition that may explain the symptoms of fatigue; (2) psychiatric illnesses with psychotic features; (3)
substance abuse; (4) dementia; and (5) eating disorders or severe obesity (Fukada et al., 1994).

However, even though the Fukuda et al. (1994) description of CFS is said to be a ‘consensus definition’, the criteria are still somewhat controversial (Sharpe, 1997). Kumar and Lynch (1997) outline several criticisms of the Fukuda et al. (1994) criteria. Firstly, the duration criterion for fatigue is arbitrary and is not based on epidemiological data. Secondly, it may be difficult to establish a definite onset for the symptoms of fatigue. In addition, all CFS diagnostic criteria have been criticised for emphasising fatigue while other symptoms such as joint pain may be equally if not more disabling (Kumar & Lynch, 1997). How fatigue is assessed is also problematic and there several are different measures available, but it is unclear to what extent these measures are comparable (Kumar & Lynch, 1997).

Not only have the diagnostic criteria for CFS been called into question, but the validity of the concept of CFS has also been challenged (Kumar & Lynch, 1997). Whichever CFS criteria are used, CFS patients share many of the same symptoms as other medical unexplained conditions such as fibromyalgia (Sharpe, 1997). CFS patients may also meet criteria for psychiatric disorders as defined in DSM IV (American Psychiatric Association, 1994) such as depression, anxiety and somatoform disorders (Sharpe, 1997). For example, Chalder, Power and Wessely (1996) suggest that in hospital populations up to sixty-seven percent of people with CFS may also be diagnosed with depression. In addition, studies suggest that prevalence rate for anxiety disorders and somatization disorder in CFS is approximately twenty percent (Wessely et al., 1998). This calls into question whether CFS can be considered a discrete condition. Several studies have compared CFS patients to groups of either psychiatric patients or patients with physical illness to determine whether CFS patients present with distinct symptoms. For example, Wessely and Powell (1989) used discriminant function analysis to compare groups of people with CFS, depression and neuromuscular disorders. Results showed that the CFS group could not be distinguished from the other groups on the basis on fatigue symptoms. Lynch (1996; cited in Kumar & Lynch, 1997) also compared people with CFS, depression and diabetes on measures of fatigue. People in the CFS group
described an unpredictable pattern of physical and mental fatigue that often occurred several days after activity. However, discriminant function analysis of fatigue symptoms showed that up to thirty percent of the sample were misclassified.

This overlap between the symptoms of CFS and other medical and psychiatric conditions has led some researchers to argue that people diagnosed with CFS do not represent a homogenous group. Several authors have suggested that it may be advantageous to define subgroups of CFS, for example, people with and people without a co-morbid psychiatric diagnosis (Kumar & Lynch, 1997). However, Kumar and Lynch (1997) suggest that at present it is unclear how sub-classification of CFS would offer any advantage for research into the nature and aetiology of CFS. Further research is required to determine whether sub-classification of groups of CFS patients could offer useful information regarding outcome and response to treatment (Kumar & Lynch, 1997).

A second issue of controversy in the area of CFS is the aetiology of the condition and the relative contribution of physical and psychological factors (Chalder, Cleare & Wessely, 2000). CFS sufferers themselves vehemently assert that their symptoms have an organic cause and reject psychological explanations (Wessely, 1998). Indeed, CFS support groups actively advocate physical causes and treatments for CFS (Banks & Prior, 2001). Chalder et al. (2000) suggest that this stance is fuelled by the belief that "CFS seen as a physical disease implies legitimacy whereas CFS seen as a psychological illness implies malingering" (pg. 160). However, identifying an organic cause for CFS has proven difficult. People with CFS frequently attribute the onset of their symptoms to a viral illness (Chalder et al., 2000). Several different viruses, for example the Epstein Barr virus, enteroviruses and the glandular fever virus, have been investigated as possible precursors to CFS, but the results of research studies are inconclusive (Wessely, et al., 1998). Researchers have also investigated neuroendocrine, neurobiological, and immune system functions as possible causes of CFS. While there is some evidence that there are brain abnormalities and changes in neurotransmitter and immune system function in people with CFS, these findings do not confirm that CFS has a solely organic cause (Moss-Morris, 1997). Such biological changes may also be found in a number of
psychiatric disorders and immune system dysfunction can be related to stress (Moss-Morris, 1997). In addition, Komaroff (2000) points out that not all patients who meet the criteria for CFS have biological markers and those physical causes that have been identified in some CFS patients cannot account for all the symptoms reported by sufferers.

The difficulty in identifying an organic cause of CFS has led some health professionals to conclude that the symptoms of CFS are purely psychological and are 'all in the mind' (Chalder & Williams, 1997). However, researchers have commented that the psychological versus physical debate surrounding the aetiology of CFS is artificial and unhelpful (Abbey, 1996: Wessely et al., 1998). Indeed, Abbey (1996) suggests that it reflects the dichotomization of mind and body that is a feature in western culture and medicine, despite the growing evidence that psychosocial factors play a role in a range of physical illnesses. Therefore, in order to provide a clearer understanding of CFS, aetiological models will need to incorporate a range of factors. How then have psychological theories contributed to our understanding of CFS?

**Psychodynamic Theories**

Abbey (1996) suggests that CFS has received relative little attention in the psychodynamic literature. This situation may in part arise from the fact that Freud himself had little to say about the application of psychoanalytic techniques in treating chronic fatigue or 'neurasthenia' as it was then called (Abbey, 1996). Indeed, Freud did not believe that neurasthenia was an example of a 'psychoneurosis' - that is a disorder that has its origins in earlier life circumstances (Abbey, 1996). Rather, he suggested that neurasthenia was due to factors in the current circumstances of the individual resulting in the depletion of energy (Sharpe, 1997). Freud concluded that the symptoms of fatigue had no symbolic meaning (Abbey, 1996). Thus, psychoanalytic therapy, which focuses on the identification, interpretation, and expression of unconscious conflicts would not be applicable to understanding or treating chronic fatigue.

Despite Freud's dismissal of CFS as a condition that could not be understood in
psychodynamic terms, modern theorists have attempted to develop psychodynamic models of CFS. Central to recent psychodynamic theories of CFS is the idea that both psychiatric and organic illnesses develop in ‘vulnerable’ individuals (Taerk & Gnam, 1994). Taylor (1992: cited in Taerk & Gnam, 1994) suggests that vulnerable individuals have an increased susceptibility to disease. This susceptibility arises because individuals have deficits in their ability to regulate their internal psychological and physiological states. Difficulties in regulating internal psychological and physiological states are in turn thought to stem from a disruption in the mother-child relationships. In an attempt to compensate for these deficits, individuals become overly dependent on others. When these dependent relationships are disrupted, the person becomes ill.

Taerk and Gnam (1994) have incorporated the idea of illness susceptibility into a psychodynamic model of CFS. In this model, the combination of loss or separation from the person or ‘object’ on whom the individual has become dependent and a viral infection, intensify individuals’ difficulties in regulating internal states. This leads to CFS symptoms and also secondary symptoms of depression and anxiety. Taerk and Gnam (1994) suggest that the theory is supported by evidence that suggests early traumatic events, such as separation, compromise immune system functioning thereby making people susceptible to viral illness. Further evidence to support the theory is presented in two case studies where psychoanalytic techniques were used to help the individuals internalise skills for regulating internal states thereby reducing symptoms.

Several criticisms of this psychodynamic model of CFS can be made. Firstly, Taerk and Gnam (1994) themselves concede that conclusions about the validity of the psychodynamic model can not be drawn from two case studies. Secondly, the different components of the theory are not described in detail and there is little evidence cited to support the model. Indeed, psychodynamic theories are notoriously difficult to investigate empirically (Bateman & Holmes, 1994). Thirdly, Taerk and Gnam (1994) do not adequately explain how symptoms of CFS may be maintained. Lastly, Sharpe (1997) suggests that the emphasis in psychodynamic approaches on internal psychological processes may be unacceptable to CFS sufferers and therefore
psychoanalytic therapy would not be a popular treatment option. Indeed, an extensive literature search did not identify any reports of psychodynamic treatment trials for CFS.

Nonetheless, Abbey (1996) argues that psychodynamic theories of chronic fatigue syndrome have contributed to our understanding of the condition by emphasising the interplay between mind and body. However, this approach is not unique to psychodynamic approaches to CFS.

Systemic Theories
While psychodynamic theories focus on the individual, systemic theories consider how a person's symptoms are affected by the systems within which the person operates, for example the family, society, and the wider culture. Systemic approaches have been widely applied to understanding the role of social networks in how people adapt to and cope with chronic illness (Abbey, 1996). Research suggests that the social networks of chronically ill people can be dramatically effected (Heijmans, de Ridder & Bensing, 1999). For example, illness in a family member can lead to changes in family members' roles and functioning or shift the balance of power in family (Abbey, 1996: Wright & Cottrell, 1997). In turn, the response of family and friends to illness may influence a person’s beliefs about health and illness, health related behaviours, symptom expression and even the course of the illness (Wright & Cottrell, 1997: Heijmans et al., 1999).

Unfortunately, there is a lack of research on the impact CFS on family systems (Abbey, 1996) and there does not appear to be an integrated systemic theory of CFS. Nevertheless, research does suggests that interpersonal and social factors are important in CFS (Sharpe, 1997). The role of family factors in the development and maintenance of CFS is suggested by studies which show that children with CFS often have a family member who also has CFS (Wessely et al., 1998). For example Levine, Kueger and Straus (1989: cited in Wright & Cottrell, 1997) found that in a sample of thirty-two children with CFS, fifty percent had an adult family member who also had CFS. However, there has not been extensive investigation into how family factors such as attitudes and beliefs about illness may influence the
development and maintenance of CFS in children (Wright & Cottrell, 1997).

In the adult population of CFS sufferers, various interpersonal and social factors have been investigated. For example, Cope, Mann, Pelosi and David (1996: cited in Chalder & Williams, 1997) examined the role of social support in CFS. When compared to a control group, the CFS group were less satisfied with their family and social relationships. This finding suggests that perceived lack of social support contributes to CFS (Chalder & Williams, 1997). However, Chalder (1998: cited in Chalder & Williams, 1997) found that high social support actually increased the impact of life events on CFS symptoms. Chalder and Williams (1997) therefore suggest that too much support can be just as detrimental as too little. These authors go on to hypothesise that family and friends of people with CFS may attempt to be supportive, but in fact offer the 'wrong' sort of advice. Family members and friends may hold strong beliefs about physical causes of CFS (Sharpe, 1997) and encourage the person with CFS to rest, thereby making CFS symptoms worse. There is no empirical evidence to directly support this hypothesis (Chalder & Williams, 1997). However, the impact of family members' beliefs about CFS is highlighted in a study by Heijmans et al. (1999) where the illness beliefs of CFS suffers and their spouses were compared. Results showed that when spouses' were more optimistic compared to their partners about the outcome of the illness, the functioning of the CFS sufferer was better. When spouses' minimised the role of organic factors in CFS (in relation to their partner's perceptions), sufferers functioned less well.

Family responses to CFS symptoms have also been investigated. Schamling, Smith and Buchwald (2000) examined the effect of partners' responses to illness behaviour in a sample of one hundred and nine people with CFS. Results indicated that the more frequently partners expressed concern in response to reports of fatigue by CFS sufferers, the greater the reports of fatigue and pain severity. In addition, although not systematically studied, researchers have commented on the impact on CFS of wider social systems such as the media, CFS support groups and the medical profession. For example, in a qualitative study, Clements, Sharpe, Simkin, Borrill and Hawton (1997) interviewed sixty-six CFS sufferers about their beliefs about their symptoms and how these beliefs were formed. Four themes concerning how illness
beliefs are formed were identified. These were personal reflection, media reports and self-help literature, health professionals and the influence of others.

In summary, there is some evidence that a systemic perspective on CFS may aid our understanding of how social contexts contribute to the development and maintenance of CFS. However, further research is needed to investigate the role of family attitudes and behaviours and the attitudes of health professionals and the media in CFS. In addition, there is little mention of the family in treatment approaches for CFS in adults other than to suggest that family members may be used as co-therapists in cognitive behavioural therapy (Abbey, 1996). However, the evidence that exists suggests that therapy approaches for CFS need to pay more attention to family attitudes and responses to CFS symptoms.

**Cognitive Behavioural Theories**

In contrast to psychodynamic and systemic models, cognitive behavioural models of CFS are better defined and have been more rigorously investigated. Cognitive behavioural models of CFS take as their starting point generic cognitive behavioural models of illness that suggest that peoples' interpretations of symptoms may play an important role in maintaining and exacerbating symptoms (Sharpe, 1996). Firstly, 'catastrophic' interpretations of symptoms may lead people to experience emotional distress (Sharpe, 1996). Secondly, if people believe that they are powerless to exert any control over their symptoms, this may lead to the use of maladaptive coping strategies (Sharpe, 1996). Thirdly, emotional distress and maladaptive coping strategies may themselves intensify symptoms (Sharpe, 1996). Cognitive behavioural models have been successfully used to understand adjustment and coping in medical conditions such as cancer and rheumatoid arthritis and other medically unexplained syndromes such as irritable bowel syndrome, fibromyalgia, chronic pain and noncardiac chest pain (Abbey, 1996).

Wessely, David, Butler and Chalder (1989) have proposed a cognitive behavioural model of CFS where an organic illness (e.g. viral infection) precipitates a number of psychological reactions, which then maintain the symptoms of fatigue. It is suggested that in the recovery phase following a virus, people commonly experience
feelings of fatigue as a result of physical deconditioning (Wessely et al., 1989). If such symptoms are interpreted as evidence of an ongoing physical health problem, it is likely that sufferers will attempt to alleviate symptoms by resting and avoiding activity (Wessely et al., 1989). However, such coping strategies lead to an increase in physical deconditioning and a worsening of symptoms. (Wessely, et al., 1989). This lack of control over symptoms can lead to feelings of frustration, anxiety and depression, which can in turn generate further physical symptoms (Wessely, Butler, Chalder & David, 1991).

However, the Wessely et al. (1989, 1991) model of CFS has a number of shortcomings. Firstly, it is argued that the model does not adequately explain all the clinical features of CFS (Surawy, Hackmann, Hawton & Sharpe, 1995). For example, people with CFS often describe a behaviour pattern of periods of rest accompanied by short burst of activity (Sharpe, 1997). The Wessely et al. (1989, 1991) model does not explain why this pattern may exist (Sharpe, 1997). Secondly, there is an emphasis on how CFS symptoms are maintained in the Wessely et al. (1989, 1991) model, but aetiology of CFS and how psychological factors may play a role is not adequately addressed (Surawy et al., 1995).

Surawy et al. (1995) have expanded on the cognitive behavioural model of Wessely et al. (1989). In this more complex cognitive behavioural model, Surawy et al. (1995) describe predisposing, precipitating and maintaining factors for CFS. Surawy et al. (1995) suggest that a predisposing factor in the development of CFS are beliefs and assumptions concerning the need to achieve and to meet high standards. For example, Surawy et al. (1995) found that people with CFS held assumptions such as “If I don’t meet all my responsibilities to others all the time I am a failure” (pg. 537). When individuals are faced with a ‘critical incident’ that makes it difficult to continue with previous levels of activity, such as excessive stress or an acute illness, beliefs concerning the need for achievement are activated (Surawy et al., 1995). An initial reaction in the face of such difficulties may be for the person to try harder to meet their goals. However, at some point, this behaviour becomes too much for the person to cope with and they enter a state of chronic exhaustion, frustration and demoralisation. Surawy et al. (1995) go on to suggest that for people who hold such
high expectations of coping and achievement, the only acceptable explanation for their symptoms is a physical disease. Explanations such as depression or difficulties in coping with a stressful situation would imply that they are somehow weak or at fault for developing the condition (Surawy et al., 1995). For example, Surawy et al. (1995) point to chronic fatigue sufferers who state “It can’t be depression because I’m not that sort of person” (pg. 538). Once CFS symptoms have been interpreted as a sign of a physical disease, this leads the person to rest and avoid activity, as suggested in the Wessely et al. (1989) model. However, Surawy et al. (1995) also suggest that such a reduction in activity levels is experienced as anxiety provoking given sufferers’ beliefs about achievement. Consequently, people with CFS engage in bursts of activity in an attempt to meet their expectations. However, these activity bursts exacerbate symptoms and this reinforces their belief that they have a serious illness and causes further emotional distress (Surawy et al., 1995).

There is evidence to suggest that people with CFS do hold beliefs about the need for high standards for performance and perfectionism. For example, White and Schweitzer (2000) compared forty-four people with CFS to forty-four healthy people using the Multidimensional Perfectionism Scale. People with CFS reported significantly higher levels of perfectionism than those in the control group. In particular, people with CFS endorsed more items on the ‘doubts about actions’ and ‘concern over mistakes’ subscales of the MPS. These scales included statements such as “if I fail at work/school, I am a failure as a person” and “Even when I do something very carefully, I often feel that it is not quite right” (White & Schweitzer, 2000). The hypothesis that stress and illness can precipitate CFS symptoms is also supported. A number of studies have demonstrated that compared to control groups, people with CFS report more stressful life events and periods of illness prior to the onset of symptoms (Chalder et al., 2000).

In addition, studies provide evidence that beliefs people hold about their symptoms contribute to both the onset and maintenance of CFS (Moss-Morris, 1997). For example, Sharpe, Hawton, Seagrott and Pasvol (1992) found that in people with chronic fatigue and CFS, functional impairment over a two year period was
significantly associated with beliefs that their symptoms had a viral cause and that avoiding exercise was beneficial. In addition, Petrie, Moss-Morris and Weinman (1995) investigated the role of catastrophic beliefs about increasing activity levels in a sample of CFS patients. Results showed that more catastrophic beliefs about the consequences of activity were associated with higher levels of fatigue and more impaired occupational and social functioning. Qualitative research has also highlighted the nature of illness beliefs and coping behaviours in CFS patients. Clements et al. (1997) found that CFS sufferers tended to attribute their symptoms to a physical cause and placed an emphasis on rest and reducing activities as strategies to cope with CFS symptoms. In addition, sufferers also talked about trying to overcome the CFS symptoms by periodically attempting bouts of activity which inevitably resulted in the need for total rest (Clements et al., 1997).

Additional support for cognitive behavioural models of CFS comes from studies of cognitive behaviour therapy (CBT) for CFS. CBT has been found to be more effectively than standard medical care (Sharpe, Hawton, Simkin, Surawy, Hackman, Klimes, Peto, Warrell & Seagrott, 1996), relaxation therapy (Deale, Chalder, Marks & Wessely, 1997) and no treatment (Butler, Chalder, Ron & Wessely, 1991). However, in two other trials, CBT was found to be no more effective in decreasing functional impairment than standard medical care (Lloyd, Hickie & Brockman, 1993) or no treatment (Friedberg & Krupp, 1994). Moss-Morris (1997) suggests that this result may be in part due to different CBT approaches being used in these studies. While the three successful trials used CBT methods based on the cognitive behavioural models described above, the two unsuccessful trials of CBT used a different approach. In the CBT approaches employed by Lloyd et al. (1993) and Freidberg and Krupp (1994), the beliefs that sufferers held about CFS and their symptoms were not addressed. It therefore appears that the effective ingredient in CBT for CFS is the challenging of patients' beliefs (Moss-Morris, 1997). Nevertheless, even in the trials where CBT had a significant overall effect, not all participants improved and across trials most participants continued to experience symptoms of fatigue (Sharpe, 1998).

Overall, there is growing evidence to support cognitive behavioural models of CFS.
However, methodological difficulties with the existing research limit the conclusions that can be drawn. For example, research studies frequently use different criteria for CFS, different measures of disability and fatigue, and CFS samples differ on variables such as illness duration. Further research is needed to investigate the predisposing, precipitating and maintaining factors suggested by cognitive behavioural models using standardised CFS criteria and outcome measures. In addition, it would be beneficial to establish predictors for outcome of CBT of CFS (Sharpe, 1998).

Despite these limitations, cognitive behavioural models contribute to our understanding of CFS in a number of ways. Firstly, cognitive behavioural models highlight the importance of people’s illness beliefs and how these may contribute to the development and maintenance of CFS. Secondly, cognitive behavioural models may provide an explanation as to why there is a high incidence of psychiatric disorder in CFS. For example, Moss-Morris (1997) suggests that catastrophic and negative beliefs about CFS, and a lack of control over symptoms may cause high levels of depression. Thirdly, the cognitive behavioural model of Surawy et al. (1995) aids our understanding as to why CFS sufferers vehemently assert that there is an organic cause for their symptoms. Within the cognitive behavioural model this type of attribution is seen as stemming from CFS sufferers’ beliefs about achievement and failure. Physical illness attributions are seen as a way reducing blame and preserving self-esteem (Surawy et al., 1995). Finally, cognitive behavioural models of CFS may help to highlight differences between CFS and other disorders with which the diagnostic criteria overlap. As previously stated studies have shown that it is difficult to differentiate between CFS, depression and physical illness on the basis of fatigue symptoms (Wessely & Powell, 1989: Lynch, 1996). Moss-Morris (1997) suggests that investigating peoples’ beliefs about their symptoms may be a more effective way to distinguish between CFS and other disorders. For example, Moss-Morris and Petrie (1996: cited in Moss-Morris, 1997) compared a group of CFS patients to a group of people with depression. People with CFS were significantly more likely to attribute their symptoms to a physical, external cause while people with depression were more likely to attribute their symptoms to internal causes.
Conclusion
Chronic fatigue syndrome (CFS) poses substantial difficulties for sufferers. Not only do they have to cope with a range of disabling symptoms, but also with their problems being frequently dismissed as 'all in the mind' (Chalder & Williams, 1997) or given derogatory terms such as 'yuppie flu' (Moss-Morris, 1997). CFS also presents considerable difficulties for researchers and clinicians. The publication of the first diagnostic for CFS by Holmes et al. (1988) has been called a “milestone in the acceptance of the syndrome and the beginning of a new generation of scientific studies” (Wessely et al., 1998: pg. 130). However, there still exists considerable controversy surrounding the definition of CFS and the extent to which the symptoms of CFS overlap with other disorders such as chronic pain, fibromyalgia, anxiety and depression (Wessely et al., 1998: Kumar & Lynch, 1997).

In addition, the aetiology of CFS is the subject of much debate. A large amount of research has been aimed at identifying an organic cause of CFS symptoms. However, many researchers have called for models of CFS to consider the psychological, social and biological aspects of the condition (Wessely et al., 1998). To this end, this essay has considered how psychodynamic, systemic and cognitive behavioural theories have contributed to our understanding of CFS.

There has been relatively little attention paid to CFS in both the psychodynamic and systemic literature (Abbey, 1996). The psychodynamic theory of CFS proposed by Taerk and Gnam (1994) attempts to integrate both psychological and biological factors of CFS, however the theory is limited by the lack of evidence to support it. There is evidence to suggest that a systemic perspective may aid our understanding of how family factors and wider social attitudes contribute to the development and maintenance of CFS. However, systemic theories are also limited by a lack of research.

It has been argued here that cognitive behavioural models have made the most substantial contribution to our understanding of CFS. Such models, in particular that of Surawy et al. (1995), set out predisposing, precipitating and maintaining factors
for CFS for there is some support from empirical studies. By highlighting the role of illness beliefs in CFS, cognitive behavioural models provide an explanation as to why CFS sufferers may find it easier to attribute their symptoms to physical causes (Surawy et al., 1997) and why there is a higher incidence of psychiatric disorder associated with CFS (Moss-Morris, 1997). Examination of illness beliefs in CFS may also help to distinguish between CFS and other similar disorders (Moss-Morris, 1997). In addition, cognitive behavioural models have also given rise to the only form of treatment for CFS that has been demonstrated to provide some benefit (Moss-Morris, 1997).

However, our understanding of CFS is far from complete. For example, further research is needed to identify predictors of outcome for cognitive behaviour therapy (Sharpe, 1997), and to clarify how family factors may contribute to the development and maintenance of CFS.
References


Kumar, S. & Lynch, S. (1997) Chronic fatigue syndrome: its development and


Clinical Section

This section contains summaries of clinical experience gained in the four core placements and two specialist placements. Also contained in this section is a summary of the five case reports written for each core placement, plus one specialist placement. The five clinical case reports are submitted in full in Volume Two of this portfolio, along with placement contracts, supervisor evaluation forms and full records of clinical activity.
Summary of Clinical Experience
Adult Mental Health Placement

Placements Details

*Dates:* October 1998 to March 1999

* Supervisor: Ms Jenny Trust, Chartered Clinical Psychologist

*NHS Trust:* Surrey Hampshire Borders NHS Trust

*Base:* Aldershot CMHT

Client demographics

- Individual work with 10 clients (4 male, 6 female) ranging in age from 21 to 61
- Group work with 7 clients (6 male, 1 female) ranging in age from 25 to 59

Presenting Problems / Issues

- Anxiety
- Depression
- Chronic Pain
- Family and relationship problems
- OCD
- Phobia
- Cognitive impairment following head injury
- Sexual Abuse
- Bereavement
- Schizophrenia (group work)

Settings

- CMHT outpatient
- Day Hospital

Assessment Procedures

- Assessment Interviews
• Questionnaires including: Hospital Anxiety and Depression Scale, Beck Depression Inventory, Beck Hopelessness Scale, Beck Anxiety Inventory, Impact of Events Scale, McGill Pain Questionnaire, Crown Crisp Inventory, Padua Inventory and Maudsley Obsessive Compulsive Scale
• Neuropsychological Assessment using WAIS-R, WMS-R, Schonell Reading Test, Rey 15 Item and Behavioural Assessment of the Dysexecutive Syndrome

Interventions
• Cognitive Behaviour Therapy
• Integrative models (CBT / Psychodynamic)
• Psychoeducation

Other Experience
• Contributed to the development of an information leaflet for clients referred to CMHT clinical psychologist
• Observed CBT anxiety management group and psychodynamically informed music therapy group
• Observed CPA reviews in the CMHT and inpatient ward
• Met with and observed work of other professionals (Occupational Therapist, CPN, Psychiatrist and Family Therapist)
• Attended an Eating Disorder Workshop
• Met with the Trust Professional Lead for Clinical Psychology and co-ordinator of Psychological Therapies to discuss psychological services within the Trust, professional and organisational issues
• Contributed to Assistant Psychologist’s Workshop
People with Learning Disabilities Placement

Placements Details

Dates: April 1999 to September 1999

Supervisor: Dr Elaine Alves, Consultant Clinical Psychologist

NHS Trust: Bournewood Community NHS Trust

Base: St Peter’s Hospital, Chertsey

Clients demographics

- Individual work with 7 clients (4 female, 3 male) ranging in age from 31 to 68

Presenting Problems / Issues

- Anxiety
- Family problems
- Challenging behaviour
- Emotional and physical abuse
- Assessment of dementia in people with Down’s syndrome
- Psychosis
- Assessment of placement suitability

Settings

- Community based outpatient
- Day centres
- Group homes
- Inpatient unit

Assessments

- Assessment interviews with staff / carers using behavioural model
- Functional analysis (charts, diaries, direct observation)
People with Learning Disabilities Placement Summary

- Questionnaires including: Hospital Anxiety and Depression Scale, Motivational Assessment Scale, PAS-ADD, Negative Symptoms of Schizophrenia Scale, Brief Symptom Inventory
- Neuropsychological Assessments using, WAIS, CAPE, MEAMS, AMIPB, WAIS-III, Bournewood Downs Dementia Assessment.
- Daily Living Skills assessment using HALO

Interventions
- Behavioural interventions
- Cognitive Behaviour Therapy
- Staff support sessions

Other experience
- Presentation to psychology department
- Visits to various services for people with learning disabilities including group homes, day centres and challenging behaviour unit
- Attended departmental seminars
- Met with other professionals working with people with learning disabilities (clinical nurse specialist, speech therapist, psychiatrist)
- Observed psychiatric outpatient clinic
- Attended IPP meeting for learning disabled client living in the community
Children, Adolescents and Families Placement

Placements Details

Dates: October 1999 to March 2000

Supervisor: Mrs Ruth Armstrong, Consultant Clinical Psychologist

NHS Trust: South West London Community NHS Trust

Base: Sutton Hospital

Client demographics

- Individual work with 12 clients (6 female, 6 male) ranging in age from 2½ to 16

Presenting Problems / Issues

- Adjustment to Epilepsy
- Phobia
- Enuresis
- Encopresis
- Bereavement
- Assessment of cognitive functioning to inform school placement
- Learning Disabilities / Developmental delay
- Self injurious behaviour
- Family problems including sibling jealousy, parental management issues and relationship difficulties between parents and child
- Anxiety
- Somatic symptoms

Settings

- Community based outpatient
- Home visits

Assessments

- Assessment interviews with CBT, behavioural and systemic frameworks
• Functional analysis
• Questionnaires including: Parenting Stress Index, Behaviour Rating Scales, Vineland Adaptive Behaviour Scales, Spence Children’s Anxiety Scale, Children’s Depression Inventory, Bene Anthony Family Relations Test,
• Neuropsychological Assessments using: WISC-III, WORD, Rivermead Behavioural Memory Test (children’s version)

Interventions
• Behavioural, CBT and systemic interventions
• Psychoeducation

Other experience
• Case presentation to psychology department
• Presentation to Child Expert Witness Group on practice effects in repeated neuropsychological assessment
• Attended meetings of Child Expert Witness Group
• Meetings with other professionals (Family Therapist, CPN, Social Worker, Child Psychotherapist, Children’s Inpatient Unit Manager)
• Attended a Child Protection case conference
• Observed development assessment (Ruth Griffith) carried out by a paediatrician
• Attended presentation on Chronic Fatigue Syndrome in Children
• Observed administration of Rorschach Ink Blot assessment
• Observed a multidisciplinary team ADHD clinic
Older Adults Placement Summary

Older Adults Placement

Placements Details

Dates: April 2000 to September 2000

Supervisor: Mrs Clare Crellin, Chartered Clinical Psychologist

NHS Trust: Mid Sussex NHS Trust

Base: Linwood Community Mental Health Centre

Client demographics

- Individual work with 13 clients (11 female, 2 male) ranging in age from 67 to 88

Presenting Problems / Issues

- Cognitive impairment
- Depression
- Anxiety
- Phobia
- Behaviour difficulties on inpatient ward
- Adjustment post stroke
- Hypocondriasis
- Bereavement

Settings

- Community outpatient
- Day Unit
- Inpatient
- Home Visits
- GP surgery

Assessments

- Assessment interviews using a structured interview schedule
Older Adults Placement Summary

• Questionnaires including: Beck Anxiety Inventory, Beck Depression Inventory II, Beck Hopelessness Scale, Impact of Events Scale, Hospital Anxiety and Depression Scale and Millon Clinical Multiaxial Inventory-II

• Neuropsychological Assessments using: WAIS-III, WMS-III, NART, FAS, Category Naming, Graded Naming Test, Description Naming Test and CAMDEX.

• Evaluation of outcome using CORE assessments for Primary Care and Mental Health Services

Interventions

• CBT interventions
• Psychoeducation
• Integrative CBT and psychoanalytic interventions

Other experience

• Attended Psychology Department team meetings and presentations.
• Attended Adult and Older Adult Psychology team and EMI team meetings
• Attended ward round for older adult inpatient ward
• Attended two sessions of a dementia workshop for care staff run by members of the EMI team
• Presentation to EMI team on memory groups for older adults with dementia (with another trainee)
• Involved in the development of a memory group for older adults
• Involved in the revision of a structured assessment interview schedule
• Involved in setting up Primary Care Sessions for older adults at GP surgery
• Developed audit sheet for EMI clinical psychology service
• Involved in piloting the use of the CORE assessments for Primary Care and Mental Health Services for EMI clinical psychology referrals.
Specialist Placement in Clinical Health Psychology

**Placements Details**

*Dates:* October 2000 to March 2001  
*Supervisor:* Dr Michelle Sowden  
*NHS Trust:* Surrey Hampshire Borders NHS Trust  
*Base:* Frimley Park Hospital

**Client demographics**
- Individual work with 8 clients (10 female, 3 male) ranging in age from 31 to 52
- Joint work with supervisor with 5 clients (1 male, 4 female) ranging in age from 27 to 66
- Group work (2 sessions) with 9 clients (2 male, 7 female) ranging in age from 31 to 62

**Presenting Problems / Issues**
- Chronic Pain (group work)  
- Anxiety  
- Depression  
- Sexual Abuse  
- Somatic Symptoms  
- Gynaecological Problems / sexual problems  
- Issues relating to management and adjustment to conditions including: Diabetes, Lupus, Epilepsy, Multiple Sclerosis and Crohn’s disease  
- Psychogenic Amnesia

**Settings**
- Hospital based outpatient

**Assessments**
- Assessment interviews within mainly CBT framework
Specialist Placement (1) Summary

• Questionnaires including: Beck Anxiety Inventory, Beck Depression Inventory, Beck Hopelessness Scale, General Health Questionnaire, Problems with Diabetes Questionnaire, Perceived Stress Scale, Visual Analogue Scales.

Interventions
• Individual, couple and group CBT

Other experience
• Attended Department of Psychological Medicine team meetings and presentations
• Presentation of Major Research Project to Trust Psychology Research Group
• Presentation of conference attended to Department of Psychological Medicine
• Produced document on options for waiting list management of referrals to clinical psychology
• Met with Specialist Nurses (diabetes, breast cancer, terminal cancer, cardiac rehabilitation) in the hospital
• Visited specialist cancer centre
• Assisted in the development of interview questions and was involved in interviewing a candidate for an assistant psychologist’s post.
Specialist Placement in Neuropsychology

Placements Details

*Dates*: April 2001 to September 2001

*Supervisor*: Dr Drew Alcott

*Organisation*: Priory Health Care and Bournewood Community NHS Trust

*Base*: Unsted Park Rehabilitation Centre

Client demographics

- Individual work with 13 clients (10 male, 3 female) ranging in age from 20 to 75

Presenting Problems / Issues

- Acquired brain injury as a result of road traffic accidents, assault, CVA, and subarachnoid haemorrhage
- Cognitive impairment due to degenerative conditions such as dementia and multiple sclerosis
- Assessment of cognitive difficulties including memory problems, executive functioning impairments and visual neglect
- Anxiety and Depression
- Impaired awareness of difficulties
- Confabulation
- Difficulties adjusting to brain injury

Settings

- Inpatient unit
- Community outpatient

Assessments

- Assessment interviews

*Experience gained from April 2001 to July 2001*
• Neuropsychological assessments including: AMIPB, WAIS-III, Behavioural Assessment of the Dysexecutive Syndrome, Hayling and Brixton Tests, VOSP, Modified Wisconsin Card Sorting Test, NART, FAS, Cognitive Estimates Test, Abbreviated Mental State Examination, Token Test.

Interventions
• CBT and behavioural interventions
• Feedback of neuropsychological assessment results

Other experience
• Attended clinical review meetings and goal planning sessions
• Attended case conferences
• Observed group work (cognitive rehabilitation group, communication group and brain injury group)
• Met with and observed work of other professionals (Speech and Language Therapist, Occupational Therapist, Physiotherapist)
• Visited other services for people with acquired brain injury including; Headway, Wolfson Rehabilitation Hospital, Ticehurst Hospital, and Royal Hospital for Neuro-disability.
• Attended presentation: “Neuropsychiatric aspects of head injury”
• Attended conference: “Rehabilitation Dysexecutive Disorders”
Clinical Case Report Summaries
Adult Mental Health Case Report Summary

Cognitive Behavioural Assessment and Intervention for a 41 year old woman with Depression

March 1999

Year 1
Some of the details of this case have been changed to maintain client confidentiality

Reason for Referral
Mrs Brown was a 41 year old woman with depression, referred by her Community Psychiatric Nurse.

History of Presenting Problem
Mrs Brown reported that she had first experienced symptoms of depression five years previously, following the break up of a long-term relationship. At this point she attempted suicide and was referred to a psychiatrist, but reported that her symptoms of depression persisted. A year prior to her referral to clinical psychology, Mrs Brown experienced a second episode of acute depression and was again referred to a psychiatrist and CPN. Mrs Brown reported that she had found it helpful to talk about her difficulties with the CPN, but that her symptoms of depression had not improved significantly. She was discharged by her CPN when she received an appointment with the trainee clinical psychologist.

Assessment
The assessment involved interviews with Mrs Brown that covered information about the nature and course of her depression. The Beck Depression Inventory (BDI) and Beck Hopelessness Scale (BHS) were also used. The BDI indicated that Mrs Brown was suffering from severe symptoms of depression and her score on the BHS suggested that she was experiencing a high level of hopelessness. Although she reported suicidal ideation, Mrs Brown stated that she would not act on these thoughts. During the assessment phase, information was also gained about Mrs Brown’s family, psychiatric, and employment history. She highlighted job and financial stress as a current contributing factor to her depression. In addition, Mrs Brown reported a number of beliefs developed from childhood regarding the importance of coping with her problems on her own and the importance of personal and financial stability.
Formulation
Mrs Brown’s depression was formulated within Beck’s Cognitive Model of depression. Several critical incidents were identified as triggering Mrs Brown’s beliefs about coping with her problems on her own and the importance of personal and financial stability. This appeared to lead to the emergence of negative automatic thoughts and the symptoms of depression reported by Mrs Brown. Her depression seemed to be maintained by work stress, loneliness and financial difficulties. In addition, Mrs Brown had difficulty identifying possible ways of dealing with the practical difficulties she faced and this appeared to exacerbate her feelings of hopelessness and powerlessness.

Intervention
The intervention was aimed at helping Mrs Brown to use problem-solving strategies to tackle her difficulties at work and with finances and to identify and modify negative automatic thoughts maintaining her low mood. In addition, she was encouraged to increase her involvement in pleasurable activities to help her overcome her lack of motivation and to help her evaluate negative automatic thoughts such as “I am not doing anything worthwhile”.

Outcome and Critique
Mrs Brown was able to tackle some of the practical difficulties that she had identified as sources of stress at the initial assessment. She had begun to increase her activities and had re-established several social relationships that helped ease her feelings of loneliness. Mrs Brown also reported that by monitoring and identifying her negative thoughts, she was able to look at situations from different perspectives. Consequently, by the middle of the therapy sessions, Mrs Brown’s scores on the BDI had dropped from the severe range to the mild range. However, this improvement was not maintained and she relapsed at the end of the intervention.

Issues relating to the provision of further clinical psychology input for Mrs Brown and aspects of the intervention are discussed.
People with Learning Disabilities Case Report Summary

Behavioural Assessment and Intervention for a Learning Disabled Client with Behaviour Problems

September 1999

Year 1
Some of the details of this case have been changed to maintain client confidentiality

Reason for Referral
Miss Amber was a 46 year old woman with severe learning disabilities and limited verbal communication skills. Staff at the group home where she lived requested clinical psychology input as they reported having difficulty managing a variety of problem behaviours displayed by Miss Amber.

History of Presenting Problem
Staff reported that Miss Amber’s problem behaviours included stealing food, pushing and hitting other residents in the group home, socially inappropriate behaviours such as walking around the home with her clothes undone and interrupting staff meetings at the home. These behaviours had been present for approximately eight months prior to the referral.

Assessment
Drawing on information from Miss Amber’s key worker and psychology notes from previous referrals, Miss Amber’s behaviour was formulated within an operant model of behaviour. It was hypothesised that the main function of Miss Amber’s behaviour was to gain attention and that this was reinforced by staff in the group home. Further assessments and functional analyses were carried out to (1) test the initial formulation (2) provide baseline data to evaluate the intervention and (3) aid with the development of an intervention package. Assessments measures completed by staff at the group home included the Motivation Assessment Scale and an Antecedent, Behaviour and Consequence (ABC) Chart. Miss Amber was also directly observed in a range of settings.

Formulation and Intervention
Analysis of the data from assessments supported the main points of the initial formulation. An intervention package was then devised to (1) moderate the antecedents and consequences of the client’s behaviour and (2) help the staff group to gain a better understanding of the client’s problems. To this end a set of behaviour
management guidelines were developed to help the staff team manage Miss Amber’s behaviour more appropriately.

Outcome and Critique

Subsequent observation of Miss Amber and the staff team suggested that Mrs Amber’s behaviour problems were more appropriately managed, however there was no objective change in the frequency of behaviour problems reported. Possible reasons for this outcome are discussed, in particular the impact of major changes in the staff team prior to implementation of the intervention. Aspects of the assessment process and additional components that could have been included in the intervention are also reviewed.
Assessment of an eight year old girl with somatic symptoms using a Psychosomatic model

March 2000

Year 2
Children, Adolescents and Families Case Report Summary

Some of the details of this case have been changed to maintain client confidentiality

Reason for Referral
Jane Smith was an 8 year old girl, referred for assessment by a Consultant Anaesthetist in Pain Management.

History of Presenting Problem
Two years prior to the referral Jane had developed severe pain in her groin and hip area, for which no medical explanation could be found. The pain in Jane’s groin and hip had since resolved, but at the time of assessment she was experiencing headaches and had a poor appetite. Due to the nature of Jane’s previous and current somatic symptoms, the assessment aimed not only to gain an understanding of the predisposing, precipitating and maintaining factors for the problem, but also to address concerns about the possibility of sexual abuse and the presence of an eating disorder.

Assessment
The assessment procedure included interviews with Jane and her family. In addition, the Bene Anthony Family Relations Test, the Spence Children’s Anxiety Scale, the Children’s Depression Inventory, Pain ratings and Visual Analogue Scales of Jane’s current worries were used. The purpose of these assessments was to gain additional information regarding factors involved in the development and maintenance of the problem and to provide baseline data against which to evaluate the intervention.

Formulation
The assessment revealed no evidence of sexual abuse or the presence of an eating disorder. Jane’s difficulties were formulated within Lask and Fosson’s (1989) Psychosomatic Model. The formulation emphasised the role of Jane’s high levels of anxiety, significant family stress, the family’s history of ill health and Jane’s developmental level in the development of her somatic symptoms. In addition, it was hypothesised that Mr and Mrs Smith’s parenting style contributed the development and maintenance of Jane’s symptoms.
**Intervention**

Based on the formulation, an intervention plan consisting of individual and family sessions was developed. The intervention was to focus on helping Jane to express her anxieties and to use psychoeducation and anxiety management techniques to help Jane understand and control her anxiety. Family sessions were planned to help Mr and Mrs Smith contain Jane’s anxiety more appropriately. The planned intervention was to be carried out by the placement supervisor.

**Prognosis and Critique**

Factors that may have influenced the family’s engagement and progress in therapy are discussed. In addition, limitations of the assessment process and Psychosomatic Model are commented on.
Older Adults Case Report Summary

Neuropsychological Assessment of a 73 year old man with suspected dementia

September 2000

Year 2
Some of the details of this case have been changed to maintain client confidentiality

**Reason for Referral**
Mr May was a 73 year old man who had been provisionally diagnosed as suffering from atypical dementia, by a Consultant Neurologist. Neuropsychological assessment was requested to aid diagnosis.

**History of Presenting Problem**
Mr May reported noticing a gradual worsening of his memory for approximately eighteen months prior to assessment including word-finding difficulties, and problems spelling previously familiar words, remembering names and recognising faces.

**Assessment**
Interviews were conducted with Mr May and his wife to gain information regarding Mr May’s social, educational, occupational and medical history. Based on this information, it was hypothesised that Mr May was suffering from either Alzheimer’s disease or semantic dementia. Several neuropsychological tests were selected to test this hypothesis. The Wechsler Adult Intelligence Scale – 3rd edition (WAIS-III) was used to assess current intellectual functioning. An estimate of Mr May’s premorbid functioning was provided by the National Adult Reading Test, with appropriate corrections for use with the WAIS-III. Memory functioning was assessed using the Wechsler Memory Scale – 3rd edition (WMS-III) and semantic memory was assessed using FAS Letter Fluency Test, Category Fluency Test, Graded Naming Test and the Description Naming Test.

**Formulation**
Neuropsychological test results suggested that Mr May had suffered a general decline in intellectual functioning consistent with a diagnosis of degenerative dementia. In addition, general impairments in memory functioning and a relatively greater decline in non-verbal aspects of cognitive functioning suggested that the likely diagnosis was Alzheimer’s disease.
Outcome and Recommendations
The results of the assessment were fed back to Mr May and his wife and they were informed that the likely cause of Mr May's memory problems was Alzheimer's disease. However, to confirm this diagnosis a follow-up assessment was arranged for nine months time. Mr and Mrs May were given information on coping strategies for memory problems and Mr May was advised to consult his general practitioner regarding a driving assessment. In addition, Mr May was referred to an Aricept treatment programme.
Specialist Placement Case Report Summary
(Clinical Health Psychology)

Cognitive-behavioural assessment and intervention with a 45 year old woman with Multiple Sclerosis

March 2001

Year 3
Some of the details of this case have been changed to maintain client confidentiality

**Reason for Referral**
Mrs White was a 45 year old woman with Multiple Sclerosis (MS). She was referred by her GP as she appeared to be having difficulties coming to terms with MS and the physical limitations it imposed on her.

**History of Presenting Problem**
Mrs White had been diagnosed with MS twelve years previously but she reported that initially she had coped with the illness and had not suffered major physical problems. Her condition deteriorated two years ago and at the time of assessment she was in a wheelchair and had significant weakness in both arms. Mrs White was reliant on help from her husband and social services carers to carry out self-care tasks, however the type and amount of help that she received from others was often at odds with what she felt she required. This caused Mrs White considerable distress as her relationship with her husband was poor and she believed that she could not discuss difficulties with him. In addition, she was reluctant to allow other people to 'get close' and this frequently resulted in difficulties with her social services carers and left Mrs White feeling that she had little control over her own life.

**Assessment**
During the assessment interviews, information was gained from Mrs White regarding her personal and family history, psychiatric history and issues relating MS. An assessment of her mood and coping style was carried out via interview, as self-report measures were difficult for Mrs White to complete due to her physical disabilities. Information gained during the interview suggested that Mrs White was experiencing moderate symptoms of depression and that her coping style was characterised by avoidance of difficulties and conflict with significant others e.g. her husband and her carers.
Specialist Placement Case Report Summary

Formulation
Mrs White's difficulties were formulated with a cognitive-behavioural model that emphasised how her presenting problems were influenced by her negative automatic thoughts and beliefs that others would respond unfavourably to her requests for more appropriate help. Thus, her style of coping was to withdraw from others, thereby making it more difficult for her husband and carers to meet her needs.

Intervention
The intervention was aimed at helping Mrs White to identify and challenge her negative automatic thoughts. Mrs White was encouraged to set up behavioural experiments to test her beliefs and negative automatic thoughts about various situations. Problem-solving strategies for practical difficulties were also discussed. Dysfunctional beliefs regarding the meaning of acceptance and adjustment to MS were highlighted as a significant issue for Mrs White and these were addressed in therapy sessions. However, it was also acknowledged feelings of grief and sadness about the losses Mrs White had sustained as a result of the MS were realistic. Psychoeducation regarding the role of grief in response to illness was included in the intervention.

Outcome
At the end of the therapy sessions, Mrs White reported that she felt more able to tackle the difficulties she faced and had made some significant changes in how she interacted with the various professionals involved in her care. Mrs White also reported that her feelings of hopelessness and low mood had improved.
Research Section

This section contains three pieces of work. The Service Related Research Project was carried out in Year 1, during the Adult Mental Health Placement. The Literature Review was completed in Year 2 and is intended as an introduction to the Major Research Project. The Major Research project was carried out in Years 2 and 3.
Service Related Research Project

Evaluation of the ‘Living with Schizophrenia’ group

July 1999

Year 1
Abstract

Objectives: To evaluate the effectiveness of a 6 week CBT group for people with psychosis, in terms of reducing symptom impact, anxiety and depression and increasing knowledge about schizophrenia. Additional aims were to determine the acceptability of group therapy and to identify how the group may be developed.

Design: Suitable clients with schizophrenia or schizoaffective disorder were invited to attend the group. Assessments were completed pre and post group and at 4 week follow-up.

Setting: Community Day Centre.

Participants: 7 clients with persistent positive or negative symptoms of schizophrenia.

Outcome Measures: Knowledge Scale, Hospital Anxiety and Depression Scale, Symptom Rating Scale Views about Schizophrenia Scale and a Satisfaction Questionnaire.

Results: Post group there was an increase in knowledge about schizophrenia, reduced anxiety, reductions in the impact of targeted symptoms and increased coping and control. There was little change in depression. At follow-up, gains were maintained for symptom reduction, increased coping and control. The format and content of the group was well received by participants.

Conclusions: Although conclusions are limited by the small sample size, the group appears to be an acceptable and potentially beneficial form of therapy for people with psychosis. Suggested changes to the group include increasing the number of sessions or focussing one topic rather than several.
Introduction

There is now strong evidence to suggest that cognitive behavioural therapy (CBT) may have an important role to play in the treatment of psychosis. Traditional CBT techniques such as belief modification and reality testing have been successfully used to address distorted beliefs underlying delusions and hallucinations leading to a reduction in the impact of such symptoms (Haddock, Tarrier, Spaulding, Yusupoff, Kinney & McCarthy 1998). In addition, CBT approaches such as coping strategy enhancement and problem solving have been developed to improve the way individuals cope with psychotic symptoms. Such techniques have been demonstrated to significantly reduce positive symptoms (Tarrier, Yusupoff, Kinney, Gledhill, Haddock & Morris, 1998).

Research evaluating the effectiveness of CBT for psychosis has concentrated mainly on individual CBT interventions (e.g. Kuipers, Garety, Fowler, Dunn, Bebbington, Freeman & Hadley, 1997) with group CBT approaches being widely ignored (Gledhill, Lobban & Sellwood, 1998). However, CBT for schizophrenia in a group format may have a number of benefits over individual interventions (Gledhill et. al., 1998). People with schizophrenia may believe that psychotic symptoms are unique to them, thus by discussing symptoms in a group, feelings of stigma and anxiety may be reduced. In addition, group work can provide an ideal setting for peer support, reality testing and education (Kahn & Kahn, 1992). Secondly, group work may improve coping strategies via generalisation and modelling. Thirdly, group therapy may permit the delivery of CBT to more clients, which has important implications for mental health services, whose resources are limited.

In the light of these considerations a group entitled “Living with Schizophrenia” was developed, from a therapy programme outlined by Gledhill et. al. (1998), by a clinical psychologist working at a community day centre. The group was based on CBT techniques – mainly coping strategy enhancement (Tarrier, Beckett, Harwood, Baker, Yusupoff & Ugarteburu, 1993), and psychoeducation (Fowler, Garety & Kuipers, 1995). These techniques were applied within in a normalising framework
(Turkington, & Kingdon, 1996) which aims to reduce the stigma and anxiety associated with the experience of psychotic symptoms.

Research suggests that such techniques may help reduce the impact of positive symptoms, decrease anxiety and depression, and improve understanding of psychosis (Fowler, et al., 1995).

Aims

The "Living with Schizophrenia" group had been run once before in the day centre, but had yet to be evaluated. Given the emphasis on evidence based practice in the NHS (Department of Health, 1998), the aims of this research were therefore -

- To evaluate the effectiveness of the group in terms of reducing the impact of clients' targeted symptoms, increasing ability to cope with and control a targeted symptom, increasing knowledge about schizophrenia, and reducing anxiety and depression. It was predicted that these improvements would be maintained at a 4-week follow-up.
- To determine the acceptability to clients of a group format.
- To identify how the group may be developed in terms of content, structure, and length.

Method

Participants

Referrals to the group were invited from CMHT's and key workers in the catchment area of the day centre two months prior to the start of the group. A psychiatrist and a clinical psychologist then assessed potential participants according to the inclusion and exclusion criteria shown in Table 1.
Table 1. Inclusion and exclusion criteria for the ‘Living with Schizophrenia’ group.

<table>
<thead>
<tr>
<th>Inclusion criteria</th>
<th>Exclusion criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Primary diagnosis of schizophrenia / schizoaffective disorder</td>
<td>• Acute psychosis preventing rationale dialogue</td>
</tr>
<tr>
<td>• Persistent positive or negative symptoms</td>
<td>• Disruptive in group settings</td>
</tr>
<tr>
<td>• Ability to share difficulties verbally</td>
<td>• Limited attention span</td>
</tr>
<tr>
<td>• Stabilised on neuroleptic medication</td>
<td>• History of violence</td>
</tr>
<tr>
<td>• Able to concentrate in a group setting</td>
<td></td>
</tr>
<tr>
<td>• Have a named community worker</td>
<td></td>
</tr>
<tr>
<td>• Registered with a GP in the day centre catchment area</td>
<td></td>
</tr>
</tbody>
</table>

Seven clients (six men and one woman) were invited to attend the group and all accepted. Table 2 contains descriptions of the participants.

Table 2. Description of Participants

<table>
<thead>
<tr>
<th>Code</th>
<th>Sex</th>
<th>Age</th>
<th>Diagnosis</th>
<th>Duration of Disorder (years)</th>
<th>No. of hospital admissions</th>
<th>Time since last admis.</th>
<th>Current medication</th>
<th>Time known to service</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>F</td>
<td>37</td>
<td>Schizophrenia</td>
<td>20</td>
<td>2 + long stay ward for 10 years</td>
<td>3 years</td>
<td>Risperidone Zudopenthixol Procyclidine</td>
<td>2 years</td>
</tr>
<tr>
<td>B</td>
<td>M</td>
<td>25</td>
<td>Schizoaffective disorder</td>
<td>6</td>
<td>3</td>
<td>22 months</td>
<td>Lithium Carbonate Sertinole Thyroxine Carbamazipine</td>
<td>3 months</td>
</tr>
<tr>
<td>C</td>
<td>M</td>
<td>51</td>
<td>Schizophrenia</td>
<td>22</td>
<td>5</td>
<td>5 months</td>
<td>Risperidone Clomipramine</td>
<td>1 year</td>
</tr>
<tr>
<td>D</td>
<td>M</td>
<td>40</td>
<td>Schizoaffective disorder</td>
<td>24</td>
<td>5</td>
<td>2 years</td>
<td>Lithium Melleril</td>
<td>4 months</td>
</tr>
<tr>
<td>E</td>
<td>M</td>
<td>59</td>
<td>Schizophrenia</td>
<td>23</td>
<td>0</td>
<td>-</td>
<td>Trifluperazine Procyclidine</td>
<td>1 year</td>
</tr>
<tr>
<td>F</td>
<td>M</td>
<td>29</td>
<td>Schizophrenia</td>
<td>8</td>
<td>6</td>
<td>1 year</td>
<td>Clozaril Carbamezepine Sulpindlle</td>
<td>8 months</td>
</tr>
<tr>
<td>G</td>
<td>M</td>
<td>40</td>
<td>Schizophrenia</td>
<td>19</td>
<td>3</td>
<td>13 years</td>
<td>Depixol Disipal Olanzepine Diazepam</td>
<td>10 months</td>
</tr>
</tbody>
</table>
Measures & Procedure

Each participant was asked to complete the following questionnaires at the start of the group, at the end of the group and at four-week follow-up:

- Knowledge Scale (Smith & Birchwood, 1987)(appendix 1) – a multiple-choice questionnaire aimed at assessing clients’ understanding of schizophrenia in terms of knowledge of appropriate terminology, symptoms, course and prognosis. Three questions applicable only to clients living with their families were removed
- Hospital Anxiety and Depression Scale (appendix 2) – Based on the original scale by Zigmond and Sniath (1983) but modified by Powell (1992) to be more user friendly.
- Views about schizophrenia scale (Gledhill et al., 1998) (appendix 3) – assesses satisfaction with having a diagnosis of schizophrenia and perceived understanding of schizophrenia.

A Symptom Rating Scale (Gledhill et al., 1998) (appendix 4) was introduced in the second week after an explanation of symptom monitoring and was completed each week thereafter and at follow-up. Participants were asked to identify a target symptom that was causing them the greatest distress. The Symptom Rating Scale was then used to assess the impact of the symptom - frequency, distress, preoccupation and conviction (only rated if target symptom was a delusion) and the person’s perceived ability to cope with and control the symptom.

A Satisfaction questionnaire (Gledhill, et al., 1998: plus one additional question)(appendix 5) was completed at the end of sixth session of the group.

Group Therapy Programme

The group consisted of six weekly meetings lasting for approximately one hour. It was run by a clinical psychologist and a clinical psychologist in training. Table 3 provides a summary of therapy sessions. At the end of each session, participants were given handouts containing summaries of the information covered in the session.
Table 3. Summary description of the group.

<table>
<thead>
<tr>
<th>Session</th>
<th>Content of session</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Introductions and ground rules. Aims of the group. Initial ratings.</td>
</tr>
<tr>
<td>2</td>
<td>What is schizophrenia? : Discussion on positive and negative symptoms and introduction to symptom monitoring. Causes of schizophrenia.</td>
</tr>
<tr>
<td>3</td>
<td>Treatment &amp; management of schizophrenia. Medication and psychological approaches.</td>
</tr>
<tr>
<td>4</td>
<td>Coping Strategies and Coping Strategy Enhancement. Self monitoring</td>
</tr>
<tr>
<td>5</td>
<td>Review of self monitoring and the effectiveness of strategies. Factors that influence coping. Early signs of relapse</td>
</tr>
<tr>
<td>Follow up</td>
<td>Follow up assessments and debriefing four weeks after the 6th session</td>
</tr>
</tbody>
</table>

Results

Attendance for the six therapy sessions was generally good with a seventy-two percent attendance rate. Two participants (A & D) missed one session and one participant missed two sessions (C). Two participants (F & G) only attended two sessions. Participant G had a history of non-attendance at the day centre. Participant F reported that he felt uncomfortable in a group setting and during the two sessions he attended, frequently had to leave the room. The data from these two participants were excluded from the analysis as only baseline data were available. Additional missing datum was due to participant A having to leave sessions early for depot injection appointments and participant C feeling unable to fill out the questionnaires due to the small sample size, it was not possible to run a statistical analysis on the data. Descriptive statistics are reported below and data on individual scores for each of the rating scales may be found in appendix 6.

Knowledge Scale

Overall, there was an increase in Knowledge Scale scores at the end of the group, but this gain was not maintained at follow-up. Mean Knowledge scale scores for the five
participants are shown in Table 4.

Table 4. Mean Knowledge Scale scores

<table>
<thead>
<tr>
<th>Knowledge Scale (n=5)</th>
<th>Pre mean</th>
<th>Post mean</th>
<th>Follow-up mean</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>13.8</td>
<td>15.4</td>
<td>11.8</td>
</tr>
</tbody>
</table>

For one participant (A) scores on the Knowledge Scale were worse post group and at follow up compared to pre group scores. For two of the participants (C & D), scores on the Knowledge Scale were lower at follow up than at baseline. This was marked for participant C, whose follow up score was ten points below that recorded at baseline. However, this gentleman reported that he was beginning to feel unwell at the time of follow up and staff also reported a deterioration in his mental state.

**Hospital Anxiety and Depression Questionnaire**

Scores for four participants are reported here as one participant (C) did not complete the questionnaire at follow-up. Means for anxiety and depression scores are shown in Table 5.

Table 5. Mean HADS scores

<table>
<thead>
<tr>
<th></th>
<th>Pre mean</th>
<th>Post mean</th>
<th>Follow-up mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>HADS – anxiety (n=4)*</td>
<td>10.5</td>
<td>8</td>
<td>8.25</td>
</tr>
<tr>
<td>HADS – depression (n=4)*</td>
<td>7.5</td>
<td>7</td>
<td>8.5</td>
</tr>
</tbody>
</table>

*a score of 8 or below indicates levels of anxiety or depression within the normal range*

Overall, there was a reduction in anxiety scores at the end of the group for three out of four participants. Of these three, two participants (B & E) reported an increase in anxiety at follow-up. However, for participant B anxiety levels were still below baseline levels and for participant E anxiety was still in the normal range (i.e. below 8 points). Participant (A) reported an increase in anxiety of one point during the group, but at follow-up anxiety had returned to baseline levels.
There was a small overall decrease in depression scores from pre group to post group. Only one participant showed an increase in depression levels post group. At follow-up, there was a general increase in depression scores with both participants B and D reporting higher depression scores than at follow-up.

**Symptom Rating Scale**

Means for four participants for which there is pre, post, and follow-up data is shown in Table 6. Data for participant C is not included, as he did not feel able to identify a symptom at the start of the group.

<table>
<thead>
<tr>
<th>Table 6. Mean Symptom Rating Scale scores</th>
<th>Pre mean</th>
<th>Post mean</th>
<th>Follow-up mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>Frequency (n = 4)</td>
<td>5.75</td>
<td>4.75</td>
<td>4.25</td>
</tr>
<tr>
<td>Distress (n = 4)</td>
<td>5</td>
<td>4.5</td>
<td>4.25</td>
</tr>
<tr>
<td>Preoccupation (n = 4)</td>
<td>4.5</td>
<td>4</td>
<td>4.5</td>
</tr>
<tr>
<td>Control * (n = 4)</td>
<td>2.75</td>
<td>4</td>
<td>3.25</td>
</tr>
<tr>
<td>Coping * (n = 4)</td>
<td>3</td>
<td>3.5</td>
<td>3.75</td>
</tr>
</tbody>
</table>

* high scores indicate better ability to control and cope with symptoms.

Two of the four participants (D & E) showed reductions in frequency and distress caused by their target symptoms at the end of the group which was maintained or further reduced at follow-up. Participant A showed a decrease in frequency and distress only at follow-up. Participant B reported no change on these dimensions and consistently rated both frequency and distress at the highest level.

Ratings for preoccupation with target symptoms changed the least over the group. However, Chadwick, Birchwood and Trower (1996) suggest that when clients are asked to actively work on their symptoms during CBT interventions one would expect preoccupation to change little or even increase (as with participant A) over the course of therapy.
The conviction rating only applied to one participant (A). There was no change in conviction rating from baseline to post group, but at follow-up she rated her conviction in her delusion had dropped one point.

On the coping dimension, there was an overall increase from pre to post group and a further increase at follow-up. This mainly reflects the marked increase in perceived ability to cope with the target symptom reported by participant A. The results for the other participants is more variable with participant B reporting no change, participant D reporting a one point increase and participant E reporting a one point drop from pre group level at follow-up. Ratings of perceived control over the target symptom showed an increase for baseline levels post group. Although there was a decrease in control at follow up, this was still above baseline levels. As with coping scores, the pattern of means for control reflects the changes in control for participant A. Participant B reported no change in coping or control. Participant D reported an increase in perceived coping during the group, but at post treatment, rated coping ability at the same level as baseline. However, there was a slight increase in coping at follow up. Participant E reported steady levels of coping through out the group, but reported a two point decrease in coping ability at follow up.

Views about Schizophrenia

One participant’s (B) views about schizophrenia changed from being “not happy” to feeling neutral. Three participant’s (C, D, E) views remained unchanged – two “not happy” and one “neutral”. One participant (A) showed a change from “not happy” to “neutral” during the group but this change was not maintained at follow up.

Three participants (B, D & E) reported an increase in perceived understanding of schizophrenia at follow up – two participants (B & D) understanding went from understanding “a little” to understanding “some” and one participant (E) went from understanding nothing to understanding “a little”. One client (C) showed no increase in understanding and perceived that he knew “little” throughout the group and at follow up. One participant (A) reported that she understood less about
schizophrenia. At baseline she perceived that she knew “quite a lot” but at post group and follow up this had changed to understanding “a little”.

One participant (A) remained unchanged in her understanding of her symptoms, one participant (B) showed an increase and one participant (D) reported a decrease in understanding at the end of the group, but at follow up reported levels of understanding similar to that recorded at baseline. Two participants (C&E) reported a decrease in understanding about their illness. This was especially marked for participant C and probably reflects the fact that he experienced a relapse at the end of the group.

**Satisfaction Questionnaire**

Tables 7a and 7b show data from the satisfaction questionnaire.

Table 7a. Satisfaction Questionnaire data

<table>
<thead>
<tr>
<th></th>
<th>Yes</th>
<th>No</th>
<th>Unsure</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. I found the group enjoyable</td>
<td>5</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>2. I feel that I benefited in some way by attending the group</td>
<td>5</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>3. There were some things about the way that the group was run that I did not like</td>
<td>1*</td>
<td>4</td>
<td>-</td>
</tr>
<tr>
<td>4. I feel that I benefited from meeting people who had similar problems to my own</td>
<td>5</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>5. I found it difficult to discuss my problems in the presence of others</td>
<td>3</td>
<td>2</td>
<td>-</td>
</tr>
<tr>
<td>6. I would have preferred to have been seen by a psychologist on my own</td>
<td>-</td>
<td>4</td>
<td>1</td>
</tr>
<tr>
<td>7. I feel more able to cope with my problems since attending the group</td>
<td>3</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>8. I would like to be involved in another group like this in the future</td>
<td>3</td>
<td>2</td>
<td>-</td>
</tr>
</tbody>
</table>

*One client commented that he would have liked more information about schizophrenia
Table 7b. Satisfaction Questionnaire data

<table>
<thead>
<tr>
<th>Rating</th>
<th>Count</th>
</tr>
</thead>
<tbody>
<tr>
<td>Very helpful</td>
<td>1</td>
</tr>
<tr>
<td>Helpful</td>
<td>4</td>
</tr>
<tr>
<td>Neutral</td>
<td>0</td>
</tr>
<tr>
<td>Unhelpful</td>
<td>0</td>
</tr>
<tr>
<td>Very Unhelpful</td>
<td>0</td>
</tr>
</tbody>
</table>

All participants reported that they had enjoyed the group, found it helpful and felt that they had benefited from it in some way. Several people said that they had found it difficult to talk about their problems in front of others, but said that they would not have preferred to see a psychologist on their own.

When asked what they found most helpful about the group, comments included

- “the amount of helpful information given out in the group”
- “learning about schizophrenia”
- “listening and talking to other people in the group”
- “talking about the symptoms of schizophrenia and medication”
- “hearing that other people had the same problems as me”
- “being able to join-in in a group that was interested in my problems

There were no aspects of the group that the participants did not like. However, several clients commented that a six-week group was too short and that they would have liked time to go into some to some of the topics in more detail.

Discussion

Overall, the results show the ‘Living with Schizophrenia’ group achieved it’s aim of reducing the impact of a targeted symptom, reducing anxiety and increasing individuals’ knowledge about schizophrenia at least for the duration of the group.
However, the areas in which improvements were found were variable for each participant and the group had least impact on ratings of depression.

Although the changes symptom impact, anxiety and knowledge were small they are comparable to findings of previous research on group CBT for psychosis (Gledhill et al., 1998). In addition, for people with a long history of psychosis even small changes in functioning may have a significant effect (Kuipers et al., 1997).

At follow-up the gains in reduction of symptom impact and increases in coping and control were overall maintained which suggests that participants continued to utilise techniques learnt in the group. Specific information about schizophrenia – as measured by the Knowledge Scale – did not appear to be retained, although three out of the five participants perceived their general understanding of schizophrenia had improved. A reduction in anxiety was maintained for only one participant.

Group treatment seemed to be an acceptable form of treatment as evidenced by the positive comments on the Satisfaction Questionnaire and from the comments made during the group. Although non-attendance and drop out are common problems in studies evaluating CBT for schizophrenia, the ‘Living with Schizophrenia’ group was overall well attended. All group members participated well and were able, at least to some extent, to share their experiences of psychosis with the group. This was particularly marked for participant A. She had been known to the service for two years but had previously said little about her positive symptoms. In the group she was able to verbalise her delusion and hallucinations. Other clients were also able to share information that they had previously not discussed which suggests that the group was a safe environment. This aspect of the group seemed to be especially helpful of participants. Only one client felt unable to tolerate group work, saying that he felt too anxious to participate.

The content and structure of the group seemed to be appropriate for the participants and led to lively discussions. This suggests that the topics chosen for the group were pertinent to the clients. Several group members commented that they would have liked to receive the information discussed in the group when they were diagnosed.
with schizophrenia / schizoaffective disorder and this may have helped them come to terms with their illness more easily. It is therefore worth considering targeting future ‘Living with Schizophrenia’ groups at people with recent onset psychosis.

In terms of the length of the group, there was a large amount of information covered in six hourly sessions. The view of participants and therapists was that a longer group might have been appropriate. This would have allowed the group to go into the material presented in more depth. It should also be noted that in trials of individual CBT, significant and lasting improvements in functioning were achieved with intensive therapy. For example, in one study (Kuipers et al., 1997) the average number of sessions was eighteen, with some clients receiving up to fifty sessions. However, due to service constraints it would not be possible to have a group of this length at the day centre. An alternative would be for future groups to focus on one component of CBT for psychosis, for example coping strategy enhancement or psychoeducation.

A disadvantage of group CBT for psychosis is that it does not allow for individualised formulation and intervention (Gledhill et al., 1998). If this had been possible it may have aided individuals’ understanding of their own particular symptoms and may have helped the application of coping strategies. For future groups, effectiveness may be improved by an extended individual assessment prior to the group. An alternative may be to use the group as an introduction to CBT principles i.e. monitoring and discussion of symptoms which could lead into individual work.

There are a number of criticisms that can be made about the design of the study. It was not possible, because of service constraints, to use a clinician rated tool to assess clients pre and post group. If this had been done, an objective measure of symptom severity could have been made. It was also not possible to include a control group in the evaluation. In addition, because the ratings were completed in sessions with the group facilitators there is the possibility of bias. However, comments from day centre staff about the functioning of clients supports the benefits reported by clients.
Despite these criticisms, this evaluation has demonstrated that the ‘Living with Schizophrenia’ group benefited the participants in terms of reducing the impact of a target symptom, increasing coping and control and understanding of schizophrenia. There were also short-term gains in knowledge and anxiety reduction. In addition, the group has an advantage over individual therapy in terms of therapist time and by providing the opportunity for peer support and normalisation of symptom experience.

Therefore, it would appear that the ‘Living with Schizophrenia’ group is potentially beneficial form of therapy and should continue to be offered to day centre clients. However, implementing the above recommendations may enhance the group.
References


Appendix 1
Knowledge About Schizophrenia

Below are some questions about the causes and treatment of schizophrenia. Please read each question and put a tick in the box next to the answer that you think is the right one.

1. Who can become schizophrenic?
   □ Anyone
   □ Men only
   □ People with personality disorders
   □ Criminals
   □ Don’t know

2. The usual age when the illness first attacks is:
   □ Anytime
   □ Middle Age
   □ In early twenties
   □ Childhood
   □ Don’t know

3. The chance of developing schizophrenia is:
   □ 1 in 1000
   □ 1 in 500
   □ 1 in 100
   □ 1 in 200
   □ Don’t know

4. If you are a child of someone who has schizophrenia the chances of you also having schizophrenia are:
   □ The same as anyone else
   □ Higher than anyone else
   □ Lower than anyone else
   □ A 99% possibility
   □ Don’t know

5. An attack of schizophrenia may be triggered by:
   □ A knock on the head
   □ Difficulties at birth
   □ Physical illness
   □ Stress
   □ Don’t know

6. Which of the following is most common in schizophrenia?
   □ To have one attack & recover completely
   □ To have several attacks but with periods of feeling better in between
   □ To be permanently ill with no periods of recover at all.
   □ To have one attack but not completely recover to what you were before
   □ Don’t know

Please turn to the next page
7. Which of the following do you believe are common symptoms in schizophrenia? 
(There is more than one correct answer here, so all the boxes you think are right)

- Hearing voices
- Lack of energy
- Incontinence
- Delusions
- Headaches
- Irritability
- Loss of appetite
- Lack of affection
- Sleep problems
- Overactivity
- Withdrawal
- Don’t know

8. Which of the following are negative symptoms of schizophrenia? 
(There is more than one correct answer here, so all the boxes you think are right)

- Hearing voices
- Withdrawal
- Lack of affection
- Lack of energy
- Thought disorder
- Delusions
- Irritability
- Don’t know

9. A positive symptoms of schizophrenia is:

- A symptom that is definitely due to schizophrenia and not due to anything else
- A symptom that is used to diagnose schizophrenia
- When something is added to a person’s normal behaviour
- When there is a loss from the person’s normal behaviour
- Don’t know

10. When schizophrenic symptoms reappear and get much worse this is called:

- Relapse
- Omission
- Remission
- Prolapse
- Don’t know

11. When a person with schizophrenia is admitted to hospital under a ‘section’ this means:

- Voluntary admission
- Compulsory admission
- Admission with the person’s consent
- Admission by the police
- Don’t know

Please turn to the next page
12. The average length of stay in hospital for a first attack of schizophrenia is:
- 3-6 weeks
- 6 months
- 12 weeks
- One year
- Don’t know

13. Medication can help reduce (remove symptoms) in what percentage of patients
- 25% (quarter)
- 75% (three quarters)
- 50% (half)
- 100% (all)
- Don’t know

14. The main medication given to remove schizophrenic symptoms are:
- Antihistamines
- Narcotics
- Neuroleptics
- Tranquillisers
- Don’t know

15. If a schizophrenic patient is taking their medication, the risk of getting a 2nd attack of schizophrenia within one year is reduced from 75% to –
- 70%
- 50%
- 10%
- 30%
- Don’t know

16. Rehabilitation is a word for –
- Giving medication
- Helping a person to settle back to normal life out of hospital
- Helping the person to find accommodation
- Hospital treatment
- Don’t know

17. Medication is more effective for:
- Positive symptoms
- Negative symptoms only
- All symptoms equally
- Mainly negative symptoms
- Don’t know

Please turn to the next page
18. Which of the following are often associated with the onset of schizophrenia?
(There is more than one correct answer here, so all the boxes you think are right)
- Too much stress
- Poor diet
- Inability to get angry and express your feelings directly
- Runs in the family
- Biological problems, body chemicals
- Personality type – just the kind of person
- A split personality
- Family problems as a child
- An upsetting experience, for example the death of an important person.
- Don’t know

19. To help a person recover from schizophrenia the family / friends / staff should try to:
(There is more than one correct answer here, so all the boxes you think are right)
- Leave the person alone
- Try to get him / her to do things for themselves
- Do as much for the person as possible
- Encourage him / her to mix with other people
- Let the person do what they want
- Not burden the person with housework
- Ensure that he / she takes their medication
- Don’t know

20. Which of the following are unhelpful to a person with schizophrenia
(There is more than one correct answer here, so all the boxes you think are right)
- Too much pressure on the person
- Nagging by the family
- Sitting around all day
- Stopping taking medication
- Giving the person responsibility
- Treating the person like an adult
- Don’t know

21. If you notice side effects of the medication that you are taking you should
- Wait to see if the side effects go away
- Ask a doctor’s advice
- Come off medication altogether
- Take a lower dose of the medication than prescribed
- Don’t know
Appendix 2

Hospital Anxiety & Depression Scale

Please indicate how you are feeling now, or how you have been feeling in the last day or two, by ticking the column to the right of each of the following statements:

<table>
<thead>
<tr>
<th></th>
<th>Yes definitely</th>
<th>Yes sometimes</th>
<th>No, not much</th>
<th>No, not at all</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>I wake early and then sleep badly for the rest of the night.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>I get very frightened or have panic feelings for apparently no reason at all.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>I feel miserable and sad.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>I feel anxious when I go out of the house on my own.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>I have lost interest in things.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>I get palpitations, or a sensation of 'butterflies' in my stomach or chest.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>I have a good appetite.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>I feel scared or frightened.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>I feel life is not worth living.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>I still enjoy the things I used to.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>I am restless and can't keep still.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>I am more irritable than usual.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>13</td>
<td>I feel as if I have slowed down.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>14</td>
<td>Worrying thoughts constantly go through my mind.</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

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Appendix 3

Views about Schizophrenia

1. How do you feel about having a diagnosis of schizophrenia?
(Please tick one box)

- very unhappy
- not happy
- neutral
- happy
- very happy

2. How much do you understand about schizophrenia?
(Please tick one box)

- nothing
- a little
- some
- quite a lot
- a lot

3. Do you feel that you understand your illness / symptoms / experiences
(Please tick one box)

- not a lot
- a little
- some
- quite a lot
- a lot
Appendix 4
Symptom Rating Scale

Name: ____________________________ Date: ____________________________

Main symptom / experience ____________________________________________

With respect to your main symptom over the last week, please rate the following:

1. **Frequency**: How often have you experienced it?

   
   
   
   
   
   
   
   1  2  3  4  5  6  7
   
   not at all                              all the time

   What is your explanation for it?

2. **Conviction**: How much do you believe this?

   
   
   
   
   
   
   
   1  2  3  4  5  6  7
   
   not at all                              extremely so

3. **Distress**: How much distress does it cause you?

   
   
   
   
   
   
   
   1  2  3  4  5  6  7
   
   none at all                             extreme

   amount

Please turn to the next page
4. **Preoccupation**: How preoccupied by it are you?

1  2  3  4  5  6  7

not at all          all the time

5. **Control**: How much control do you feel you have over it?

1  2  3  4  5  6  7

None              complete
At all

6. **Coping**: How much have you felt able to cope with it?

1  2  3  4  5  6  7

not at all          completely
Appendix 5

Satisfaction Questionnaire

Thank you for coming to the Living with Schizophrenia group at...

It would be helpful if we could have some feedback on how you found it. Please complete the following questions by circling the most appropriate answer.

1. I found the group enjoyable
   Yes / No

2. I feel that I benefited in some way by attending the group
   Yes / No

3a. There were some things about the way that the group was run that I didn't like
   Yes / No

3b. If you answered ‘yes’ to the above question, what was it that you did not like?

4. I feel that I benefited from meeting people who had similar problems to my own
   Yes / No

5. I found it difficult to discuss my problems in the presence of others
   Yes / No

6. I would have preferred to have been seen by a psychologist on my own
   Yes / No

7. I feel more able to cope with my problems since attending the group
   Yes / No

8. I would like to be involved in another group like this in the future
   Yes / No

Please turn to the next page...
9. Which part of the group did you find most helpful?

10. Overall, how helpful did you find the group?
    (please tick one box)
    
    - Very helpful □
    - Helpful □
    - Neutral □
    - Unhelpful □
    - Very Unhelpful □

11. Are there any other comments that you would like to make about the group?  
(Please use the back of the sheet of you do not have enough room)
Appendix 6

Graph of Knowledge Scale Scores for all participants at baseline, post treatment and follow-up

Graph of HADS Anxiety Scores for all participants at baseline, post treatment and follow-up

Graph of HADS Depression Scores for all participants at baseline, post treatment and follow-up

Missing data = X
Appendix 6

Data on views about having a diagnosis of schizophrenia

<table>
<thead>
<tr>
<th>Participant</th>
<th>Baseline</th>
<th>Post</th>
<th>Follow-up</th>
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<tbody>
<tr>
<td>A</td>
<td>not happy</td>
<td>neutral</td>
<td>not happy</td>
</tr>
<tr>
<td>B</td>
<td>not happy</td>
<td>neutral</td>
<td>neutral</td>
</tr>
<tr>
<td>C</td>
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<td>not happy</td>
<td>not happy</td>
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<tr>
<td>D</td>
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<td>neutral</td>
<td>neutral</td>
</tr>
<tr>
<td>E</td>
<td>not happy</td>
<td>not happy</td>
<td>not happy</td>
</tr>
<tr>
<td>F</td>
<td>very unhappy</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>G</td>
<td>neutral</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

Graph showing how well schizophrenia understood by participants

Graph showing how well participants understood their own symptoms

missing data = X
Appendix 6

Graph of Symptom Rating Scale scores for Participant A
Target symptom: Believing that God had made her into a god

- **Frequency**
  - Extreme: 7
  - Amount: 6
  - Sessions: 2, 3, 4, 5, 6, follow-up

- **Distress**
  - Extreme: 7
  - Amount: 4
  - Sessions: 2, 3, X, X, 6, follow-up

- **Conviction**
  - Extreme: 7
  - Amount: 4
  - Sessions: 2, 3, X, X, 6, follow-up

- **Preoccupation**
  - All the time: 7
  - Amount: 4
  - Sessions: 2, 3, X, X, 6, follow-up

- **Coping**
  - Completely: 7
  - How able to cope with symptom: 5
  - Sessions: 2, 3, X, X, 6, follow-up

- **Control**
  - Complete: 7
  - Amount: 4
  - Sessions: 2, 3, X, X, 6, follow-up

Missing Data = X
Appendix 6

Graph of Symptom Rating Scale scores for Participant B
Target Symptom: Communication problems & isolation

Frequency

Distress

Preoccupation

Coping

Control

Missing Data = X
Appendix 6

Graph of Symptom Rating Scale scores for Participant C

Target symptom: Feeling out of touch with God

Missing Data = X
Appendix 6

Graph of Symptom Rating Scale scores for Participant D
Target Symptom: Concentration problems

- Frequency
- Distress
- Preoccupation
- Coping
- Control
Appendix 6

Graph of Symptom Rating Scale scores for Participant E
Target Symptom: Hearing Voices telling him to hurry up

Frequency

<table>
<thead>
<tr>
<th>Frequency</th>
<th>Sessions</th>
<th>Follow up</th>
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</thead>
<tbody>
<tr>
<td>All the time</td>
<td>7</td>
<td></td>
</tr>
<tr>
<td>How often symptom experienced</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Not all</td>
<td>2</td>
<td>3</td>
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Distress

<table>
<thead>
<tr>
<th>Distress caused by symptom</th>
</tr>
</thead>
<tbody>
<tr>
<td>Extreme amount</td>
</tr>
<tr>
<td>Not at all</td>
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Preoccupation

<table>
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<tr>
<th>Preoccupation</th>
<th>Sessions</th>
<th>Follow up</th>
</tr>
</thead>
<tbody>
<tr>
<td>All the time</td>
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<td></td>
</tr>
<tr>
<td>How preoccupied by symptom</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Not at all</td>
<td>2</td>
<td>3</td>
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Coping

<table>
<thead>
<tr>
<th>How able to cope with symptom</th>
</tr>
</thead>
<tbody>
<tr>
<td>Complete</td>
</tr>
<tr>
<td>Not at all</td>
</tr>
</tbody>
</table>

Control

<table>
<thead>
<tr>
<th>Control over symptom</th>
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</thead>
<tbody>
<tr>
<td>Complete</td>
</tr>
<tr>
<td>Not at all</td>
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</table>

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Appendix 6

Graph of Symptom Rating Scale scores for Participant F
Target Symptom: Anxiety

Frequency

Distress

Preoccupation

Coping

Control

Missing Data = X
Appendix 6

Graph of Symptom Rating Scale scores for Participant G
Target Symptom: Hearing voices - critical comments from his family

Frequency

<table>
<thead>
<tr>
<th>Sessions</th>
<th>All the time</th>
<th>Not at all</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>7</td>
<td>1</td>
</tr>
</tbody>
</table>

Distress

<table>
<thead>
<tr>
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<th>None at all</th>
<th>Some at all</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>6</td>
<td>1</td>
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Preoccupation

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<tr>
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<th>Not at all</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>6</td>
<td>1</td>
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</table>

Coping

<table>
<thead>
<tr>
<th>Sessions</th>
<th>Not at all</th>
<th>Completely</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>2</td>
<td>1</td>
</tr>
</tbody>
</table>

Control

<table>
<thead>
<tr>
<th>Sessions</th>
<th>Not at all</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>2</td>
</tr>
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</table>

Missing Data = X
CONFIDENTIAL

25th June 1999

Claire Monteith
Clinical Psychologist in Training
c/o PsychD in Clinical Psychology
University of Surrey
Guildford
Surrey

Dear Claire

Re: 'Living with Schizophrenia' Presentation

Just a quick note to thank you for the presentation that you gave to Willow House staff earlier today on the evaluation of the above group that you co facilitated here earlier this year. The presentation was well received by staff and generated useful discussion afterwards.

Thank you once again

Best Wishes

Amanda Ediriweera
Clinical Psychologist
Literature Review

Cognitive models of posttraumatic stress disorder – A review

August 2000

Year 2
Introduction

It has long been recognised that exposure to traumatic events can lead to problematic behavioural and emotional reactions. These reactions have been documented throughout history by writers including Samuel Pepys and Charles Dickens (Turnbull, 1998), but it was not until the late 19th century that the impact of trauma received attention in the psychological literature (Jones & Barlow, 1990). Psychological difficulties following trauma were then described using numerous labels including ‘nervous shock’, ‘shell shock’ and ‘survivor syndrome’ that suggested varying sets of symptoms according to the type of event experienced (Foa, Steketee & Rothbaum, 1989). However, with the publication of the classification system DSM-III (American Psychiatric Association, 1980) the similarity of symptoms after exposure to a range of traumatic events was recognised (Foa et al., 1989) and the term ‘posttraumatic stress disorder’ was established.

Posttraumatic stress disorder (PTSD) is now acknowledged as a common reaction to traumatic events such as war, assault, disaster, accidents (Ehlers & Clark, 2000) and life threatening illness (Mayou & Smith, 1997). DSM-IV (American Psychiatric Association, 1994) defines the symptoms of PTSD as: (1) persistent re-experiencing of the trauma, for example intrusive thoughts, images and dreams; (2) persistent avoidance of stimuli associated with the event including places and activities and emotional numbing and; (3) persistent symptoms of increased arousal for example sleep disturbance and difficulties concentrating. These symptoms must be present for more than one month and PTSD is termed ‘acute’ if symptom duration is less than three months and ‘chronic’ if symptoms persist for more than three months.

Both acute and chronic PTSD are relatively common in the general population with estimates of lifetime prevalence ranging from four to twelve percent (Yule, Williams & Joseph, 1999). However, in specific populations for example, war veterans and disaster survivors PTSD prevalence may be as high as fifty percent (Yule et al., 1999). In addition, PTSD symptoms are not only distressing but may also have a profound impact on an individual’s life. Chronic PTSD sufferers commonly report difficulties with social relationships, an increase in physical health problems, substance abuse and high levels of other psychiatric disorders for example,
depression (Yule et al., 1999). Therefore, there is a need for researchers and clinicians to understand how and why PTSD develops, not only to aid assessment and treatment techniques, but also to inform strategies to prevent the development of PTSD after experiencing a traumatic event.

**Issues that need to be addressed by etiological models of PTSD**

Central to any etiological model of PTSD must be an explanation of the three main symptom clusters of re-experiencing phenomena, avoidance and hyperarousal (Dalgleish, 1999). However, there are several other important aspects of PTSD that must be accounted for. Firstly, Jones and Barlow (1990) point out that PTSD is one of the few psychiatric disorders where the trigger for the development of symptoms is clearly identifiable. That is “witnessing or experiencing an event where there is actual or threatened death or injury and where the person responded with intense fear or horror” (American Psychiatric Association, 1994). However, the development of PTSD is more complex than simply being exposed to traumatic event. For example, not everyone who has experienced a car accident will develop PTSD (Jones & Barlow, 1990). How can the absence of PTSD be explained in individuals who have experienced similar traumas to those who have developed PTSD? Secondly, etiological models need to explain why some individuals initially cope very well in the aftermath of trauma only to develop PTSD months or years after the event – so called delayed onset PTSD (Dalgleish, 1999). Thirdly, Steil and Ehlers (2000) suggest that the occurrence of PTSD symptoms in the immediate aftermath of a traumatic event can be seen as a normal adaptation process. If this is the case, how and why do these symptoms persist and become maladaptive? Fourthly, research has demonstrated that a number of variables such as the level of social support following trauma, a history of psychological problems and prior experience of trauma can influence development of PTSD (Dalgleish, 1999). Etiological models need to explain how these factors serve to exacerbate or protect against the development of PTSD. Lastly, theories need to be able to suggest how treatment approaches may be implemented (Dalgleish, 1999).
Towards a cognitive approach to PTSD

Given the complexity of PTSD, numerous theories have been developed, for example, biological models (Van der Kolk, Boyd, Krystal & Greenberg, 1984: Kolb, 1987) and behavioural models (Keane, Zimmerling & Caddell, 1985). However, both biological models and behavioural models currently fail to explain key aspects of PTSD. For example, biological models fail to explain the significance of factors such as social support in the development of PTSD (Jones & Barlow, 1990). While behavioural models do not explain the occurrence of re-experiencing symptoms that are central to PTSD (Foa et al., 1989). In addition, both behavioural and biological models have difficulty in accounting adequately for individual differences in reactions to trauma (Jones and Barlow, 1990). Indeed, this is one of the most problematic areas for models of PTSD (Dalgleish, 1999).

In order to address the problem of differing reactions to trauma, several authors have drawn parallels between PTSD and what is known about the role of cognitive factors in other anxiety disorders (Jones & Barlow, 1990: Ehlers & Steil, 1995). For example, the meaning that individuals' attach to events has been shown to be involved in the development and maintenance of panic disorder (Clark, 1989) and obsessive-compulsive disorder (Salkovskis, 1985). Therefore, Ehlers and Steil (1995) suggest that how an individual perceives a traumatic event and the meaning it has, may also play a role in the development and maintenance of PTSD.

Several cognitive models of PTSD have been developed and Brewin, Dalgleish and Joseph (1996) argue that the cognitive approach to PTSD is the “most fully developed and offers the greatest explanatory and predictive power” (pg. 673). This review will attempt to demonstrate how cognitive models of PTSD have evolved. Throughout the review, the focus of the discussion will be the theoretical aspects of the models presented and how well the models explain some of the key features of PTSD. The research base supporting cognitive models of PTSD will be discussed in the introduction to the major research project, where relevant.
Before reviewing cognitive models, research highlighting the role of cognitive factors in PTSD will be discussed, as all cognitive models suggest that such factors are central to understanding responses to trauma.

**Cognitive factors involved in the development of PTSD**

*Uncontrollability and Unpredictability*

Several authors suggest that perceived uncontrollability and unpredictability of trauma is important in the development of PTSD (e.g. Jones and Barlow, 1990). According to attribution theory, people need to be able to predict the future and to perceive some control over events (Joseph, 1999). Thus "for the victim of an uncontrollable, unpredictable important stressor, daily life is marked by a sense of impending doom, because threat pertinent to his or her survival can neither be predicted or controlled" (Foa, Zinbarg & Rothbaum, 1992 pg. 231). Perceived uncontrollability and unpredictability of trauma has been demonstrated to be associated with PTSD severity in several studies. For example, in a retrospective study, victims of physical or sexual assault with PTSD were more likely to report feeling out of control during the assault than victims without PTSD (Dunmore, Clark and Ehlers, 1999). In addition, prospective studies of individuals involved in a nuclear accident (Baum, Cohen & Hall, 1993: cited in Ehlers & Steil, 1995) and women following childbirth (Czarnocka & Slade, 2000) also found an association between perceptions of control and PTSD severity.

*Perceived Threat to Life*

Similarly, the perceived threat of the trauma is thought to play a role in predicting PTSD severity and may be at least as important as the actual danger posed by an event (Foa et al., 1992). Perceived threat to life has been found to be associated with PTSD in retrospective studies of rape victims (Kilpatrick et al., 1989: cited in Ehlers & Steil, 1995), survivors of assault (Dunmore, et al., 1999) and road traffic accident victims (Blanchard, Hickling, Mitnick, Taylor, Loss & Buckley, 1995; Steil & Ehlers, 2000). Perceived threat to life was also associated with PTSD severity in a prospective study of assault victims (Dunmore, Clark & Ehlers, in press). Interestingly, there is also evidence to suggest that perceptions of threat may change over time and this can influence PTSD symptoms. For example, Foa et al. (1989)
describe how a rape victim developed PTSD only after learning that the perpetrator killed his next victim.

**Causal Attributions**

Joseph (1999) suggests that when individuals are faced with uncontrollable, unpredictable and life threatening events, they are motivated to develop an understanding of why the trauma happened to them. However, explanations of the cause of the trauma developed by trauma survivors may be linked with the development of PTSD. Ehlers and Steil (1995) suggest that individuals who attribute the trauma to internal causes (i.e. something that they did) are more likely to experience PTSD. Joseph, Brewin, Yule and Williams (1991) investigated the role of causal attributions in disaster survivors. Internal attributions for trauma were more significantly correlated with PTSD symptoms compared to external attributions. Internal causal attributions for trauma have also been found to be associated with PTSD in survivors of child sexual abuse (Wenniger & Ehlers, 1998) and victims of sexual and physical assault (Dunmore, Clark & Ehlers, 1997,1999).

**Global Beliefs**

As well as investigating individuals' appraisals of traumatic events, researchers have also examined the role of more global beliefs in PTSD. Several authors have suggested that PTSD may develop if trauma changes an individual's basic assumptions about the fairness and safety of the world and leads them to hold more negative beliefs (Janoff-Bulman & Frieze, 1983: Foa et al., 1989). In addition, Foa and Riggs (1993) suggest that trauma may also confirm an individuals pre-existing negative beliefs. The role of negative global beliefs in PTSD has been demonstrated in several studies. For example, PTSD symptoms were associated with negative beliefs concerning safety, trust and self-esteem in two samples of child sexual abuse survivors (Wenniger & Ehlers, 1998). Similarly, Dunmore et al. (1997, 1999 and in press) found that global, negative beliefs were associated with persistent PTSD and there was evidence to suggest that trauma can act to both shatter and confirm previous beliefs.
Appraisals of Trauma Sequelae

Studies have shown that the distress caused by PTSD symptoms varies between individuals and it is suggested that this is because people attach different meanings to PTSD symptoms (Ehlers & Steil, 1995). In particular, Ehlers and Steil (1995) suggest that if an individual interprets the occurrence of intrusive recollections negatively, for example, “I am going crazy”, they will be more likely to develop PTSD. Negative interpretations of symptoms of PTSD have been demonstrated to play a role in PTSD after road traffic accidents (Ehlers & Steil, 2000) assault (Dunmore, Clark & Ehlers, 1997, 1999, in press), and in ambulance personnel (Clohessy & Ehlers, 1999).

In addition, appraisals of the consequences of the trauma have been demonstrated to be involved in the development of PTSD. The perception that trauma has caused permanent change, for example beliefs such as ‘My life has been destroyed’, have been investigated in assault victims (Dunmore et al., 1999, in press) and former political prisoners (Ehlers, Maercker & Boos, 2000). In these studies, perceived permanent change was significantly associated with PTSD. Perceptions of permanent change have also been found to be associated with poorer outcome following exposure therapy for PTSD (Ehlers, Clark, Dunmore, Jaycox, Meadows, & Foa, 1998). Nonetheless, Ehlers et al. (2000) acknowledge that many survivors of trauma experience actual permanent change, for example chronic injury. However, studies have compared survivors with and without PTSD, suggesting that perceived permanent change contributes to the development of PTSD over and above actual change in life circumstances (Ehlers et al., 2000).

A third aspect of trauma aftermath that has been investigated is how survivors of trauma perceive the reactions of significant others. Several studies suggest that level social support may be an important factor in reducing the impact of trauma (e.g. Kilpatrick, Veronen & Best, 1985). However, perceived social support may be more important than actual levels of social support available. For example, Dunmore et al. (1997, 1999, in press) found that negative perceptions of other people’s reactions e.g. “I feel like other people are ashamed of me now” were associated with PTSD.
Coping Strategies

How an individual copes with the symptoms of PTSD may also influence the development and maintenance of the disorder (Jones & Barlow, 1990). Maladaptive coping strategies associated with PTSD include not talking or thinking about the trauma, rumination about the trauma, safety behaviours, drug and alcohol abuse and suppressing thoughts about trauma (Ehlers & Steil, 1995). It is suggested that these types of coping behaviours serve to maintain PTSD symptoms because they prevent the individual from reassessing the trauma and changing their maladaptive beliefs (Ehlers & Steil, 1995). In addition, several coping strategies actually have the effect of increasing the frequency of symptoms. For example, Shipherd and Beck (1999) found that thought suppression significantly increased the frequency of intrusive thoughts in survivors of sexual assault with chronic PTSD compared to those without PTSD. Dunmore et al. (1997, 1999, in press), and Clohessy and Ehlers (1999) have also demonstrated the association between a variety of dysfunctional coping strategies and PTSD.

Cognitive Models of PTSD

The evidence presented above suggests that cognitive factors have an important role to play in the development and maintenance of PTSD. This review will now consider how such factors have been incorporated into cognitive models of PTSD.

Janoff-Bulman's Shattered Assumptions Theory

Janoff-Bulman’s shattered assumptions theory (Janoff-Bulman & Frieze, 1983; Janoff-Bulman, 1989) suggests that ordinarily people make sense of their experiences using a set of basic assumptions about themselves and the world. These basic assumptions consist of beliefs about personal invulnerability, the perception of the world as meaningful and a positive view of the self (Janoff-Bulman & Frieze, 1983). During the course of a lifetime, basic assumptions about the self and the world undergo change as new information is encountered. These changes are usually gradual and as such do not present a threat to the individual’s belief system (Janoff-Bulman, 1989). However, it is suggested that trauma forcibly and drastically challenges an individual’s basic assumptions and this causes intense stress and
anxiety. In order to minimise distress the individual must try to either incorporate the new information provided by the trauma into their pre-existing assumptions or revise their beliefs to 'fit' (Janoff-Bulman, 1989). In order to accomplish this task, individuals may use a number of strategies including self-blame, re-interpretations of the event and denial (Janoff-Bulman & Frieze, 1983). Janoff-Bulman also incorporates the earlier work of Horowitz (1982) and suggests that recurrent intrusive thoughts are also a mechanism by which individuals attempt to process trauma information. These strategies may therefore be viewed as adaptive and minimise the likelihood of psychological breakdown (Janoff-Bulman, 1989). However, for some individuals thinking about the trauma and experiencing intrusive thoughts is too distressing. Therefore, they alternate between processing the trauma and avoidance. This means that they are unable to successfully incorporate the new information provided by trauma and PTSD develops (Janoff-Bulman, 1992).

Janoff-Bulman’s shattered assumptions theory represents an important step forward in our understanding of PTSD and the role that prior beliefs play. However, there are a number of problems with this model. Dalgleish (1999) points out that the model does not explain how core assumptions about the self and the world are shattered. In addition, Dalgleish (1999) raises the question of why the three assumptions highlighted by Janoff-Bulman are central, when there are presumably a range of different beliefs that may be affected by trauma. A third problem for the model is the finding that individuals with PTSD are more likely to have a premorbid history of psychological problems (Kilpatrick et al., 1985). Research suggests that dysfunctional beliefs about the self and the world are also prominent in other psychological problems (Beck, 1976). If individuals already hold assumptions about personal vulnerability and negative self-perception, how can the shattering of assumptions be involved in the development of PTSD for these individuals (Dalgleish, 1999)?

**Foas Information Processing Theory**

Foa and her colleagues have developed an information processing theory of PTSD that draws on Lang’s (1977) theory of fear. Lang’s theory suggests that information about feared stimuli is stored in a network in long term memory. This network holds
information about: (1) the feared situation; (2) verbal, physiological and behavioural responses to fear and; (3) the meaning of the stimulus and responses and how these are linked (Foa et al., 1989). This network of information is seen as a ‘program’ for escape and avoidance behaviour (Foa et al., 1989: Dalgleish, 1999). Also stored in the fear network is information about the danger and threat posed by stimuli (Foa et al., 1989). Foa et al. (1989) argue that in PTSD, stimuli and responses become stored in the fear network when basic concepts of safety are violated and the world is seen as less predictable or controllable. Because of this change in beliefs about safety, the fear structure involved in PTSD includes information about a large number of stimuli (Foa et al., 1989). Activation of the fear network, by exposure to reminders of the trauma, causes the symptoms of PTSD such as heightened arousal and re-experiencing of the trauma. Avoidance strategies are then used to suppress the fear generated. Foa et al. (1989) argue that adjustment to trauma may only be achieved if the information in the fear network is incorporated into existing memory. For this to happen all the components of the fear memory must be activated and new information that is incompatible with the fear network must be incorporated. However, as the fear network in PTSD is large and sufferers avoid stimuli that activate it, the fear network is difficult to dismantle and symptoms persist (Foa et al., 1989). In addition, Foa and Riggs (1993) suggest several factors that may explain why PTSD persists in some individuals. These are the individual’s beliefs about the world and self prior to trauma, the disorganised nature of the trauma memory and post trauma experiences. These factors work together to impede processing of the trauma. For example, post trauma misinterpretations of PTSD symptoms or the reactions of others may feed into and maintain the fear network (Jaycox & Foa, 1996).

A strength of Foa’s information processing theory is that it incorporates and expands upon the ideas Janoff-Bulman (1989). In addition, it incorporates many of the cognitive factors that have been highlighted as important in the development and maintenance of PTSD, such as perceived uncontrollability and unpredictability and causal attributions. There is also an attempt to explain individual differences in reaction to trauma. However, the theory does not fully explain how new information
may be incorporated into the fear network (Dalgleish, 1999) and delayed onset PTSD (Jones & Barlow, 1990).

**Cognitive Action Theory**

The cognitive action theory of Chemtob, Roitblat, Hamada, Carlson and Twentyman (1988) also draws on the work of Lang (1977). Similar to Foa’s theory, Chemtob et al. (1988) suggest that information about the world (thoughts, images, actions, emotions and behaviours) is stored and processed in ‘networks’. Networks consist of ‘nodes’ that carry information and these nodes are linked with each other hierarchically. Thus, nodes that carry more simple information are lower down the network than nodes that carry more sophisticated, abstract information. Chemtob et al. (1988) suggest that learning takes place when new nodes are formed or when new connections between nodes are made. Chemtob et al. (1988) use this the concept of networks to describe a model PTSD based on research with war veterans. They suggest that during combat individuals establish memory networks consisting of information about threat, threat expectancy and responses to threat. This network is adaptive in a combat situation as it promotes survival, however it is maladaptive in non-combat situations. Chemtob et al. (1988) suggest that it is the preservation of these threat-related networks outside combat situations that is responsible for the development of symptoms of PTSD. In addition, a number of characteristics of the networks of people with PTSD contribute to the maintenance of the disorder by increasing the ease with which the network is activated.

Chemtob et al.’s (1988) cognitive action theory provides a detailed account of the structure and workings of the fear network central to information processing theories of PTSD. However, it is somewhat limited as a model of PTSD because of its emphasis on PTSD following combat (Jones & Barlow, 1990). In addition, cognitive action theory does not specify why some soldiers develop PTSD and not others (Dalgleish, 1999). In addition, the theory does not incorporate cognitive factors or other important variables such as social support (Dalgleish, 1999).
**Dual Representation Theory**

The dual representation theory of PTSD (Brewin, Dalgleish & Joseph, 1996) combines elements from both Janoff-Bulman and information processing theories. The theory proposes that trauma information is stored in two types of memory. 'Verbally Accessible Memories' (VAM's) are retrievable memories and contain information similar to that stored in the fear network proposed by Foa et al. (1989). For example, sensory, response and meaning information about the trauma (Dalgleish, 1999). 'Situationally Accessible Memories' (SAM's) contain detailed sensory and physiological information about the trauma that cannot be deliberately accessed by the individual and are only activated when triggered by stimuli associated with the traumatic event. Both VAM and SAM are involved in producing PTSD symptoms and therefore, emotional processing of both VAM and SAM memories are necessary for successful adjustment to trauma. In order to do this Brewin et al. (1996) argue that individuals must integrate the information available via VAM's with their pre-existing beliefs (similar to Janoff-Bulman). To modify the content of SAM system, Brewin et al. (1996) suggest that new non-threatening information must be integrated with the trauma memories and this can only happen when the SAM system is activated (similar to Foa). If an individual is able to successfully process the information in both the VAM and SAM systems, it is argued that PTSD will not develop (Brewin et al., 1996). Several factors may aid successful processing for example, small discrepancies between trauma information and prior assumptions, good social support and an ability to tolerate initial symptoms (Brewin et al., 1996). However, if emotional processing is not complete, PTSD will develop. Brewin et al. (1996) suggest a variety of reasons why processing may be hindered. These include large discrepancies between trauma information and prior assumptions, poor social support and an inability to prevent intrusions of SAM's into consciousness. Brewin et al. (1996) also incorporate the findings of Ehlers and Steil (1995) and suggest that misinterpretations of PTSD symptoms may also lead to a failure of emotional processing because they maintain negative beliefs about the self and the world. In addition, delayed onset PTSD is explained by proposing that emotional processing of trauma information may be inhibited by avoidance. However, the information remains available and may be reactivated at a later date (Brewin et al., 1996)
Dalgleish (1999) argues that the strength of dual representation theory of PTSD is that it incorporates many of the cognitive factors of known importance in PTSD and builds on previous models. However, the theory does not appear to fully specify how new information is integrated with the trauma memories (Dalgleish, 1999).

**Ehlers and Clark’s Cognitive Model of PTSD**

It can be seen that as the cognitive approach to PTSD has developed, cognitive theories have become more sophisticated and each new cognitive model of PTSD provides fresh insights into the nature of the disorder. However, models of PTSD proposed by Janoff-Bulman (1989), Foa et al. (1989), Chemtob et al. (1988) and Brewin et al. (1996) fail to provide sufficient detail regarding the cognitive factors and processes involved in the development and maintenance of the disorder.

A cognitive model of PTSD developed by Ehlers and Clark (2000) draws and expands on the theories described above in an attempt to synthesise current knowledge about the role of cognitive factors in PTSD. Ehlers and Clark (2000) point out that with all other anxiety disorders, cognitive models suggest that anxiety is related to some future threat. However, in PTSD distress is linked to an event that has already happened. Therefore, Ehlers and Clark (2000) argue that PTSD occurs if an individual processes the traumatic event in such a way that leads to a sense of current threat. Ehlers and Clark (2000) propose that a sense of current threat is generated by several processes. Firstly, individuals who develop PTSD think about the trauma and its sequelae in excessively negative terms. Ehlers and Clark (2000) identify several types of negative appraisals: (1) Negative appraisals of the traumatic event and the actions taken during the event, for example ‘It was my fault’, ‘bad things always happen to me’; (2) Negative appraisals of the trauma sequelae including initial PTSD symptoms, other people’s reactions and the consequences of the trauma for example ‘I will never be able to lead a normal life again’. Such appraisals produce negative emotions such as anxiety, depression and anger.

Secondly, Ehlers and Clark (2000) suggest that the development of PTSD is linked to the way in which the trauma is encoded in memory. It is argued that in normal
processing, information is incorporated into autobiographical memory and organised by theme and time period. This means that memories can be intentionally recalled, but that involuntary recollections are less likely. However, Ehlers and Clark (2000) suggest that in PTSD, the trauma memory is poorly elaborated and not integrated with other autobiographical memories. Thus, individuals have difficulty in intentionally recalling aspects of the traumatic event but experience frequent involuntary intrusions. In addition, Ehlers and Clark (2000) suggest that during trauma certain stimuli, for example sound and smell, become associated with danger. These associations become encoded in the trauma memory and mean that re-experiencing symptoms are easily triggered by physical cues. Individuals may not be aware of what the triggers are and this makes it difficult to learn that such triggers no longer signal danger.

Ehlers and Clark (2000) also suggest several factors that cause PTSD to persist. Firstly, the nature of the trauma memory and the appraisals of the trauma interact to maintain each other and the sense of threat that they generate. For example, Ehlers and Clark (2000) suggest that when individuals with PTSD recall the trauma, their memories are coloured by their excessively negative appraisals of the event and details of the event that confirm their appraisals are selectively attended to. Alternatively, the fragmented nature of the memory for the traumatic event may itself be interpreted in negative terms. For example, being unable to remember aspects of the event may be interpreted by the individual as signifying that there is something seriously wrong with them (e.g. brain damage) and this can contribute to negative appraisals of the trauma (Ehlers & Clark, 2000). Secondly, individuals with PTSD engage in a number of cognitive and behavioural coping strategies such as avoidance and thought suppression. These strategies are maladaptive because they directly produce PTSD symptoms and prevent change in the negative appraisals and the trauma memory because they stop the individual being exposed to new information (Ehlers & Clark, 2000).

In order to explain individual differences in reactions to trauma, Ehlers and Clark (2000) suggest that several factors influence the way in which trauma is processed. It is argued that the type of cognitive processing that individuals engage in during the
trauma may influence appraisals of a traumatic event and the encoding traumatic memories. For example, 'mental defeat' describes a type of processing where the individual perceives a total loss of control and feelings of dehumanisation during the trauma. It is suggested that individuals who experience mental defeat are more likely to negatively interpret the trauma and its sequelae (Ehlers & Clark, 2000). Similarly, trauma memories may be poorly organised if an individual is unable to process information at the time of trauma due to confusion, dissociation and emotional numbing (Ehlers & Clark, 2000). In addition, it is argued that appraisals of trauma, trauma memories, the type of coping strategies used and the type of cognitive processing engaged in during the trauma might all be influenced by a variety of background factors. These include characteristics of the trauma and previous experiences and prior beliefs of the individual. For example, if someone is the victim of previous assaults they may be more likely to interpret a subsequent assault as evidence that they are to blame for the trauma.

Having outlined the main features of the model, Ehlers and Clark (2000) go on to suggest that therapy for people with PTSD should be targeted in three main areas. That is helping the individual to modify negative appraisals of the trauma and its sequelae, to stop using dysfunctional coping strategies and to integrate the trauma memory into autobiographical memory.

A strength of the Ehlers and Clark (2000) model is that it can explain not only the symptoms central to PTSD but also a number of other features of the disorder. For example, Ehlers and Clark (2000) suggest that delayed onset PTSD occurs when a later event serves to change the meaning of the event for the individual and causes it to be interpreted as more threatening. Ehlers and Clark (2000) also argue that the model provides an explanation for PTSD phenomena such as anniversary reactions, feeling 'frozen in time', experiencing a sense of impending doom and having no benefit from talking or thinking about the trauma. Although other cognitive models also claim to provide explanations for the various features of PTSD, none provide such a detailed account as Ehlers and Clark (2000) of the cognitive factors and processes involved in the development maintenance of PTSD. In addition, as Ehlers and Clark (2000) have explicitly outlined the features of their cognitive model, it has
allowed many of the predictions made by the model to be tested. Indeed, there is now a growing body of evidence to support various aspects of the model for example Dunmore et al. (1997, 1999, in press).

Thus, the Ehlers and Clark (2000) model appears to be a more comprehensive explanation of PTSD than previous theories. In addition, the model draws together much of the research into the role of cognitive factors in PTSD.

Conclusions

In the twenty years since PTSD was first officially defined in DSM-III (American Psychiatric Association, 1980), an enormous amount of research has been directed at understanding how and why PTSD develops. Following the trend in both academic and clinical psychology, there has been much interest in cognitive factors and their role in determining how individuals respond to traumatic events. Research has demonstrated the importance of cognitive factors in determining various aspects of PTSD and the challenge has been for investigators to integrate these research findings into coherent theories. Although a number of cognitive theories have been proposed, it has been argued that currently the Ehlers and Clark (2000) model most successfully explains what is known about PTSD. However, aspects of this model are untested and it remains to be seen if this model can account for PTSD across a range of situations and populations.

Indeed, one area of recent interest is the development of PTSD after life threatening illnesses such as cancer (Smith, Redd, Peyser & Vogl, 1999), subarachnoid haemorrhage (Berry, 1998) and myocardial infarction (Bennett & Brooke, 1999). To date research in this area has been largely focussed on determining the prevalence of PTSD in such illnesses. However, there is a need to investigate factors that influence the development of PTSD after life threatening illness. An area for future research would be to determine whether Ehlers and Clark’s (2000) cognitive model of PTSD can be used to aid our understanding of and ability to predict the development of PTSD following life threatening illness.
Literature Review

References


Major Research Project

An investigation into negative appraisals and dysfunctional coping strategies associated with posttraumatic stress disorder symptoms following myocardial infarction

July, 2001

Year 3
Abstract

Main objectives: To investigate associations between negative perceptions of permanent change, negative appraisals of symptoms and use of dysfunctional cognitive and behavioural coping strategies and posttraumatic stress disorder symptoms following myocardial infarction.

Design: Cross-sectional design

Setting: Two large general hospitals with cardiac rehabilitation services

Participants: Seventy-four people who were assessed four to twelve weeks after an myocardial infarction.

Main outcome measures: Posttraumatic stress disorder symptoms were assessed using the Posttraumatic Stress Diagnostic Scale (PDS: Foa et al., 1997). Participants completed questionnaires designed to assess potential factors associated with posttraumatic stress disorder symptoms including appraisals of symptoms, perceptions of permanent change following myocardial infarction and use of dysfunctional cognitive and behavioural coping strategies. Anxiety, depression, social dysfunction and somatic symptoms were also assessed using the General Health Questionnaire (GHQ-28: Goldberg & Hillier, 1979)

Results: 12 (16%) participants met DSM-IV criteria for acute PTSD. Experience of previous traumatic events, previous psychological problems, perceived severity of MI, perceived threat to life of MI, negative perceptions of permanent change, negative appraisals of symptoms, and use of dysfunctional cognitive and behavioural coping strategies were all associated with PTSD symptom severity following MI.

Conclusions: These results are consistent with Ehlers and Clark’ s (2000) model of PTSD which emphasises the role of appraisals of trauma sequelae and dysfunctional coping strategies in the development and maintenance of PTSD. In addition, the significant proportion of participants experiencing either acute PTSD or subsyndromal PTSD symptoms following MI, suggests the possibility of PTSD symptoms following MI needs to be taken into account when considering the provision of cardiac rehabilitation services.
Introduction

Annually, approximately 270,000 people in the United Kingdom will experience a heart attack or myocardial infarction (MI) (British Heart Foundation, 2000). It is estimated that of this number approximately 150,000 people will survive the acute stage of MI (British Heart Foundation, 2000). However, people who have had an MI have an increased risk of subsequent fatal and non-fatal cardiac events (Frasure-Smith, Lesperance & Talajic, 1995). Therefore, cardiac rehabilitation services aim to enable people to achieve and maintain healthy lifestyles in order to reduce the recurrence of MI or other cardiac complications (NHS Centre for Reviews and Dissemination, 1998). The majority of cardiac rehabilitation programmes focus on improving the physical health of patients through exercise programmes and education regarding the benefits of stopping smoking and maintaining a healthy diet (NHS Centre for Reviews and Dissemination, 1998).

However, it is widely acknowledged that psychological factors significantly contribute to recovery following MI (Petrie & Weinman, 1997). For example, symptoms of anxiety and depression following MI have been shown to be associated with subsequent MI, increased hospitalisation costs and increased mortality (NHS Centre for Reviews and Dissemination, 1998). Anxiety, depression, irritability and somatic symptoms following MI are also associated with failure to return to work and decreased levels of social, leisure and sexual activities (Melamed, Heruti, Shiloh, Zeidan & David, 1999). Furthermore, psychological problems following MI may contribute to psychosocial adjustment over and above the actual severity of the MI (Melamed et al., 1999). Therefore, it is clinically important to understand the psychological impact of MI (Lowe, Norman & Bennett, 2000), to identify patients who are showing signs of distress following MI, and to provide appropriate and effective interventions (Mayou, Gill, Thompson, Day, Hicks, Volmink & Neil, 2000).

Psychological impact of Myocardial Infarction

Research has highlighted that the psychological impact of MI varies considerably from person to person. For example, in a large-scale prospective study of 203 MI patients, Havik and Maeland (1990) identified six subgroups of patients with
differing patterns of emotional reactions. One of these subgroups (n=69) comprised MI patients who displayed only low levels of anxiety, depression and irritability during hospital admission, two weeks, six weeks, six months and three to five years after discharge (Havik & Maeland, 1990). Indeed, some people report positive effects of MI (Petrie, Buick, Weinman & Booth, 1999). However, for a significant proportion of people, symptoms of anxiety and depression are common sequelae of MI (Bennett, Lowe, Mayfield & Morgan, 1999). Forty to fifty percent of MI patients report moderate to severe symptoms of anxiety during hospital admission, with around a third continuing to experience anxiety at three to six months (Bennett et al., 1999). Approximately twenty percent of MI patients experience moderate to severe levels of depression immediately following MI, with a quarter reporting symptoms of depression at six months (Bennett et al., 1999). Thus, for a significant proportion of people, an MI can have profound psychological impact.

However, Shalev, Schreiber, Galai and Melmed (1993) suggest that while most clinicians are aware that people may experience symptoms of anxiety and depression following a life-threatening illness, the severity and impact of such reactions may not be fully recognised. In addition, Shalev et al. (1993) point out that research studies (and cardiac rehabilitation programmes) often utilise global measures of symptomatology to assess the impact of physical illness. Such measures may be useful in identifying individuals experiencing distress, but may have little clinical utility in suggesting what the most appropriate intervention might be. Shalev et al. (1993) therefore suggest that when considering the impact of physical illness, it may be more clinically relevant to be able to comment about the presence of specific psychiatric disorders, for example panic disorder or obsessive-compulsive disorder.

Indeed, in the literature on the psychological impact of MI, there has recently been interest in using the concept of posttraumatic stress disorder (PTSD) to describe possible reactions to MI (e.g. Bennett & Brooke, 1999, Shalev et al., 1993).

**Myocardial Infarction and PTSD**

PTSD is a psychiatric disorder characterised by nightmares and intrusive thoughts about a traumatic event, accompanied by increased arousal and by avoidance of
reminders of the trauma (American Psychiatric Association, 1994). PTSD is defined as ‘acute’ if the symptom duration is less than three months and ‘chronic’ if symptoms persist for longer than three months (American Psychiatric Association, 1994).

PTSD was first defined in 1980 (DSM III, American Psychiatric Association, 1980) as a possible consequence of traumatic events, such as war, that were “outside the normal range of human experience”. However, changes in the diagnostic criteria for PTSD in DSM-IV (American Psychiatric Association, 1994) now place more emphasis on a person’s reactions to an event rather than the type of event. Thus, a traumatic experience is defined as one where “the person experienced, witnessed or was confronted with an event or events that involved actual or threatened death or serious injury, or threat to the physical integrity of self or others” (American Psychiatric Association, 1994).

PTSD is now widely acknowledged as a possible consequence of traumatic events such as road traffic accidents and assault (Ehlers & Clark, 2000). For example, the prevalence rate for PTSD following road traffic accidents is estimated at between ten and forty-six percent (Ehlers, Mayou & Bryant, 1998). However, an MI may be as equally traumatic as these ‘external’ traumas. For example, Bennett (1993) describes an MI as “a physically devastating event. It is frightening, involving immediate threat to life and loss of control. It can be extremely painful. Sufferers are often surrounded by worried bystanders, rushed dramatically to hospital where they are surrounded by modern medical paraphernalia, monitored by ECG, routinely observed by nurses and given painkilling medications that sedate and confuse” (pg. 7).

Thus, an MI is sudden, unexpected, uncontrollable and carries the potential threat of death (Bennett & Brooke, 1999; Alonzo, 2000), factors that have been shown to be important in the development of PTSD following other types of trauma (Resnick, Kilpatrick, Best & Kramer, 1992: Copland, Literature Review, this volume, pg. 175).

Indeed, Kutz, Shabtai, Solomon Neumann and David (1994) suggest that if the emotional sequelae of MI are scrutinised, the symptoms commonly reported in the
Major Research Project

literature are those that can be associated with a diagnosis of PTSD. These include nightmares, self-isolation and social withdrawal, memory and concentration problems, tension and irritability (Kutz et al., 1994).

Prevalence and impact of PTSD following Myocardial Infarction

PTSD following MI was first reported in detail by Kutz, Garb and David (1988). Kutz et al. (1988) presented a series of four case reports of individuals meeting DSM-III criteria (American Psychiatric Association, 1980) for PTSD following MI. These authors also reported unpublished data from their clinical practice suggesting that fifteen percent of patients suffer chronic PTSD following MI. An additional five to ten percent were judged to suffer from acute PTSD (Kutz et al., 1988). Subsequent studies have generally supported this prevalence rate.

Doerfler, Pbert and DeCosimo (1994) assessed PTSD symptoms in a sample of 50 men following MI (n = 27) or cardiac surgery (CABG)(n = 23), six to twelve months following the event. Measures used to assess PTSD symptomatology were the Reaction Index (RI: Frederick, 1985: cited in Doerfler et al., 1994), the Impact of Events Scale (IES: Horowitz, Wilner & Alvarez, 1979), the Inventory to Diagnose Depression (IDD: Zimmerman & Coryell, 1987) and the Trait Anger Scale (TAS: Spielberger, 1980). Four individuals (3 MI and 1 CABG: 8%) met DSM-III-R criteria for PTSD (American Psychiatric Association, 1987). An additional five individuals (10%) reached criteria for PTSD on the RI. However, PTSD was diagnosed either on the basis of an amalgam of scores on the RI, IES and TAS, which were then checked against DSM-III-R criteria for PTSD (American Psychiatric Association, 1987) or on the RI which does not cover all the criteria stipulated in DSM-III-R. In addition, the severity, duration and onset of PTSD symptoms were not assessed. Therefore, it is not clear whether the PTSD symptoms experienced by the individuals in this study were directly related to MI or CABG (Bennett & Brooke, 1999).

In a more rigorous study, Kutz, Shabtai, Solomon Neumann and David (1994) assessed 100 patients six to eighteen months following MI. PTSD symptoms were assessed using the PTSD Inventory (Solomon, Mikulincer & Jacob, 1987) a self-
report questionnaire based on DSM-III criteria for PTSD (American Psychiatric Association, 1980). The descriptions of symptoms were adapted for use with MI patients. Background information, including information on previous traumatic events, rehabilitation outcome, quality of life since the MI and subjective experiences during the MI was obtained via questionnaires and semi-structured interviews. PTSD was diagnosed in twenty-five percent of the sample. Nine percent of patients were retrospectively diagnosed as suffering from acute PTSD symptoms that lasted less than six months and was not present at time of assessment, while sixteen percent of the sample were found to be suffering from chronic PTSD.

Bennett and Brooke (1999) reported data for 47 patients who had experienced an MI six to twelve months previously. PTSD symptomatology was assessed using the Posttraumatic Diagnostic Scale (Foa, Cashman, Jaycox & Perry, 1997) and the Impact of Events Scale (Horowitz et al., 1979). In this study, four individuals (10%) met criteria for PTSD. Unfortunately, data was not reported for individuals experiencing symptoms of PTSD, but who did not reach full diagnostic criteria.

However, one study found lower rates of PTSD symptoms following MI. Van Driel and Op den Velde (1995) used the SCID-PTSD interview (Spitzer, Williams & Gibbons, 1986) to assess PTSD symptoms in a sample of 23 MI patients within two weeks of MI (time 1) and twenty-two to twenty-six months after MI (time 2). At time 1, one person (4%) showed signs of acute stress disorder as defined in DSM-IV (American Psychiatric Association, 1994). Several other people reported PTSD symptoms, but these were not sufficient to warrant a diagnosis of acute stress disorder. Of the eighteen surviving participants interviewed at time 2, one person (5%) experienced symptoms of partial PTSD and several reported sub-threshold symptoms of PTSD.

Reasons for the lower prevalence rate for PTSD following MI found in this study are unclear. It is possible that lower prevalence rates were found by van Driel and Op den Velde (1995) as these researchers used a diagnostic interview (SCID-PTSD interview: Spitzer, et al., 1986) to assess PTSD symptoms, whereas PTSD symptoms were assessed via self-report in the other studies. The SCID-PTSD interview
(Spitzer, et al., 1986; Spitzer, Williams, Gibbon & First, 1990) is considered the ‘gold standard’ for PTSD diagnosis (Foa, 1995) and therefore is a more rigorous method for assessing PTSD prevalence rates. However, Bennett and Brooke (1999) used a self-report measure (PDS; Foa et al., 1997) that has been shown to have satisfactory diagnostic agreement with the SCID-R-PTSD interview (Spitzer, et al., 1990). A second possible reason for low lower prevalence rates found by van Driel and Op den Velde (1995) is that the time since MI at which PTSD symptoms were assessed is longer than in other studies.

Nevertheless, despite the relatively low prevalence rate of PTSD found in this study, van Driel and Op den Velde (1995) stress that MI is “an example of a sudden, potentially shocking and life-endangering event, which may profoundly alter life-style and future expectations” (pg. 152). They go on to argue that PTSD should be considered as a possible sequelae of medical events that are “associated with intense and inescapable distress, lack of control and perceived or actual threat to life” (pg. 158).

Table 1. shows a summary of prevalence rates for PTSD symptoms following MI found in previous studies.

<table>
<thead>
<tr>
<th>Study</th>
<th>% of participants reaching diagnostic criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Acute Stress Disorder</td>
</tr>
<tr>
<td>Kutz et al. (1988)</td>
<td>not assessed</td>
</tr>
<tr>
<td>Doefler et al. (1994) (n=50)*</td>
<td>not assessed</td>
</tr>
<tr>
<td>Kutz et al. (1994) (n=100)</td>
<td>not assessed</td>
</tr>
<tr>
<td>Van Driel &amp; Op den Velde (1995)</td>
<td>4%</td>
</tr>
<tr>
<td></td>
<td>(n=23)</td>
</tr>
<tr>
<td>Bennett &amp; Brooke (1999)</td>
<td>not assessed</td>
</tr>
<tr>
<td></td>
<td>(n=47)</td>
</tr>
</tbody>
</table>

* includes 23 CABG patients
Given the research on prevalence, Green, Epstien, Krupnick and Rowland, (1997) conclude that a small but significant proportion of MI survivors may experience PTSD and a larger number may experience subsyndromal levels of PTSD symptoms.

PTSD symptoms following MI are not only distressing but may also have healthcare implications. Alonzo (2000) suggests that PTSD symptoms following chronic illnesses, including MI, may impact on an individual’s ability to cope with the sequelae of the illness. For example, PTSD symptoms may make a person less capable of appropriately managing difficulties associated with the illness, thus placing that individual at risk (Alonzo, 2000).

In the context of managing the consequences of MI, Alonzo and Reynolds (1998) and Alonzo (2000) consider how people who have experienced an MI respond when experiencing a subsequent MI or other cardiac related problems. Evidence suggests that survival rates following MI are greatly increased if individuals seek prompt medical care (ideally within an hour of onset of MI) (Alonzo, 2000). However people experiencing a second or further MI generally take longer to seek medical assistance (Alonzo, 2000). Alonzo and Reynolds (1998) and Alonzo (2000) suggest that a possible explanation for this is that PTSD symptoms associated with a first MI, in particular avoidance reactions, may make people less willing to seek medical attention for additional cardiac problems. In addition, Shalev et al. (1993) and Mayou and Smith (1997) suggest that PTSD symptoms following physical illness may be associated with poor adherence to treatment or rehabilitation programmes and avoidance of medical consultations.

On the other hand, in some cases, PTSD symptoms may be associated with the over-use of health care services. For example, frequent visits to accident and emergency departments because the person may wrongly interpret the symptoms of PTSD, such as hyperarousal or re-experiencing, as a sign that another heart attack is about to happen (Green et al., 1997). Kutz et al. (1994) found that in a sample of 100 MI patients, forty-seven percent of those who reported frequent visits to accident and emergency departments were suffering from PTSD. In addition, there was no relationship between the frequency of these visits and objective measures of cardiac
function (Kutz et al., 1994).

In addition to the health care implications, Kutz et al. (1994) also suggest that PTSD following MI may be associated with a number of psychosocial difficulties. Kutz et al. (1994) compared 25 people with PTSD post MI, with 75 people without PTSD post MI on a range of 'rehabilitation factors'. Results showed that at six to eighteen months post MI, people with PTSD were significantly less likely to have returned to work and significantly more likely to have experienced reduced social activity, avoidance of social encounters, report feeling 'handicapped', fear of staying alone and fear of driving compared to MI patients without PTSD. In addition, for the PTSD group, there was no significant relationship between post MI functioning and degree of cardiac complications (Kutz et al., 1994).

Given the potential impact of PTSD symptoms following MI, identification and management of PTSD symptoms is an important clinical issue.

**Management and identification of factors associated with PTSD following Myocardial Infarction**

Psychological debriefing interventions are aimed at minimising the immediate impact of traumatic events and preventing the later development of psychiatric symptoms, in particular PTSD symptoms (Rose, Wessely & Bisson, 2001). However, there is evidence from studies of debriefing interventions after traumatic events such as childbirth, road traffic accidents, miscarriage and violent crime, that debriefing is ineffective and may exacerbate problems in the longer term (Kenardy, 2000; Mayou, Ehlers & Hobbs, 2000; Rose et al., 2001).

Therefore, Bennett and Brooke (1999) highlight the importance of identifying factors associated with the PTSD symptoms following MI. This would aid in the development of a profile of those patients who are at risk. Such people could then be closely monitored and appropriately targeted treatment can then be offered if necessary. Therefore, an understanding of the circumstances under which PTSD develops following MI may lead to improved treatment and outcome.
To date, only two published studies have investigated factors associated with PTSD following MI. Kutz et al. (1994) compared people with PTSD following MI (n=25) with people without PTSD following MI (n=75). Sociodemographic variables included age, education, ethnic background, economic status and pre-MI employment. PTSD was significantly associated with ethnic background, although low numbers of female and single participants excluded analysis of gender and marital status. Previous traumatic events including, experience of prior MI, previous hospitalisation for cardiac problems, past injury, previous life threatening illness, experience of war, PTSD prior to MI and psychiatric history were also compared for the two groups. Prior MI, prior hospitalisation for cardiac problems and PTSD prior to MI were significantly related to PTSD following MI. When the two groups were compared on their subjective experience of MI (intensity of pain, anticipated permanent disability, fear, loss of consciousness, or need for resuscitation) only anticipated permanent disability was significantly associated with PTSD following MI. No significant differences were found between the two groups on objective measures of MI severity e.g. degree of cardiac complications and need for cardiopulmonary resuscitation.

Bennett and Brooke (1999) investigated the association between PTSD symptom severity as measured on the Impact of Events Scale (IES: Horowitz et al., 1979) and the Posttraumatic Diagnostic Scale (PDS: Foa et al., 1997) and a number of psychological variables associated with PTSD in other populations. Variables included negative affect (measured by the Positive and Negative Affect Schedule: PANAS: Watson, Clark & Tellegen, 1988), alexithymia (measured by the Toronto Alexithymia Scale: TAS: Taylor, Ryan & Bagby, 1985) and social support (measured by the Duke Social Support Questionnaire: DUKE: Broadhead, Gehlbach, DeGruy & Kaplan, 1988). In addition, awareness of MI at the time of the event was measured on a Likert type scale. Regression analyses showed that higher scores on alexithymia and awareness at the time of MI, lower age and low social support were each significantly predictive of PTSD symptom severity.

The Kutz et al. (1994) and Bennett and Brooke (1999) studies provide preliminary data on possible predictors of PTSD following MI. However, Bennett and Brooke
(1999) conclude that further research is needed to identify additional factors that are associated with PTSD following MI.

One potential area of further study is to investigate the association of cognitive factors, in particular the way that individuals conceptualise and cope with their experience, and PTSD following MI. Generally, within the literature on adjustment following MI, emphasis is placed on the importance of an individual's perceptions and beliefs about their MI (Petrie & Weinman, 1997) and the type of coping strategies employed (Bennett et al., 1999). Within the literature on PTSD, appraisals of the traumatic event and its sequela and the use of dysfunctional coping strategies have been demonstrated to associated with PTSD following traumatic events such as road traffic accidents and assault (Ehlers & Clarke, 2000; Copland, Literature Review, this volume, pg. 177-178).

Bennett, Conway & Clatworthy (submitted) investigated the contribution of cognitive factors to the development of PTSD following MI. 70 people who were hospitalised following MI were recruited to the study. Initially, participants were asked to complete the Impact of Events Scale (IES: Horowitz et al., 1979), the Global Mood Scale (GMS: Denollet, 1993) and the Cognitive Appraisals Questionnaire (CAQ: Conway (unpublished): cited in Bennett et al., submitted). The CAQ measured: (1) degree of surprise experienced during the MI; (2) how frightened participants felt; (3) the intensity of their emotions; (4) how important the event was to them; (5) whether they felt helpless; (6) whether they felt their life was in danger and (7) the presence of dissociative symptoms. Participants were followed-up three months later and asked to complete the IES, GMS and the Posttraumatic Diagnostic Scale (Foas et al. 1997). 39 people completed the questionnaires at follow-up and seven percent of those received a diagnosis of PTSD. The results showed a strong relationship between negative appraisals of the MI (measured by the CAQ), negative emotions and dissociation and subsequent PTSD symptoms (measured by the PDS & IES).

In addition, Kutz et al. (1994) found that anticipation of permanent disability during the acute MI phase was associated with the development of PTSD symptoms. Thus,
Kutz et al. (1994) suggested that “the consequence of MI imagined by the patient during the coronary event is the most significant subjective factor in determining future PTSD” (pg. 54).

Thus, there is some evidence to suggest that appraisals of the MI and its consequences may be an important factor in identifying people who may be at risk of developing PTSD post MI. However, current research on factors associated with PTSD following MI is limited and does not appear to be based on any clear theoretical framework — a criticism that has been made generally about research on psychological factors and adjustment to MI (Petrie & Weiman, 1997).

The role of appraisals and coping strategies in Ehlers and Clark’s (2000) model of PTSD
Ehlers and Clark (2000) have recently proposed a cognitive model of PTSD that emphasises the role of appraisals of the traumatic event, its sequelae and dysfunctional coping strategies (Copland, Literature Review, this volume, pg. 183-186).

Ehlers and Clark (2000) point out that with all other anxiety disorders, cognitive models suggest that anxiety is related to some future threat. However, in PTSD distress is linked to an event that has already happened. Therefore, Ehlers and Clark (2000) argue that PTSD occurs if an individual processes the traumatic event in such a way that leads to a sense of current threat. Ehlers and Clark (2000) suggest that one way in which a sense of current threat is generated is that individuals who develop PTSD think about the trauma and its sequelae in excessively negative terms. Ehlers and Clark (2000) identify several types of negative appraisals thought to be important in the development and maintenance of PTSD. Negative appraisals of the traumatic event may include over exaggerating the possibility of future traumatic events or perceiving the trauma as evidence that “bad things always happen to me” (Ehlers & Clark, 2000). Appraisals of the way that an individual acted and the emotions experienced at the time of the event may also effect the way a traumatic event is perceived (Ehlers & Clark, 2000). Ehlers and Clark (2000) also suggest that negative appraisals of the trauma sequelae contribute to the development of PTSD. Negative interpretations of PTSD symptoms may include thoughts such as “I am
going mad.” Likewise, trauma survivors may interpret other people’s reactions as meaning that “I cannot rely on other people” or the consequences of the trauma may be interpreted as signs of permanent and negative change such as “I will never be able to lead a normal life again.”

Ehlers and Clark (2000) go on to suggest that such negative appraisals of the traumatic event and its sequelae are maintained as individuals with PTSD engage in a number of cognitive and behavioural coping strategies such as avoidance and thought suppression. These strategies are maladaptive because they directly produce PTSD symptoms and prevent change in the negative appraisals because they stop the individual being exposed to new information (Ehlers & Clark, 2000). In addition, Ehlers and Clark (2000) outline factors that may influence individuals’ appraisals of traumatic events and the coping strategies used. These include the type of cognitive processing that individuals engage in during the trauma, characteristics of the trauma and previous experiences and prior beliefs of the individual (Ehlers & Clark, 2000).

Empirical support for the role of appraisals and dysfunctional coping as outlined by Ehlers and Clark’s (2000) model of PTSD

Recent research has provided evidence to support the role of appraisals (of emotions and actions during the trauma, PTSD symptoms, perceptions of other’s responses and perceptions of permanent change) and dysfunctional coping strategies in the development and maintenance of PTSD (Ehlers & Clark, 2000).

Dunmore, Clark and Ehlers (1997) interviewed 11 people with persistent PTSD following an assault and 9 people who had meet PTSD criteria in the month after an assault but who had since recovered. The interview included information regarding appraisals of actions during the event, appraisals of other people’s responses, appraisals of PTSD symptoms and appraisals of permanent change at the time of the assault or shortly after. Results showed that people in the persistent PTSD group were more likely to report negative appraisals of their actions during the event, negative appraisals of other’s responses, negative appraisals of PTSD symptoms and negative perceptions of permanent change than people who had recovered (Dunmore et al., 1997). However, the conclusions that can be drawn from this study are limited
because of the small sample size and the use of non-blind ratings of the interview material (Dunmore et al., 1997). In addition, participants were asked to recall details of a traumatic event that occurred an average of eight years previously for the persistent PTSD group and two years previously for the recovered PTSD group. Thus, the results may have been confounded by recall bias.

In a more comprehensive study, Dunmore, Clark and Ehlers (1999) assessed 92 people who had experienced physical or sexual assault. Participants were categorised according to their scores on the PTSD Symptom Scale (PSS-SR: Foa, Riggs, Dancu & Rothbaum, 1993) in the month after the assault and at the time of assessment (at least three months after the assault). Twenty-eight people did not meet PTSD criteria at either time point (no PTSD group), forty-four people met PTSD criteria at both time points (persistent PTSD group) and twenty people met PTSD criteria in the month after the assault only (recovered PTSD group). All participants completed a battery of questionnaires designed specifically for the study assessing thoughts, emotions and actions experienced during the assault, appraisals of symptoms, other people's reactions and perceptions of permanent change in the month after the assault and coping strategies used in the month after the assault. Firstly the no PTSD and PTSD (both recovered and persistent) groups were compared to determine which cognitive factors were associated with the development of PTSD. Results showed that the PTSD group were significantly more likely to report experiencing negative thoughts during the assault (mental defeat, confusion, detachment) and to appraise their emotions during the assault in a more negative way. The PTSD group reported significantly more negative appraisals of their symptoms and other people's reactions and were more likely to believe that the assault had had a permanent negative impact on their lives. In addition, the PTSD group were significantly more likely to have engaged in dysfunctional coping strategies. Apart from confusion and appraisal of actions during the assault, all other cognitive and behavioural variables significantly contributed to severity of PTSD symptoms when previous history and perceived and objective assault severity was controlled for.

Dunmore et al. (1999) also compared the persistent PTSD and recovered PTSD
groups to investigate which variables may predict persistent PTSD. The persistent PTSD group reported significantly higher levels of mental defeat and detachment during the assault, more negative appraisals of their emotions, and there was a trend towards more negative appraisals of their actions. The persistent PTSD group also differed significantly from the recovered PTSD group in terms of negative appraisals of symptoms and negative perceptions of permanent change, but no differences were found in terms of perceptions of other people’s responses. The persistent PTSD group was also more likely to engage in some types of dysfunctional coping strategies.

In a prospective study, Dunmore, Clark and Ehlers (in press) interviewed 57 people within four months of an assault and again at six and nine months to investigate the relationship between various cognitive factors and PTSD severity across time. The cognitive factors investigated were similar to those in the Dunmore et al. (1999) study. Several variables – mental defeat, mental confusion and detachment at the time of the assault, negative appraisals of symptoms, negative appraisals of other people’s responses, perceived negative permanent change, avoidance/safety seeking coping strategies and negative beliefs after the assault – were significantly associated with PTSD symptom severity at all three time points, after controlling variables related to previous history and assault severity.

Additional support for the role of dysfunctional coping strategies and appraisals of the traumatic event and its sequelae in the development and maintenance of PTSD, as outlined by Ehlers and Clark (2000), is provided by research on other samples. For example, road traffic accident victims (Ehlers & Steil, 1995; Steil & Ehlers, 2000; Ehlers, Mayou & Bryant, 1998), ambulance staff (Clohessy & Ehlers, 1999) former political prisoners (Ehlers, Maercker & Boos, 2000) and the analysis of treatment outcome (Ehlers, Clark, Dunmore, Jaycox, Meadows & Foa, 1998).

With respect to negative perceptions of permanent change, Ehlers et al. (2000) acknowledge that many survivors of trauma do experience actual permanent change, for example chronic injury. However, studies have compared survivors with and without PTSD, suggesting that perceived permanent change contributes to the
development of PTSD over and above actual change in life circumstances (Ehlers et al., 2000). For example, Ehlers, et al. (2000) interviewed 81 former political prisoners, 32 with chronic PTSD, 20 with remitted PTSD and 29 without PTSD. Those with PTSD scored higher on permanent change than those without PTSD. In a regression analysis, perceptions of permanent change explained unique variance in PTSD symptom severity, even after measures of objective and subjective trauma severity were controlled for.

In addition, Ehlers et al. (1998) compared 20 women who had experienced sexual assault and who had received exposure therapy for PTSD symptoms. Participants were divided into two groups—good outcome (n=10) and poor outcome (n=10). The groups did not differ on any significant variables except that the poor outcome group had experienced a longer assault than the good outcome group. Transcripts of treatment sessions were rated for evidence that the participants reported permanent change to their lives as a result of the assault. Results showed that perceptions of permanent change correlated negatively with improvement in PTSD symptoms.

Summary

From the literature reviewed, it would appear that experiencing an MI can have a significant impact on the individual. An MI is sudden, unexpected, uncontrollable and is potentially life threatening and as a result may act as a trigger for PTSD symptoms in some people. While researchers have begun to investigate factors associated with PTSD symptoms following MI, the literature in this area is still scant and limited by the lack of use of a clear theoretical framework. However, research on the development and maintenance of PTSD symptoms in other populations has highlighted the important role of appraisals and coping strategies (e.g. Copland, Literature Review, this volume, pg. 183-186). The Ehlers and Clark (2000) model of PTSD provides a framework for understanding how appraisals and coping strategies contribute to the development and maintenance of the disorder. Ehlers and Clark’s (2000) model may therefore aid our understanding of the factors associated with PTSD symptoms following MI.
Aims
The aims of the present study were to: (1) collect information regarding the prevalence of PTSD symptoms following MI; and (2) investigate factors associated with PTSD symptoms following an MI. In particular, the aim was to explore the association between negative appraisals and dysfunctional coping strategies and PTSD symptoms following MI.

To investigate factors associated with PTSD symptoms following MI, the following variables were included in the study:

- **Demographic data and individuals' experiences prior to MI**
  Based on previous studies of PTSD following traumatic events including assault (e.g. Dunmore et al., 1999, in press), childbirth (Czarnocka & Slade, 2000) diagnosis of cancer (Smith, Redd, Peyser & Vogl, 1999) and HIV (Kelly, Raphael, Judd, Kernutt, Burnett & Burrows, 1998) and previous research on MI (e.g. Kutz et al., 1994; Bennett & Brooke, 1999), factors relating to demographic variables and individuals' experiences prior to MI were investigated. These included prior hospitalisation for cardiac problems (Kutz et al., 1994), previous psychological problems (Dunmore et al., 1999; Czarnocka & Slade) and experience of previous traumatic events (Smith et al., 1999; Kelly et al., 1998).

- **Subjective experiences during MI**
  Previous studies suggest that perceived severity of the trauma and perceived threat to life during trauma are associated with PTSD symptoms in other populations (Dunmore et al., 1999; Copland, Literature Review, this volume, pg. 175). Therefore measures of perceived severity of MI and perceived threat to life at the time of MI were included in this study.

- **Negative appraisals and dysfunctional coping strategies**
  Several factors outlined in Ehlers and Clark's (2000) model of PTSD were investigated in this study. These were: (1) appraisals of symptoms; (2) perceptions of permanent change and; (3) use of dysfunctional cognitive and behavioural coping
strategies.

**Hypotheses**
The main hypotheses of the study were –

1. More severe PTSD symptoms following MI will be associated with negative perceptions of permanent change.

2. More severe PTSD symptoms following MI will be associated with negative appraisals of symptoms.

3. More severe PTSD symptoms following MI will be associated with use of dysfunctional cognitive and behavioural coping strategies.

**Method**

*Design*
The study used a cross-sectional design. Individuals who had experienced an MI were assessed using self-report questionnaires covering demographic information, previous experiences, PTSD symptoms, anxiety, depression, somatic symptoms, social dysfunction and cognitive factors, hypothesised to be associated with PTSD symptoms.

*Procedure*
Ethics committee approval for the study was granted from two general hospitals (Appendix 1 & 2). Consent was then gained from the relevant consultant cardiologists and physicians to recruit people under their care who had experienced an MI. At both hospital sites all people who had experienced an MI were referred to cardiac rehabilitation services when medically fit. Therefore, consecutive referrals for people who had experienced an MI were identified from the cardiac rehabilitation records at the two participating hospitals. Exclusion criteria for the study were: (1) people with a learning disability; (2) people with dementia or cognitive impairment; (3) people who do not speak English and therefore would have difficulty filling out
the questionnaires; (4) people with a concurrent serious illness and; (5) people who had experienced a previous MI.

All people eligible to take part in the study had experienced an MI at least four weeks prior to being asked to participate in the study and not more than three months. This time-period was chosen for several reasons. Firstly, at this point all participants would still be in contact with hospital services. Therefore any participant who was found to be suffering from PTSD or significant distress could be monitored more closely by the cardiac rehabilitation staff or referred on to the appropriate services. Secondly, DSM-IV diagnostic criteria for acute PTSD require that the symptoms have been present for a minimum of four weeks post trauma (American Psychiatric Association, 1994). Thirdly, the aim of the study was to identify factors associated with PTSD symptoms that may be present shortly after MI, while people are in a rehabilitation programme.

At the two hospital sites all people who met the inclusion criteria were asked to participate in the study. Due to differences in cardiac rehabilitation services at the two hospitals, different recruitment procedures were used. At site one, potential participants were recruited to the study when they attended cardiac rehabilitation sessions. The purpose of the study was explained and it was stressed that participation was entirely voluntary. Those who agreed to participate in the study were given an information sheet (Appendix 3), a consent form (Appendix 4) to sign and a questionnaire pack. Once completed, the consent form and questionnaires could either be handed back in a sealed envelope to cardiac rehabilitation staff or returned to the researcher in a prepaid envelope. The cardiac rehabilitation nurses gave individuals who were eligible for the study, but who did not attend cardiac rehabilitation sessions, the research packs on routine home visits. Potential participants who had not returned the questionnaires within three weeks were contacted again by the researcher. This was done to maximise response rate. No further contact was made with potential participants who did not return the questionnaires thereafter.

At site two, the researcher checked with the cardiac rehabilitation nurse and general
practitioners that people eligible for the study continued to survive. Research packs were then sent to potential participants. The research pack contained a letter briefly explaining the purpose of the research and stating that participation was voluntary (Appendix 5). The packs also contained an information sheet (Appendix 6), consent form (Appendix 7) and questionnaire pack (the same as for site one). Completed questionnaires could be returned to the researcher in a pre-paid envelope. A reminder letter (Appendix 8) was sent to potential participants who had not returned the questionnaires within three weeks. As with site one, no further contact was made with potential participants who did not return the questionnaires. Figure 1 shows a summary of the recruitment procedures used for both hospital sites.

The researcher contacted participants who indicated on the questionnaires that they were suffering from significant distress. The researcher asked individuals if they were receiving support for their difficulties and if appropriate, sought permission to alert cardiac rehabilitation staff, who could then arrange suitable intervention.
Figure 1. Summary of recruitment procedures used across both hospital sites.

- Ethical approval and consent from cardiologists and physicians obtained for site 1 & 2
- At site 1 & 2, suitable participants identified from cardiac rehabilitation records
  - At site 1, potential participants given research packs at cardiac rehabilitation sessions by researcher OR on home visit by cardiac rehabilitation nurse
  - At site 2, potential participants sent research packs

Participants

Across the two hospital sites, 146 suitable participants were identified (46 from site one, 100 from site two). 74 people completed and returned the questionnaire packs (25 from site one, 49 from site two), with a total response rate of 51%. This return rate is similar to return rates in other questionnaire-based studies of PTSD following MI (Doerfler et al., 1994; Bennett & Brooke, 1999). Analysis of the characteristics of responders versus non-responders is shown in the Results section (pg. 220).
Measures

Table 2 shows a summary of the questionnaires given to participants

Table 2. Summary of questionnaires

- Background Information Questionnaire
- Part 1 of the Posttraumatic Diagnostic Scale (Foa et al., 1997)
- General Health Questionnaire – 28 (Goldberg & Hillier, 1979)
- Parts 3 & 4 Posttraumatic Diagnostic Scale (Foa et al., 1997)
- Subjective Experience of MI
- Appraisals of symptoms
- Perceptions of permanent change
- Dysfunctional cognitive and behavioural coping strategies

Background information and previous experiences

Two questionnaires were used to obtain demographic information and details of previous experiences that have been demonstrated to be associated with PTSD following MI and other types of traumatic events. A Background Information Questionnaire (Appendix 9) asked participants about demographic data including age, gender, marital status, ethnic background, educational qualifications and employment status. In addition, information was requested regarding psychological difficulties before MI and previous hospital admissions for cardiac related problems.

To assess previous exposure to traumatic events, participants completed Part 1 of the Posttraumatic Diagnostic Scale (PDS: Foa, et. al., 1997). Minor adjustments to the wording of Part 1 of the PDS were made for this study. In its original form, Part 1 of the PDS lists eleven traumatic events and has an option for participants to add other traumatic events that they have experienced that are not listed in the questionnaire. For the present study, Item 11 of the PDS was changed from “life threatening illness” to “life threatening illness other than a heart attack” (Other Traumatic Events Questionnaire, Appendix 10).

General Health Questionnaire (GHQ-28; Goldberg & Hillier, 1979) (Appendix 11). The 28-item version of the General Health Questionnaire assesses the presence and severity of psychiatric symptoms, other than PTSD. It is an extensively used assessment with trauma victims (Turner & Lee, 1998) and is a reliable and extensively validated screening tool for psychiatric symptoms in medical outpatients.
and inpatients (Goldberg, 1995; Goldberg, Gater, Ustun, Sartorius, Piccinelli, Gureje & Rutter, 1997). The GHQ-28 provides a total score and scores for four subscales – somatic symptoms, anxiety and insomnia, social dysfunction, and depression. There are two scoring methods available for the GHQ-28 (Turner & Lee, 1998). The GHQ scoring method (0-0-1-1) allows the questionnaire to be used as a screening test for psychiatric symptoms (Goldberg et al., 1997). This method was used to aid in the identification of participants who may have been in need of additional support and whom the researcher contacted to seek permission to alert cardiac rehabilitation staff. A score of seven or above was taken to indicate the presence of a psychiatric disorder (Goldberg et al., 1997). The Likert method of scoring the GHQ (0-1-2-3) was used for statistical analyses as this scoring method assesses the severity of reported symptoms (Goldberg et al., 1997).

Posttraumatic Diagnostic Scale (PDS: Foa, Cashman, Jaycox, Perry, 1997)
The presence and severity of posttraumatic stress disorder symptoms was assessed using Parts 3 and 4 of the Posttraumatic Diagnostic Scale (PDS: Foa, Cashman, Jaycox, Perry, 1997). The PDS is a self-report measure of PTSD developed from the DSM-IV PTSD criteria (Foa et al., 1997). It is a well-validated scale for assessing PTSD symptoms and has satisfactory agreement with the Structured Clinical Interview for PTSD (SCID-R-PTSD: Spitzer, Williams, Gibbon & First, 1990) (kappa = 0.65, agreement = 82%, sensitivity = 0.89 and specificity = 0.75: Foa et al., 1997). In addition, the PDS has been used in previous research on PTSD and MI (Bennett & Brooke, 1999).

To ensure that participants rated PTSD symptoms related to their MI, they were asked to rate parts 3 and 4 of the PDS, with respect to their MI. The wording of the instructions and the items were modified to make this clear (Appendix 12).

Part 3 of the PDS consists of seventeen items corresponding to the cluster of PTSD symptoms listed in DSM-IV (American Psychiatric Association, 1994). Five items relate to re-experiencing symptoms, seven to avoidance, and five to arousal. The frequency with which each symptom has been experienced in the last four weeks is rated on a four point scale (0 = not at all or only one time, 1= once a week of
less/once in a while, 2 = 2 to 4 times a week / half the time, 3 = 5 or more times a week / almost always). A symptom severity score is obtained by summing the score on items 1-17 of Part 3. The maximum score on the PDS for total PTSD symptom severity score is 51. The alpha coefficient for internal consistency of the total symptom severity score is 0.92 (re-experiencing $\alpha = 0.78$: avoidance $\alpha = 0.84$ arousal $\alpha = 0.84$) (Foa et al., 1997). Foa (1995) suggests that total PTSD symptom severity scores can be classified into the following categories; 0 = no PTSD symptoms, 1-10 = mild symptoms, 11-20 = moderate symptoms, 21-35 = moderate to severe symptoms and 36-51 = severe symptoms.

Two questions ask about duration of symptoms to assess whether the individual reaches criteria for acute PTSD (symptom duration 1-3 months) or chronic PTSD (symptom duration more than 3 months). As symptom duration of more than 3 months would indicate that participants were rating symptoms that had been present prior to MI, it was planned to exclude any participants who indicated that the symptoms they endorsed had been present for more than 3 months. However, no participants reported symptom duration of more than 3 months.

Part 4 of the PDS assesses to what extent the symptoms described in part 3 have interfered with different aspects of functioning in the last four weeks and is required to make a diagnosis of PTSD. Areas of functioning covered are work, household duties, friendships, leisure activities, family relationships, sex life, general satisfaction with life and overall level of functioning in all areas. Foa (1995) suggests that people indicating that PTSD symptoms have affected their overall functioning or the 7 other areas of functioning covered can be classified as suffering from a 'severe' level of impairment. People indicating 3-6 areas of life affected = moderate impairment, 1-2 areas affected = mild impairment, 0 areas affected = no impairment.

A diagnosis of acute PTSD requires that the traumatic event includes the presence of: (1) physical injury or the perception of threat to life; (2) a sense of feeling terrified or helpless during the event and; (3) the endorsement of at least one re-experiencing
symptom, three avoidance symptoms and two arousal symptoms (Foa et al., 1997). In addition, these symptoms must be present for at least one month and must impair at least one area of functioning (Foa et al., 1997).

Subjective Experience of MI (Appendix 13)
To assess the subjective experience of MI and to gain information required for acute PTSD diagnostic criteria, participants rated the perceived severity of the MI, the extent to which they believed their life was in danger, how helpless and how terrified they felt at the time the MI took place. Perceived severity of the MI, perceived threat to life and degree of helpless and terror experienced at the time of MI were each rated on an 11 point scale ranging from 0-100, adapted from previous research on PTSD (Dunmore et al., 1999; in press). Each scale was marked in units of 10.

Diagnostic criteria for PTSD require that during the event the person experienced (1) physical injury or the perception of threat to life and; (2) a sense of feeling terrified or helpless. Therefore, for the purposes of determining the prevalence rate of acute PTSD, perception of threat to life, helplessness and terror were transformed into dichotomous variables. A score of 10 was the first point above 0 on the scales and represented at least some degree of perceived threat to life, helplessness or terror. Therefore, perception of threat to life, helplessness and terror were coded as present if rated as 10 or more on the 0-100 scale. For statistical analyses, scores of 0-100 were used for perceived severity and perceived threat to life.

Cognition and behaviour questionnaires
No standardised questionnaires were available to measure negative perceptions of permanent change, negative appraisals of symptoms and dysfunctional cognitive and behavioural coping strategies. Therefore, three questionnaires were developed to assess these factors.

Items for the questionnaires on appraisals of symptoms and dysfunctional coping strategies, were taken from measures used in previous research on PTSD and victims of physical/sexual assault (Dunmore et al., 1997, 1999, in press), ambulance workers (Clohessy & Ehlers, 1999) and road traffic accident victims (Steil & Ehlers, 2000).
Items from previous questionnaires that were related to specific types of event e.g. assault were not used. For the dysfunctional coping strategies questionnaire, items were included to assess the different types of dysfunctional cognitive and behavioural coping strategies that have been associated with PTSD symptoms e.g. avoidance, thought suppression, distraction, detachment, and 'undoing'/rumination (Steil & Ehlers, 2000; Dunmore et al., 1999 & in press; Clohessy & Ehlers, 1999). For the appraisals of symptoms, items were included to assess both general reactions following a traumatic event and appraisals of specific PTSD symptoms (Dunmore et al., 1999 & in press).

The questionnaire on perceptions of permanent change following MI was developed with reference to the literature on psychological adjustment following MI (Bennett, 1990; Petrie & Weinman, 1997). Items were chosen to reflect areas which MI patients may perceive as threatened or negatively affected by MI. For example, Bennett (1990) asked 64 people 2-12 weeks post MI, about aspects of their life they perceived as threatened by their MI. Five categories were identified as potential sources of threat following MI. These were physical problems, medication/self-care, work, finances and social activities.

Draft versions of the three questionnaires were piloted on a small sample (n = 6) of people who had experienced a MI and reviewed by two cardiac rehabilitation nurses to check for ease of completion, face validity, and relevance to MI patients. Items were excluded from the final versions of the questionnaires or modified according to feedback if (1) people indicated that the wording of the item was confusing or (2) the item was difficult to rate. Details of the final versions of the questionnaires are given below.

**Appraisals of symptoms** (Appendix 14)

A 10-item questionnaire (alpha = 0.73) assessed participants' appraisals of symptoms following MI. Items were worded in the form of statements, for example “My reactions since the heart attack mean that I cannot cope”. Participants were asked to rate how much they had agreed with each item over the last four weeks. Items were rated on a 7 point scale (0 = totally disagree, 1 = disagree very much, 2 = disagree
slightly, 3 = neutral, 4 = agree slightly, 5 = agree very much, 6 = totally agree). Scores on the individual items were summed to provide a total score for appraisals of symptoms. A higher score indicated more negative appraisals of symptoms.

*Perceptions of permanent change* (Appendix 15)
An 8-item (alpha = 0.91) questionnaire assessed participants’ beliefs that the MI would have a permanent, negative impact on their life. The areas covered by the questionnaire were physical health, medication and self-care, work, general activities e.g. sports and housework, relationships and social activities. In addition, one question asked about overall permanent change to life. Items were worded in terms of statements for example, “I will have financial problems.” Participants were asked to rate their agreement with items on a 7 point scale (0 = totally disagree, 1 = disagree very much, 2 = disagree slightly, 3 = neutral, 4 = agree slightly, 5 = agree very much, 6 = totally agree). Scores on the individual items were summed to provide a total score for perceptions of permanent change, with higher scores indicating more negative perceptions of permanent change following MI.

*Dysfunctional cognitive and behavioural coping strategies* (Appendix 16)
A 14-item questionnaire (alpha = 0.85) assessed how often participants had used dysfunctional cognitive and behavioural coping strategies in the past month. Participants rated how frequently they had engaged in each coping strategy on four point scale (0 = never, 1 = sometimes, 2 = often, 3 = always). Two items asked about avoidance of activities, e.g. “I have avoided stressful events”. Five items dealt with forms of cognitive avoidance (distraction, suppression, avoidance) e.g. “I have tried to distract myself from distressing thoughts”. Three items asked about with detachment, e.g. “I have tried to numb my emotions” and two items dealt with ‘undoing’ or attempts to mentally alter the event, e.g. “I have thought about ways in which the heart attack might have been prevented”. In addition, two items asked whether people had disclosed information about the MI to others, which is seen a positive coping strategy in relation to traumatic events (Clohessy & Ehlers, 1999). These items were “I have allowed myself to show my feelings to others” and “I have talked to family and friends about the heart attack and about what I was feeling”. Scores for these items were reversed when calculating a total score for use of
dysfunctional coping strategies. A higher total score indicates more frequent use of dysfunctional cognitive and behavioural coping strategies.

Results
For all statistical analyses, parametric tests were used when data were; (1) measured on a continuous scale and (2) were normally distributed. When data did not meet criteria for parametric tests, the equivalent two-tailed non-parametric tests were used. If equivalent non-parametric tests were not available, data was suitably transformed. Missing data was excluded on an analysis by analysis basis.

Although directional main hypotheses were generated for the study, two-tailed tests were used as the study was exploratory and the aim was to investigate the nature of the relationships between various factors and PTSD symptoms following MI. In addition, Howell (1992) argues that two-tailed tests are always more appropriate.

Comparison of responders versus non-responders
Responders (n = 74) versus non-responders (n = 72) were compared on data available from cardiac rehabilitation records using independent t-tests and Mann-Whitney U tests for continuous data and chi-square tests ($\chi^2$) for categorical data, to establish whether the two groups differed. For non-responders across the two sites, complete data was available for age and gender. Unfortunately, in the cardiac rehabilitation records, data for martial status and ethnic background was incomplete for many non-responders and was therefore not analysed. At site one, Hospital Anxiety and Depression Scale (HADS: Zigmond & Sniath, 1983) scores were available for both responders and non-responders at the time-period during which questionnaires were distributed. Therefore, HADS anxiety and depression scores for non-responders and responders from site one were compared.

Table 3 shows data for age and gender for non-responders and responders across the two hospital sites and results of the statistical analyses. Table 4 shows data for HADS anxiety and depression scores for non-responders and responders at site one and results of the statistical analyses.
Table 3. Data for non-responders and responders on age and gender and \( t / \chi^2 \) statistics and p-values for non-responders compared to responders on appropriate statistical tests.

<table>
<thead>
<tr>
<th></th>
<th>Non-responders (n= 72)</th>
<th>Responders (n= 74)</th>
<th>( t / \chi^2 ) statistic (p-value)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age (n= 151)</strong></td>
<td>M= 65.78, SD = 12.75, Range = 39-90</td>
<td>M= 61.99, SD = 11.21, Range = 40-85</td>
<td>( t = 1.909 ) (p=0.058)</td>
</tr>
<tr>
<td><strong>Gender (n= 151)</strong></td>
<td>Male = 49, Female = 23</td>
<td>Male = 56, Female = 18</td>
<td>( \chi^2 = 0.700 ) (p=0.403)</td>
</tr>
</tbody>
</table>

Table 4. Data for non-responders and responders on HADS anxiety and depression scores and \( t \)-test/Mann Whitney \( U \) results and p-values for non-responders compared to responders on appropriate statistical tests.

<table>
<thead>
<tr>
<th></th>
<th>Non-responders (n= 18)</th>
<th>Responders (n= 21)</th>
<th>( t )-test / Mann Whitney ( U ) (p-value)</th>
</tr>
</thead>
<tbody>
<tr>
<td><em><em>HADS score (n= 39</em>)</em>*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>anxiety</strong></td>
<td>M= 8.89, SD = 5.25, Range = 1-19</td>
<td>M= 5.71, SD = 4.56, Range = 0-15</td>
<td>( t = 2.043 ) (p = 0.048)</td>
</tr>
<tr>
<td><strong>depression</strong></td>
<td>Median = 6, Range =0-16</td>
<td>Median = 2, Range = 0-12</td>
<td>( z = -1.563 ) (p = 0.118)</td>
</tr>
</tbody>
</table>

*HADS not completed by 7 participants

There were no significant differences between non-responders and responders from both sites in terms of age and gender. For site one, there were no significant differences between non-responders and responders on depression scores at time of assessment.

There was a significant difference between non-responders and responders from site one on anxiety scores at time of assessment. Non-responders from site one reported higher HADS anxiety scores at the time of assessment than responders.

**Characteristics of sample**

For the 74 participants who completed questionnaire packs, a summary of demographic data are shown in Table 5.
Table 5. Demographic characteristics of the participants (N=74, unless otherwise stated)

<table>
<thead>
<tr>
<th>Sex (n (%))</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>56 (78%)</td>
</tr>
<tr>
<td>Female</td>
<td>18 (24%)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Age in years (mean (SD))</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>62.14 (11.28)</td>
</tr>
<tr>
<td>Range:</td>
<td>40 - 85</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Time in weeks since MI (mean (SD))</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>7.88 (2.37)</td>
</tr>
<tr>
<td>Range:</td>
<td>4 - 12</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Marital status (n (%))</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Single</td>
<td>5 (7%)</td>
</tr>
<tr>
<td>Married / cohabiting</td>
<td>52 (70%)</td>
</tr>
<tr>
<td>Widowed</td>
<td>11 (15%)</td>
</tr>
<tr>
<td>Divorced / separated</td>
<td>6 (8%)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Ethnic Background (n (%))</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>White UK</td>
<td>67 (90%)</td>
</tr>
<tr>
<td>White Other</td>
<td>2 (2.5%)</td>
</tr>
<tr>
<td>Indian</td>
<td>3 (4%)</td>
</tr>
<tr>
<td>Other</td>
<td>2 (2.5%)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Education (n (%))</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>No qualifications</td>
<td>33 (46%)</td>
</tr>
<tr>
<td>School examinations (GCSE, O / A level)</td>
<td>25 (35%)</td>
</tr>
<tr>
<td>Diploma</td>
<td>4 (5%)</td>
</tr>
<tr>
<td>Degree or above</td>
<td>9 (13%)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Employment Status (n (%))</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Full / part time work</td>
<td>26 (35%)</td>
</tr>
<tr>
<td>Receiving disability allowance</td>
<td>4 (5%)</td>
</tr>
<tr>
<td>Studying</td>
<td>1 (1%)</td>
</tr>
<tr>
<td>Retired</td>
<td>39 (53%)</td>
</tr>
<tr>
<td>Other (e.g. carer)</td>
<td>4 (5%)</td>
</tr>
</tbody>
</table>

The mean age, age range and ratio of males to females in this sample are comparable to participant samples in previous research on PTSD following MI (Kutz et al., 1994; Bennett & Brooke, 1999) and research on adjustment following MI (Bennett et al., 1999; Lowe et al., 2000). In addition, the ratio of males to females in this study corresponds to the ratio of males to females experiencing MI in the general population (British Heart Foundation, 2000).

Data on participants’ experiences prior to MI are shown in Table 6.
Table 6. Data on participant’s experiences prior to (most recent) MI (N=74, unless otherwise stated).

<table>
<thead>
<tr>
<th>Experience</th>
<th>Yes (n(%))</th>
<th>No (n(%))</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hospitalised for heart problems other than MI</td>
<td>6 (8%)</td>
<td>68 (92%)</td>
</tr>
<tr>
<td>Experience of traumatic events other than MI</td>
<td>41 (55%)</td>
<td>33 (44%)</td>
</tr>
<tr>
<td>Psychological difficulties prior to MI</td>
<td>10 (14%)</td>
<td>64 (86%)</td>
</tr>
<tr>
<td>Received help for prior psychological difficulties</td>
<td>4 (40%)</td>
<td>6 (60%)</td>
</tr>
</tbody>
</table>

*a N = 10

Prevalence of PTSD symptoms
The mean score for total PTSD symptom severity on the PDS was 8.99 (SD = 9.47, range 0 - 41). The mean scores for the PTSD symptom subscales were: re-experiencing symptoms M = 2.35 (SD=2.82, range 0-12), arousal symptoms M = 3.01 (SD=3.34, range 0-15) and avoidance symptoms M = 3.62 (SD=4.49, range 0-19).

The mean number of PTSD symptoms reported was 5.34 (SD=4.48, range 0 – 17). The most commonly reported PTSD symptoms were having trouble sleeping (54%), feeling emotionally upset when reminded of the MI (54%), having upsetting thoughts or images about the MI (43%), having less interest in activities (42%) and feeling that plans for the future will not come true (42%).

12 participants (16%) met DSM-IV criteria (American Psychiatric Association, 1994) for acute PTSD. Therefore, participants reported: (1) perceived threat to life during MI; (2) a sense of feeling terrified or helpless during MI; (3) at least one re-experiencing symptom, three avoidance symptoms and two arousal symptoms; (4) the presence of symptoms for at least one month and (5) that symptoms impaired at least one area of functioning (American Psychiatric Association, 1994; Foa et al., 1997). Of these 12 participants, 6 (50%) reported moderate PTSD symptoms, 3
(25%) reported moderate to severe symptoms and 3 (25%) reported severe symptoms (Foa, 1995). 9 (75%) participants who fulfilled criteria for acute PTSD, reported that PTSD symptoms were associated with severe impairment in functioning affecting all areas of life, 1 (8%) reported moderate impairment and 2 (17%) reported mild impairment (Foa, 1995). Characteristics of the 12 participants reaching diagnostic criteria for acute PTSD are shown in Appendix 17.

For the remaining 62 participants who did not reach acute PTSD criteria, 11 participants did not report any PTSD symptoms. 51 participants (68% of total sample) reported some PTSD symptoms. Of these 51 participants, 38 participants reported mild symptoms, 11 reported moderate symptoms, 2 reported moderate to severe symptoms (Foa, 1995). For the 51 participants who reported at least mild PTSD symptom severity, the level of impairment associated with symptoms was explored. 18 participants reported severe impairment in functioning, 6 reported moderate impairment, 11 mild impairment and 16 no impairment (Foa, 1995).

**Prevalence of other psychiatric symptoms**

The mean total score on the GHQ-28 was 20.24 (SD = 14.85, range 5 – 71). The scores for the GHQ-28 subscales were: anxiety and insomnia, mean = 4.82 (SD = 4.88, range 0 – 21); depression, mean = 1.85 (SD = 4.14, range 0 – 18); social dysfunction, mean = 9.30 (SD = 3.9, range 0 – 21) and somatic symptoms, mean = 4.62 (SD = 4.25, range 0 – 21).

Using screening scores for the GHQ-28, 21 (28%) participants met criteria for psychiatric symptoms (a score of 7 or more: Goldberg et al., 1997).

**Relationship between PTSD symptoms and other psychiatric symptoms**

A Spearman's rank order correlation showed a significant association between GHQ-28 total scores and PTSD symptom severity as measured by the PDS ($r_s = 0.785$, $p < 0.0005$). Spearman's rank order correlations were also significant between PTSD symptom severity and the GHQ-28 subscales for anxiety and insomnia ($r_s = 0.751$, $p < 0.0005$), depression ($r_s = 0.620$, $p < 0.0005$), social dysfunction ($r_s = 0.552$, $p < 0.0005$) and somatic symptoms ($r_s = 0.545$, $p < 0.0005$).
Of the 12 participants meeting criteria for acute PTSD, 7 (58%) were identified as psychiatric cases using the GHQ-28. However, 5 (42%) participants meeting criteria for acute PTSD were not classified as cases on the GHQ-28. For the 62 participants who did not meet acute PTSD criteria, 14 met criteria for caseness on the GHQ-28.

Relationship between demographic data, participants' experiences prior to MI and PTSD symptoms.
Several analyses were carried out to determine whether there were any relationships between demographic data and participants' experiences prior to MI and PTSD symptom severity as measured by the total symptom severity score on the PDS.

For marital status, ethnic background, education level and employment status the categories were collapsed into two categories owing to small numbers of participants in some categories. For example, for marital status the categories 'single', 'married/cohabiting', 'widowed' and 'divorced/separated' were collapsed into two categories - 'married/cohabiting' and 'not married/cohabiting.' Therefore, Mann-Whitney U tests were used to analyse categorical demographic and participants' experiences prior to MI data. Table 7 shows the results of the Mann-Whitney U tests.
Table 7. Mann-Whitney U test results

<table>
<thead>
<tr>
<th></th>
<th>Median symptom severity score</th>
<th>Mann-Whitney U</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Gender</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male (n=56)</td>
<td>6</td>
<td>z =-0.019</td>
<td>p=0.985</td>
</tr>
<tr>
<td>Female (n=18)</td>
<td>6</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Marital Status</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Married / cohabiting (n=52)</td>
<td>7</td>
<td>z =-0.071</td>
<td>p=0.943</td>
</tr>
<tr>
<td>Not married / cohabiting (n=22)</td>
<td>6</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Ethnic background</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>White UK (n=67)</td>
<td>6</td>
<td>z =-0.463</td>
<td>p=0.643</td>
</tr>
<tr>
<td>Other (n=7)</td>
<td>11</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Education level</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No formal qualifications (n=33)</td>
<td>8</td>
<td>z =-0.02</td>
<td>p=0.997</td>
</tr>
<tr>
<td>'O' level of above (n=38)</td>
<td>6</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Employment status</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Working full / part time (n=26)</td>
<td>6</td>
<td>z =-0.136</td>
<td>p=0.892</td>
</tr>
<tr>
<td>Not working (n=48)</td>
<td>7</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Prior hospitalisation for cardiac related problems</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes (n=6)</td>
<td>13</td>
<td>z =-0.814</td>
<td>p=0.416</td>
</tr>
<tr>
<td>No (n=68)</td>
<td>6</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Experience of traumatic events other than MI</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes (n=41)</td>
<td>8</td>
<td>z =-2.803</td>
<td>p=0.005</td>
</tr>
<tr>
<td>No (n=33)</td>
<td>4</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Psychological difficulties prior to MI</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes (n=10)</td>
<td>17</td>
<td>z =-2.711</td>
<td>p=0.007</td>
</tr>
<tr>
<td>No (n=64)</td>
<td>6</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* n= 71

There were no significant differences in PTSD symptom severity scores for gender, marital status, ethnic background, education level, employment status or having been hospitalised for cardiac related problems.

There was a significant difference in PTSD symptom severity scores for people who had previously experienced traumatic events other than MI and those who had not. Participants who had previously experienced traumatic events other than MI reported higher PTSD symptom severity scores (median = 8) than participants who had not experienced prior traumatic events (median = 4). There was also a significant difference in PTSD symptom severity scores for people who reported psychological...
problems prior to MI and those who did not. Participants reported who psychological problems prior to MI had higher PTSD symptom severity scores (median = 17) than participants who did not report previous psychological problems (median = 6).

For continuous demographic data e.g. participants' age and the time in weeks since MI, Spearman's rank order correlations were used to examine the relationship between these variables and PTSD symptom severity, as measured by the total symptom severity score on the PDS. There was no significant relationship between participants' ages ($r_s = -0.070$, $p = 0.551$) or time since MI ($r_s = 0.227$, $p = 0.052$) and PTSD symptom severity scores.

**Relationship between PTSD symptom severity and subjective experiences during MI.**

Spearman's rank order correlations were used to examine the relationship between participants' perceived severity of MI and perceived threat to life, at the time MI occurred, and PTSD symptom severity, as measured by the total symptom severity score on the PDS.

There were significant, positive correlations between PTSD symptom severity and perceived severity of MI at the time it occurred ($r_s = 0.292$, $p = 0.013$) and perceived threat to life at time of MI ($r_s = 0.377$, $p = 0.001$).

**Test of main hypotheses - Correlations between negative appraisals, dysfunctional coping strategies and PTSD symptom severity scores.**

Firstly, Spearman's rank order correlations were used to examine the relationship between perceptions of permanent change, appraisals of symptoms, use of dysfunctional cognitive and behavioural coping strategies and PTSD symptom severity, as measured by the total symptom severity score on the PDS.

There were significant positive correlations between PTSD symptom severity and perceptions of permanent change ($r_s = 0.595$, $p < 0.0005$), appraisals of symptoms ($r_s = 0.486$, $p < 0.0005$) and use of dysfunctional cognitive and behavioural coping...
strategies ($r_s = 0.666, p<0.0005$). Thus, as predicted in the main hypotheses for the study, the severity of PTSD symptoms was significantly associated with negative perceptions of permanent change, negative appraisals of symptoms and more frequent use of dysfunctional cognitive and behavioural coping strategies.

However, in other trauma populations, depression and PTSD symptoms are often co-morbid (Yule, Williams & Joseph, 1999). Depression is also associated with MI (Bennett et al., 1999). Indeed, in this study, there was a significant association between GHQ-28 depression scores and PTSD symptom severity. Therefore, it is possible that the significant correlations between PTSD symptom severity and the three main variables investigated in this study could partly attributed to the presence of depressive symptoms. Thus, partial correlations were calculated between negative perceptions of permanent change, negative appraisals of symptoms, use of dysfunctional cognitive and behavioural coping and total PTSD symptom severity, controlling for GHQ-28 depression scores. For total PTSD symptom severity scores, a logarithmic transformation was applied to normalise the distributions (Tabachnick & Fidell, 1996). Although GHQ-28 depression scores were positively skewed, transformations either did not normalise the distribution or made it negatively skewed. Therefore, GHQ-28 depression scores were not transformed (Tabachnick & Fidell, 1996).

The partial correlations between total PTSD symptom severity and perceptions of permanent change ($r_p=0.443, p<0.0005$), appraisals of symptoms ($r_p=0.240, p=0.042$) and dysfunctional cognitive and behavioural coping strategies ($r_p=0.558, p<0.0005$), were all significant when controlling for GHQ-28 depression scores. Thus, the associations between PTSD symptom severity and the three main variables investigated in this study were not solely attributable to the presence of depressive symptoms.

Finally, both Dunmore et al., (in press) and Steil and Ehlers (2000) point out that there is some overlap between the cognitive and behavioural coping strategies assumed to be important in the development and maintenance of PTSD and avoidance symptoms of PTSD. Therefore, the partial correlation between (log of)
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total PTSD symptom severity and use of dysfunctional cognitive and behavioural coping strategies was calculated, controlling for GHQ-28 depression scores and (log of) the avoidance subscale of the PDS. This partial correlation was significant ($r_p=0.2873$, $p=0.017$). Thus, use of dysfunctional cognitive and behavioural coping strategies was associated with PTSD symptoms, other than those concerned with avoidance.

**Multiple regression analysis**

Firstly, to determine how much of the variance in PTSD symptom severity could be explained by the three main variables investigated in this study (perceptions of permanent change, appraisals of symptoms and use of dysfunctional cognitive and behavioural coping strategies), a multiple regression analysis was carried out. Standard multiple regression analysis (method enter) was used chosen because of the relatively small sample size (Brace, Kemp & Snelgar, 2000).

For PTSD symptom severity, as measured by the total symptom severity score on the PDS, a logarithmic transformation was applied, to improve the normality, linearity and homoscedasticity of residuals (Tabachnick & Fidell, 1996). Perceptions of permanent change, appraisals of symptoms and use of dysfunctional coping strategies were not transformed as these variables were normally distributed. Using the SPSS casewise diagnostic, no outliers were found.

Perceptions of permanent change, appraisals of symptoms and use of dysfunctional coping strategies were entered into the multiple regression equation simultaneously. Together these variables explained 48.8% ($R^2$) of the variance in PTSD symptom severity ($R^2=0.488$, Adjusted $R^2 = 0.465$, $F_{3,67} = 21.309$, $p<0.0005$). Perceptions of permanent change (standardised $\beta = 0.312$, $p=0.018$) and use of dysfunctional cognitive and behavioural coping strategies (standardised $\beta = 0.469$, $p<0.0005$) each explained unique variance in PTSD symptom severity. Despite a significant bivariate correlation between (log of) PTSD symptom severity and appraisals of symptoms ($r=0.486$, $p=<0.0005$), appraisals of symptoms did not make a unique contribution to the variance in PTSD symptom severity (standardised $\beta = 0.024$,
p=0.840). This may be due to the significant correlations between appraisals of symptoms and the other factors in the equation, suggesting that the variance in PTSD symptom severity scores explained by appraisals of symptoms is shared with the other factors (Green, Salkind & Akey, 2000). Table 8 shows the Pearson’s correlations between the three main variables.

Table 8. Pearson’s correlations between perceptions of permanent change, appraisals of symptoms and dysfunctional cognitive and behavioural coping strategies.

<table>
<thead>
<tr>
<th>Variable</th>
<th>1</th>
<th>2</th>
<th>3</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Perceptions of permanent change</td>
<td>-</td>
<td>0.684 ((p&lt;0.0005))</td>
<td>0.555 ((p&lt;0.0005))</td>
</tr>
<tr>
<td>2. Appraisals of symptoms</td>
<td>-</td>
<td>-</td>
<td>0.403 ((p&lt;0.0005))</td>
</tr>
<tr>
<td>3. Coping strategies</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

A second multiple regression analysis was carried out to determine the amount of variance in total PTSD symptom severity that was explained by the three main variables, over and above GHQ-28 depression scores and other significant factors. Other significant factors were those which had been found to be significantly associated with PTSD symptom severity in the previous analyses. These were perceived severity of MI and perceived threat to life at the time of MI, experience of previous traumas and psychological problems prior to MI.

For total PTSD symptom severity scores and perceived threat to life scores, logarithmic transformations were applied for this analysis to normalise the distributions and to improve the normality, linearity and homoscedasticity of residuals (Tabachnick & Fidell, 1996). As before, GHQ-28 depression scores were not transformed as transformations either did not normalise the distribution or made it negatively skewed. All other continuous variables were normally distributed. The variable ‘psychological problems prior to MI’ had a large, uneven distribution of participants. When this is the case, Tabachnick and Fidell (1996) suggest that inclusion of such variables in a regression equation can produce outliers in the solution. However, using the SPSS casewise diagnostic, no outliers were found.
when 'psychological problems prior to MI' was included in the regression equation.

GHQ-28 depression scores were entered into the equation first, then the variables perceived severity of MI, perceived threat to life, experience of previous traumas and previous psychological problems were simultaneously forced into the regression equation, using method enter (Tabachnick & Fidell, 1996). Perceptions of permanent change, appraisals of symptoms and use of dysfunctional coping strategies were then simultaneously added to the equation in the final step.

With GHQ-28 depression scores in the regression equation, 22% of the variance in PTSD symptom was explained ($R^2 = 0.220$, Adjusted $R^2 = 0.208$, $F_{1,67} = 18.862$, $p<0.0005$). In the second step, perceived severity of MI, perceived threat to life, experience of previous traumas and previous psychological problems accounted for an additional, significant proportion of the variance after controlling for GHQ-28 depression scores ($R^2$ change $= 0.208$, $F_{4,63} = 5.739$, $p<0.0005$). With a combination of GHQ-28 depression scores, perceived severity of MI, perceived threat to life, experience of previous traumas and previous psychological problems included in the equation, 42.8% of the variance in PTSD symptom severity was explained ($R^2 = 0.428$, Adjusted $R^2 = 0.383$, $F_{5,62} = 9.431$, $p<0.0005$).

In the final step, when perceptions of permanent change, appraisals of symptoms and use of dysfunctional coping strategies were added to the equation, 58.5% of the variance in PTSD total symptom severity was explained ($R^2 = 0.585$, Adjusted $R^2 = 0.529$, $F_{8,60} = 10.554$, $p<0.0005$). Perceptions of permanent change, appraisals of symptoms and use of dysfunctional coping strategies accounted for a significant proportion of the variance in PTSD symptom severity, when controlling for GHQ-28 depression scores, perceived severity of MI, perceived threat to life, experience of previous traumas and previous psychological problems ($R^2$ change $= 0.157$, $F_{3,60} = 7.335$, $p<0.0005$).

However, only use of dysfunctional coping strategies (standardised $\beta = 0.346$, $p=0.001$) explained unique variance in PTSD symptom severity. Appraisals of
symptoms (standardised $\beta = 0.031$, $p=0.809$) and perceptions of permanent change (standardised $\beta = 0.208$, $p=0.110$) did not explain any unique variance in PTSD symptom severity once GHQ-28 depression scores, perceived severity of MI, perceived threat to life, experience of previous traumas and previous psychological problems had been controlled for.

**Discussion**

The aims of the present study were: (1) to collect information regarding the prevalence of PTSD symptoms following MI; and (2) to investigate factors associated with PTSD symptoms following MI. In particular, to explore associations between perceptions of permanent change, appraisals of symptoms and use of dysfunctional cognitive and behavioural coping strategies and PTSD symptoms following MI.

**Prevalence and impact of PTSD symptoms following MI**

12 participants (16%) met DSM-IV (American Psychiatric Association, 1994) criteria for acute PTSD following MI, as measured by the PDS (Foa et al., 1997). All 12 participants reported moderate to severe PTSD symptoms and for the majority (75%), PTSD symptoms were associated with severe impairment of functioning, affecting all areas of life.

The prevalence rate of 16% for acute PTSD following MI in this study is slightly higher than the prevalence rate of 5-10% for acute PTSD reported by Kutz et al. (1988) and 9% reported by Kutz et al. (1994). However, Kutz et al. (1988) describe acute PTSD as symptoms which “subsequently subsided within the year following the coronary event” (pg. 169) and therefore cannot be directly compared with the results of this study. Likewise, in the Kutz et al. (1994) study, acute PTSD was defined as symptoms lasting less that six months and a diagnosis was made retrospectively. Other published studies (e.g. Doefler et al., 1994; Van Driel & Op den Velde, 1995; Bennett & Brooke, 1999) have reported prevalence rates for chronic PTSD following MI (symptom duration of more than three months) of between 5% and 16%. There is no data available on the typical duration or recovery...
rates for acute and chronic PTSD symptoms following MI. However, given that PTSD symptoms spontaneously remit somewhat over time in other populations (Ehlers & Clark, 2000: Dunmore et al., in press), the prevalence rate of 16% for acute PTSD in this study is not inconsistent with studies that report rates of chronic PTSD following MI of between 5% and 16%.

In this study, of the remaining 62 participants who did not reach DSM-IV (American Psychiatric Association, 1994) criteria for acute PTSD, 51 (68% of total sample) reported mild to moderate/severe symptoms of PTSD. For approximately half of these participants, PTSD symptoms were associated with moderate to severe impairment in functioning.

Thus, the results of this study support Green et al.'s (1997) conclusion that following MI small but significant proportions of people are at risk of developing PTSD and that a larger number experience subsyndromal levels of PTSD symptoms. In addition, the severity of PTSD symptoms and levels of impairment associated with PTSD symptoms for both participants with acute PTSD and participants with subsyndromal levels of PTSD in this study, highlights clinical significance of PTSD symptoms following MI.

However, due to a moderate response rate of 51%, it is difficult to determine the actual prevalence rate of acute and subsyndromal PTSD symptoms following MI. The prevalence rate of PTSD symptoms may have been underestimated. As Bennett and Brooke (1999) suggest people with PTSD symptoms may not have completed questionnaires in order to avoid being reminded of their MI. This possibility was tentatively explored by comparing responders and non-responders from site one on the Hospital Anxiety and Depression Scale (Zigmond & Sniath, 1983), administered routinely as part of the cardiac rehabilitation programme. There was a significant difference between responders and non-responders on HADS anxiety scale scores, with non-responders reporting higher HADS anxiety scores. This suggests that people experiencing anxiety, and possibly PTSD symptoms following MI may have opted not to complete the questionnaires. Indeed, several people commented to the researcher that they were declining to take part in the study as they were too
‘stressed’ or ‘upset’ by their MI or preferred not to think about their MI.

**Prevalence of other psychiatric symptoms and detection of PTSD symptoms following MI**

21 (28%) participants met criteria for the presence of psychiatric symptoms following MI (anxiety and insomnia, depression, social dysfunction, somatic symptoms) as measured by the General health Questionnaire-28 (Goldberg & Hillier, 1978). This is similar to prevalence rates for psychiatric symptoms in the first three months after MI found in other studies (Terry, 1992; Bennett et al., 1999).

There was some overlap between those participants identified as reaching criteria for acute PTSD following MI and those reaching caseness on the GHQ-28. However, 5 (42%) participants who met criteria for acute PTSD following MI were not identified as ‘cases’ on the GHQ-28. Thus, general screening measures for psychological distress, employed in research on MI and in cardiac rehabilitation services may ‘miss’ some people with significant symptoms of PTSD - a problem highlighted by Shalev et al. (1993). Moreover, in a national survey of cardiac rehabilitation programmes in the UK, only a quarter used validated assessments of anxiety and depression (NHS Centre for Reviews and Dissemination, 1998). Thus, more consistent use of psychological assessments is needed in order to detect people suffering from psychiatric symptoms, including PTSD following MI. In addition, given the difficulty in identifying people with PTSD symptoms with general screening measures, use of specific assessments of PTSD symptom may also need to be considered. However, routine assessment of posttraumatic stress disorder symptoms following MI, may not be appropriate until more is known about the nature, duration and impact of such symptoms.

**Factors associated with PTSD symptoms following MI**

*Demographic data and previous experiences*

The association between age, gender, marital status, ethnic background, educational level, employment status, prior hospitalisation for cardiac related problems, previous experience of traumatic events other than MI, previous psychological problems and PTSD symptom severity was investigated.
Only experience of previous psychological problems prior to MI and experience of other traumatic events other than MI were significantly associated with PTSD symptom severity. Younger age of participants (Bennett & Brooke, 1999), prior hospitalisation for cardiac problems and ethnic background (Kutz et al., 1994) have been found to be associated with PTSD symptoms following MI in other studies. However, in this study there was no significant association between PTSD symptom severity and age, ethnic background or prior hospitalisation for cardiac problems. There may be several reasons for this. Firstly, numbers of participants in this study who had experienced prior hospitalisation for cardiac problems or who were from ethnic backgrounds other than White UK were low. Secondly, previous research on factors associated with PTSD symptoms following MI is limited and younger age of participants and prior hospitalisation for cardiac problems and ethnic background have been found to be associated with PTSD symptoms following MI in only one study each. Therefore, at present no conclusions can be drawn about the relative importance of such factors in PTSD symptoms following MI. Furthermore, there are inconsistent findings from studies in other trauma populations regarding the association between age, ethnic background and PTSD symptoms (e.g. Foa & Riggs, 1993).

The association between experience of previous psychological problems prior to MI and experience of other traumatic events other than MI and PTSD symptom severity is consistent with research in other populations. For example, studies have demonstrated an association between previous psychological problems and both acute and chronic PTSD symptoms following different types of trauma including, assault (Dunmore et al, 1999; in press) and childbirth (Czarnocka & Slade, 2000). An association between experience of previous traumatic events and PTSD has also been found in other populations, such as assault victims (Dunmore et al, 1999 & in press), cancer patients (Smith et al., 1999) and people diagnosed with HIV (Kelly et al., 1998). It is also in line with theoretical models of PTSD that suggest previous psychological problems and previous experience of trauma make a person more ‘vulnerable’ to developing PTSD symptoms following a subsequent trauma (Ehlers & Clark, 2000; Foa & Riggs, 1993). For example, Ehlers and Clark (2000) and Foa
and Riggs (1993) suggest that a subsequent trauma may reactivate emotions, beliefs and memories associated with a previous trauma. Likewise, people who have experienced psychological problems may already hold negative beliefs about themselves. A traumatic experience may activate these pre-existing beliefs and contribute to the development of PTSD symptoms (Foa & Riggs, 1993).

When considering the association between previous psychological problems, previous traumatic events and PTSD symptom severity following MI found in this study, several points can be raised. Firstly, 55% of the participants in this study reported experiencing traumatic events other than MI. Although this figure may seem high, it is consistent with the estimated lifetime prevalence rate for traumatic events in the general population (Alonzo, 2000, Yule et al., 1999). Secondly, 9 out of the 10 participants who reported psychological problems prior to MI also reported experiencing previous traumatic events. Although no data was collected regarding the type of psychological problems experienced, it is possible these people experienced PTSD symptoms prior to their MI. The MI may then have exacerbated these pre-existing symptoms (Alonzo, 2000; Hamner, 1994). However, it seems unlikely that pre-existing PTSD symptoms, exacerbated by MI could solely account for PTSD symptom severity following MI found in this study. For example, when examining the characteristics for the 12 participants reaching diagnostic criteria for acute PTSD (Appendix 17), only 4 of these people reported previous psychological problems.

**Subjective experiences during MI**

In relation to perception of severity of MI and perceived threat to life at time of MI, higher scores on both measures were significantly associated with PTSD symptom severity. This finding is in line with research on PTSD in other populations. For example, perceived threat to life has been found to be associated with PTSD symptoms in assault victims (Dunmore et al., in press) and people involved in road traffic accidents (Ehlers, et al., 1998). In addition, the association between perceived severity of MI and perceived threat to life at time of MI and PTSD symptom severity highlights the link between how an event is appraised, what meaning it has for that individual and degree of distress experienced (Ehlers & Steil, 1995; Joseph, 1999).
**Perceptions of permanent change, appraisals of symptoms and use of dysfunctional cognitive and behavioural coping strategies**

The main aim of the study was to investigate the association between perceptions of permanent change, appraisals of symptoms and use of dysfunctional cognitive and behavioural coping strategies, as outlined in Ehlers and Clark's (2000) model of PTSD, and PTSD symptom severity following MI.

However, as with previous studies (Yule et al., 1999) PTSD symptom severity was associated with symptoms of depression. Similar to cognitive models of PTSD, cognitive models of depression highlight the importance of negative thoughts about the self, the world and the future (Beck, 1976). This raised the possibility that the relationships between the three cognitive factors and PTSD symptom severity, was mediated by the presence of symptoms of depression. This possibility was investigated by examining the correlations between negative perceptions of permanent change, negative appraisals of symptoms and more frequent use of dysfunctional cognitive and behavioural coping strategies and PTSD symptoms when controlling for symptoms of depression.

**Main hypothesis 1 - More severe PTSD symptoms will be associated with negative perceptions of permanent change**

Negative perceptions of permanent change were significantly associated with PTSD symptom severity both before and after symptoms of depression were controlled for. Furthermore, negative perceptions of permanent change following MI significantly predicted PTSD symptom severity over and above what could be predicted by negative appraisals of symptoms and use of dysfunctional cognitive and behavioural strategies.

The association between negative perceptions of permanent change following MI and PTSD symptom severity is consistent with previous research on assault victims (Dunmore et al., in press). In addition, Kutz et al. (1988) found that people who anticipated significant disability during the early stages of recovery following MI were more likely to develop PTSD symptoms, regardless of the objective severity of
the MI. Conversely, people who initially denied any disability following MI were less likely to develop PTSD symptoms. This corresponds with the finding that initial denial of the impact of MI corresponds to lower levels of anxiety and depression in the longer term (Havik & Maeland, 1990). In addition, Janoff-Bulman (1989) suggests that some level of denial following traumatic events is an adaptive response as denial may allow the individual time to slowly adjust to a trauma. It then follows that people who hold negative appraisals of the trauma and believe that it will have a permanent negative impact on their life will be less successful in adjusting to the trauma and may go on to experience PTSD symptoms. Dunmore et al. (in press) suggest that negative perceptions of permanent change may link to symptoms of diminished interest, a sense of forshortened future and difficulty in accepting and processing the traumatic event. Furthermore, Ehlers and Clark (2000) suggest that negative perceptions of permanent change serve to create a sense of current and continued threat from the traumatic event. It is this sense of current threat that Ehlers and Clark (2000) argue is central to the development and maintenance of PTSD.

However, perceptions of permanent change did not contribute unique variance to PTSD symptom severity over and above than explained by appraisals of symptoms and use of dysfunctional coping strategies, once GHQ-28 depression scores, perceived severity of MI, perceived threat to life, experience of previous traumas and previous psychological problems had been controlled for. This suggests that the variance in PTSD symptom severity explained by negative perceptions of permanent change is shared with other factors.

**Main Hypothesis 2 – More severe PTSD symptoms will be associated with negative appraisals of symptoms**

With regards to appraisals of symptoms, both appraisals of post-MI reactions in general and specific PTSD symptoms were assessed. Negative appraisals of symptoms were significantly associated with PTSD symptom severity both before and after symptoms of depression were controlled for.

The significant correlation between negative appraisals of symptoms and PTSD symptom severity following MI is in line with research on assault victims (Dunmore
et al., in press), ambulance workers (Clohessy & Ehlers, 1999) and motor vehicle accident victims (Steil & Ehlers, 2000). As Ehlers and Clark (2000) suggest, symptoms such as intrusive recollections, irritability, mood swings, and lack of concentration are common reactions in the immediate aftermath of a traumatic event. However, if individuals do not view these symptoms as a part of a normal reaction and instead interpret them as evidence that they are 'going crazy' or cannot cope, this is likely to increase levels of distress (Ehlers & Clark, 2000). As with negative perceptions of permanent change, negative appraisals of symptoms may serve to maintain a sense of current threat from the traumatic event (Ehlers & Clark, 2000). In addition, negative appraisals of symptoms may motivate people to engage in dysfunctional coping strategies, which actually enhance PTSD symptoms (Ehlers & Clark, 2000).

However, appraisals of symptoms did not explain any unique variance in PTSD symptom severity over and above that explained by perceptions of permanent change and use of dysfunctional cognitive and behavioural coping strategies, both before and after controlling for other significant variables. There may be several reasons for this. It could be argued that the types of appraisals measured in this study were not as meaningful to MI patients as other trauma populations or that generally appraisals of symptoms is not as relevant to PTSD in MI patients as in other populations. However, this interpretation seems unlikely, given the significant bivariate correlation found between PTSD symptom severity following MI and negative appraisals of symptoms. A more likely explanation is suggested by examining the intercorrelations between the three main variables investigated in this study. Negative appraisals of symptoms were significantly correlated with perceptions of permanent change and use of dysfunctional cognitive and behavioural coping strategies. This suggests that the variance in PTSD symptom severity scores explained by appraisals of symptoms was shared with the other factors (Green, et al., 2000). Indeed, Ehlers and Clark (2000) and Steil and Ehlers (2000) suggest that the prime mechanism whereby negative appraisals of symptoms are associated with PTSD symptoms is that negative appraisals of symptoms motivate people to engage in dysfunctional coping strategies. In addition, the two studies which have investigated appraisals of symptoms, along with other cognitive factors, tested the
amount of variance explained by each variable separately e.g. separate regression equations were run for each variable (Dunmore et al., 1999, in press). Therefore, there is no previous data with which to compare the relative importance of appraisals of symptoms compared to perceptions of permanent change and use of dysfunctional cognitive and behavioural coping strategies.

**Main hypothesis 3 – More severe PTSD symptoms will be associated with use of dysfunctional cognitive and behavioural coping strategies**

The dysfunctional coping strategies investigated in this study included behavioural and cognitive avoidance (distraction, suppression), detachment and 'undoing' or rumination. More frequent use of dysfunctional cognitive and behavioural coping strategies was significantly associated with PTSD symptom severity both before and after symptoms of depression were controlled for. In addition, use of dysfunctional cognitive and behavioural coping strategies explained unique variance in PTSD symptom severity over and above that explained by perceptions of permanent change and use of dysfunctional cognitive and behavioural coping strategies, both before and after controlling for other significant variables.

The association between use of dysfunctional cognitive and behavioural coping strategies and PTSD symptom severity is consistent with previous research on PTSD. Dysfunctional coping strategies have been found to be associated with PTSD symptoms in victims of assault (Dunmore et al, 1999, in press) motor vehicle accident victims (Ehlers et al., 1998; Steil & Ehlers, 2000).

However, both Dunmore et al., (in press) and Steil and Ehlers (2000) argue that there is some overlap between PTSD symptoms and coping strategies e.g. avoidance coping strategies and PTSD symptoms related to avoidance. Therefore, the possibility that cognitive and behavioural avoidance items in the dysfunctional cognitive and behavioural coping strategies questionnaire were solely responsible for the significant correlation between dysfunctional coping strategies and PTSD symptom severity was explored. When controlling for avoidance symptoms on the PDS, the association between other PTSD symptoms and dysfunctional cognitive and behavioural coping strategies remained significant. This is consistent with
suggestion that dysfunctional coping strategies are associated with re-experiencing symptoms in PTSD (Steil & Ehlers, 2000). In addition, Ehlers and Clark (2000) suggest that dysfunctional coping strategies are associated with PTSD symptoms not only by directly producing PTSD symptoms but also by (1) preventing change in negative appraisals of the trauma and its sequelae and; (2) preventing change in the nature of the trauma memory.

**Limitations of study**

*Sample size and statistical analyses*

As previously discussed in the section on prevalence of PTSD symptoms following MI, the response rate for the study was moderate and it is possible that people with PTSD symptoms declined to take part. This may limit the generalizability of the findings presented.

The relatively small sample size in this study may also limit the generalizability. In particular, the results of the multiple regression analyses may have been affected by the sample size as the ratio of cases-to-number of factors in the regression equation was smaller than generally recommended (Tabachnick & Fidell, 1996). However, as the nature of the study was essentially exploratory, multiple regression analyses were carried out to further examine the relationships between PTSD symptom severity following MI and perceptions of permanent change, appraisals of symptoms and cognitive and behavioural coping strategies, when controlling for other significant factors.

*Research design*

The use of a cross-sectional research design prevents any conclusions being drawn about the role of perceptions of permanent change following MI, appraisals of symptoms and use of dysfunctional cognitive and behavioural coping strategies in the development of PTSD symptoms following MI. What can be said is that there is evidence from this study that these factors may be associated with PTSD symptoms following MI. In particular, use of dysfunctional cognitive and behavioural coping strategies was associated with PTSD symptom severity when controlling for other significant factors. However, further research is needed to confirm the associations
between perceptions of permanent change following MI, appraisals of symptoms, use of dysfunctional cognitive and behavioural coping strategies and PTSD symptoms following MI. In addition, prospective research is needed to investigate whether these variables are predictive of both acute and chronic PTSD following MI.

Assessment of PTSD symptoms

PTSD symptoms were assessed using the PDS, which is a self-report questionnaire. Although the PDS has satisfactory agreement with the Structured Clinical Interview for PTSD (SCID-R-PTSD: Spitzer, Williams, Gibbon & First, 1990), it is possible that ratings were influenced by response bias. As Foa (1995) points out, the PDS is a tool to aid diagnosis of PTSD and is not intended replace a structured diagnostic interview. Therefore, it would have been desirable to confirm the prevalence rate for acute PTSD via interview. However, the study was mainly concerned with factors associated with PTSD symptom severity. Therefore, the use of the PDS was judged as adequate for this purpose.

Questionnaires measuring perceptions of permanent change, appraisals of symptoms and dysfunctional coping strategies.

The exploratory nature of this study required that cognitive and behaviour factors hypothesised to be associated with PTSD symptoms be measured by questionnaires developed specifically for this study. The questionnaires for perceptions of permanent change and use of behavioural and cognitive coping strategies had good internal consistency (0.91 and 0.85 respectively) (Barker, Pistrang & Elliot, 1994). The questionnaire for appraisals of symptoms had adequate internal consistency (0.73) (Barker et al., 1994). All measures had face validity and were developed from measures used in previous research studies. However, other forms of validity were not assessed. Thus, the use of these questionnaires may limit the generalizability of the research findings.

With regards to the questionnaire measuring perceptions of permanent change, the items in this questionnaire focussed on perceived change in lifestyle e.g. changes in physical health, self-care, work, relationship and activities. However, it would have been useful to also include items assessing perceptions of negative change in
participants' sense of self. For example, Janoff-Bulman (1989) argues that the impact of trauma on an individual's sense of self worth may be an important factor in adjusting to trauma.

Objective measure of MI severity

Unfortunately, no objective measure of MI severity was included in the study. In the design stage of the research, enquires were made as to appropriate measures of MI severity. Several possibilities were identified by consultant cardiologists at the two participating hospitals, including the Killip class (a standardised assessment based on chest X-ray, heart and lung sounds and signs of shock), peak cardiac enzyme levels and left ventricular injection fraction. However, at the point of data collection several difficulties were encountered. Firstly, Killip class needs to be rated by a medical professional at the time the person is admitted to hospital and would therefore require additional work for clinical staff. Secondly, peak cardiac enzyme levels and left ventricular injection fraction data were not readily available for all participants. Thirdly, measures of severity available differed between participants and across the two participating hospital sites. Therefore, an objective measure of MI severity was not included in this study and the contribution of actual MI severity to PTSD symptoms following MI could not be investigated. However, in other samples, for example road-traffic accident victims (Ehlers, Mayou & Bryant, 1998), there is inconsistent evidence regarding the relationship between objective trauma severity and PTSD symptoms. In addition, research on MI suggests that actual severity of the MI is not strongly associated with either PTSD symptoms specifically (Kutz et al., 1994) or general psychological adjustment following MI (Terry, 1992).

Nevertheless, it would have been desirable to control for objective severity of MI when assessing the relationship between the three main variables investigated in this study and PTSD symptom severity, in particular perceptions of permanent change.

Clinical implications

Bearing in mind the limitations, the results of this study suggest several clinical implications for the identification and treatment of PTSD following MI.
The prevalence rate for both acute PTSD and subsyndromal PTSD symptoms following MI and the level of impairment associated with these symptoms suggests that identification of people ‘at risk’ from developing PTSD is an important clinical issue. The findings of this study suggest that people with a prior history of trauma and psychological problems, and who perceive the MI to be serious and life threatening may experience PTSD symptoms following MI. In addition, people who hold negative appraisals about their symptoms and future and who engage in dysfunctional coping strategies may also experience PTSD symptoms. If these results are supported by further research, it may be useful to assess such factors in cardiac rehabilitation programmes to aid in the identification of people at risk of developing PTSD symptoms.

This study employed questionnaires to assess negative perceptions of permanent change, negative appraisals of symptoms and use of dysfunctional cognitive and behavioural coping strategies. These measures appeared relevant to MI patients and scores on these measures were related to PTSD symptom severity. However, in order to gain clinically relevant data, an individual’s idiosyncratic appraisals and coping strategies would also need to be assessed. In order to gain information relating to appraisals, Ehlers and Clark (2000) suggest asking individuals about the kinds of difficulties that have been more distressing since the traumatic event and to explore the people’s beliefs about their symptoms and their future. In addition, White (2000) suggests that when considering PTSD symptoms following life-threatening illness, it may be helpful to ask specific questions about the person’s beliefs relating to the impact of PTSD symptoms on the course of the illness. To identify use of dysfunctional cognitive and behavioural coping strategies, Ehlers and Clark (2000) suggest that it is useful to ask how the individual is attempting to put the event behind them. This might include asking what they think is the best way of coping with the trauma, what kinds of things they avoid, how they deal with re-experiencing symptoms or what they think might happen if they allow themselves to think about the trauma (Ehlers & Clark, 2000).

With regard to treatment, Ehlers and Clark (2000) suggest given the evidence for an association between negative appraisals of trauma sequelae, dysfunctional coping
strategies and PTSD, treatment approaches for PTSD would need to target these areas. Ehlers and Clark (2000) suggest that the application of cognitive-behaviour therapy (CBT) techniques would be appropriate to try to achieve change in these areas. For example, psychoeducation about symptoms commonly experienced after a traumatic event may help an individual to modify negative appraisals of symptoms (Ehlers & Clark, 2000). Likewise ‘reliving’ the traumatic event e.g. describing the experience in detail in words or in writing may be useful in identifying an individual’s negative appraisals (Ehlers & Clark, 2000). Cognitive restructuring techniques can then be used to modify these appraisals and the person then attempts to incorporate these modified appraisals into subsequent reliving sessions (Ehlers & Clark, 2000). Reliving may also be a power technique to ‘test’ negative appraisals related to the consequences of thinking in detail about the traumatic event (Ehlers & Clark, 2000).

The association between negative perceptions of permanent change, negative appraisals of symptoms and use of dysfunctional cognitive and behavioural coping strategies and PTSD symptoms following MI found in this study, would suggest that attention would also need to be paid to these areas when considering interventions for this client group. CBT is a widely used and effective intervention for people with difficulties adjusting to a range of medical problems (White, 2000). Therefore, CBT techniques used in the treatment of PTSD in other trauma populations, could be used with people suffering from PTSD following MI. While the literature on interventions with people who have PTSD following MI is scant, that which does exist, highlights the application of cognitive-behavioural techniques in helping to modify people’s appraisals of MI and its consequences (Hartman & Burgess, 1985). In addition, Kutz et al. (1988) describe three cases in which techniques such as graded increase in activities, guided imagery, in vivo exposure and relaxation were used to reduce the severity and impact of PTSD symptoms following MI. However, further research is needed on the application of CBT techniques for PTSD following MI.

In this section, potential areas for assessment and treatment of PTSD following MI have been discussed. However, even if the clinical value of such strategies are
demonstrated in future research, the question arises as to how assessment and treatment strategies may be implemented. The provision of NHS clinical psychology services for people with physical health problems varies greatly according to region and medical speciality (White, 2000). In a national survey of cardiac rehabilitation programmes in the UK, only 21% had some input from a clinical psychologist (NHS Centre for Reviews and Dissemination, 1998). Indeed, for the two hospital sites participating in this research project, only one had direct access to a clinical psychologist.

Areas for future research

This study investigated several of factors shown to be associated with PTSD in other populations. The main focus of the study was on appraisals of the sequelae of MI e.g. negative perceptions of permanent change, negative appraisals of symptoms and use of dysfunctional cognitive and behavioural coping strategies. Further research is needed to confirm the association between these variables and PTSD symptoms following MI and to address the limitations of this study, outlined above.

However, given the potential application of Ehlers and Clark's (2000) model of PTSD in understanding PTSD symptoms following MI, future research could explore other types of appraisals outlined in the Ehlers and Clark (2000) model of PTSD. For example, negative appraisals of behaviour such as self-blame, guilt, and internal causal attributions for the trauma are associated with PTSD symptoms (Dunmore et al, 1999; Joseph, Brewin, Yule & Williams, 1991). Such appraisals may seem more relevant to people who have experienced traumatic events such as assault. However, there evidence to suggest that people who have had an MI also experience a sense of guilt and self-blame (Petrie & Weinman, 1997). For example, in a qualitative study, Johnson and Morse (1990) found that MI patients frequently believed that they had had a MI because of something they did ‘wrong’ - “It’s the old story, I guess...I’ve worked hard. I’ve put in long hours, lots of stress. I’ve worked for this heart attack and I got it.....I guess you could say I got what I deserved” (pg. 129).

Likewise, appraisals of other people’s reactions following MI may be an area for future research on PTSD symptoms following MI. Bennett and Brooke (1999) found
that lower availability of social support following MI was associated with PTSD symptoms. However, the nature and quality of social support and how the support is perceived may be as important as the amount and availability of social support (Joseph, 1999). In addition, when considering PTSD symptoms following life threatening illness or medical procedures, appraisals of medical staff’s reactions and provision of support may also be important. For example, Czarnocka and Slade (2000) found that the perceived supportiveness of staff and the degree to which women felt that their wishes and views were listened to by staff was significantly associated with PTSD symptoms following childbirth.

Conclusions

An MI can have a significant impact on the individual. While research has focussed on anxiety and depression following MI, relatively little attention has been paid to the possibility that an MI may act as a trigger for PTSD symptoms in some people (Alonzo, 1999). However, several studies suggest that a significant proportion of people may experience PTSD symptoms following MI. The results of this study support this conclusion. The nature and impact of PTSD symptoms following MI, may therefore need to be taken into account when considering the provision of cardiac rehabilitation services. Identification of factors associated with PTSD following MI would aid in the development of a profile of those patients who are at risk. Such people could then be closely monitored and offered treatment where necessary. In this study, previous psychological problems, prior experience of trauma, perceived severity and threat to life at the time of MI, negative appraisals of symptoms, negative perceptions of permanent change and use of dysfunctional cognitive and behavioural coping strategies were found to be associated with PTSD symptom severity following MI. These findings are consistent with research on PTSD after other types of trauma and with cognitive models of PTSD. However, more research is necessary to further our understanding of the role that negative appraisals of symptoms, negative perceptions of permanent change and use of dysfunctional cognitive and behavioural coping strategies play in PTSD following MI.
References


Medicine, 31, 23-30.


Dear Mrs Copland

Re: An investigation into cognitive appraisals and coping strategies associated with posttraumatic stress disorder (PTSD) following myocardial infarction - 0033.11

Thank you for your letter of 4 July 2000. The points raised by the Committee have now been dealt and I am happy to approve the commencement of the study along with the two changes.

Wearing my chaplaincy 'hat', I think the question about religious belief and practice are an interesting addition. It might help to include C/E Anglican and Free Church among religious denominations, rather than Protestant. But doing that, or not, does not affect our approval.

With all good wishes.

Yours sincerely

Canon Ian Ainsworth-Smith
Chairman
Local Research Ethics Committee

Please Note: All research should be conducted in accordance with the guidelines of the Ethical Committee; the reference number allocated to the project should be used in all correspondence with the Committee and the Committee should be informed:

(a) when the project is complete.
(b) what stage the project is at one year from today's date.
(c) if any alterations are made to the treatment or protocol which might have affected ethical approval being granted.
(d) all investigators whose projects have been approved by this Committee are required to report at once any adverse experience affecting subjects in the study and at the same time state the current total number of Serious Adverse Events that have occurred.
Appendix 2

NORTH WEST SURREY LOCAL RESEARCH ETHICS COMMITTEE

The Ridgewood Centre
Old Bisley Road
Frimley
Camberley
Surrey
GU16 5QE

Direct Line: 01276 605325
Fax: 01276 605496
DX: 118800 Frimley 2
Chairperson: Mr David Harrison
Administrator: Ms Sarah-Jane Richards
E-Mail: sjrich.wsha@fsmail.net

Date: 5th December 2000

Mrs Claire Copland
Trainee Clinical Psychologist
Frimley Park Hospital
DX 119050
FRIMLEY 3

Dear Mrs Copland

PRO/120/00 - An investigation into cognitive appraisals and coping strategies associated with post-traumatic stress disorder (PTSD) following myocardial infarction

Thank you for submitting and presenting the above protocol at the North West Surrey ethics committee meeting held on the 1st December 2000. I am pleased to confirm that the committee have granted your study and application full ethical approval.

For your information the following documentation was reviewed:

- Research Proposal
- CV (Claire Copland)
- Background Information Questionnaire
- General Health Questionnaire
- Approval Letter from St Georges Healthcare LREC
- Introduction Letter
- Information Sheet
- Consent Form
- Approval Letter from Dr MJ Boyd (Consultant Physician @ FPH)
- Approval Letter from Diane Curran (Cardiac Rehab Sister @ FPH)
- Certificate of Professional Liability Insurance

Please notify the committee in advance of any significant proposed deviation from the original protocol. Would you also report any unusual or unexpected results, which raise questions about the safety of the research once the study is under way. The committee would be interested in the final results of your study and wish you every success in carrying it out.

Yours sincerely

[Signature]

David Harrison
CHAIRMAN

cc: Dr Emma Dunmore, Lecturer in Clinical Psychology, University of Surrey
An Investigation into Psychological Factors associated with adjustment after a Heart Attack

You are being invited to take part in the above research study. 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. Please ask if there is anything that is not clear or if you would like more information.

What is the purpose of the study?

Having a heart attack is a very stressful event. Afterwards people can experience many different feelings, thoughts, and emotions. Some of these feelings, thoughts, and emotions are helpful, but in other cases, they can make life more difficult. This research aims to investigate how people adjust after having a heart attack and what thoughts and coping strategies make the adjustment more difficult.

Specifically this project is investigating -

- The thoughts people have during a heart attack and about its consequences.
- How people react to the different emotions that can be experienced following a heart attack
- How people cope with difficult emotions and thoughts following a heart attack.

The results of the research will help us to understanding how people react after a heart attack and how best to help people after this kind of stressful event.

Why have you been chosen?

You have been invited to take part in this study because you have experienced a heart attack. Approximately 100 other people who have had a heart attack and who have been treated at St George's Hospital will also be invited to take part in the study over the next few months.

Your participation in this research project is entirely voluntary and you can decide not to take part without giving a reason. This will not affect your medical care in any way.

What does the study involve?
The study involves filling out some questionnaires about:

- Your background (for example your age, marital status, occupation etc)
- How you are feeling now and if heart attack has affected your life (for example your work, relationships etc)
- What you thought about your heart attack at the time it happened
- What you think the consequences of having a heart attack will be
- How you have reacted to your thoughts and feelings after your heart attack
- How you have coped with your thoughts and feelings after your heart attack
- Other stressful or traumatic events that you have experienced in your life

Please turn over

St George's Cardiovascular Research Group
Promoting cardiovascular research into prevention and treatment of diseases of the heart, brain and circulation
Each of the questionnaires has instructions on how to complete them. Filling out the questionnaire should about 20 minutes. We would also like to access your medical records so that we can gather additional information about your heart attack, for example the severity of your heart attack and what treatment you received.

All information which is collected about you during the course of the research will be kept strictly confidential. If you indicate in the questionnaires that you are having difficulty coping with your heart attack, we will seek your permission to contact your consultant cardiologist or cardiac rehabilitation nurse so that he or she can decide whether you would benefit from additional support.

Any information about you which leaves the hospital will have your name and address removed so that you cannot be recognised from it.

What are the possible disadvantages of taking part in the research?
Because having a heart attack is a potentially stressful event, you may find some of the questions upsetting. Therefore, there is absolutely no need to answer any questions that you do not want to answer.

If you would like more information about the project or if you would like to discuss any of the issues raised by the research, please contact me or my colleague, Dr Emma Dunmore, Chartered Clinical Psychologist, at the address below. We are unable to offer a counselling service, but can provide you with a list of local support agencies should you wish to talk to someone in more detail.

Thank you very much.

Claire Copland
PsychD Clinical Psychology
University of Surrey
Guildford, Surrey
GU1 5XH.
Telephone: 01483 259441

Dr Emma Dunmore
PsychD Clinical Psychology
University of Surrey
Guildford, Surrey
GU1 5XH.
Telephone: 01483 259441
Appendix 4

Consent Form

Title of Project: An Investigation into Psychological Factors associated with adjustment after a Heart Attack

Researcher: Claire Copland

I confirm that I have read and understand the information sheet dated March 2000 (Version 1) provided for the above study and have had the opportunity to ask questions.

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

I understand that sections of any of my medical notes may be looked at by responsible individuals from the University of Surrey, or from regulatory authorities where it is relevant to my taking part in the research. I give permission for these individuals to have access to my records.

I agree to take part in the above study.

Name (Please print) Date Signature

St George's Cardiovascular Research Group
Promoting cardiovascular research into prevention and treatment of diseases of the heart, brain and circulation

Cardiological Sciences
St George's Hospital Medical School
Cranmer Terrace
London SW17 0RE

Study number: 00.33.11
Participant Identification Number:

Please read and complete this consent form before you fill out the questionnaires.
Dear

We are writing to ask if you would take part in an investigation currently being conducted at the University of Surrey. The aim of this study is to improve our understanding of how people adjust to a heart attack and what thoughts and coping strategies are involved in this process. By investigating how people react after a heart attack, we ultimately hope to improve the help offered to people after this kind of stressful event.

The research is being conducted in collaboration with the Cardiac Unit at Frimley Park Hospital, who have agreed to allow us to send the enclosed questionnaires to people they have treated. The questionnaires should take about 20 minutes to complete and can be returned to us in the prepaid envelope provided.

Participation in this research is entirely voluntary and before you decide to take part, it is important that you read the attached information sheet so that you understand more about the research.

Please take time to read the information carefully. If you have any questions about the research please contact us at the University of Surrey (telephone number: 01483 259441)

Thank you very much for your time.

Yours sincerely

Claire Copland
Trainee Clinical Psychologist

Dr Emma Dunmore
Chartered Clinical Psychologist
Appendix 6

Information Sheet October 2000

An Investigation into Psychological Factors associated with adjustment after a Heart Attack

You are being invited to take part in the above research study. 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. Please contact us if there is anything that is not clear or if you would like more information.

What is the purpose of the study?
Having a heart attack is a very stressful event. Afterwards people can experience many different feelings, thoughts, and emotions. Some of these feelings, thoughts, and emotions are helpful, but in other cases, they can make life more difficult. This research aims to investigate how people adjust after having a heart attack and what thoughts and coping strategies make the adjustment more difficult.

Specifically this project is investigating -
• The thoughts people have during a heart attack and about its consequences.
• How people react to the different emotions that can be experienced following a heart attack
• How people cope with difficult emotions and thoughts following a heart attack.

The results of the research will help us to understand how people react after a heart attack and how best to help people after this kind of stressful event.

Why have you been chosen?
You have been invited to take part in this study because you have experienced a heart attack. Approximately 100 other people who have had a heart attack and who have been treated at St George's Hospital, London or Frimley Park Hospital, Frimley, will also be invited to take part in the study over the next few months.

Your participation in this research project is entirely voluntary and you can decide not to take part without giving a reason. This will not affect your medical care in any way.

What does the study involve?
The study involves filling out some questionnaires about:
• Your background (for example your age, marital status, occupation etc)
• How you are feeling now and if heart attack has affected your life (for example your work, relationships etc)
• What you thought about your heart attack at the time
• What you think the consequences of having a heart attack will be
• How you have reacted to your thoughts and feelings after your heart attack
• How you have coped with your thoughts and feelings after your heart attack
• Other stressful or traumatic events that you have experienced in your life

Please turn over
Each of the questionnaires has instructions on how to complete them. Filling out the questionnaire should take about 20 minutes. The questionnaires can then be returned to the University of Surrey in the enclosed prepaid envelope.

In addition, we would also like to access your medical records so that we can gather additional information about your heart attack, for example the severity of your heart attack and what treatment you received.

All information that is collected about you during the course of the research will be kept strictly confidential. However, if you indicate in the questionnaires that you are having difficulty coping with your heart attack, we will seek your permission to contact the cardiac rehabilitation nurse at Frimley Park Hospital.

Any information about you which leaves the hospital will have your name and address removed so that you cannot be recognised from it.

What are the possible disadvantages of taking part in the research?
Because having a heart attack is a potentially stressful event, you may find some of the questions upsetting. Therefore, there is absolutely no need to answer any questions that you do not want to answer.

If you would like more information about the project or if you would like to discuss any of the issues raised by the research, please contact us at the address below. We are unable to offer a counselling service, but can provide you with a list of local support agencies should you wish to talk to someone in more detail.

If you decide to take part in the research...
If you decide that you would like to take part in the research project, please fill out the following consent form, before you fill in the questionnaires. The completed consent form and the questionnaires can then be returned in the enclosed prepaid envelope.

Thank you very much for your time

Claire Copland
Trainee Clinical Psychologist
PsychD Clinical Psychology
University of Surrey
Guildford, Surrey
GU1 5XH
Telephone: 01483 259441

Dr Emma Dunmore
Chartered Clinical Psychologist
PsychD Clinical Psychology
University of Surrey
Guildford, Surrey
GU1 5XH
Telephone: 01483 259441
Participant Number:

**Consent Form**

*Please read and complete this consent form before you fill out the questionnaires*

**Title of Project:** An Investigation into Psychological Factors associated with adjustment after a Heart Attack

**Researcher:** Claire Copland

1. I confirm that I have read and understand the information sheet dated October 2000 provided for the above study and have had the opportunity to ask questions

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 or legal rights being affected

3. I understand that sections of any of my medical notes may be looked at by responsible individuals from the University of Surrey, or from regulatory authorities where it is relevant to my taking part in the research. I give permission for these individuals to have access to my records.

4. I agree to take part in the above study

---

**Signature**

**Date**
Dear

Thank you for reading the information sheet and questionnaires we sent you for the research project “An Investigation into Psychological Factors associated with adjustment after a Heart Attack”.

If you have already sent back the questionnaires, thank you very much for your time. If you have not sent back the questionnaires yet, we would still appreciate your help with this research and you can contact us on 01483 259441 if you have any questions. Of course, your participation in this research is entirely voluntary and if you have decided that you do not wish to fill out the questionnaires, we respect your decision. We will not contact you again regarding the research project.

Thank you again for your time

Yours sincerely

Claire Copland
Trainee Clinical Psychologist

Dr Emma Dunmore
Chartered Clinical Psychologist
Appendix 9

Participant Number:

Background Information

I would like to get some basic information about you so that I can get an idea the type of people who have taken part in my research.

The information that you give can not be used to identify you in any way. However, if you do not want to answer some of the questions, please do not feel that you have to.

1. Age: ________________
2. Gender (circle one): Male / Female
3. Date of Heart Attack: ________________
4. Have you ever been hospitalised for any heart problems other than a heart attack? 
   No ☐ Yes ☐ if yes, please specify the problem that you were in hospital for ________________

5. What is your current married status? (tick the appropriate answer)
   Single ☐ Married / cohabiting ☐ Widowed ☐ Divorced / separated ☐ Other (please specify) ________________

6. Which (if any) of the following terms best describes your ethnic background? (tick the appropriate answer)
   Black - African ☐ Black-Caribbean ☐ Black-other ☐ Pakistani ☐ Bangladeshi ☐ White UK ☐ White Other ☐ Other (please specify) ________________

7. What is your highest educational qualification? (tick the appropriate answer)
   None ☐ GCSE (s) / O-levels / CSE (s) ☐ A-level(s) ☐ Diploma (HND, SRN, etc) ☐ Degree ☐ Postgraduate degree / diploma ☐

8. What is your current employment status? (tick the appropriate answer)
   Full time work ☐ Studying ☐ Part time work ☐ Registered Unemployed ☐ Receiving disability allowance ☐ Retired ☐ Other (please specify) ________________

Please turn over
Appendix 9

9. What is your job now? (if unemployed / retired, what was your last job?)

10. Before your heart attack, did you have any psychological problems (e.g., anxiety, depression, alcohol misuse)?

   Yes □
   No □

   If yes, did you receive help for your problem (for example from a psychiatrist, psychologist, or counsellor)?

   Yes □
   No □
Appendix 10

Participant Number:

Other Traumatic Events

Many people have lived through or witnessed a very stressful and traumatic event at some point in their lives. Below is a list of traumatic events. Please put a checkmark in the box next to ALL the events that have happened to you or that you have witnessed.

1. □ Serious accident, fire or explosion (for example, an industrial, farm, car, plane or boating accident)
2. □ Natural disaster (for example, tornado, hurricane, flood or major earthquake)
3. □ Non sexual assault by a family member or someone you know (for example, being mugged, physically attacked, shot, stabbed or held at gunpoint)
4. □ Non sexual assault by a stranger (for example, being mugged, physically attacked, shot, stabbed or held at gunpoint)
5. □ Sexual assault by a family member or someone you know (for example, rape or attempted rape)
6. □ Sexual assault by a stranger (for example, rape or attempted rape)
7. □ Military combat or a war zone
8. □ Sexual contact when you were younger than 18 with someone who was 5 or more years older than you (for example contact with genitals, breasts)
9. □ Imprisonment (for example, prison inmate, prisoner of war, hostage)
10. □ Torture
11. □ Life threatening illness other than a heart attack
12. □ Other traumatic event.

Please specify________________________________________________________________________

_____________________________________________________________________________________

_____________________________________________________________________________________
Appendix 11

General Health Questionnaire
(GHQ-28)

Please read this carefully. We should like to know if you have had any medical complaints and how your health has been in general, over the past few weeks. Please answer ALL the questions on the following pages simply by underlining the answer which you think most nearly applies to you. Remember that we want to know about present and recent complaints, not those that you had in the past.

It is important that you try to answer ALL the questions.

Thank you very much for your co-operation.

Have you recently:

<table>
<thead>
<tr>
<th></th>
<th>Better than usual</th>
<th>Same as usual</th>
<th>Worse than usual</th>
<th>Much worse than usual</th>
</tr>
</thead>
<tbody>
<tr>
<td>A1</td>
<td>Been feeling perfectly well and in good health?</td>
<td>Not at all</td>
<td>No more than usual</td>
<td>Rather more than usual</td>
</tr>
<tr>
<td>A2</td>
<td>Been feeling in need of a good tonic?</td>
<td>Not at all</td>
<td>No more than usual</td>
<td>Rather more than usual</td>
</tr>
<tr>
<td>A3</td>
<td>Been feeling run down and out of sorts?</td>
<td>Not at all</td>
<td>No more than usual</td>
<td>Rather more than usual</td>
</tr>
<tr>
<td>A4</td>
<td>Felt that you are ill?</td>
<td>Not at all</td>
<td>No more than usual</td>
<td>Rather more than usual</td>
</tr>
<tr>
<td>A5</td>
<td>Been getting any pains in your head?</td>
<td>Not at all</td>
<td>No more than usual</td>
<td>Rather more than usual</td>
</tr>
<tr>
<td>A6</td>
<td>Been getting a feeling of tightness or pressure in your head?</td>
<td>Not at all</td>
<td>No more than usual</td>
<td>Rather more than usual</td>
</tr>
<tr>
<td>A7</td>
<td>Been having hot or cold spells?</td>
<td>Not at all</td>
<td>No more than usual</td>
<td>Rather more than usual</td>
</tr>
<tr>
<td>B1</td>
<td>Lost much sleep over worry?</td>
<td>Not at all</td>
<td>No more than usual</td>
<td>Rather more than usual</td>
</tr>
<tr>
<td>B2</td>
<td>Had difficulty in staying asleep once you are off?</td>
<td>Not at all</td>
<td>No more than usual</td>
<td>Rather more than usual</td>
</tr>
<tr>
<td>B3</td>
<td>Felt constantly under strain?</td>
<td>Not at all</td>
<td>No more than usual</td>
<td>Rather more than usual</td>
</tr>
<tr>
<td>B4</td>
<td>Been getting edgy and bad-tempered?</td>
<td>Not at all</td>
<td>No more than usual</td>
<td>Rather more than usual</td>
</tr>
<tr>
<td>B5</td>
<td>Been getting scared or panicky for no good reason?</td>
<td>Not at all</td>
<td>No more than usual</td>
<td>Rather more than usual</td>
</tr>
<tr>
<td>B6</td>
<td>Found everything getting on top of you?</td>
<td>Not at all</td>
<td>No more than usual</td>
<td>Rather more than usual</td>
</tr>
<tr>
<td>B7</td>
<td>Been feeling nervous and strung-up all the time?</td>
<td>Not at all</td>
<td>No more than usual</td>
<td>Rather more than usual</td>
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</table>
## Appendix 11

Have you recently:

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<tbody>
<tr>
<td>C1</td>
<td>Been managing to keep yourself busy and occupied?</td>
<td>More so than usual</td>
<td>Same as usual</td>
<td>Rather less than usual</td>
</tr>
<tr>
<td>C2</td>
<td>Been taking longer over the things you do?</td>
<td>Quicker than usual</td>
<td>Same as usual</td>
<td>Longer than usual</td>
</tr>
<tr>
<td>C3</td>
<td>Felt on the whole you were doing things well?</td>
<td>Better than usual</td>
<td>About the same</td>
<td>Less well than usual</td>
</tr>
<tr>
<td>C4</td>
<td>Been satisfied with the way you've carried out your task?</td>
<td>More satisfied</td>
<td>About same as usual</td>
<td>Less satisfied than usual</td>
</tr>
<tr>
<td>C5</td>
<td>Felt that you are playing a useful part in things?</td>
<td>More so than usual</td>
<td>Same as usual</td>
<td>Less useful than usual</td>
</tr>
<tr>
<td>C6</td>
<td>Felt capable of making decisions about things?</td>
<td>More so than usual</td>
<td>Same as usual</td>
<td>Less so than usual</td>
</tr>
<tr>
<td>C7</td>
<td>Been able to enjoy your normal day-to-day activities?</td>
<td>More so than usual</td>
<td>Same as usual</td>
<td>Less so than usual</td>
</tr>
</tbody>
</table>

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</thead>
<tbody>
<tr>
<td>D1</td>
<td>Been thinking of yourself as a worthless person?</td>
<td>Not at all</td>
<td>No more than usual</td>
<td>Rather more than usual</td>
</tr>
<tr>
<td>D2</td>
<td>Felt that life is entirely hopeless?</td>
<td>Not at all</td>
<td>No more than usual</td>
<td>Rather more than usual</td>
</tr>
<tr>
<td>D3</td>
<td>Felt that life isn't worth living?</td>
<td>Not at all</td>
<td>No more than usual</td>
<td>Rather more than usual</td>
</tr>
<tr>
<td>D4</td>
<td>Thought of the possibility that you might make away with yourself?</td>
<td>Definitely not</td>
<td>I don't think so</td>
<td>Has crossed my mind</td>
</tr>
<tr>
<td>D5</td>
<td>Found at times you couldn't do anything because your nerves were too bad?</td>
<td>Not at all</td>
<td>No more than usual</td>
<td>Rather more than usual</td>
</tr>
<tr>
<td>D6</td>
<td>Found yourself wishing you were dead and away from it all?</td>
<td>Not at all</td>
<td>No more than usual</td>
<td>Rather more than usual</td>
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<td>D7</td>
<td>Found that the idea of taking your own life kept coming into your mind?</td>
<td>Definitely not</td>
<td>I don't think so</td>
<td>Has crossed my mind</td>
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271
Below is a list of problems that people sometimes have after experiencing a traumatic event such as a heart attack. Please read each one carefully and rate each problem with respect to your heart attack. Circle the number (0-3) that best described how often that problem has bothered you in the PAST MONTH.

0 = Not at all or only one time  
1 = once a week or less / once in a while  
2 = 2 to 4 times a week / half the time  
3 = 5 or more times a week / almost always

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<tr>
<th>Problem</th>
<th>0</th>
<th>1</th>
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<tr>
<td>Having upsetting thoughts or images about the heart attack</td>
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<td>Having bad dreams or nightmares about the heart attack</td>
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<td>Reliving the heart attack, acting or feeling as if it was happening again</td>
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<td>Feeling emotionally upset when you were reminded of the heart attack (for example feeling scared, angry, sad, guilty etc)</td>
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<td>Experiencing physical reactions when you were reminded of the heart attack (for example, breaking out in a sweat, heart beating fast)</td>
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<td>Trying not to think about, talk about or have feelings about the heart attack</td>
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<td>Trying to avoid activities, people or places that remind you of the heart attack</td>
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<td>Not being able to remember an important part of what happened during the heart attack</td>
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<td>Having much less interest or participating less often in important activities</td>
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<td>Feeling distant or cut off from people around you</td>
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<td>Feeling emotionally numb (for example being unable to cry or unable to have loving feelings)</td>
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<td>Feeling as if your future plans or hopes will not come true (for example, you will not have a career, marriage, children, or a long life.)</td>
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<td>Having trouble falling or staying asleep</td>
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<td>Feeling irritable or having fits or anger</td>
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<td>Having trouble concentrating (for example drifting in and out of conversation, losing track of a story on television, forgetting what you read)</td>
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<td>Being overly alert (for example checking to see who is around you, being uncomfortable with your back to a door)</td>
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<td>Being jumpy or easily startled (for example when someone walks up behind you)</td>
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How long have you experienced the problems that you have reported above? (circle one)  
1. Less than 1 month  
2. 1 to 3 months  
3. More than 3 months

How long after the heart attack did these problems begin? (Circle one)  
1. Less than 6 months  
2. 6 months or more

Please indicate if the problems you rated above have interfered with any of the following areas of your life DURING THE PAST MONTH. Circle Yes or No.

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<td>Household chores and duties</td>
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<td>Relationships with friends</td>
<td>Yes</td>
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<td>Fun and leisure activities</td>
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<td>Relationships with your family</td>
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<td>Sex Life</td>
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<td>General satisfaction with life</td>
<td>Yes</td>
<td>No</td>
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<tr>
<td>Overall level of functioning in all areas of your life</td>
<td>Yes</td>
<td>No</td>
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Appendix 13

Severity of Heart Attack

Please read the following questions and rate how you felt about the heart attack at the time that it happened.

Circle the number on the scale that best represents how you felt.

1. At the time of the heart attack, how severe did you think that your heart attack was?
   0 10 20 30 40 50 60 70 80 90 100
   Mild Extremely severe

2. At the time of the heart attack, to what extent did you think that your life was in danger?
   0 10 20 30 40 50 60 70 80 90 100
   I did not believe I was completely convinced I would die that I would die
   helpless

3. At the time of the heart attack, to what extent did you feel helpless?
   0 10 20 30 40 50 60 70 80 90 100
   I did not feel helpless I felt completely helpless

4. At the time of the heart attack, to what extent did you feel terrified?
   0 10 20 30 40 50 60 70 80 90 100
   I did not feel terrified I felt totally terrified
Appendix 14

Participant Number:

Thoughts After a Heart Attack

This questionnaire lists different thoughts which people may have after a heart attack. Read each statement and decide how much you have agreed or disagreed with each statement in the last four weeks. For each of the statements, please show your answer by putting a circle round the word which best describes how much you have agreed with the statement. Because people react to a heart attack in many different ways, there are no right or wrong answers.

1. If I get angry, it will be dangerous to my health
   - Totally Agree
   - Very Agree
   - Neutral
   - Disagree
   - Slightly
   - Disagree

2. My reactions since the heart attack mean that I cannot cope
   - Totally Agree
   - Very Agree
   - Neutral
   - Disagree
   - Slightly
   - Disagree

3. If I cannot control my thoughts about the heart attack it means that I am weak
   - Totally Agree
   - Very Agree
   - Neutral
   - Disagree
   - Slightly
   - Disagree

4. If I cannot control my thoughts or feelings about the heart attack, I will go crazy
   - Totally Agree
   - Very Agree
   - Neutral
   - Disagree
   - Slightly
   - Disagree

5. It is normal to take a long time to recovery from a heart attack
   - Totally Agree
   - Very Agree
   - Neutral
   - Disagree
   - Slightly
   - Disagree

6. Something terrible will happen if I do not control my thoughts about the heart attack
   - Totally Agree
   - Very Agree
   - Neutral
   - Disagree
   - Slightly
   - Disagree

7. If I cannot remember something about the heart attack then it is because I would find it unbearable
   - Totally Agree
   - Very Agree
   - Neutral
   - Disagree
   - Slightly
   - Disagree

8. I should be over this by now
   - Totally Agree
   - Very Agree
   - Neutral
   - Disagree
   - Slightly
   - Disagree

9. Other people would cope better with a heart attack than me
   - Totally Agree
   - Very Agree
   - Neutral
   - Disagree
   - Slightly
   - Disagree

10. My thoughts and feelings mean that I am going to have another heart attack
    - Totally Agree
    - Very Agree
    - Neutral
    - Disagree
    - Slightly
    - Disagree

* score on item 5 reversed
Appendix 15

Consequences of Heart Attack

This questionnaire lists different thoughts that people might have about the consequences of having a heart attack.

Please read the statements and decide how much you agree with each one in the last four weeks. For each of the statements, please show your answer by putting a circle round the word which best describes how much you agree with the statement. Because people react to a heart attack in many different ways, there are no right or wrong answers.

1. My life has been permanently changed by my heart attack

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<th>Totally Agree</th>
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2. I will have physical disabilities that will constantly effect me

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3. I will have persistent problems dealing with medication and self care

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4. I will not be able to return to the same level of work as before

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5. I will not be able take part in my normal activities for example sports and housework

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6. I will have problems with relationships

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7. I will have problems with social activities

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8. I will have financial problems

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275
Appendix 16

Participant Number:

Coping Strategies after a Heart Attack

Below is a list of coping strategies that people may use after a heart attack.

Please think about the past 4 weeks. Circle the word that best describes how often you have used or tried to use each type of coping strategy, even if you were unable to succeed. Because people react to a heart attack in many different ways, there are no right or wrong answers.

1. I have avoided stressful events
   - Never
   - Sometimes
   - Often
   - Always

2. I have tried to distance myself from my emotions
   - Never
   - Sometimes
   - Often
   - Always

3. I have tried to numb my emotions
   - Never
   - Sometimes
   - Often
   - Always

4. I have tried hard not to think about the heart attack
   - Never
   - Sometimes
   - Often
   - Always

5. I have tried to distract myself from distressing thoughts
   - Never
   - Sometimes
   - Often
   - Always

6. I have tried to push thoughts to the back of my mind.
   - Never
   - Sometimes
   - Often
   - Always

7. I have tried hard to keep my thoughts and emotions under control
   - Never
   - Sometimes
   - Often
   - Always

8. I have allowed myself to show my feelings to others
   - Never
   - Sometimes
   - Often
   - Always

9. I have tried to erase the memory of the heart attack
   - Never
   - Sometimes
   - Often
   - Always

10. I have thought about ways in which the heart attack might have been prevented
    - Never
    - Sometimes
    - Often
    - Always

11. I have talked to family and friends about the heart attack and about what I was feeling
    - Never
    - Sometimes
    - Often
    - Always

12. I have ruminated about why a heart attack happened to me
    - Never
    - Sometimes
    - Often
    - Always

13. I have allowed myself to become detached from what is going on around
    - Never
    - Sometimes
    - Often
    - Always

14. I have avoided looking at TV programmes or newspaper articles about heart attacks
    - Never
    - Sometimes
    - Often
    - Always
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