

**INTRUSIVE MEMORIES AND TRUAMA-RELATED SYMPTOMS IN
INDIVIDUALS PRESENTING WITH DENTAL ANXIETY**

BY

AMANDA JACQUELINE FORBES

AUGUST 2001

**SUBMITTED IN PART FULFILMENT OF THE DEGREE OF DOCTORATE
IN CLINICAL PSYCHOLOGY AT THE UNIVERSITY OF EDINBURGH**



For
Karl

Contents

Page No.

Contents	i
Acknowledgements	vi
Declaration	vii
Abstract	viii
Chapter 1.	
Dental Anxiety	1
1.1 Characteristics of Dental Anxiety	1
1.2 Treating Dental Anxiety	8
1.3 A General Overview of Fear Acquisition, Development and Maintenance.	11
1.4 Contemporary Model of Conditioning	19
1.5 The Panic and Alarm Theory of Fear Acquisition	24
1.6 Evolutionary Models of Fear Acquisition	27
1.7 Cognitive Models of Fear Acquisition and Maintenance	29
1.8 Neurobiological Model of Fear Acquisition	32
1.9 Dental Anxiety; Acquisition and Maintenance	35
Aims of Study 1	42
Hypotheses	42
Chapter 2.	
Study 1: Comparison of Dental Anxiety Measures and Direct and Indirect Pathways of Fear Acquisition	44
2.1 Design	44
2.2 Sample	44
2.3 Measures	45
2.4 Clinical Interview	47
2.5 Analysis	47
Chapter 3.	
Results: Study 1 Comparison of Dental Anxiety Measures and Direct and Indirect Pathways of Fear Acquisition	48
3.1 Demographic Data	48
3.1.1 Participants	48
3.1.2 Exploratory Data Analysis of the MDAS, DFS Behavioural Avoidance and Physiological Arousal Scales	48

Chapter 4.	
Discussion: Study 1 Comparison of Dental Anxiety Measures and Direct and Indirect Pathways of Fear Acquisition	55
Chapter 5.	
History and Models of Post Traumatic Stress Disorder and Application to Medical and Dental Fears	60
5.1 Introduction and History of PTSD	60
5.2 Epidemiology of PTSD	68
5.3 Predisposing Factors	71
5.4 Comorbidity	74
5.5 Models of PTSD	76
5.5.1 Psychoanalytical Model of PTSD	76
5.5.2 Cognitive Models of PTSD	78
5.5.3 Horowitz's Theory of PTSD	79
5.5.4 The Cognitive Appraisal Model: Janoff-Bulman	81
5.5.5 Foa's Fear Network	82
5.5.6 Dual Representation Theory	83
5.5.7 The Schematic, Propositional, Associative and Analogical Representational Systems (SPAARS)	85
5.5.8 Ehlers and Clark's Cognitive Model of PTSD	90
5.5.9 Psychobiology of PTSD	97
5.5.10 Integrative Cognitive Neuroscience Account of PTSD	102
5.6 Treatment of PTSD	105
5.7 PTSD Related to Dental and Medical Events	112
Aims of Study	118
Hypotheses	118
Chapter 6	
Study 2: Intrusive Memories and Trauma Related Symptoms and the Differences between Individuals with and without Intrusive Memories on Measures of Dental Anxiety, General Psychopathology and Beliefs Related to Dental Care	119
6.1 Design	119
6.2 Sample	119
6.3 Sample Size	121
6.4 Measures	122
6.5 Procedure	126
6.6 Analysis	127

Chapter 7	
Results: Study 2	
Intrusive Memories and Trauma Related Symptoms and the Differences between Individuals with and without Intrusive Memories on Measures of Dental Anxiety, General Psychopathology and Beliefs Related to Dental Care	129
7.1 Demographic Data	129
7.1.1 Participants	129
Chapter 8	
Discussion: Study 2	
Intrusive Memories and Trauma Related Symptoms and the Differences between Individuals with and without Intrusive Memories on Measures of Dental Anxiety, General Psychopathology and Beliefs Related to Dental Care	144
8.1 Summary of Research	144
8.2 Discussion of Research Findings	145
8.3 Methodological Problems	152
8.4 Study Limitations	154
8.5 Ethical Considerations	155
8.6 Future Research	156
8.7 Conclusions	158
References	160
Appendix 1: MDAS	181
Appendix 2: Histogram MDAS	182
Appendix 3: Histogram DFS physiological scores	183
Appendix 4: Histogram DFS behavioural avoidance	184
Appendix 5: DCC	185
Appendix 6: SIP	186
Appendix 7: Patient Information Sheet	187
Appendix 8: Scattergrams Study 2	188

List of Tables.

Table 1.	Acquisition of Phobias Dental and Blood Phobics	37
Table 2.	Kolmogorov-Smirnov Test of Normality: Study 1	49
Table 3.	Mean Age for Direct and Indirect Conditioning Groups and Independent-samples t-test for age: Study 1	50
Table 4.	Mean Scores for Dental Anxiety and Independent-samples t-test between Direct and Indirect Conditioning Groups, Study 1	51
Table 5.	Mean Scores for DFS physiological arousal and Independent Samples t-test Direct and Indirect conditioning groups, Study 1	52
Table 6.	Mean DFS scores for behavioural avoidance and independent Samples t-test, study 1	53
Table 7.	Mean age, age at trauma and dental avoidance history for the Intrusive memory and Non-Intrusive memory group, Study 2	131
Table 8.	Kolmogorov-Smirnov test of normality, Trauma measures	132
Table 9.	Kolmogorov-Smirnov test of normality, general measures	133
Table 10.	Comparison of means scores for age and dental anxiety level for responders and non-responders.	134
Table 11.	Mean scores for trauma measures, intrusive memory group	135
Table 12.	Pearson-Product-Moment Correlations between IES (intrusion Scale) and IES (avoidance), SIPS (intrusion, avoidance, increased arousal and total)	136
Table 13.	Mean MDAS scores intrusive memory and non-intrusive memory group and independent-samples t-test MDAS between the two groups	138
Table 14.	Mean DCC scores intrusive memory and non-intrusive memory group and independent-samples t-test DCC between the two groups	139

Table 15. Mean BSIGLOB scores intrusive memory and non-intrusive memory group and independent-samples t-test BSIGLOB between the two groups	140
Table 16. Mean BSIPST scores intrusive memory and non-intrusive memory group and independent-samples t-test BSIPST between the two groups	141
Table 17. Mean BSIPSDI scores intrusive memory and non-intrusive memory group and independent-samples t-test BSIPSDI between the two groups	142

List of Figures

Figure 1. Example of a Dental Fear Conditioning Response	36
Figure 2. Horowitz's Model of PTSD	80
Figure 3. Diagram of the Dual Representation Model of PTSD	85
Figure 4. The basic architecture of the SPAARS model	86

Acknowledgements

I would like to thank Dr Pauline McGoldrick for her advice and words of support. Professor Mick Power for his valuable and speedy feedback. Associate Professor Ad de Jongh for his interest, support and encouragement.

I am indebted to all the participants who agreed to take part in this study, and to the staff at the dental hospital who made me feel welcome.

I would also like to thank Miss Penelope Fraser, for her support, patience and words of encouragement during my clinical placement.

To my fellow class-mates a big thank-you for making the last three years a pleasant experience, and of course to Ailie and Emma, a big thank-you for your support, encouragement and humour.

I would also like to thank Gill and the boys for never losing faith and for support especially through the difficult times.

DECLARATION

“ I certify that this is a true and accurate account of the work carried out. This thesis has been composed by myself and the work herein is my own”.

Signed.

Amanda Jacqueline Forbes

Abstract

Research indicates that between 36-40% of people are afraid to visit the dentist, 20% are highly anxious and 5% avoid dental treatments due to severe anxiety (Lindsay & Jackson, 1993). Dental anxiety is known to be a major barrier to optimal health care with individuals fearful of impending invasive procedures often presenting only when in severe pain. Dental anxiety has been found to be based on past memories of experiences of pain and loss of control in the dental setting.

Evidence from the literature suggests that some individuals with dental anxiety also present with additional psychological problems, such as panic disorder. De Jongh, Muris, ter Horst and Duyx (1995) also reported that individuals with dental anxiety also exhibited more catastrophizing thoughts related to dental treatment. A recent study by De Jongh, Aartman & Brand (manuscript submitted for publication) reported that intrusive memories of past distressing dental experiences were associated with trauma-related symptoms. However no structured assessment for Post-Traumatic Stress Disorder (PTSD) was carried out.

This study aimed to identify the association between intrusive memories related to dental experiences and trauma-related symptomatology and to investigate differences in psychopathology and frequency of thought content in individuals presenting with and without intrusive memories. The proportion of individuals with intrusive memories who would meet criteria for PTSD was also investigated. Results will be given and conclusions reached.

Chapter 1. Dental Anxiety

Dental anxiety is a major barrier to optimal oral health care with patients fearful of impending invasive procedures often presenting only when in severe pain. Dental anxiety can also interfere with dental treatment compliance and make control of pain and completion of treatment difficult for the patient and the dental practitioner (Walker & Cooper, 1998).

1.1 Characteristics of Dental Anxiety

Fear of the dentist is experienced to varying degrees. Research indicates that between 36-40 per cent of people are afraid to visit the dentist, 20 per cent are highly anxious and 5 per cent avoid dental treatments due to severe anxiety (Lindsay & Jackson, 1993, Stouthard & Hoogstraten, 1990). However the true extent of dental anxiety is unknown as many of those with difficulties either avoid treatment or only attend for symptomatic care (McGoldrick, Levitt, De Jongh, Mason & Evans, submitted for publication). A recent adult dental health survey carried out in the United Kingdom reported that amongst dentate adults 23 per cent of men and 40 per cent of women reported being “definitely nervous” of some types of dental treatment (Walker & Cooper, 1998). In a review of behavioural research in dentistry between 1987-1992, ter Horst and de Wit (1993) found that women reporting more dental anxiety than men was almost universal. Liddell & Locker (1993) reported that dental anxiety peaks during early adolescence and declines with increasing age.

Dental anxiety is frequently based on past memories of experiences of pain and loss of control in the dental setting (Kent, 1987; Mazey & Mito, 1993). Locker, Shapiro and Liddell (1996) found that negative dental experiences, in particular, painful experiences, were related to dental anxiety. McNeil and Berryman (1989) reported that pain, mutilation and being closed-in were found to be three of the most important components of dental anxiety. A study carried out by Rice and Liddell (1998) also found that discomfort, pain and invasive procedures were most often identified as reasons for negative attitudes towards dentistry.

The dentist-patient relationship has also been identified as a factor contributing to the aetiology and maintenance of dental anxiety. Bernstein, Kleinknecht and Alexander (1979) reported that amongst a sample of high-dental fear students, half of the group cited the perceived manner of the dentist as a factor accounting for their present fears. Kent (1998) suggested that this study appears to show that the manner of the dentist may have an independent effect on the students' feelings. Cold or uninterested behaviour was enough to make some students feel negatively about dentistry, whereas caring and warm behaviour displayed by the dentist could have an interactive effect and obviate the long-term effects of painful experiences. Milgrom, Vignehsa and Weinstein (1992) found that adolescents were more likely to be highly anxious of dental treatment if they believed that their dentist was unsympathetic. In a study carried out by Weiner, Forgione, Weiner and Hwang (2000) dentists who were rated as exhibiting a variety of negative behaviours and attitudes were more likely to increase levels of concern in patients. The authors argue that negative dental

practitioner behaviours may act as fear-provoking stimuli, leading to increased fear and avoidance of dental treatment.

Milgrom, Weinstein and Getz (1995) describe approach-avoidance conflicts operating in individuals with dental phobia. Based on Dollard & Miller's (1950) work, they describe conflicts between the consequences of neglecting dental care and the anxiety associated with attending. According to this model the patient is faced with two competing tendencies with respect to a single situation i.e. motivated to approach the dentist, yet also wanting to avoid the experience. This creates a state of conflict. Dollard and Miller (1950) suggested that these two tendencies change in strength as the person in conflict moves closer or further away from the desired but feared situation. If further away in time and distance the approach tendency is stronger than the avoidance. As the appointment time approaches the patient becomes more anxious and the avoidance tendency is stronger than the approach tendency. Milgrom et al (1985) argue that this approach-avoidance relationship may explain why some patients make appointments then cancel them close to the appointment time.

The most serious degree of dental fear is specific phobia (Moore, Brødsgaard & Birn (1991). These patients are known to avoid the dentist for many years and the avoidance of dental treatment is strongly associated with extreme deterioration of oral health (Cohen, 1985). A recent qualitative study (Cohen, Fiske, & Newton, 2000) also found that patients with severe dental anxiety reported problems with both family and workplace relationships. These patients described loss of authority at

work following disclosing their dental anxiety to work colleagues and poor job interview performance because of impaired speech. They also described difficulties in the development and maintenance of family relationships. The authors note that health is more than the absence of clinical disease and also involves the ability to eat, speak and socialise without feeling discomfort or embarrassment. A study by Nuttall, Steele, Pine, White and Pitts (2001) found that in a population of 6,204 adults over half (51 per cent) reported that they had been affected by their oral health. The authors found that in the 12 months preceding the study the most commonly experienced impact on quality of life was oral pain. The next most frequently experienced problems arising from oral condition were found to be psychological. These included feeling tense, self-conscious and embarrassed about their oral condition. Eight per cent of those in the study reported that they had been severely affected by their oral health in that they felt their life was less satisfying or that they were totally unable to function at some time in the preceding year as a result of their oral condition.

De Jongh, Bongaarts, Vermeule, Visser, De Vos and Makkes (1998) reported that in reviews of the dental literature, dental fears and blood-injury-injection (BII) phobia are often dealt with together. Edelman (1992) suggests that dental phobia is a subtype of blood-injury phobia. However a number of studies have found that dental phobia should be considered as independent of BII phobia (De Jongh et al 1998; Locker, Shapiro & Liddell, 1997). Dental phobics were found to demonstrate fear of specific dental stimuli or procedures and more general aspects of the dental treatment situation i.e. anticipation of encounters with specific dental-related stimuli, loss of

control in the dental setting and pain. They were found to endorse less BII fears such as an excessive fear of blood and injections (De Jongh et al 1998).

Kent, Rubin, Getz and Humphries (1996) designed a scale to assess the social and psychological effects of dental phobia. They found that dental phobics were more likely to endorse items, which indicated social and cognitive consequences e.g. *“I feel that people will laugh at me if I tell them about my fears about dentistry”* and *“the need to see a dentist is constantly on my mind”*.

There is evidence from the literature that patients presenting with dental fears are not a homogeneous group (Liddell and Gosse 1998). It has been reported that some patients presenting with high dental anxiety may also have other psychological difficulties such as multiple phobias, panic disorder, and general anxiety disorder (Aartman, 2000, Roy-Byrne, Milgrom, Khoon-Mei, Weinstein & Katon, 1994).

Kaaklo, Coldwell, Getz, Milgrom, Roy-Byrne & Ramsay al (2000) reported that amongst a group of patients with dental injection phobia, over half had an additional current Axis I diagnosis other than dental injection phobia, mainly anxiety, mood or adjustment disorder. Subjects presenting with additional Axis I diagnoses reported higher dental anxiety, greater severity of injection fear cognitions and poorer relationships with dental professional. Kaaklo et al (2000) suggest that further investigations are required to explore the treatment possibilities for patients with and without additional current diagnoses.

Milgrom, Weinstein, Kleinknecht and Getz (1985) developed an aetiological categorisation system (The Seattle System) to explain dental anxiety. The four categories were 1) conditioned fear of specific dental stimuli; 2) anxiety about somatic reactions during treatment e.g. panic attacks, an allergic reaction; 3) trait anxiety or multiphobic symptoms and 4) distrust of dental personnel. However the authors did not make clear how the last three categories could have their aetiological basis explained. It is possible that a conditioned fear response could account for categories two, three and four. For example, Davey (1992) suggests that panic disorder may be acquired as a result from the conditioning of panic to either internal stimuli (dizziness) or external stimuli (situation). Speirs and Barsby (1995) reported that hyperventilation is common amongst the dentally anxious population whilst in a dental setting. Dizziness is known to be an effect of hyperventilation, therefore it is conceivable that panic disorder could be conditioned in this situation. In addition Weiner et al (2000) have proposed that dental practitioner behaviours may act to provoke fear responses accounting for the development of the fourth category in Milgrom et al's (1985) categorisation.

Moore et al (1991) used the Seattle System of classification to reflect DSM-III-R (APA, 1982) diagnostic categorisation then subsequently DSM-IV (APA, 1987, 1994) categorisation with patients presenting with dental anxiety. They found that the existence of multiple phobias and general anxiety complicated the presentation of dental fear in about one third of their patients. They also found that the majority of the patients in the study suffered from social embarrassment about their dental fear and their inability to do something about it.

In a study carried out by Roy-Byrne, Milgrom, Khoon-Mei, Weinstein and Katon (1994) 60 per cent of their dentally anxious patients were found to have a simple dental phobia, whilst the rest of their sample presented with anxiety-based or mood disorders according to DSM-III-R criteria (APA, 1982). They also found that half of the individuals who presented with the diagnosis of simple phobia had a past psychiatric history.

Enneking, Milgrom, Weinstein and Getz (1992) found that patients presenting with non-specific dental fears (all stimuli including making a dental appointment are seen as terrifying) compared to patients with specific dental fears (injection) reported more concerns about a medical catastrophe during dental treatment and were more likely to relapse. It was observed that the non-specific patient group reflected symptoms and behaviours more readily associated with generalised anxiety disorder, such as being easily fatigued, sleep disturbances and irritability.

Berggren & Carlsson (1985) and Moore, (1991) argued that dentally anxious patients presenting with other psychological difficulties have greater difficulties in treatment than individuals presenting with specific dental anxiety. Berggren and Carlsson (1985) found that patients with dental anxiety who were also anxious about a number of situations and objects (not dentally related) were less likely to benefit from behavioural therapy than those who presented with a specific dental anxiety.

In summary, the extent of dental anxiety in the population is reported to be unknown. However where the problem is identified the literature suggests that it has serious consequences on health, social and workplace relationships. For some individuals with dental anxiety, additional psychological difficulties have been identified. It is not clear from the literature if these additional psychological difficulties develop as a consequence of the dental anxiety e.g. self-consciousness, increased tension (Nuttall, et al, 2001), poor family relationships (Cohen et al, 2000), or that dental anxiety develops in individuals predisposed to the development of general psychological disorders. It has been proposed that additional psychological difficulties presenting with dental anxiety leads to difficulties in treatment intervention. The next section discusses the treatment of dental anxiety and the types of methods used.

1.2 Treating Dental Anxiety

Various treatments exist for patients with anxiety, however according to McGoldrick & Durham, (2000) psychological therapies are reported to have been applied less to dental anxiety relative to other anxiety disorders. The various treatments that have been implemented in the treatment of dental anxiety include psychological treatments such as, cognitive behavioural therapies (De Jongh, Muris, ter Horst & Duyx 1995a), and pharmacotherapy, such as conscious sedation with nitrous oxide, intravenous sedation with one or more sedative drugs and general anaesthesia (Kaufman & Jastik, 1995). A recent study by McGoldrick et al (manuscript submitted for publication) found that referrals to a dental anxiety clinic were more likely to have been for pharmacological intervention than psychological management. However for the majority of patients receiving pharmacological

treatment, re-referral was common, suggesting that the application of this type of treatment had only short-term benefits. The authors argue that due to the amnesic effects of the drugs given during intravenous sedation, habituation of the anxiety response does not take place. The patients then will have had no opportunity to increase self-efficacy and build confidence to cope with future dental procedures.

According to Aartman (2000) the results of studies carried out comparing pharmacological versus psychological interventions are inconclusive, although there has been a slight advantage reported for the behavioural interventions. A number of studies have reported a decrease in post-treatment dental anxiety scores following behavioural interventions (Milgrom et al 1995, Hakeberg, Berggren & Carlsson 1990).

Johren, Jackowsky, Gangler, Satory and Thom (2000) carried out a clinical trial to examine short and medium term reduction of dental fear in patients with dental phobia who were requiring oral surgery. Groups were compared between one-session psychological treatment (stress management training and imaginal exposure), oral sedation (pharmacological) and control group (no intervention). Following intervention and prior to surgery both interventions caused the level of reported dental fear to decrease compared to the control group. At follow-up two months later the oral sedation group showed a return to reported baseline fear level whereas the psychologically treated group showed further improvement. At one year follow-up only the group who had received psychological treatment reported sustained reduction of levels of fear.

Studies which have examined the process of providing the dental phobic patient with a means of control during dental procedures have also been found to be successful (Kent, 1998). Various procedures have been used such as information (Thrash, Marx and Box, 1982) and stop signals (Wardle, 1983). Stop signals e.g. raising a hand, have been used as a control mechanism. Milgrom et al (1995) reported that where the patient can indicate to the dentist to stop treatment at a particular point this increases the patient's control, thus reducing the patient's helplessness in the dental situation. Milgrom et al (1995) argue that the patients perception of control is critical for successful intervention. This is in accord with Bandura's (1982) argument that successive treatments are those where control and self-efficacy are enhanced. Litt, Nye and Shafer (1993) compared a self-efficacy group (given false feedback about their ability to relax) and a relaxation group. The self-efficacy group, were found to have increased beliefs in their ability to cope with dental procedures. It was also found that staff's blind ratings of distress during the procedure were less for the self-efficacy group than the relaxation group.

Currently McGoldrick and Durham (2001) are conducting a systematic Cochrane review of psychotherapy-based treatments for dental anxiety, as there are no authoritative guidelines currently available to inform rational clinical decision making in this area.

1.3 A General Overview of Fear Acquisition, Development and Maintenance.

Conditioning Models:

Before considering the acquisition, development and maintenance of dental fears¹ dental anxiety and its variants, a brief background of general fear acquisition and maintenance will be presented.

The acquisition of human fears have mostly been explained in terms of conditioning models and can be traced back to the work of Pavlov (1927). Classical conditioning is a process whereby associating a Conditioned Stimulus (CS) with an Unconditioned Stimulus (UCS) results in a Conditioned Response (CR) to the Conditioned Stimulus (CS). In their most rudimentary form Pavlovian conditioning accounts maintain that fear is a conditioned emotional response (CER) to a stimulus or event that is acquired via pairings with an aversive or traumatic unconditioned stimulus (UCS) (Forsyth, Daleiden & Chorpita, 2000; Rescorla, 1988,).

Watson and Raynor (1920) paired a pet rat (the conditioned stimulus, CS) with a loud noise (the unconditioned stimulus, UCS) in an attempt to condition a fear of the rat in an 11-month boy. They reported that the boy would begin to cry (the conditioned response, CR) when the rat was introduced to the room. Davey (1997)

¹ *(Rachman (1998) draws a distinction between the terms anxiety and fear. He describes anxiety as the tense anticipation of a threatening but vague event, where the person has difficulty in identifying the cause of the uneasy tension. Fear he describes as a combination of tension and unpleasant anticipation where the emotional reaction is to a specific perceived danger. However he does go on to suggest that the distinction between fear and anxiety especially in clinical terms may not be readily discernible. Often the terms anxiety and phobias are used to describe the same psychological problem i.e. social anxiety/social phobia even where there is a specific focus to the problem. Authors often use the terms, fear and anxiety interchangeably and for this thesis the terms used by the original authors will be used so as not to change the emphasis of their work.*

reports that a series of failures to replicate this study led to scepticism about the usefulness of conditioning models.

However according to Menzies and Clarke (1995) an instrumental contingency existed between Little Albert's behaviour and the presentation of the loud noise. The noise was only presented when Albert reached out and touched the rat. Menzies and Clark (1995) argue that it was not until Mowrer's (1939) extension of Watson and Raynor's account that instrumental learning was seen to take an important role in the development and maintenance of phobias.

Mowrer's (1939) two-stage theory of anxiety was influential in the acceptance of conditioning as an important factor in fear acquisition. Mowrer believed that anxiety was a learned response, which occurred to conditioned stimuli that have been followed by situations of pain or injury (UCS). He argued that fear would motivate humans to avoid these situations, and that the reduction of fear would serve to reinforce behaviours such as avoidance as it would bring about a sense of relief. For Mowrer the initial acquisition of fear was due to Pavlovian conditioning. Laboratory research carried out by Malloy and Levis (1988) lends support to Mowrer's two-stage theory. They found that, when pairings of light (CS) and shock (UCS) are given, followed by an opportunity to react with an escape response, persistent avoidance behaviour occurs.

A number of important features have evolved from the conditioning model. It was believed that neutral stimuli associated with fear or pain developed to become fearful

conditioned stimuli. The number of repetitions was believed to strengthen the fear as was the intensity of the fear or pain experienced in the presence of the stimuli. This model also highlighted temporal contiguity between the CS and UCS.

The conditioning theory was supported by a number of sources. A vast number of experiments were carried out on laboratory animals where fear reactions were generated by exposing animals to neutral and aversive stimuli. After a few pairings of an innocuous CS (usually a light or tone) and a noxious UCS (such as a brief shock to the feet) a constellation of CRs, characteristic of fear, (changes in heart rate and arterial blood pressure, somatosensory immobility, potentiated acoustic startle and pupillary dilation) have been observed when the CS is presented alone (Maren & Fanselow, 1996).

In humans fear reactions have been observed under combat conditions where the fear has resulted from traumatic stimulation (Rachman 1998). Sanderson, Laverty & Campbell (1963) conducted an experiment in which subjects were given a drug, which produced a temporary suspension of breathing. Most of the subjects were found to have developed intense fears of the stimulus encountered in or connected with the setting where they received the drug.

However although it was generally believed that the conditioning model could explain fear acquisition, a number of problems were identified that the model could not adequately demonstrate.

Davey (1997) argues that one of the major criticisms of the conditioning model of phobias is that a simple contiguity-based model does not appear to have the power to predict the acquisition of a phobia. It is now recognised that conditioned responses can develop when the CS and UCS are separated in time (Rachman, 1998). Observations of non-contiguous conditioning have been reported from experiments in food aversions. Animals given a novel food then made ill minutes or even hours later can form a strong aversion to the food (Garcia & Koelling, 1966).

Menzies and Clark (1995) report that one of the common attacks on the classical conditioning account is on the infrequency of one-trial learning in the laboratory. Seligman (1971) argued that one-trial learning may explain the acute onset of many phobias and may be obtained if extremely traumatic UCSs are employed. However Sturgis and Scott (1984) and Eysenck (1979) claim that one-trial learning is rare in laboratory studies. Recent work by Marks (1987) however did find that conditioning of skin conductance could be obtained in a single trial using relatively mild electric shock and a variety of CSs.

Rachman (1977) and Emmelkamp (1982) reported that many people who present with phobias are unable to recall any aversive or traumatic experience at the onset of their phobia. It has also been observed that not all people who experience a traumatic experience develop subsequent fears (Aitkin, Lister & Main, 1981). Hallam and Rachman (1976) conducted a study involving an electrical UCS, which they predicted would result in the development of a conditioned fear reaction. One group received 205 shock trials over ten sessions and another group received 20 shocks

during a single session. The authors failed to confirm their hypothesis. Therefore neither the number of repetitions of the UCS or intensity of the UCS produced a fear reaction. This being contrary to the original conditioning model which suggested that both the number of repetitions and the intensity of the UCS strengthen the conditioned response.

Incubation, where fear increases over non-reinforced presentations of the CS is noted to occur frequently in clinical populations (Eysenck, 1979). It has not been reported to occur as frequently in laboratory experiments. The Sanderson et al (1963) study is reported to be one of the few examples of laboratory fear incubation (Rachman, 1998). The conditioning model would not predict incubation, where an unreinforced presentation of the CS would be expected to produce a decrement in fear. Therefore it is possible that in the clinical population, some other processes may influence an increase in fear. This issue will be further discussed in section 1.4, in the discussion of contemporary models of conditioning.

Davey (1997) also raises the problem with the conditioning model where it assumes that all stimuli can be transformed into a fear signal. The assumption of equipotentiality (Seligman & Hager, 1972) has not been found in either general population or psychiatric studies (Rachman, 1990). Davey (1997) reported that people tend to develop phobias of animals, heights and water more readily than fears of knives, electric outlets and guns. Menzies and Clark (1995) observe that the distribution of phobias consists of objects or situations that represent serious dangers to pretechnical rather than modern man. Therefore these fears may have served an

adaptive function to our ancestors, and are now part of our evolutionary biological inheritance.

It has also been suggested that phobias can be acquired through vicarious transmission of fear. Bandura (1969) demonstrated that both the processes of observational learning and modelling could influence emotional responses. Rachman (1968) proposed that fears could be acquired either directly or indirectly (vicarious transmission and transmission of information). He proposed that severe fears were acquired through direct conditioning and moderate fears through indirect conditioning. He further proposed that the anxiety correlates of fear would differ with direct conditioning experiences having more behavioural and physiological correlates and indirect conditioning experiences having elevated cognitive correlates, such as higher levels of negative cognitions (Rachman, 1976).

Rachman (1977) proposes evidence for indirect conditioning obtained from the second world war. It was observed that children were more likely to develop fears if their mothers were also fearful. More recently Ost (1987) reported that the second most common pathway to fear was vicarious (observing) experiences and the least common was via instruction/transmission of information in a group of patients referred with different phobias. Ost (1987) noted that phobias acquired through the least common method were acquired at an earlier age. Ollendick and King (1991) also found that childhood fears were often acquired from threatening information.

In a series of studies Mineka and Cook (1993) reported that laboratory reared rhesus monkeys that did not initially exhibit a fear of snakes acquired an intense and persistent fear of snakes after several sessions of watching models (monkeys that exhibited an intense fear of snakes). However the authors report that it is not clear what mechanisms are involved in the process of fear acquisition mediated by observational learning.

According to Mineka and Cook (1993) the mechanisms of observational learning might not be very different from those thought to be involved in direct conditioning. Bandura (1969) argued that both direct and vicarious conditioning processes are governed by the same basic principles of associative learning, but that they differ in the source of emotional arousal.

Ost and Hugdahl (1981) offer support for Rachman's (1977) hypothesis that phobias could be acquired through both direct and indirect pathways (vicarious and instruction/information). They found evidence for direct and indirect conditioning pathways. In studies examining several clinical groups (animal phobics, social phobics and claustrophobics, blood and dental phobics). Ost and Hugdahl (1985) found all three pathways could account for the acquisition of phobias. In all their studies however they found that the number of directly conditioned cases to be significantly higher than those who identified indirect pathways to account for their fears.

However Menzies and Clarke (1995) report that only amongst the animal phobics (direct conditioning group) in Ost and Hugdahls (1981) work was there a difference in severity of anxiety between direct and indirect conditioning pathways of fear acquisition. The animal phobics (direct conditioning) reported higher physiological reactions compared to subjective reactions when in the presence of the fearful object. Amongst the social and claustrophobic groups higher scores were observed on the subjective components (negative thoughts and anticipatory worry) irrespective of the acquired pathway. The claustrophobic indirect group, were also found to have the strongest behavioural reaction (time enclosed in a small test chamber and measure of heart-rate). This finding being contrary to Rachman's (1977) hypothesis, which suggests that the indirectly conditioned group would have displayed a less stronger behavioural reaction than the directly conditioned group.

Menzies and Clark (1995) argue that more research is required in relation to other phobias to test Rachman's hypothesis further.

In summary, classical conditioning has been recognised as one of the main theories to explain fear acquisition and can account for the motivating behaviour that reduces the strength of that fear. Much of the research to support this came from animal research, with some evidence coming from human research and the effects of traumatic stimulation. However a number of problems have been identified with the classical conditioning theory, such as its lack of predictive value as to the conditions under which a phobia would not be acquired, and the lack of recall of an aversive experience that would explain the fear. The classical conditioning approach was also

unable to explain the process of incubation, and the uneven distribution of fears breaches the view that all stimuli can be transformed into a fear signal. Alternative learning routes to fear acquisition have also been identified. These difficulties have led to the development of a contemporary model of conditioning, which is discussed in the next section.

1.4 Contemporary Model of Conditioning:

A recent contemporary model of conditioning has been proposed (Davey 1992). Davey (1992) argues that humans only exhibit a differential CR when they are able to verbalise the CS-UCS contingency (exhibit contingency awareness). He points to research findings where the relationship between the CS and UCS is masked (by distraction), and subjects fail to exhibit a conditioning response. Davey (1992) suggests that this implies a type of “cognitive relational learning”.

Davey (1989) has also argued that UCS revaluation may also have considerable importance in the modulation of human CR strength. He argues that UCS revaluation may be achieved by direct experience, for example an individual may reassess an aversive UCS more favourably, through, habituation. UCS revaluation may also be inflated; due to experiencing a similar UCS of greater intensity. White and Davey (1989) manipulated UCS evaluation in an electrodermal conditioning task, by inflating the UCS. After pairings of CS-UCS, the tone UCS was inflated by exposing the participants to similar tones of increased intensity. Presentations of the CS, subsequent to this resulted in an increased magnitude CR. The authors argue that the CR was mediated by a CS-UCS associative link.

Davey (1992) also argues that the UCS can be reevaluated by socially or verbally transmitted information. Davey and Mckenna (1983) found that informing participants that future UCS presentations would be less intense, elicited weaker CR responses (electrodermal).

Another way in which the UCS can be evaluated is the persons reaction to the CS or UCS (Davey, 1992). Davey (1989) and Russell and Davey (1991) found that when participants believed they were emitting a strong CR, when they are not (false feedback), they exhibit a resistance to extinction compared to subjects who believe they are emitting a weak CR.

Other factors which can influence the CS-UCS relationship have been identified as situational information (current information about the contingency) and prior expectations about the covariation. Alloy and Tabachnik (1984) found that where situational information is unambiguous and prior expectations are low, participants could detect event contingencies accurately. However a covariation bias can arise which distorts the perception of the covariation (Davey, 1992). Davey (1992) argues that covariation bias is important as it shows that the relationship between CS-UCS need not depend solely on the situational information contained in the CS-UCS contingency, but critically may depend as much on any prior expectation that the subject holds about the stimuli. According to Davey (1992) this may explain why not all stimuli can be transformed into a fear signal. Tomarken, Minelka and Cook (1989) argue that the distribution of fears to certain objects (prepared stimuli- see

section 1.6) may be due to covariation bias. In an experiment they exposed participants to slides of prepared and unprepared stimuli which was followed by shock, tone or nothing. The relationship between the slides and outcome were random however the participants overestimated the contingency between slides of prepared stimuli and shock. It is suggested that this experiment provides evidence that prior expectations influence the strength or course of conditioning in humans by generating a covariation bias which influences perception of the CS-UCS relationship (Davey, 1992). However Davey was unable to conclude as to the source of the expectancy bias and argues that it may be “pre-wired” as a result of natural selection (this is similar to Seligman’s argument see section 1.6).

Sensory preconditioning is another factor, which according to Davey (1992) can influence the CS-UCS contingency. In animal studies, when an animal is exposed to pairings of two neutral stimuli e.g. brief light (CS1) followed by a tone (CS2), there are no behavioural changes noted. However if the animal is then given pairings of CS2 with a UCS, subsequent presentation of CS1 alone will elicit a CR (Davey, 1992). Dickinson (1980) refers to this as behaviourally silent learning as it is only with further tests that it becomes known that learning occurred in the initial part of the procedure.

Davey (1992) argues that these factors can help explain the problems encountered by the classical conditioning model. He suggests that the lack of predictive value can be accounted for by UCS evaluation. As CRs are mediated by an internal representation of the UCS, the strength of any CR will depend on the individual’s evaluation of the

UCS. The conditioning experience may not result in a fear CR if the individual is able to devalue the aversiveness of the traumatic experience. The contemporary conditioning theory would therefore predict that pairings of a situation with trauma would not necessarily lead to a phobia if the traumatic UCS were devalued in some way immediately after the experience (Davey, 1992).

Davey (1992) also argues that sensory preconditioning can explain why some individuals appear unable to recall any trauma at the time of the first appearance of their fear to the phobic stimulus. He argues that it is conceivable for an individual to learn an association between a CS and UCS when the UCS is unaversive (sensory preconditioning). At a later stage through UCS revaluation the aversiveness of the UCS may become inflated subsequently.

The contemporary conditioning model is also believed to be able to account for incubation effects. Davey (1992) argues that evaluation of the CR can be influenced by the evaluation of the UCS, specifically through inflation occurring between successive presentations of the CS. UCS rehearsal is one way in which he suggests that the process of incubation can occur. Marks (1987) has reported that following a trauma, many individuals rehearse the event in their minds. Davey (1992) argues that rehearsal could lead to inflation of the UCS prior to subsequent encounters with the CS.

Davey (1992) also argues that his model can explain why fears appear to attach to some events and situations and not others. His explanation for this is based on the

findings that the CS-UCS relationship can be determined by pre-existing beliefs. The uneven distribution of fears will be based on outcome expectancy biases, if beliefs about the relationships between certain events and traumatic outcomes are held, expectancy biases according to Davey (1982) will operate to produce an uneven distribution of fears.

Davey's (1992) analysis also considers direct and indirect acquisition processes. Indirect conditioning is known to influence behaviour by producing outcome expectancies (Bandura, 1977). As has already been identified outcome expectancies have an important role in covariation and in determining the strength of the conditioned responses. Therefore according to the contemporary model indirect experiences may influence the nature of learning when the individual does not encounter a direct experience. Expectancies can also produce differential CRs in the absence of a direct experience with an UCS, therefore the nature of the CR will depend on the information encoded in the activated UCS representation (Davey, 1992). According to Davey (1992) direct and indirect conditioning are functionally and dynamically similar. He argues that they obey similar associative rules, but differ only to the extent to which they are different learning procedures (direct experience with an UCS may encode different information into a UCS representation than an indirect experience, with this information influencing the strength of the CR). However Davey (1992) does suggest that more work is required on the relationship between direct and indirect conditioning, especially measures of response strength and persistence and the nature and content of the UCS representations generated by both types of learning.

In summary the contemporary conditioning model appears to offer explanations to account for the limitations found in the classical conditioning literature. Individuals presenting with anxiety disorders are known to present with cognitive biases centred around threat and harm (Beck, Emery & Greenberg, 1985) therefore it is conceivable that these biases could account for inflation of the UCS through revaluation.

1.5 The Panic and Alarm Theory of Fear Acquisition:

According to the panic and alarm theory of fear acquisition, exposure to an environmental UCS is neither necessary nor sufficient for fear conditioning (Forsyth & Eifert, 1995). Barlow (1988) describes fear as a biological response, which can be described in terms of true, false and learned alarms. True alarms are derived from the biological “fight or flight” mechanism. These responses are associated with increased oxygen intake and increased blood flow. They are also associated with verbal evaluations of harm or threat. False alarms are believed to occur in the absence of real harm or threat. Barlow, Brown and Craske (1994) describe panic attacks as a common example of false alarms. Panic attacks in anxiety disorders are a central defining diagnostic feature according to DSM-IV diagnosis criteria.

Barlow (1988) suggests that the repeated occurrence of false alarms may result in learned alarms through their association with an internal or external cue. It has been suggested that learned alarms may become associated with interoceptive cues (the perception of events within the body, such as nausea, vertigo and visceral sensations) through a process of interoceptive conditioning. This applies to a learned relation

between internal somatic cues and other sensations of the alarm response (Forsyth and Eifert 1995).

Forsyth and Eifert (1995) suggest that the type of conditioning relevant to phobias may be exteroceptive (an environmental CS and an interoceptive UCS). Merckelbach, Rutter, van den Hout and Hoekstra (1989) suggested that all conditioning experiences consisted of frightful internal bodily sensations. McNally and Steketee (1985) reported that amongst a group of animal phobics it was not the fear of the animal per se but the consequences that the animal had come to represent, specifically aversive bodily sensations and panic. According to McNally and Laska (1992) these aversive bodily sensations are capable of producing Post-Traumatic Stress Disorder (PTSD) like symptoms in some patients.

Forsyth, Eifert and Thomson (1996) have reported preliminary data, which they argue provides experimental support that abrupt, autonomic bodily responses can produce, fear conditioning in the absence of an identifiable direct experience with environmental pain or trauma. They developed an interoexteroceptive conditioning preparation using high concentrations of CO₂ enriched air (the psychophysiological effects of breathing CO₂ are characteristic of patients with panic disorder e.g. tachycardia, breathlessness, dizziness) paired with animated environmental and bodily video stimuli. This is in contrast to the exteroexteroceptive type that involves pairings of environmental CSs and environmental UCSs such as shock or noise. In their study they demonstrated that repeated pairings of 20sec inhalations of 20 per cent CO₂ enriched air (UCS) produced abrupt autonomic responses in normals with

changes in self-reported distress and fear (electrodermal and cardiac domains) to fear relevant stimuli (snake and heart) compared to fear irrelevant (flowers and sperm) CSs. They argued that this study provided experimental support for the view that anxiogenic responses can produce fear conditioning in the absence of an identifiable direct experience with pain or trauma. Forsyth et al (1996) also observed that participants who showed little response to the UCS also showed little evidence of conditioning. This observation was taken to highlight that the presence of a nonthreatening stimulus (NS)-UCS pairing was not sufficient to produce conditioning (as suggested by the original Pavlovian model). It was therefore suggested that response (UCR) intensity may be an important factor to consider in accounting for individual differences in fear conditioning (Forsyth et al, 2000). The authors point out however that they are not implying that a UCS is not involved in conditioning but that they dispute findings that an identifiable UCS is the only evidence for direct conditioning.

Forsyth et al (2000) further examined the UCS-UCR intensity responses by using two UCS intensities (13 per cent and 20 per cent CO₂) paired with the following CSs (snake, heart and flowers). It was hypothesised that individual differences in response intensity would account for more of the variance in fear conditioning than UCS intensity. The UCR was expected to mediate the UCS → CR relation (Forsyth et al, 2000). It was found that greater UCR intensity predicted greater conditioned fear, this effect was found to be robust across response domains and CSs (snakes and heart). The authors argue that this finding may explain why some

individuals develop PTSD and others do not when exposed to the same traumatic event.

1.6 Evolutionary Models of Fear Acquisition:

Fear acquisition has also been explained in terms of evolutionary models. Much of the basis for this approach has developed from the finding of the non-random distribution of fears. It is thought that fear and anxiety play a survival role (Merckelbach & de Jong, 1997) and that fearfulness is partially under genetic control (Stevenson, Batten & Cherner, 1992). Three evolutionary approaches are described by Merckelbach and de Jong (1997). The first focuses on one fear, blood-injection-injury phobia (BII). It is argued that the vasovagal syncope observed in this type of phobia is related to tonic immobility, death feigning and freezing seen in animal adaptive behaviours (Marks & Nesse, 1994). Secondly it is believed that the purpose of the vasovagal syncope is to slow down the circulation which is adaptive if the organism is loosing blood (Barlow, 1988). However in a study carried out by De Jongh et al (1998) no association between fainting and BII phobia was found.

The second evolutionary approach is based on a non-associative account of fear acquisition. Menzies & Clark (1995) argue that no direct or indirect traumatic pairing with the feared stimulus is required to explain phobic onset. They suggest that evolutionary pressures have endowed certain stimulus configurations “prepotent” cues (e.g. fear of heights, odors, and novelty) with fear evoking functions. These cues were viewed, as threatening to the species and innate fear reactions to them would have increased survival.

The third evolutionary approach evolved from the work of Seligman (1971). He argued that the non-random distribution of fears could be accounted for by a biological predisposition to acquire certain fears following associative learning (conditioning). Seligman's "preparedness" theory combines both conditioning theory and non-associative learning. Seligman (1971) argued that conditioning accounts of phobias should be considered from within an evolutionary framework. His basis for his premise is the argument that evolutionary pressures had selected for an adaptive predisposition to associate pre-technologically dangerous stimuli with aversive consequences and this could account for the disparity that fears appeared to attach to some objects and situations e.g. snakes more than others e.g. cars. Although he recognised that fears could also attach to unprepared stimuli, he believed that the learning process was different for prepared and unprepared stimuli. He proposed that fears to prepared stimuli were rapidly acquired, were resistant to extinction and resistant to the influence of cognitive factors such as instructions and information (Seligman, 1971).

He argued that individuals who acquired such fears would be more likely to survive, reproduce and pass on genetic information. Therefore there was an adaptive value in the development of certain fears.

Menzies and Clark (1995) argue that this model could account for some of the difficulties found with the original conditioning model. They argue that it could

account for the quick development of phobias with nonreinforced exposure, it could also account for the difficulties with fear extinction. Ohman, Fredrickson, Hugdahl and Rimmo (1976) reported that electrodermal responses conditioned to evolutionary fear-relevant stimuli (snakes, spiders) are more resistant to extinction than responses conditioned to evolutionary-fear-neutral stimuli (houses, flowers and mushrooms). McNally and Foa, (1986) have not obtained evidence to support these findings. Davey (1992) also reported that little evidence exists for rapid acquisition of prepared fears in laboratory studies using human subjects.

The evolutionary models, place their emphasis on genetic influences and fear acquisition, however as already discussed prior expectation of the CS-UCS contingency, which may arise from cultural influences may also be influential in the acquisition of fear. Thus possibly weakening the biological premise of the evolutionary models. Although evolutionary models of fear acquisition have not been proposed to account for dental anxiety it is possible that non-associative influences may be involved in the acquisition of the disorder.

1.7 Cognitive Models of Fear Acquisition and Maintenance:

The importance of the role of beliefs and expectations in the aetiology and maintenance of anxiety disorders are well researched. The main premise of the cognitive model is that dysfunction arises from an individual's interpretation of events with behavioural responses arising from these interpretations seen as important in the maintenance of emotional problems (Wells, 1998). Although other

cognitive approaches have been developed (Ellis 1962), the model of anxiety proposed by Beck (1976) will be considered here.

Beck's (1976) cognitive theory of emotional disorders suggest that anxiety is accompanied by distortions in thinking specifically centred around harm and danger, this bias in thinking also maintains the emotional response. It is this fixation on danger and harm and an associated underestimation of ability to cope which activates the underlying danger schemas² (Beck, Emery & Greenberg, 1985). Anxiety schemas contain assumptions and beliefs about danger to one's personal domain (Beck et al 1985). Negative automatic thoughts (NATs) are appraisals or interpretations of events and according to Beck et al (1985) can occur outside of the focus of immediate awareness but are amenable to consciousness. NATs can occur in both imaginal and verbal form. The same authors suggest that in specific phobias individuals associate a situation or object with danger and hold assumptions concerning the negative events that could occur when exposed to the stimulus.

Dysfunctional assumptions and beliefs are believed to result from early experiences. Rachman (1998) observed that cognitive models of anxiety accept that the acquisition of fears may be as a result of maladaptive learning, including conditioning. However the emphasis is on the individuals interpretation of the event whether the fear was acquired by conditioning or not.

² Beck evoked the concept of schemas. These represent the sum of previous experiences, serving as templates that direct attention, influence encoding, interpretation of stimuli and facilitate recall

Merckelbach et al (1996) argue that specific phobia is not characterised by a general cognitive dysfunction e.g. deficits in memory, motor deficits. They argue that cognitive dysfunctions in specific phobias are restricted to certain processes i.e. attentional and judgmental. Attentional biases have been demonstrated in specific phobia using the modified Stroop task (subjects are required to name the colour of a word while ignoring the meaning of the word). Lavy, van den Hout and Arntz (1993) found that spider phobics recorded longer colour naming latencies when they were presented with spider related words compared to non-spider related words. Merckelbach et al (1996) explain this finding in terms of the spider phobic subjects automatically directing their attention to the content of the threatening words, which interferes with their main task. The same authors suggest that attentional bias has clinical consequences. MacLeod and Hagan (1992) found that an attentional bias towards threat-related material was a good predictor of emotional distress elicited by stressful life events.

Merckelbach et al (1996) argue that two types of judgmental bias operate in the maintenance of specific phobias. The first is covariation bias, which is the tendency to overestimate the association between phobic stimuli and aversive outcomes (Merckelbach et al, 1996; Tomarken, Sutton & Minelka, 1995). This has been demonstrated experimentally (de Jong, Merckelbach & Arntz, 1995). Under experimental conditions phobic subjects overestimate the contingency between phobic stimuli and aversive outcomes, as discussed in the Tomarken, Minelka and Cook (1989) study. Residual covariation bias following treatment for spider phobics has been found to predict higher spider fear at 2 year follow-up (de Jong et al, 1995;

Merchelbach et al 1996). According to Merchelbach et al (1996) the tendency to attribute aversive experiences to the phobic object will sustain phobic fear.

The second judgmental bias believed to occur in specific phobias is that of emotional reasoning (Beck & Emery, 1985). Arntz, Rauner and van den Hout (1995) argue that fearful subjects tend to infer danger from fear, i.e. anxiety symptoms imply the presence of danger. According to Merchelbach et al (1996) emotional reasoning may serve to legitimise the phobic fear and may maintain the phobia.

A study by Thorpe and Salkovskis (1995) also found evidence that conscious cognitions were present and central in a wide range of specific phobias and were important in the maintenance of the disorder.

1.8 Neurobiological Model of Fear Acquisition:

Studies carried out by Kluver and Bucy (1937) gave the first insight into the neurobiological substrates of fear acquisition. Following temporal lobe resections in monkeys, it was observed that their behaviour was characterised by visual agnosia, hypersexuality, reduced neophobia and loss of fear. Later work indicated that the reduced fear in resected monkeys was due specifically to damage in the amygdala (Maren & Fanselow, 1996). The amygdala is thought responsible for initiating a variety of hard-wired responses to threat (Brewin, 2001). These include the release of stress hormones, activation of the sympathetic nervous system, and the behavioural responses such as fight/flight and freezing (Armony & LeDoux, 1997). It is believed that the amygdala integrates internal representations of the world in the form of

memory images with emotional experiences associated with those memories (Armony & LeDoux, 1997). Following assigning meaning to sensory information, the amygdala guides emotional behaviour by projections to the hypothalamus, hippocampus and basal forebrain (le Doux, 1995; van der Kolk, 1994).

LeDoux (1995) suggests that within the amygdala there are two systems, which have specific roles with regard to fear acquisition. The basolateral complex (BLA) of the amygdala is thought to be a substrate for sensory convergence from both cortical and subcortical areas and is a locus for CS-UCS association during fear conditioning. In contrast the central nucleus of the amygdala which receives projections from the BLA projects to brain areas involved in the generation of fear responses, such as the lateral hypothalamus (Maren & Fanselow, (1996). Destruction of neurones in the BLA or central amygdala has been found to be detrimental to both the acquisition and expression of conditioned fear (LeDoux 1995). The thalamus-amygdala pathway (the thalamus carries out a quick analysis of the sensory input which then activates the amygdala which generates an emotional response). According to LeDoux (1995) this is a “quick and dirty” route, which in certain situations allows rapid responses to threatening stimuli, which it may be argued, have an adaptive value.

Urbach-Wiethe disease is a rare disorder that results in bilateral degeneration of the amygdala. Bechara et al (1995) studied a patient with this disorder and found that compared with normal control patients there was no evidence of a fear response following either a visual or auditory CS-UCS pairing (loud noise). Although this patient displayed a fear conditioning deficit, she was found to be able to accurately

recall the training procedures. A study carried out with a patient with combined amygdala and hippocampal damage found that both fear conditioning and recall of the procedure was disrupted (Maren & Fanselow, 1996).

The hippocampal system is thought to record in memory the spatial and temporal dimensions of experience, playing an important role in the categorisation and storage of incoming stimuli in memory (van der Kolk, 1997). According to van der Kolk (1994) proper functioning of the hippocampal system is necessary for declarative memory.

Maren and Fanslow (1996) suggest that the amygdaloid system plays a critical role in fear conditioning and the hippocampal system mediates declarative memory for the events associated with training. Further evidence that suggests the amygdala participates in fear acquisition is the finding of high densities of binding sites to substances that modulate fear and aggression such as the benzodiazepines (Niehoff & Kuhar, 1983) and serotonin (Hensman, Guimaraes, Wang & Deakin, 1991).

Most processing of sensory input according to van der Kolk (1994) occurs outside of conscious awareness. Therefore it appears that these brain structures are significantly involved in fear acquisition and response at the associative level.

There does appear to be evidence that both the amygdala and hippocampus are involved in conditioned fear and memory. It is possible that other anatomical areas are also involved in the mediation of fear and fear memories. A better understanding

of brain systems that are involved in fear mediation may lead to more effective pharmacological strategies for treating clinical fears. Drawing on both biological and psychological knowledge may allow a better understanding of the processes involved in fear acquisition, maintenance and treatment.

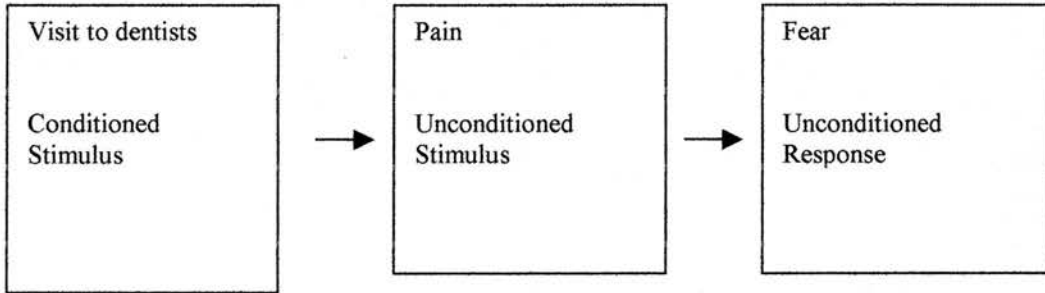
1.9 Dental Anxiety: Acquisition and Maintenance

Lautch (1971) who conducted one of the earliest studies into dental anxiety suggested that dental fears are the result of learned associations between traumatic experiences, dental care and the role of classical conditioning. He studied 34 dental phobic patients and 34 control subjects. He reported that all the 34 dental phobics had experienced a previous traumatic dental experience, mostly physical pain however some patients reported a feeling of oncoming disaster. Ten of the non-phobic subjects also reported a previous comparable traumatic dental experience. He also found that the majority of phobic patients in his study developed dental phobia in childhood and adolescence. All the dental phobic patients could recall the traumatic experience vividly and according to the author any reference to dentistry either in thought or reality produced vivid images of the traumatic experience and heightened anxiety.

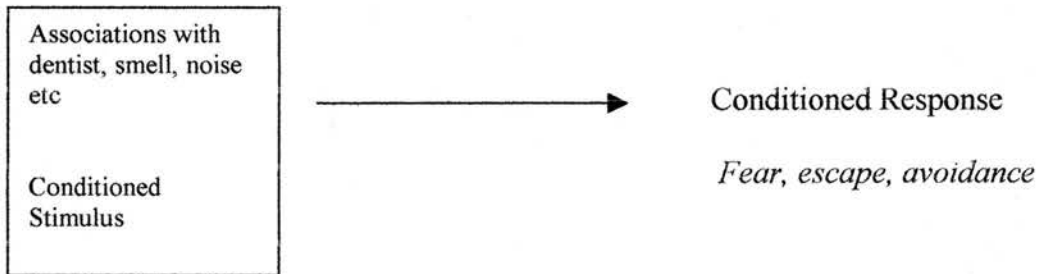
An example of a dental fear conditioning response is presented in figure 1.

Figure 1. Example of a Dental Fear Conditioning Response.

During conditioning



After Conditioning



A number of other earlier studies have also investigated the origin of dental phobia. Kleinknecht, Klepac and Alexander (1973) studied 487 students and found that 17 per cent reported negative expectancies from others as the perceived origin of their dental fears, 13.5 per cent had experienced painful dental work and 17 per cent reported poor dental management, physical abuse by the dentist and perceived mistakes as the origin of their fears. Bernstein, Kleinknecht and Alexander (1979)

investigated 225 students, amongst the students who reported a fear of the dentist, 22 per cent attributed their fear to a single traumatic incident occurring in the dental chair, 19 per cent reported vicarious factors and 50 per cent reported negative dentist behaviours.

Ost and Hugdahl (1985) as reported earlier carried out an investigation into the origin of dental and blood phobias amongst a clinical population. Their findings of classification of the acquisition of phobias are presented in Table 1.

Table 1. Acquisition of phobias of dental and blood phobics

WAY OF ACQUISITION	DENTAL	BLOOD
Direct conditioning	n 35 (68.6%)	n 10 (45.5%)
Vicarious experiences	n 6 (11.8%)	n 7 (31.8%)
Instruction/Information	n 3 (5.9%)	n 2 (9.1%)
No Recall	n 7 (13.7%)	n3 (13.6%)

The same authors also investigated Rachman's (1978) hypothesis that different pathways of fear would show differences in anxiety components. No significant differences were found between dental phobics (direct and indirect conditioning) on the measures of physiological reactions (heart-rate), negative thoughts (regarding the worst phobic situation) or anticipatory worry. Phobias acquired through indirect pathways were found to have higher, but non-significant scores on a specific dental

fear questionnaire (Dental Anxiety Scale) than those phobias acquired via direct conditioning pathways. This is contrary to Rachman's prediction.

Ost and Hugdahl (1985) also found that 25 per cent of the dental phobics experienced mild-to-moderate forms of generalised anxiety that was not associated with their phobic situation. This was compared to 9 per cent of the blood phobics.

Kent (1998) suggests that in general, a classical conditioning model is probably the most helpful in understanding the onset of dental anxiety and dental phobia. However he does suggest difficulties with the classical conditioning processes related to dental fears. One difficulty is that many individuals who are not highly dentally anxious or dental phobic have also experienced a situation where classical conditioning occurred (as found in Lutch's original study). Vassend (1993) found that 30 per cent of a sample of non-phobics had experienced intense pain at some point in their dental histories. Davey (1989) found that a group of individuals who reported a traumatic painful dental experience but did not develop dental anxiety had a history of trauma free dental treatments prior to the traumatic episode. This finding was accounted for by latent inhibition.

A number of recent studies provide evidence for the role of direct conditioning experiences in the acquisition of dental fear. De Jongh, et al (1995a), and Locker, Liddell, Dempster and Shapiro (1999) report that the majority of patients with clinically significant dental fears identify the onset of their fears to one or more traumatic dental event (painful treatment or treatment associated with terror).

De Jongh et al (1995a) found that amongst a group of undergraduate students the extent to which earlier dental treatments were perceived as painful, the extent to which earlier dental treatments were perceived as traumatic, and the frequency of painful dental experiences were all related to levels of dental anxiety. The authors also found that the dentally anxious students exhibit a high occurrence of negative thoughts. They were found to exhibit more catastrophizing thoughts, experience more difficulties in suppressing their negative thoughts and experience greater difficulty implementing relaxation and distraction techniques during dental procedures. This finding that negative cognitions play an important part in the presentation of dental anxiety supports the work by Thorpe and Salkovskis (1995) who observed that conscious cognitions are central in phobic disorders.

De Jongh, Muris, ter Zuuren, Schoemakers & Makkes (1995b) further assessed the influence of cognitive factors, which may maintain or exacerbate dental anxiety. The authors constructed The Dental Cognitions Questionnaire (DCQ) to assess the frequency and believability of beliefs pertaining to dental treatment. De Jongh et al (1995b) found that dental phobic patients reported a higher frequency of negative and catastrophic thoughts than a group of non-phobic control participants. The authors found that the thought content of dental phobic patients revealed that many beliefs related to suffocation and loss of control, which De Jongh et al (1995a) suggest resembles the contents of thoughts held by patients with panic disorder and other types of phobias. Thus adding further evidence that dental phobic patients may have characteristics of other anxiety disorders.

The dental phobic patients also reported a greater degree of belief in their negative thoughts. It was reported in this study that specific negative cognitions (loss of control, negative consequences of treatment, the dentist and condition of teeth) explained more than 70 per cent of the variance of dental phobics state anxiety ratings in the dental situation.

De Jongh, Muris, Schoenmakers & Ter Horst (1995c) argue that if treatment for dental phobia is to be effective, treatment should address the central beliefs and negative self-statements held by the patient irrespective of the level of dental anxiety. De Jongh et al (1995c) argue that the negative beliefs dental phobics hold about themselves and dental treatment are important determinants of their psychological distress. The authors carried out a study looking at one-session cognitive treatment of dental phobia. They found that compared to a group given an information intervention and a waiting list control group, a cognitive restructuring group displayed a decrease in frequency and believability of negative cognitions and a reduction in dental anxiety. However at follow-up (one-year post treatment) no differences were found between the information and cognitive restructuring group for dental anxiety. However it is possible that implementing just one session may not eliminate strongly held beliefs amongst patients who have been dentally anxious for some time. Beck et al (1985) do not recommend one session of cognitive therapy for patients with moderate or severe anxiety, with the recommendation of twenty sessions over a number of months. Further research is obviously required to address

both the identification of beliefs amongst dental anxious patients and cognitive interventions.

Indirect conditioning has also been investigated in the aetiology of dental phobia. Studies have examined the influence of these factors and have found that parental reactions especially maternal are an important aetiological factor (Horst & Wit, 1993; Milgrom, Mance, King & Weinstein, 1995). As discussed earlier, Locker et al (1999) found that a family history of dental anxiety was important and was predictive with respect to child-onset anxiety. Milgrom et al (1995) also found that direct conditioning and modeling were both important predictors of dental anxiety originating in childhood.

Townend, Dimigen and Fung (2000) also found similar influences, they reported a strong association with the conditioning pathway of dental fear acquisition, followed by the modelling pathway amongst a group of children with dental anxiety. They also found that children's fear was more strongly associated with subjective experience of pain and trauma than with objective dental pathology.

It is clear from the literature that where individuals are able to recollect the incidents that they believe contribute to their dental fears most recall experiences associated with pain or terror (De Jongh et al 1995a ; Moore et al, 1991).

De Jongh, Aartman and Brand (manuscript submitted for publication) investigated the extent to which individuals with dental phobia presented with trauma-related

symptomatology. Two groups were investigated, a group of patients with dental phobia and a group of non-dentally anxious controls. They found that the proportion of patients who reported a distressing dental procedure did not differ between the two groups. However significantly more patients in the phobic group reported intrusive memories of their distressing dental events. This study will be further discussed in chapter five and study two in relation to trauma-related symptoms in, patients with dental anxiety.

Study one will investigate pathway of conditioning and levels of dental anxiety, physiological arousal and behavioural avoidance related to dental care amongst a group of patients with dental anxiety.

Aims Of Study 1

Study 1 tested Rachman's (1968) hypothesis that direct conditioning experiences will lead to higher levels of fear than indirect conditioning experiences. This was investigated in a group of patients with dental anxiety.

Hypotheses

- 1) Direct dental conditioning experiences will lead to higher levels of dental anxiety as measured by the Modified Dental Anxiety Scale than dental anxiety acquired via indirect conditioning experiences.
- 2) Direct dental conditioning experiences will lead to higher physiological arousal associated with dental situations and procedures as measured by the Dental Fear

Survey (physiological arousal scale) than dental anxiety acquired via indirect conditioning experiences

- 3) Direct dental conditioning experiences will lead to higher levels of behavioural avoidance associated with dental situations and procedures as measured by the Dental Fear Survey (behavioural avoidance scale) than dental anxiety acquired via indirect conditioning experiences

Chapter 2: Study 1: Comparison of Dental Anxiety Measures and Direct and Indirect Pathways of Fear Acquisition.

Methods

2.1 Design: A between subjects design was used to investigate the differences between the two conditioning groups (direct and indirect) on the measures of dental anxiety, physiological arousal and behavioural avoidance related to dental situations and procedures.

2.2 Sample: The sample consisted of adult patients who attended the Dundee Dental Hospital Anxiety Clinic during University of Dundee terms October 1998-May 2000. The Dundee Dental anxiety clinic (University of Dundee and Tayside Teaching Hospital Trust) was set-up in 1998. It was developed for the clinical teaching of anxiety management for final year, undergraduate dental students. The clinic only accepts patients who are suitable for student care. A clinical psychologist, two dental surgeons and a dental assistant staff the dental anxiety clinic. The clinic runs from October to May to coincide with the university teaching terms.

Patients were referred to the anxiety clinic from both dental hospital staff (dental surgeons) and community, dental surgeons. The clinical assessment and data collection for study one was carried out by the clinical psychologist, dental surgeons and undergraduate dental students working in the dental anxiety clinic. The author

carried out no data collection for this study. The clinical psychologist, the dental surgeons or the undergraduate dental students, perform the general clinical assessment (see below). The dental staff perform the dental examination.

2.3 Measures:

The Modified Dental Anxiety Scale (MDAS) (Appendix 1) (Humphris, Morrison & Lindsay, 1995): The MDAS is a modified version of the Corah's Dental Anxiety Scale (CDAS) (1969) which has been widely used for measuring dental trait anxiety (De Jongh, et al 1995c). Although the CDAS has been widely used and information about reliability and validity is widely available, it was believed that patients could be confused by some of the questions (Humphris et al, 1995). The CDAS was found to confound answers reflecting emotions with answers describing physical experience. It was also considered that in addition a further item was essential specifically relating to local anaesthesia. Fear of a needle injection was ranked as high as the drill in terms of dental fear (Stouthard & Hoogstaaten, 1987).

The MDAS consists of 5 questions with scores added to give a general dental anxiety score. A cut-off score of 19 and above indicates the respondent as being dentally anxious. The cut-off point has been determined by a reliably defined group of dental phobics with sensitivity and specificity estimates presented (Humphris et al, 1995). The CDAS cut-off point was determined by clinical judgement without supporting data to allow assessment of the suitability of the cut-off (Corah, Gale & Illig, 1978).

The MDAS has been shown to demonstrate high levels of reliability as calculated by the use of Cronbach's alpha formula, α 's above 0.7. The sensitivity and specificity of the measure adopting the cut-off point of 19 were 0.85 and 0.91 respectively. Further research has been carried out to gain further evidence of the psychometric properties of the MDAS. In a multi-national study the overall reliability was 0.89 (Humphris, Freeman, Campbell, Tuutti & D'Souza, 2000).

Dental Fear Survey (DFS) (Kleinknecht, Klepac and Alexander, 1973). Participants are asked to rate their anxieties about twenty-seven situations related to dental fear. The first area assesses patient's avoidance of dentistry because of fear; the second area assesses degree of physiological arousal; the third area measures the degree of fear associated with dental situations and procedures; the final area provides an overall rating of the patient's fear related to dental treatment. The questionnaire is scored by adding the scores for each of the 4 areas separately plus a total score for the measure. The maximum avoidance score is 10, physiological arousal 25, procedures score, 60 overall fear, 5 and total score, 100. There is no specific cut-off but higher scores indicate more dental fear. According to Schuurs and Hoogstraten (1993) the DFS is primarily designed to detect the fear induced by the separate categories. The same authors report that the DFS has been shown to lack validity. However in one study comparing the CDAS and the DFS, phobic behaviour was found to be better identified by the DFS (Johansson & Berggren, 1992). Schuurs and Hoogstraten (1993) report test-retest reliability coefficients of 0.74 for items across participants and 0.73 for participants across all items. Schuurs & Hoogstraten, 1993 also report a split-half reliability Cronbach's $\alpha = 0.93$ and $r = 0.96$.

2.4 Clinical Interview: Clinical assessments are carried out for each patient who attends the dental anxiety clinic. This includes demographic details and dental and psychological history. Patients are asked whether they have experienced a bad dental experience, those who answer yes, are recorded as having a direct conditioning experience. Patients who answer no to this question are categorised as having an indirect experience.

The outcome of the assessment is discussed with the patient and a treatment plan is developed which includes dental, pharmacological and behavioural interventions. Some patients opt for pharmacological treatment alone, these patients are then transferred to a sedation only waiting list. Dental treatments and behavioural management of anxiety are carried out by final year, undergraduate dental students, under supervision from the clinical psychologist and dental surgeons.

2.5 Analyses: Analysis was carried out using the Statistical Package for Social Sciences/Windows (SPSS 10). Exploratory data analysis was used to determine the level of normality relating to distribution of the measured variables. An independent-samples t-test was used to identify differences between group 1 (direct conditioning) and group 2 (indirect conditioning) on measures of dental anxiety (MDAS) and physiological and behavioural avoidance (DFS).

Chapter 3: Results: Study 1 Comparison of Dental Anxiety Measures and Direct and Indirect Pathways of Fear Acquisition

The data set was checked for errors prior to analysis. No out-of-range values were found for any of the variables. Missing data were noted and the patients notes were checked accordingly. Exploratory data analysis was performed to provide information concerning the distribution of scores on continuous variables and to assess if the data met the assumptions for parametric or non-parametric statistical analysis.

3.1 Demographic Data:

3.1.1. Participants

Data were analysed from sixty-one patients, nine males and fifty-two females.

Patients were separated into two groups based on their inclusion in either the direct or indirect conditioning group. Forty-five patients were recorded as having a direct conditioning experience. Sixteen patients were identified as having an indirect conditioning experience.

3.1.2 Exploratory Data Analysis of the MDAS, DFS Behavioural Avoidance and Physiological Arousal Scales

Histograms were used to display the distribution of scores for the above measures for the direct and indirect conditioning groups (see appendix 2; MDAS, appendix 3: DFS physiological scores, appendix 4: DFS behavioural avoidance scores).

Altman and Bland (1995) suggest that relying on visual inspection of the distribution of scores to determine the assumption of normality is unreliable. The authors argue that formal statistical analysis should be performed to assess whether the sample data conforms to a normal distribution. The Kolmogorov-Smirnov statistic was performed to formally assess the assumption of normality. The results of this test are presented in table 2.

Table 2. The Kolmogorov-Smirnov Test of Normality, Study 1

Measure	condir	df	Sig
MDAS	direct	45	.076
	indirect	16	.200
DFSPHYS	direct	45	.200
	indirect	16	.200
DFSVOID	Direct	45	.083
	Indirect	16	.154

*MDASTOT scores for Modified Dental Anxiety Scale

*DFSPHYS scores for DFS physiological arousal scale

* DFSVOID scores for DFS avoidance subscale

A non-significant result (sig. value of more than 0.05) indicates normality. The results obtained from this population suggest that the assumption of normality has not been violated. As the assumption of normality has not been violated, parametric tests were applied for the analysis.

Table 3 presents the mean age for the direct and indirect conditioning group. An independent-samples t-test was conducted to compare the ages for the direct and indirect conditioning groups.

Table 3. Mean age for the Direct and Indirect Conditioning Groups and Independent-samples t-test for age, Direct and Indirect conditioning groups, Study 1

Group	N	Mean (SD)	df	t	Sig (2-tailed)	95% CI	
						lower	upper
Direct	45	34.82 (12.47)	59	.405	.687	-5.70	8.59
Indirect	16	33.38 (11.64)					

There was no statistically significant differences found for age between the direct and indirect conditioning groups, ($t(59)=.405$, $p=0.687$, two-tailed).

3.1.3 Hypotheses-Related Data

Hypotheses 1.

Hypotheses one predicted that direct dental conditioning experiences will lead to higher levels of dental anxiety than dental anxiety acquired via indirect conditioning experiences. An independent-samples t-test was conducted to compare dental anxiety scores (MDAS) between the direct and indirect conditioning groups. Table 4 presents the mean scores for dental anxiety for the direct and indirect groups and Independent-samples t-test between the conditioning groups.

Table 4. Mean Scores for Dental Anxiety Levels, Study One and Independent-samples t-test MDAS between Direct and Indirect conditioning groups, Study 1

Group	N	Mean (SD)	df	t	Sig (1-tailed)	95% CI	
						lower	upper
Direct	45	21.47 (2.30)	59	.242	.404	-1.46	1.15
Indirect	16	21.63 (2.06)					

There was no statistically significant difference found for dental anxiety between the direct and indirect conditioning groups. The direct conditioning group were not found to have higher levels of dental anxiety compared to the indirect group, ($t(59) = .242$; $p = 0.404$, one-tailed).

Hypotheses 2

Hypotheses two predicted that direct conditioning experiences will lead to higher levels of physiological arousal associated with dental situations and procedures than dental anxiety acquired via indirect conditioning experiences. An independent-samples t-test was conducted to compare DFS physiological scores between the two conditioning groups.

Table 5. Mean DFS scores for physiological arousal: Direct and Indirect conditioning groups and Independent-samples t-test for DFS physiological arousal, Direct and Indirect conditioning groups, Study 1.

Group	N	Mean (SD)	df	t	Sig (1-tailed)	95% CI	
						lower	upper
Direct	45	19.91 (3.90)	59	1.25	.115	-1.34	5.42
Indirect	16	17.88 (6.04)					

There was no statistically significant difference found for physiological arousal between the direct and indirect conditioning groups. The direct conditioning group were not found to have higher levels of physiological arousal compared to the indirect group, ($t(59)=1.25$; $p=0.115$, one-tailed).

Hypotheses 3

Hypotheses three predicted that direct conditioning experiences will lead to higher levels of behavioural avoidance associated with dental situations and procedures than dental anxiety acquired via indirect conditioning experiences. An independent-samples t-test was conducted to compare DFS behavioural avoidance scores between the two conditioning groups.

Table 6. Mean DFS scores for behavioural avoidance, Direct and Indirect conditioning groups and Independent-samples t-test for DFS behavioural avoidance, Direct and Indirect conditioning groups, Study 1.

Group	N	Mean (SD)	df	t	Sig (1-tailed)	95% CI	
						lower	upper
Direct	45	7.09 (1.69)	59	1.06	.151	-0.76	2.31
Indirect	16	6.31 (2.75)					

There was no statistically significant difference found for behavioural avoidance between the direct and indirect conditioning groups. The direct conditioning group were not found to have higher levels of behavioural avoidance than the indirect group ($t(59)=1.06$; $p=0.151$, one-tailed).

Chapter 4: Discussion Study 1: Comparison of Dental Anxiety Measures and Direct and Indirect Pathways of Fear Acquisition.

Study one was conducted to test Rachman's (1968) hypothesis that direct conditioning experiences will lead to higher levels of fear than indirect conditioning experiences. This was investigated in a group of patients with dental anxiety.

It was predicted that direct dental conditioning experiences would lead to higher levels of dental anxiety than dental anxiety acquired via indirect experiences. However this was not supported. Dental anxiety identified as being acquired via the direct pathway did not lead to higher levels of dental anxiety.

It was also predicted that direct dental conditioning experiences would lead to higher levels of physiological arousal associated with dental situations and procedures than dental anxiety acquired via the indirect pathway. Although the mean DFS physiological subscale was found to be numerically higher in the direct conditioning group, statistical analyses did not find any differences between the two groups. The hypothesis was not supported. Dental anxiety identified as being acquired via the direct pathway did not lead to higher levels of physiological arousal associated with dental stimuli and procedures.

The third hypothesis predicted that direct dental conditioning experiences would lead to higher levels of behavioural avoidance associated with dental situations and procedures than dental anxiety acquired via the indirect pathway. Although the mean DFS behavioural avoidance subscale was found to be numerically higher in the direct

conditioning group, statistical analysis did not find any difference between the two groups. The hypothesis was not supported. Dental anxiety identified as being acquired via the direct pathway did not lead to higher levels of behavioural avoidance associated with dental stimuli and procedures.

No differences were observed in the strength of the responses between the two groups in this study. A number of factors may account for the failure to support the hypotheses. Rachman (1968) proposed that direct conditioning would lead to a greater strength of response than that observed in fears acquired via the indirect pathway. He also argued that fears acquired via the indirect pathway would lead to elevated cognitive correlates (higher levels of negative cognitions). It is possible that the indirect group in this study presented with elevated scores on the three measures due to cognitive rehearsal and revaluation of the UCS, thus leading to inflation of the UCS prior to the encounter with the CS. Unfortunately, there were no measures of cognitive beliefs for the two groups available for this study. Rather than thereby making it difficult to conclude with any certainty that this may have contributed towards the failure to support the hypotheses. However as Davey (1992) suggests more work is required on the nature and content of the UCS representations generated by direct and indirect conditioning.

Another explanation that may account for the failure to support the hypotheses is that some patients identifying direct dental experiences to account for their fears may have attributed negative events to external causes. Withers and Deane (1995) argue that the identification of the direct pathway to explain the onset of fear may be more

compatible with lay explanation of the causes of fears. Retrospective accounts of acquisition of fear are also believed to be subject to memory or information bias. Therefore it is possible that some of the individuals identifying a direct conditioning experience may have acquired their fears due to an indirect experience, thereby biasing the data used for the analysis.

As found in previous research (Ost and Hugdahl, 1985) the number of direct conditioned cases was higher than those who were identified as acquiring their dental anxiety via the indirect pathway. However a limitation with the data used in study one was the classification of indirect experiences. Patients not identifying a direct conditioning experience were automatically assumed to have had an indirect dental experience, which would account for their dental anxiety. This line of reasoning is not supported by arguments put forward for example by Davey (1992) who argued that sensory preconditioning could explain why some individuals are unable to recall any direct trauma at the time of their first appearance of their fear. Also according to the non-associative account of fear acquisition, no direct or indirect traumatic pairing with the feared stimulus is required to explain phobic onset (Menzies & Clark, 1995).

In addition Rachman (1968) proposed two indirect pathways of fear conditioning, vicarious and transmission of information. Vicarious experiences have been reported as being the second most common pathway to fear acquisition. There is no identification amongst the indirect group from study one's population as to the indirect pathway.

However the data used in study one were collected for clinical purposes and therefore research protocols were not applied. A number of extraneous variables were associated with the collection of this data e.g. different individuals collected the data and there was no control over the order of the administration of the questionnaires.

These factors do limit any meaningful conclusions based on the evaluation of the data included in study one.

Another limitation in this study was the small sample size. However normality of the distribution of the scores was met for this sample and Motulsky (1995) argues that in small samples that are normally distributed nonparametric tests lack statistical power. Therefore the use of parametric tests appears justified.

Menzies and Clark (1995) have suggested that clinicians should avoid insisting that associative learning events have occurred in the patient's past and are the crucial determinants of the patient's present fear. According to the authors all models of fear acquisition should be examined by the clinician. Insisting that patients have forgotten conditioning events is not according to Menzies and Clark (1995) profitable during therapy. In addition to this argument, it can be argued that examining levels of anxiety related to pathways of conditioning may not contribute to the treatment process. Rachman (1977) did originally propose that direct conditioning would predominate in clinical cases, whereas indirect acquisition would account for mild fears. This hypothesis has not been borne out by the findings of this study where no

differences were found regardless of fear acquisition pathway between the two groups. Withers and Deane (1995) have suggested that indirect pathways of fear acquisition should also be included when investigating other psychological disorders such as from the disaster/trauma perspective.

De Jongh et al (manuscript submitted for publication) reported that intrusive memories of past traumatic dental events are associated with trauma-related symptoms. However only direct distressing experiences were addressed. Chapter five and study two will further examine the association of intrusive memories related to past distressing dental experiences and trauma-related symptoms, and will investigate if differences exist between participants with intrusive memories and those without intrusive memories on levels of general psychopathology and beliefs related to dental care. This study will include participants who report indirect pathways of fear acquisition i.e. vicarious conditioning and transmission of information.

Chapter 5. History and Models of Post-Traumatic Stress Disorder and Applications to Medical and Dental Fears.

5.1 Introduction and History of PTSD

Psychological problems arising from extreme trauma can be found in the literature since the time of Homer (Alford, 1992). Van der Kolk, Weisaeth and van der Hart (1996a) observe that an association between symptoms of hysteria and childhood histories of trauma can be traced back to 1859 and the work of Briquet. Yule, Williams and Joseph (1999) describe reactions to acute stress appearing in Shakespearean plays. The same authors describe the concept of “railway spine”. This followed the introduction of mass transport in Victorian times and was described as psychological reactions to railway disasters that seemed out of scale with the actual accident. These psychological reactions were explained as the effects of direct damage to the spine and central nervous system (CNS) (Yule et al, 1999).

Traumatic neurosis was first used by Oppenheim in 1889 and he argued that functional problems were produced by subtle changes in the molecular system in the CNS (van der Kolk et al 1996a). Traumatic neurosis was commonly found in combat soldiers and was frequently associated with cardiovascular symptoms which led to other terms being used to describe their difficulties such as “irritable heart” and “soldiers heart” (van der Kolk et al, 1996a).

During World War I traumatic stress reactions became known as “shell shock”. Van der Kolk et al (1996) note that as “shell shock” could be found in soldiers never directly exposed to gunfire, it started to become clear the causes could be related to emotional influences as well as direct traumatic experiences.

During the second World War the concept of post-trauma syndrome (Kardiner, 1941) began to be recognised. Post-trauma syndrome was recognised by irritability, outbursts of aggression, exaggerated startle response and fixations on the traumatic event (Yule et al, 1999). Kardiner (1941) also noted that patients presenting with post-trauma syndrome developed altered conceptions of the self in relation to the world, due to being fixated on the trauma.

Van der Kolk et al (1996) describe the account of Janet’s (1889) explanation of the psychological processing of trauma. Janet proposed that when people experience intense emotions their minds become incapable of matching their frightening experiences with existing cognitive schemes. As a result, their memories of the experience cannot be integrated into personal awareness, instead the memories are dissociated from consciousness and voluntary control. Janet’s (1889) formulation of the effects of trauma on the mind was based on the notion that extreme emotional arousal results in failure to integrate traumatic memories. Breuer and Freud (1893) also commented on the nature of dissociation and introduced the concept of defense hysteria (Nemiah, 1997). Breuer and Freud proposed that the ego actively repressed

memories of the traumatic event to protect itself from experiencing the painful affects associated with them (Nemiah, 1997). The psychoanalytical model of PTSD will be further discussed in section 5.4.

Van der Kolk and van der Hart (1989) reappraised the work of Janet. They proposed that Janet was the first to study dissociation systematically as the crucial psychological process with which the organism reacts to overwhelming experiences and show that traumatic memories may be expressed as sensory perceptions, affect states, and behavioural re-enactments. Janet believed that frightening or novel experiences could be split off from conscious awareness and voluntary control, and that fragments of unintegrated events may show up later as pathological automatisms (Janet believed that only a small part of the interaction between an organism and its environment occurs within conscious awareness. most experiences, values, habits and innate and acquired skills are automatically integrated into existing cognitive schema without conscious awareness, he called these automatic adaptations “automatisms”) Van der Kolk and van der Hart (1989).

It is believed that dissociation plays a role in the development of trauma-related psychological problems (Briere & Conti, 1993; Van der Kolk, van der Hart & Marmar 1996b; Shalev, Peri, Caneti & Schreiber, 1996). Dissociation is described by Van der Kolk et al (1996b) as a compartmentalization of experience, where elements of a trauma are not integrated into a unitary whole or an integrated sense of self. Van der Hart, van der Kolk and Boon (1996) utilise dissociation to refer to three distinct but related phenomena.

Primary dissociation occurs when individuals are confronted with overwhelming threat. It is suggested that sensory and emotional elements of the experience are not integrated onto personal memory and identity, thus the experience is split into isolated somatosensory elements, without integration into a personal narrative (van der Hart et al 1996; Van der Kolk & Fisler, 1995).

Secondary dissociation is believed to occur once an individual is in a dissociated state. Fromm (1965) described a dissociation between observing ego and experiencing ego. van der Hart et al (1996) describe this process as individuals mentally leaving their body at the moment of trauma and observing what happens from a distance, allowing individuals to observe the traumatic experience as spectators, and to limit their pain and distress. van der Hart et al (1996) suggest that whereas primary dissociation limits individuals cognitions regarding the reality of the experience and allows them to go on temporarily as if nothing has happened, secondary dissociation places individuals out of touch with feelings and emotions related to the trauma. Marmar, Weiss, Schlenbger (1994) refer to secondary dissociation as peritraumatic dissociation.

Tertiary dissociation is described by van der Hart et al (1996) as a process where individuals develop distinct ego states that contain the traumatic experience consisting of complex identities with distinct cognitive, affective and behavioural patterns. It is proposed that some ego states may contain the pain, fear or anger related to traumatic experiences, while other ego states remain unaware of the trauma and its concomitant affects.

Post-Traumatic Stress Disorder (PTSD) was not recognised until after the Vietnam War (Figley, 1978). In 1983 PTSD was included in the third edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM) (APA 1983,1987).

Following a traumatic episode, individuals show a variety of responses. Shalev (1992) reported that symptoms of PTSD are frequently observed during the early days following a trauma. These include, in particular intrusive memories, arousal and dissociation. According to Rothbaum and Foa (1993) early symptoms subside with time in many individuals following a traumatic experience.

The criteria for PTSD has been revised in both DSM-III-R and DSM-IV (APA 1987, 1994). PTSD criteria, has also been revised in the various revisions of the International Statistical Classification of Diseases, Injuries and Causes of Death (ICD). The current ICD-10 (1993) and DSM-IV criteria for PTSD although similar, place different emphasis on emotional numbing, with ICD-10 not viewing it as necessary for a diagnosis of PTSD (Yule et al 1999).

DSM-IV (APA, 1987,1994) characteristics for PTSD include; a) that a person has been exposed to a traumatic event, either experiencing, witnessing or being confronted with the event that involves actual or threatened death or serious injury, or threat to the person's or others physical integrity

b) the person's response to this involves fear, helplessness or horror,

c) the person re-experiences the traumatic event, which may be experienced as intrusive thoughts, nightmares, flashbacks, and intense psychological distress when exposed to reminders of the trauma

d) the person avoids stimuli associated with the trauma, avoidance can be behavioural, cognitive or emotional, numbing symptoms may also be present, which include detachment from others and restricted range of affect

e) symptoms of increased arousal including heightened startle reactions, outbursts of anger and difficulty concentrating.

A diagnosis of PTSD requires 30 days of symptom duration. It is defined as acute if duration of symptoms is less than 3 months and chronic if the duration of symptoms is 3 months or more (DSM-IV). The disorder does not have to appear within a certain time period after the trauma and can be specified as with delayed onset, if onset of symptoms is at least 6 months after the stressor (DSM-IV).

McFarlane and de Girolamo (1996) argue that problems exist characterising the nature of trauma. They suggest that central to the experience of traumatic stress are the dimensions of helplessness, powerlessness and threat to one's life, however trauma also attacks the individual's sense of self and predictability of the world. McFarlane and de Girolamo (1996) argue that there has been little discussion of

these dimensions of trauma. They criticise the development of the DSM-IV stressor criteria for its attention to stressors from within specific populations who had experienced a limited range of traumatic experiences (mainly violence). They also argue that a further problem with the stressor criteria is its implication that a different causal relationship exists between PTSD and environmental factors than between other psychiatric disorders and such factors, suggesting that individual vulnerability plays a less important role in precipitating PTSD than in other psychiatric disorders. However the prevalence of co-morbidity provides a challenge to this (see section 5.4).

McFarlane and de Girolamo (1996) argue that it is possible that PTSD symptoms can develop even after a minor stressor. Jeavons, Greenwood and de L. Horne (2000) carried out a study using a sample of individuals who had been involved in minor road traffic accidents. They reported that initial cognitions such as the individual's perceived threat to life at the time of the accident other than the objective degree of trauma had the strongest relationships to subsequent trauma. Breslau and Davis (1992) found that direct exposure was not more likely to lead to chronic symptoms of PTSD than were indirect experiences. These findings however do not run counter to DSM-IV, which indicates the importance of appraisal of the traumatic event.

McFarlane and de Girolamo's (1996) argue that it is possible that psychiatric disorders themselves can lead to secondary PTSD. They argue that as many as 50 per cent of patients who have been acutely psychotic will develop a PTSD-type

syndrome in response to their disorder. Thus arguing that inner types of experience should also be considered along with external traumatic experiences.

Briere (1998) also makes a number of points regarding the difficulties associated with the subsequent effects of the traumatic experience. He argues that the subjective response required to allow a PTSD diagnosis (fear, horror or helplessness) is open to potential difficulties. He argues that both victim and clinician may have difficulty determining if the fear, horror or helplessness was sufficiently intense to qualify for the diagnosis. Briere (1998) suggests that other negative responses to trauma may also increase the risk of posttraumatic reactions. Shame, and degradation have been identified by Wong and Cook (1992) as risk factors for traumatic reactions. Secondly, he argues that there is insufficient research to determine whether a stressor must evoke these specific responses, at a specific level of intensity. His third argument is that subjective reports of distress are affected by emotional avoidance, including dissociation. There is a potential therefore for some individuals as being viewed as nontraumatised due to alternative responses of distress that do not meet the DSM-IV criteria. Briere (1998) also proposes that the subjective responses classified in DSM-IV may be further mediated by phenomena that occur after the event. He includes the level of perceived support from others, financial or interpersonal influences, or the results of professional intervention.

In summary the diagnostic criteria for PTSD is well documented. However recent findings such as those by Jeavons et al (2000) have brought into question the nature of the subjective intensity of the stressor required for the development of the

disorder. The reporting that inner types of experience should also be considered as potential traumatic experiences raises the need for clinicians to be aware of the number of potential experiences that may be potential triggers of the disorder.

5.2 Epidemiology of PTSD

According to McFarlane and de Girolamo (1996) epidemiological studies have examined the survivors of a range of different traumatic events, however generalisations are difficult to make due to the use of different concepts, competing diagnostic systems, assessment methodologies and selective and nonrepresentative samples.

Helzer, Robbins and McEvoy (1987) carried out the first epidemiological study of PTSD and found a lifetime PTSD rate of 1.3 per cent and 0.5 per cent for women and men respectively. Breslau, Davis, Andreski and Peterson (1991) surveyed a group of young adults. Amongst those who had been exposed to a traumatic event (39 per cent), PTSD was found amongst 13.6 per cent. This gave an overall lifetime prevalence of 9.2 per cent. As part of the US Epidemiological Catchment Area study Kessler, Sonnega, Bromet, Hughes and Nelson (1995) estimated a population point prevalence rate of 1 per cent. Yule et al (1999) suggest that studies of more exposed samples give ranges from 5-15 per cent for current levels of PTSD and 4 to 12 per cent for lifetime diagnosis. Yehuda, Kahana, Schmeider, Southwick, Wilson and Giller (1994) note the high prevalence of persistent and chronic PTSD amongst

concentration camp survivors and world war two prisoners of war many years after the event.

In a study examining genetic and environmental contributions for PTSD, True et al (1993) reported that a significant genetic influence exists on PTSD symptom liability. They studied pairs of monozygotic and dizygotic combat veterans. After adjusting for combat exposure they found that genetic factors accounted for 13-30 per cent of the variance in liability for symptoms in the reexperiencing cluster, 30-34 per cent for symptoms in the avoidance cluster, and 28-32 per cent for symptoms in the arousal cluster. They also reported that no evidence could be found that shared environments could account for the development of PTSD symptoms.

McFarlane and de Girolamo (1996) argue that PTSD is a predictable consequence of traumatic events. However Yehuda and McFarlane (1995) suggest that no trauma is so severe that almost everyone exposed to the experience develops PTSD. The normality of PTSD following a severe stressor has been questioned due to the low rates of diagnosable PTSD among those who have experienced traumatic events, and the high rates of co-morbidity with other psychological difficulties among those with PTSD (Yehuda & Mc Farlane, 1995).

McMillen, North and Smith (2000) argue that both the low incidence of PTSD after some traumatic event and the psychiatric comorbidity seen in those with PTSD may reflect the stringency of PTSD symptom criteria C, the effortful avoidance and numbing criteria. They argue that meeting criteria C is more difficult for at least two

reasons. It requires a higher number of reported symptoms (3), and the symptoms in this cluster are some of the least frequently reported symptoms. A number of studies have found that the numbing symptoms have a low prevalence rate compared to higher rates of criteria B (intrusive memories) and D, symptoms of hyper arousal (North, Smith, & Spitznagel, 1994; Smith, North, McCool, Shea, 1990). McMillen et al (2000) carried out a study amongst earthquake survivors. They found that only 13 per cent of their sample met full PTSD criteria, but 48 per cent met both the reexperiencing and arousal symptom criteria, without meeting the avoidance and numbing symptoms. They did find that psychiatric comorbidity (major depression, alcohol and drug abuse) was associated mostly with avoidance and numbing symptoms. For most of their sample, intrusive memories, sleep disturbance and exaggerated startle response were the most common symptoms. Rothbaum and Foa (1993) and Stein, Walker, Hazen and Forde (1997) have developed the term “partial PTSD” to address the issue of individuals not meeting full PTSD diagnosis, but however do present with significant trauma-related distress. However as yet there are no criteria for this syndrome.

The above findings, suggest that meeting full PTSD diagnosis may be difficult due to difficulties meeting criteria C, this may call into question the accuracy of the number of individuals experiencing trauma-related symptoms and may well preclude individuals from seeking or being given appropriate clinical treatment.

5.3 Predisposing Factors:

Yehuda and McFarlane (1995) note that research indicates that a number of predisposing or antecedent factors increase the likelihood that a given stressor produces PTSD. They note that certain variables are associated with a likelihood of posttraumatic disturbance.

Breslau et al (1997) found that the prevalence of PTSD was higher for women than for men exposed to traumatic events. The risk for PTSD after exposure to traumatic events was more than 2-fold higher in women than in men. They also found that exposure to a number of traumatic events in a lifetime did not vary between the sexes. Therefore they concluded that the risk of PTSD in women could not be accounted for by a history of multiple traumatic events. However women were more likely to report rape, assault or ongoing physical or sexual abuse than men. Exposure to these categories of trauma yielded a high rate of early PTSD in women (63 per cent) but no PTSD cases in men. Women's vulnerability to PTSD was found to be greater if exposure to traumatic events occurred before the age of 15 years. Briere (1998), Kessler, Sonnega, Bromet, Hughes & Nelson (1995) suggest however that the reason that women have higher levels of PTSD is that they are more likely than men to be exposed to events that produce PTSD e.g. childhood abuse, rape and physical assault. Davidson and Fairbank (1993) also argue that strong evidence exists that a history of childhood trauma is a predisposing factor in the development of PTSD.

Brewin (2000) carried out a meta-analysis of risk factors for PTSD in which he identified a number of factors i.e. childhood abuse, family psychiatric history, low intelligence, lack of social support and life stress. However Shalev (2001) criticised Brewin's findings suggesting that when effect sizes are considered a different pattern emerges. Shalev (2001) argued that the intensity of the traumatic event and factors that follow exposure (social support and further stressors) are the strongest predictors of PTSD, this is similar to Briere's (1998) proposal. Shalev (2001) proposes that there is ample opportunity for secondary prevention of PTSD when these post-trauma factors are taken into account.

According to Bryant and Harvey (2000) identifying previous traumas and the patient's response to earlier traumatic events is important, as previous trauma is a known predisposing factor for the development of PTSD. Briere (1998) argues that individuals who have experienced previous traumas are more prone to exacerbated reactions to current traumas.

Terr (1991) draws a distinction between Type I and Type II traumatic events. Type I are believed to be short term, unexplained traumatic events, whereas Type II are believed to involve sustained and repeated events. Terr (1991) proposed that the range of reactions differ between Type I and Type II traumas with Type II reactions more likely to lead to more complex difficulties.

Marmar et al (1994) suggest that peritraumatic dissociation is a risk factor for PTSD. A number of studies have offered support for this hypothesis. Holen (1993) found

that levels of reported dissociation during a trauma was predictive of PTSD 6 months after the accident. Koopman, Classen, and Spiegel (1994) observed that dissociative symptoms at the time of the trauma more strongly predicted traumatic symptoms than did anxiety at the time of the trauma.

Recently, Bryant and Harvey (1998) reported a study in which the diagnosis of Acute Stress Disorder (ASD) was predictive of PTSD 6 months following the trauma (mild traumatic brain injury following a road traffic accident). ASD was included in DSM-IV to describe posttraumatic stress in the initial month after a trauma (Bryant & Harvey, 1998). ASD follows the same structure of PTSD i.e. it is described in terms of the stressor, reexperiencing, avoidance and arousal, however the symptom cluster that differentiates ASD from PTSD is the emphasis on dissociative symptoms (Bryant & Harvey, 2000). Either while experiencing the event or after experiencing the event, the individual must have at least three of the following to meet criteria for ASD: 1) a feeling of detachment, numbing or lack of emotional responsiveness; 2) decreased awareness of surroundings; 3) derealization; 4) depersonalization; 5) inability to remember a significant aspect of the trauma.

Other predisposing factors have also been identified that are believed to increase the risk of developing PTSD. Breslau, Davis, Andreski & Peterson (1991) found three personal characteristics to be significant risk-factors in both sexes. These were pre-existing anxiety disorders, major depressive disorders and early separation from parents.

Briere (1998) argues that traumatic event's combine with victim variables and post-trauma social responses to determine the individuals subjective interpretation of the stressor. Therefore making the determination of any specific aetiology involved in PTSD difficult.

5.4 Comorbidity

Kessler, Sonnega, Bromet, Hughes and Nelson (1995) reported that PTSD was accompanied by a higher risk of mainly depression and other anxiety disorders. Amongst disorders comorbid with PTSD they found a lifetime prevalence of 48 per cent with major depressive disorder, 30 per cent with simple phobia, 28 per cent with social phobia, there were no differences found between the sexes for these disorders. They did find that females were more likely to present with panic disorder than males (12.6 per cent versus 7.3 per cent), and agoraphobia (22.4 per cent versus 16.1 per cent). Males however were most likely to present with alcohol abuse (51 per cent versus 27.9 per cent) and drug abuse (34.5 per cent versus 26.9 per cent).

Joseph, Yule, Williams and Hodgkinson (1993) also found reported increases in the use of alcohol and cigarettes amongst survivors of a traumatic event.

There is also evidence that exposure to a traumatic event is a risk factor for medical disorders, such as cardiovascular, gastrointestinal and musculoskeletal difficulties (Friedman, 2000). Breslau and Davis (1992) found that individuals with chronic PTSD (duration of symptoms for one year or longer) reported more medical conditions than patients with acute PTSD (duration of symptoms less than one year).

Bronchitis, migraine, and gynaecological complaints among women were more commonly reported in the chronic PTSD group.

Somatization disorder has also been significantly associated with dissociation and past child abuse (Pribor, Yutzy, Dean & Wetzel, 1993) and PTSD (McFarlane, Atchinson, Rafalowicz & Papey, 1994).

As PTSD is believed to be associated with increased arousal and the inability to regulate arousal (van der Kolk, 1994) it may be predicted that co-morbid difficulties may also be present. Scaer (2001) argues that due to the activation of the autonomic nervous system in individuals presenting with PTSD, cardiac, pulmonary and bowel dysfunctions are predictable.

Shalev's (2001) proposal that lack of social support and further stressors following an exposure to a traumatic event are the strongest predictors of PTSD may also account for some of the other psychological disorders associated with it. Lack of social support and stressful life events have also been identified as a risk factor for other psychological difficulties, such as depression (Brown & Harris, 1978). Therefore a summation of these risk factors may influence and or exacerbate the PTSD difficulties and also account for the development of other disorders.

5.5 Models of PTSD

5.5.1 Psychoanalytical Model of PTSD

Freud (1905) adhered to the idea that traumatic experiences, especially when they occurred early in life, were the source of the pathogenic mental elements leading to psychological conflict and symptom formation (Nemiah, 1997). Van der Kolk, et al (1996) report that Freud adopted much of Charcot's ideas that the symptoms of hysterical patients had their origins in histories of trauma. According to Nemiah (1997) Freud viewed the personal self, or ego as possessing sufficient strength to repress the traumatic memories and affects in order to protect itself from experiencing the psychic pain associated with them. This differs from Janet's account, which suggests that the personal self is weakened by a genetically determined insufficiency of psychological energy. Therefore, when a person experiences a traumatic life event, the psychological energy expended by the painful emotional reaction to trauma further depletes the energy available to the personal self, and is unable to incorporate the memories of the events and associated feelings into its structure. According to Nemiah (1989), Janet adhered to an ego-deficit model of psychopathology, whilst Freud's formulation was based on a psychodynamic conflict model of symptom-formation.

Breuer and Freud (1893) believed that something becomes traumatic because it is dissociated and remains outside conscious awareness, referring to this state as hypnoid hysteria (Van der Kolk, McFarlane & van der Hart 1997). Freud (1905) originally proposed that an experience of sexual abuse was the specific cause of

hysteria. However, Freud (1973) later developed the concept of “defense hysteria” where he abandoned dissociation as the central process related to trauma, and proposed that repressed instinctual wishes formed the foundation of the neurosis (Van der Kolk, et al 1996). From this perspective, Freud argued that it was not the actual memories of childhood trauma that are split from consciousness, but unacceptable sexual and aggressive wishes of the child, which threatens the ego and motivates defenses against the conscious awareness of these wishes (Van der Kolk et al, 1997). Freud believed that adults reporting traumatic memories were actually presenting with the recurrence of fantasies and feelings associated with early pathogenic development distortions (Nemiah, 1997).

However during World War I, Freud (1920) revived the work of Janet and proposed that it was the overwhelming intensity of the stressor, in the absence of abreactive verbal or motor channels that led to intense affect difficulties (Van der Kolk, et al 1996).

Masson (1984) notes that the acceptance of psychoanalytic theory resulted in an absence of research on the effects of real traumatic events on children’s lives.

However according to Van der Kolk, et al (1996) it was the work of Kardiner (1941) who defined PTSD for the remainder of the 20th century. Kardiner observed that individuals presenting with traumatic neurosis developed a vigilance for and sensitivity to environmental threat. Van der Kolk, et al (1996) also note that Kardiner described from observations of his patients the “pathological traumatic syndrome”

which consisted of an altered conception of the self in relation to the world, based on being fixated on the trauma, chronic irritability, atypical dreams, startle reactions and aggressive reactions. Crucial to Kardiner's theory was the re-enactment of the original traumatic episode. Van der Kolk, et al (1996) note that although the work undertaken by Kardiner was available for practical application when World War II broke out, most of it was forgotten and required to be rediscovered during later conflicts.

5.5.2 Cognitive Models of PTSD

Dalgleish (1999) argues that a number of factors such as psychosocial factors, pre-morbid history of psychological problems, prior experience of trauma and perceived threat to the victims own life although important in bringing order to trauma research and generating new ideas remain descriptive. They do not provide a description of how these factors interact and how different reactions to trauma evolve.

He argues that theoretical approaches to PTSD must have explanatory power to meet the following conditions:

- 1) to explain the symptoms of re-experiencing, avoidance and hyperarousal
- 2) to account for the range of individual reactions to PTSD
- 3) to explain the effects of pre-morbid history of psychological problems, social support, attributional style and attitudes to emotional expression
- 4) to account for the efficacy of exposure-based treatments
- 5) to provide a coherent model of mind within which the four conditions above can be realised.

He goes on to describe a number of cognitive theories which are discussed below which he believes can offer some explanatory power to account for the five preconditions set out above.

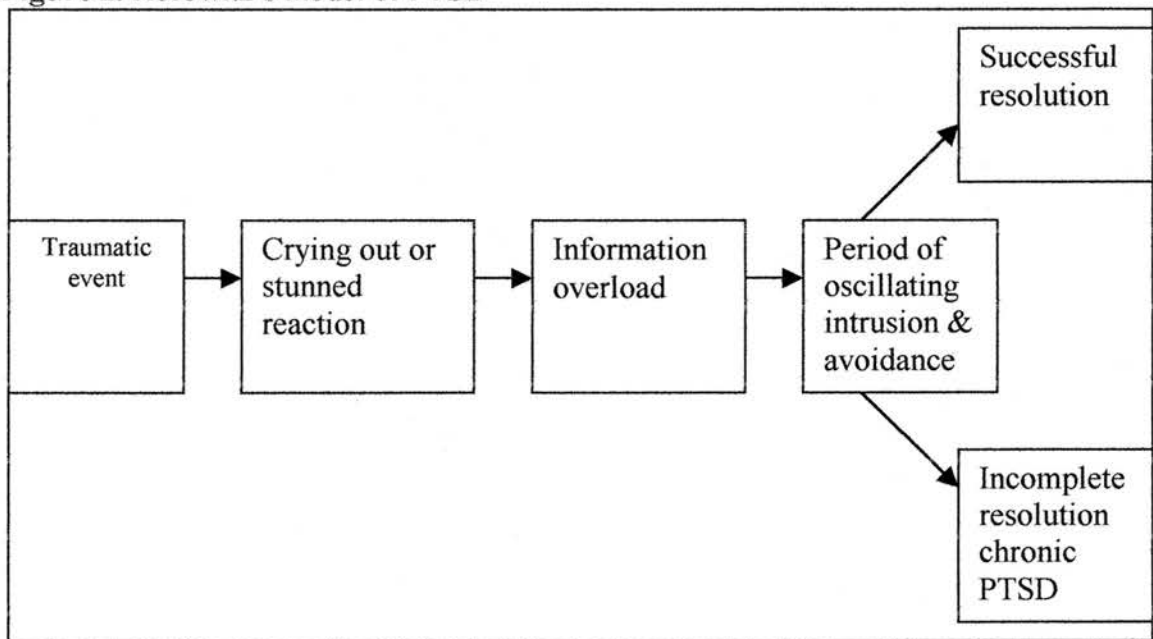
5.5.3 Horowitz's Theory of PTSD

Horowitz (1973) developed his model from psychodynamic theory. Horowitz (1973) describes that trauma related information must be processed through what he terms a "completion tendency". That is the psychological need to match new information with inner models based on older information and the revision of both until they agree (Dalglish, 1999). This is similar to Janet's argument that memories of intense emotional experiences are not consistent with existing cognitive schemes. Following a traumatic event Horowitz (1986) believed there was an initial stunned reaction, followed by a period of information overload, where thoughts, memories and images of the trauma could not be reconciled with current meaning structures. According to Dalglish (1999) there is an initial failure to "complete". A number of psychological defence mechanisms then keep the traumatic information in the unconscious, this leads to the individual experiencing a period of numbing and denial (Dalglish, 1999). Horowitz (1986) argues that the trauma related information is maintained in active memory by the completion tendency with the information breaking through the psychological defences, intruding into consciousness. This is experienced as nightmares, flashbacks and intrusive memories. It is this tension between the completion tendency and the psychological defences that leads to periods of oscillations between denial, numbing and intrusions, until the traumatic memories can be integrated with long term meaning representations (Dalglish, 1999). The

failure to integrate these memories leads to post-traumatic reactions (Horowitz, 1986).

According to Dalgleish (1999) Horowitz's explanation of PTSD can explain conditions 1, and 4, and to a limited extent part of the ways in which normal reactions to trauma can become chronic (condition 2). However he does draw attention to the limitations of this theory. He observes that this theory does not account for the finding that some individuals develop PTSD and others do not or the development of late onset PTSD (condition 2). Further, Dalgleish (1994) notes that this theory does not account for a coherent model of mind, failing to explain the nature of the existing schema structure and the way it fails to accommodate new information from the traumatic experience (condition 5).

Figure 2. Horowitz's model of PTSD



(Adapted from T. Dalgleish (1999): Chapter 10, Cognitive Theories of Post - Traumatic Stress Disorder. Page 197)

5.5.4 The Cognitive Appraisal Model: Janoff-Bulman

Janoff-Bulman (1985) developed the cognitive appraisal model of PTSD. According to this theory PTSD is the result of assumptions about the self and the world being shattered. The main formulation arising from this theory is the way in which traumatic events produce changes in the victim's thoughts and beliefs and how these changes play an important role in the emotional response to trauma (Janoff-Bulman, 1992). According to Dalgleish (1999), the assumptions referred to in this model include, the assumption of personal invulnerability; the perception of the world as meaningful or comprehensible; and the view of the self in a positive light. These assumptions provide structure and meaning, however they can be shattered when faced with a traumatic experience, leading to hyperarousal, intrusion and avoidance (Dalgleish, 1999). Dalgleish argues that a problem related to this theory is the finding that pre-morbid psychological problems are a risk factor for the development of PTSD. He argues that individuals holding pre-morbid negative assumptions are unlikely to have them shattered by a traumatic experience. Individuals holding negative assumptions are more likely to have their assumptions confirmed. This point is addressed by Ehlers and Clark (2000). They argue that prior beliefs (negative and positive) are important in that trauma victims with prior negative beliefs about themselves may view the trauma as a confirmation of these beliefs, whereas those holding positive beliefs may find them shattered as proposed by Janoff-Bulman. Dalgleish (1999) argues that Janoff-Bulman's model does not meet condition 5. He argues that there is little attempt to explain what processes are involved when the assumptions are shattered. These processes have been elaborated by Ehlers and Clark (2000) and will be discussed in section 5.5.8.

5.5.5 Foa's Fear Network

Foa and Kozak (1986) Foa, Zinbarg and Rothbaum (1992) based their model on Lang's (1977) theory of fear structures. Lang (1977) suggested that emotional images are constructed from three main classes of propositional unit, one concerned with stimulus information; one with response information and one the meaning of the situation. The fear network related to trauma centres on the stimulus information about the trauma; information about cognitive, behavioural and physiological reactions to the trauma and information which links these stimulus and response elements together (Dalglish, 1999). Foa et al (1992) argue that the trauma-related fear network can be activated by cue stimuli, which then causes information held in the network to enter consciousness. Avoidance symptoms come from the attempts to avoid and suppress activation of the network (Foa et al 1992). Foa and Riggs (1993) suggest that the trauma memory of an individual with PTSD is distinguished by representations of the world as indiscriminately dangerous and of the self as an inadequate copier. According to the fear network theory, two conditions are required for the reduction of fear. First, the fear memory must be activated, and secondly, new information must be provided, including elements that are incompatible with existing pathological elements in the structure, so that a new memory can be formed (Rothbaum and Foa, 1993). Also included in this model are the influences of unpredictability and uncontrollability which is suggested can make it difficult to assimilate into existing models in which the world is controllable and predictable.

Dalgleish (1999) suggests that Foa et al's (1992) fear network has taken a big step towards a greater understanding of how the processes underlying PTSD might operate within a cognitive system. He argues that this model meets conditions 3 and 4. He argues however that this theory may not be powerful enough to cope with the range of PTSD phenomenology or why fear networks develop in some individuals and not in others.

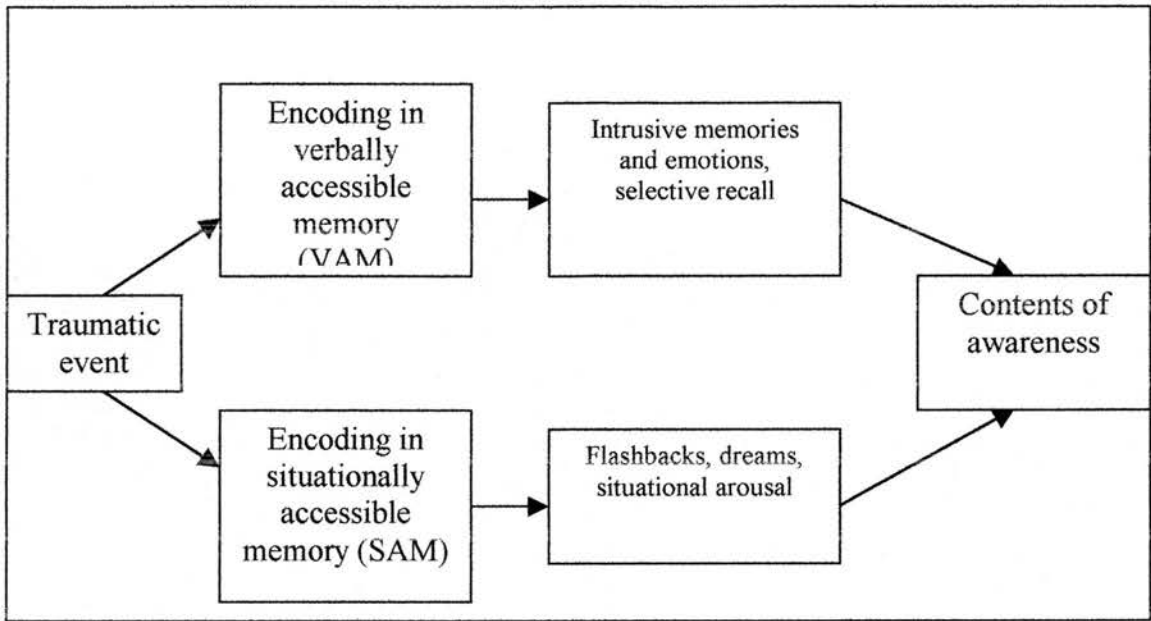
5.5.6 Dual Representation Theory

Brewin, Dalgleish and Joseph (1996) have proposed a dual representation theory for PTSD. According to the dual representation theory, memories of a personally experienced traumatic event can be of two distinct types (Brewin, 2001). The individuals conscious experience of the trauma is the first level of representation. Brewin et al (1996) refer to this as *Verbally Accessible Memories (VAMS)*. Dalgleish (1997) states that VAMS are characterised by their ability to be deliberately retrieved and progressively edited by the traumatised individual. Like Foa and Kozak's (1986), fear network theory, Dalgleish (1999) argues that VAMS, contain sensory, response and meaning information. Brewin (2001) describes VAMS as supporting ordinary autobiographical memories that can be retrieved either automatically or using deliberate strategic processes. VAMS although readily available for verbal communication are restricted due to mediated limited-capacity serial processing, such as attention (Brewin, 2001). It is suggested that the emotions that accompany VAM memories involve cognitive appraisals occurring both during the trauma and after the trauma, as the individual considers the consequences and implications of the event (Brewin, 2001).

The second level of representation consists of *Situationally Accessible Memories (SAMS)*. SAMS unlike VAMS contain information, that cannot be deliberately retrieved nor edited. SAMS according to this theory can only be accessed when aspects of the original traumatic situation cue their activation (Daggleish, 1999). Brewin et al (1996) propose that the SAM system contains information that has been obtained from more extensive, lower level perceptual processing of the traumatic scene. This would include visuospatial information that has received little conscious processing, and of the person's bodily response to it, such as autonomic and motor responses (Brewin, 2001).

Brewin (2001) argues that activation of SAM representation can account for flashbacks, which are more detailed and affect-laden than ordinary memories, and because the SAM system does not use a verbal code these memories are difficult to communicate to others and do not necessarily interact with other autobiographical knowledge. Brewin (2001) also suggests that the emotions that accompany SAM memories are restricted to those that were experienced during the trauma or subsequent moments of intense arousal. These consist of fear, helplessness and horror.

Figure 3. : A diagram of the dual representation model of PTSD



(Adapted from T. Dalgleish (1999):Chapter 10, Cognitive Theories of Post Traumatic Stress Disorder. Page 202).

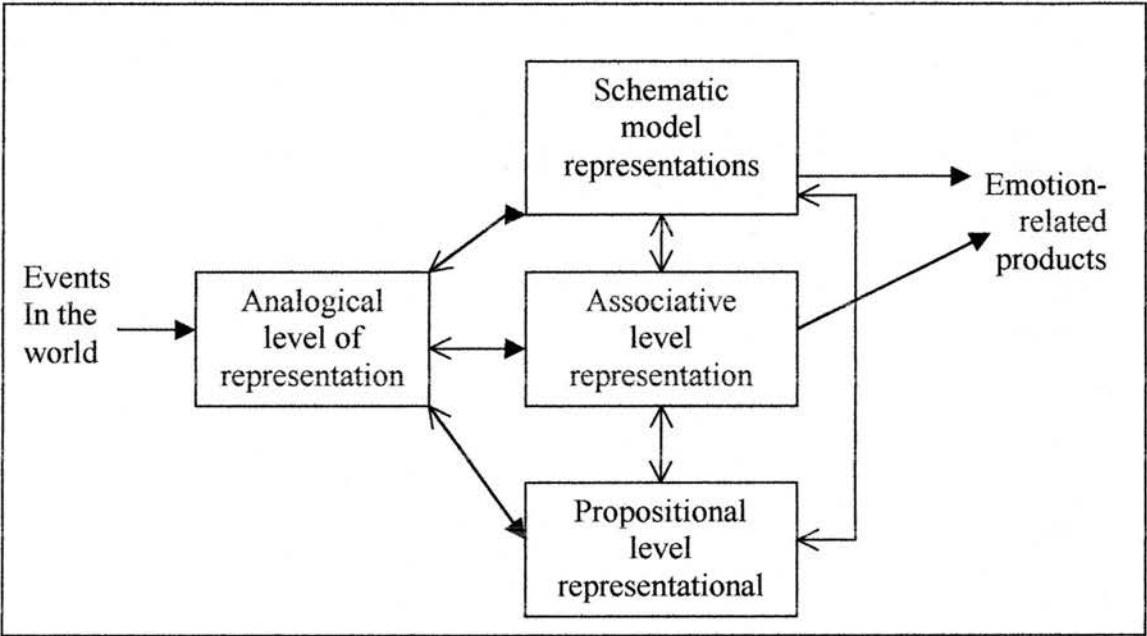
According to Dalgleish (1999) the dual representation theory offers a coherent account of the phenomenology of PTSD. He argues that this theory can account for preconditions 1, 2, 3 and 4. He also argues that the theory can offer a coherent model of mind which can incorporate the other 4 preconditions. However as Brewin (2001) suggests although the theory is consistent with clinical observations it has received limited empirical evaluation.

5.5.7 The Schematic, Propositional, Associative and Analogical Representational Systems (SPAARS)

Dalgleish (1999) considers one other cognitive approach to PTSD. This is the Schematic, Propositional, Associative and Analogical Representational Systems (SPAARS) (Power and Dalgleish, 1997). This model views emotions as adaptive

processes which reorganises the cognitive system in different ways to deal with changes in both the internal and external environment (Daggleish, 1999). The SPAARS model is an appraisal-based model of emotion comprising four levels of representation of information.

Figure 4. The basic architecture of the SPAARS model



(Adapted from T. Dagleish (1999):Chapter 10, Cognitive Theories of Post Traumatic Stress Disorder. Page 205).

The analogical representation system store information and memories in analogical form. This includes visual, olfactory, auditory, gustatory and proprioceptive images, including memories of specific events, fragments of events from an individual’s life, or representations of the properties of objects, smells and sounds (Dagleish, 1999).

Propositional representations according to Power and Dagleish (1997) are believed to be the encodings of verbal information, which also include beliefs, ideas and

objects and concepts and the relations between them in a form which is not specific to any language. This level in SPAARS is responsible for the storage of semantic facts about the world, as well as episodic memories of events in an individual's life.

The schematic model representation refers to higher-order representations of ideational content (Dalglish, 1999). This is the way in which, higher-order ideational content which, cannot be expressed in language is represented. For example Dalglish (1999) offers an explanation of this type of representation using the belief of the world as a safe place. The model of the world as a safe place from the schematic representation will contain more complexity than is thought to be captured by the propositional level. From the schematic level it will incorporate all aspects of what safety means to the individual, and guides the way in which information is processed and organised within the system (Dalglish, 1999).

The fourth level of the SPAARS model is the associative level of representation. This level of representation involves automatised emotions which are generated in a way which do not involve appraisal with respect to the individual's goals at the time of the event's occurrence (Dalglish, 1999). The associative level of representation suggests that emotions can be automatised in two ways, 1) through repetition of an event-emotion pairing and 2) when the event is biologically prepared as has been described by Seligman's theory of preparedness (as described in chapter 1).

The SPAARS model accounts for PTSD within the following framework. During a trauma, information about the event is appraised at the schematic level, in a threat-

related way leading to an experience of intense fear (Dalglish, 1999). It has then been suggested that trauma-related information is encoded and represented at the analogical, propositional and schematic levels of meaning (Power and Dalglish, 1997). This information is believed to be unintegrated with the individual's dominant schematic model of the self, world and others. This pattern of representation according to Dalglish (1999) can explain the symptoms which are characteristic of PTSD and related problems.

Re-experiencing, is accounted for by the SPAARS model from the following perspective. Following a traumatic event, the appraisal system at the schematic model level will attempt to process the unintegrated trauma-related information. This information will be appraised as incompatible and as a threat to the existing model of the self and the world (Dalglish, 1999). This continual processing of trauma-related information leads to activation and re-activation of the fear model with the individual re-experiencing the traumatic event. Flashbacks to the event are believed to occur due to the fact that little assimilation with other information in memory has occurred. According to Dalglish (1999), flashbacks can involve reactivation of unintegrated information at all levels within SPAARS. This includes the associative level, which will activate fear as a function of the repetitions during intrusions of the link between trauma-related information and fear. These intrusive phenomena will lead the individual to undertake a number of processes to avoid any reminders of the event.

Hyperarousal is explained as a consequence, of the process of the existence of unintegrated traumatic information in memory. Thus individuals whose processing

resources are continually being utilised by appraising the incompatibility of unintegrated trauma-related information in memory are likely to experience symptoms of hyperarousal (Dalgleish, 1999).

The SPAARS model also accounts for individual differences at the schematic level of representation. For example some individuals may hold models of the world as being a moderately safe place. In contrast, some individuals may hold models of the world as completely safe. According to the SPAARS model various ways of dealing with incompatible information would have been developed throughout the person's emotional history. Involving denying the incompatible information or emotionally processing the information. It is argued that the majority of individuals will, following an initial period of post-traumatic reactions, be able to integrate the traumatic information into their models of the self, others and the world. This may explain why the prevalence of PTSD following traumatic incidents is low. Those individuals with overvalued models about their world, self and others will have difficulties integrating the incompatible information and will be more at risk of developing PTSD (Dalgleish, 1999).

Dalgleish (1999) argues that the SPAARS model can account for all the pre-conditions mentioned earlier. It meets pre-condition 1 as it can account for the three central constellations of problems in PTSD. It can account for the number of individual reactions to trauma (pre-condition 2.). It can account for processes such as attributional style (pre-condition 3) and implications for therapy (pre-condition4).

Finally, according to Dalgleish it provides a coherent architecture in which PTSD can be understood (pre-condition 5).

5.5.8 Ehlers and Clarks Cognitive Model of PTSD.

Ehlers and Clark (2000) have proposed a model to explain the persistence of PTSD. Their model draws on some of the work already described by Dalgleish (1999) above including, Brewin et al (1996) and Janoff-Bulman (1992). Ehlers and Clark (2000) provide an overview of one of the main puzzles related to PTSD. That is the classification of PTSD as an anxiety disorder (DSM-IV). They argue that within cognitive models, anxiety is a result of appraisals related to impending threat, however PTSD is a disorder in which the problem is a memory for an event that has already happened. The authors suggest that the puzzle can be resolved by proposing that persistent PTSD occurs only if individuals process the traumatic event and/or its sequelae in a way which produces a sense of a serious current threat. The authors propose two processes which can lead to a sense of current threat: 1) individual differences in the appraisal of the trauma and /or its sequelae and 2) individual differences in the nature of the memory for the event and its link to other autobiographical memories. Ehlers and Clark (2000) argue that once activated, the perception of current threat is accompanied by intrusions, reexperiencing symptoms, symptoms of arousal, anxiety and other emotional responses. The perceived threat also motivates a set of behavioural and cognitive responses that reduce the perceived threat and distress in the short-term, but have the consequence of preventing cognitive change, which then leads to maintenance of the disorder.

Ehlers and Clark (2000) argue that individuals with persistent PTSD are unable to see the trauma as a time-limited event that does not have global negative implications for their future. They also propose that several types of appraisal can be made regarding the event, and that each appraisal can produce a sense of current threat. For example, it is suggested that individuals overgeneralise from the trauma event and as a consequence perceive a range of normal activities as more dangerous than they really are. Trauma survivors may also exaggerate the probability of further catastrophes, and may appraise the way they felt or behaved during the event, that may be perceived as indicating personal responsibility for the event's causality. Individuals also make appraisals of trauma sequelae, believing for example, that intrusive memories indicate that they are losing control of their mind. The consequence of this, is that many individuals attempt to suppress intrusive memories (Ehlers & Clark, 2000). Active thought suppression is known to make thoughts more likely to come to mind (Wegner, 1989), therefore this strategy for dealing with this type of appraisal is paradoxically likely to maintain it rather than ameliorate their difficulties.

Ehlers and Clark (2000) also draw attention to the nature of trauma memory and its relationship to unwanted recollections. Individuals with PTSD often report difficulties in intentionally retrieving a complete memory of the traumatic event. Intentional recall is often found to be fragmented and poorly organised, with difficulty recalling the exact temporal order of events (Foa & Riggs, 1993; van der Kolk & Fisler, 1995).

However individuals with PTSD persistently report a high frequency of involuntarily triggered intrusive memories, which Ehlers and Clark (2000) state require to be explained within existing models of PTSD. They also suggest that re-experiencing of the event has a number of important characteristics that need to be explained. These include:

- ◆ Re-experiencing mainly consists of sensory impressions, rather than elaborated thoughts. The impressions involve all modalities including physical sensations.
- ◆ The sensory impressions are experienced as if they were happening right now rather than being memories from the past and the emotions (including physical and behavioural responses) accompanying them are the same as those experienced at the time.
- ◆ The original emotions and sensory impressions are re-experienced even if the individual later acquires new information that contradicts the original impression of it.
- ◆ Individuals may re-experience physiological sensations or emotions that were associated with the traumatic event without a recollection of the event. Schacter, Norman and Koutstaal (1997) refer to this as lack of source information.
- ◆ The involuntary re-experiencing of the traumatic event is triggered by a wide range of stimuli and situations. Many of the trigger stimuli are cues that do not have a strong semantic relationship to the traumatic event, but are cues that were temporally associated with the event, e.g. physical cues similar to those before or during the event.

Ehlers and Clark (2000) propose that in persistent PTSD one of the main problems is that the trauma memory is poorly elaborated and inadequately integrated into its context in time, place, subsequent and previous information and other autobiographical memories. Brewin et al (1996) propose two routes to the retrieval of autobiographical information. The first route is via higher-order meaning-based retrieval strategies (e.g. first day at school). The second route is through direct triggering by stimuli that were associated with the event (smells). According to Ehlers and Clark (2000) the normal processing of autobiographical memories appears to have the function of reducing the ease with which memories of past experiences are unintentionally retrieved while an individual engages in everyday tasks. It is suggested that this type of organisation enhances the first retrieval route and inhibits the second. This has the effect that when an autobiographical memory enters consciousness, it comprises both specific information about the event general information about the lifetime period of the event and abstracted information about the type of event in general (Ehlers and Clark, 2000). Thus according to Ehlers and Clark (2000), this process can explain problematic intentional recall (weak semantic route to retrieval), the “here and now” quality of sensory impressions (no context in time) and the absence of links to subsequent information and the easy triggering by physically similar cues.

Ehlers and Clarks (2000) also propose strong stimulus-stimulus and stimulus-response associations influencing trauma-related difficulties. Distinct stimuli present shortly or during the event become associated with danger. Retrieval from associative memory is cue-driven and unintentional, therefore the individual may not

be aware of the triggers for reexperiencing and may not be aware that their emotional reaction is due to activation of the trauma memory. According to this proposal, failure to spot the origin of the reexperiencing symptoms makes it difficult for the patient to learn that there is no present danger when exposed to the trauma.

Strong, perceptual priming of stimuli temporally associated with the trauma, is also proposed by this model. It is argued that as a consequence of the reduced perceptual threshold, cues that were associated with the trauma and that consequently can directly trigger the trauma memory are more likely to be noticed. Baddeley (1997) suggests that individuals with PTSD elicit poor stimulus discrimination, with the result that reexperiencing symptoms occur, even if the context in which the stimulus configuration is observed is very different.

Ehlers and Clark (2000) also propose that a reciprocal relationship exists between the nature of the trauma memory and the appraisals of the trauma and its sequelae. Recall of the trauma is biased by the individual's appraisals, and selectively retrieved information which is consistent with these appraisals. They also suggest that due to the inability to recall specific or well-elaborated details of the trauma, individuals may catastrophically appraise this as a sign that something is wrong with their brain. Ehlers and Clark (2000) point out however that many of these cognitive strategies, such as selective retrieval and attention to threat may not always have an intentional quality, but are also likely to occur through automatic triggering as well as strategic responses.

Types of cognitive processing during the trauma is an essential element according to this model of PTSD. The two processes that lead to a current threat, appraisals of the trauma and its sequelae and the nature of the trauma memory are known to be influenced by the type of cognitive processing during the traumatic event. Mental defeat (Dunmore, Clark & Ehlers, 1997) has been identified as a correlate of chronic PTSD. Mental defeat is described as the perceived loss of all psychological autonomy, accompanied by the sense of not being human any longer (Ehlers & Clark, 2000). Patients presenting with mental defeat are believed to interpret the trauma as evidence for a negative view of themselves, such as not being a worthy person, or that they are permanently damaged by the trauma (Ehlers & Clark, 2000). The influence on trauma memory is believed to be dependent on the quality of processing at encoding. Roediger (1990) describes two types of encoding; conceptual and data-driven. It is believed that data-driven processing (processing the sensory impressions of the trauma) will make the trauma memory difficult to retrieve intentionally, as there has been no conceptual processing, where the meaning of the trauma is processed in an organised way and is placed into context.

Ehlers and Clark (2000) argue that their model also accounts for a number of background factors that may also influence cognitive processing during a traumatic event. These include: 1) the duration and predictability of the event; 2) previous traumatic experiences and coping styles; 3) prior beliefs and 4) state factors, such as alcohol consumption and arousal state.

Finally, Ehlers and Clark (2000) also argue that delayed onset PTSD can be explained by their model. They draw on the work of Davey (1989) who described UCS re-evaluation (chapter 1). This is a process by which the individual's evaluation of the UCS is changed over time. This can occur due to the individual acquiring novel information, which suggests that the UCS is now more aversive. Ehlers and Clark (2000) argue that delayed onset PTSD occurs because some later event gives the original trauma or its sequelae a more threatening meaning.

The model proposed by Ehlers and Clark (2000) would appear to meet the preconditions established by Dalgleish (1999). It can account for individual reactions to trauma. It includes pre-morbid histories, the theoretical basis for exposure-based treatment, and provides a coherent theory of mind in which PTSD can be understood.

The cognitive models discussed do appear to account for a number of the conditions set out by Dalgleish (1999). With the models having as their basis much of Janet's original accounts of the failure to properly integrate traumatic memories. The proposition put forward by the Dual Representation Model that SAMS can only be accessed when aspects of the original traumatic situation cue their activation does not appear to be supported by the literature or clinical observations. Keane, Zimering and Caddell (1985) reported that amongst Vietnam veterans a broad range of stimuli (not present during the original trauma) evoked anxiety and trauma-related symptoms. They argued that this was due to higher-order conditioning (the pairing of a CS with a neutral stimulus that then becomes a higher order CS, evoking a CR). They also suggested that stimulus generalisation could account for these findings (the more

similar a novel situation is to a conditioned response, the stronger the response will be to that new stimulus). Ehlers and Clark's (2000) model also proposes that the involuntary reexperiencing of the traumatic event can be triggered by a wide range of stimuli and situations.

The cognitive models of PTSD do not offer any explanations as to the neural basis of the different types of memory processes involved in the interaction of PTSD. This will be discussed in the following section

5.5.9 Psychobiology of PTSD

Van der Kolk (1996) suggests that the core disturbance in PTSD is the inability to regulate arousal. He argues that the persistence of intrusive and repetitious thoughts may be influenced by the process of kindling (a process in epilepsy where there is lowering of the seizure threshold after repeated electrical stimulation, as though the brain was learning or being trained to fit. It has been suggested that a similar model may apply to PTSD and that long-term changes in the excitability of the brain may occur making stress responses more easily triggered, which sets up a chronically disordered pattern of arousal (Freeman & Flitcroft 2000).

Van der Kolk (1996) suggests that the psychophysiological effects of trauma occur on two different levels: 1) in response to specific reminders of the trauma, and 2) in response to intense but neutral stimuli, suggesting a loss of stimulus discrimination.

Kolb and Multipassi (1982) have demonstrated that individuals with PTSD respond to specific reminders of the trauma with increases in heart rate, skin conductance and blood-pressure. Pitman, Orr and Shalev (1993) noted that this elevated arousal was observed even to traumas that occurred decades previously. Medication known to stimulate the autonomic arousal system (ANS) such as yohimbine has been shown to induce flashbacks and panic attacks in patients with PTSD, it is believed that yohimbine activates central noradrenergic neurons (Southwick, Krystal & Morgan, 1993). Lang (1979) also reported that emotionally laden mental images are accompanied by increased ANS activity.

Excessive stimulation of the CNS at the time of the traumatic event is believed to result in permanent neuronal changes, that effect learning, habituation and stimulus discrimination (Kolb, 1987). Van der Kolk (1996) notes that these neuronal changes do not depend on actual exposure to reminders of the trauma for expression. Ross, Ball & Cohen (1989) have observed abnormalities in habituation to the acoustic startle response amongst individuals with PTSD. Van der Kolk (1996) suggest that traumatised individuals have difficulty in evaluating sensory stimuli and mobilizing appropriate levels of physiological arousal.

Certain neurohormones, such as the catecholamines e.g. norepinephrine, serotonin, and glucocorticoids have been implicated in PTSD. Urinary norepinephrine (NE) levels have been found to be elevated in PTSD combat veterans compared with patients with other psychiatric disorders (Kosten, Mason, Giller, Ostroff and Harkness, 1987). A study by Shalev et al (1998) found that an increase in adrenergic

response as opposed to noradrenergic activation appeared to contribute to the development of PTSD. Basing their hypotheses on Mowrer's (1960) two-factor theory of classical conditioning, Shalev et al (1998) argued that the physiological component of the response (UCR) to trauma at the time of its occurrence may influence post-trauma outcome. They investigated heart-rate and blood pressure measures in trauma survivors at arrival to accident and emergency, one week later and at four months following the trauma. They found that subjects who developed PTSD had higher rates at both the first and second time periods. There were no differences at the four month heart-rate measure for the PTSD group and non-PTSD group. Blood-pressure, did not differentiate the groups at any of the time periods. They reported that the heart rate difference was not accounted for by rated intensity of the trauma. From within their conditioning model perspective of PTSD, they interpreted the elevated heart-rate findings in the PTSD group as reflecting higher intensity of the UCR. They also propose that excessive adrenergic activation may contribute to the development of PTSD through enhanced memory consolidation of the traumatic event (UCS).

A recent study by Bryant, Harvey, Guthrie and Moulds (in press) measured heart rates amongst individuals who had been involved in road traffic accidents. They found that heart rates obtained before the patients were discharged from hospital were significantly higher in those who later developed PTSD. These findings may be related to Forsyth and Eifert's (1995) suggestion that learned alarms may become associated with interoceptive cues through a process of interoceptive conditioning

(see chapter 1) and McNally and Lukaach's (1992) proposal that aversive bodily sensations are capable of producing PTSD.

Cortisol levels have been found to be lower in individuals with PTSD (Mason, Giller, & Kosten 1986) even although the human stress response is associated with elevated cortisol levels. McFarlane, Atchison and Yehuda (1997) found lowered serum cortisol amongst survivors of road traffic accidents who later developed PTSD. This is in contrast to other groups e.g. depressed patients who have been found to have significantly higher levels of cortisol (Scott, 2000). Yehuda, Southwick, Mason and Giller (1990) explain this paradoxical finding in PTSD due to cortisol's function as an "antistress" hormone. They argue that activation of cortisol (and catecholamines such as NE) stimulate active coping behaviours. However increased arousal in the presence of low cortisol levels provokes undifferentiated fight or flight reactions and may contribute to dysfunctional elevated arousal.

However it is not clear if the lower cortisol levels are as a consequence of a chronic heightened arousal or that individuals developing PTSD have vulnerable hypothalamic-pituitary-adrenocortical (HPA) axis.

Serotonin, has also been found to be implicated in PTSD symptomatology. Southwick et al (1993) administered a serotonin agonist (a drug that mimics a certain neurotransmitter). They found that a number of the participants experienced panic attacks, with a slightly lower number experiencing flashbacks.

It is known that when individuals are under severe stress, the secretion of these aforementioned hormones influences how memories are laid down (van der Kolk, 1997). It is believed that NE input to the amygdala is involved in the overconsolidation of traumatic memories (Le Doux, 1995). Van der Kolk (1994) argues that although physiological arousal can trigger-trauma-related memories, conversely, flashbacks may trigger a re-release of stress hormones, which further kindle the memory trace.

Van der Kolk (1994) highlights both the amygdala and the hippocampus as parts of the limbic system involved in the processing of emotionally charged memories (see chapter 1 for a review of amygdala and hippocampal function). Le Doux, Romanski and Xagoraris (1991) found in a series of animal studies that electrical stimulation of the amygdala produced conditioned fear responses. They also found that cortical lesions prevented the extinction of these responses. Penfield and Perot (1963) elicited memories through direct stimulation of structures in the temporal lobe, including the amygdala and hippocampus, with some subjects reexperiencing frightening events.

Hippocampal volume has also found to be decreased in individuals with PTSD. Bremner, Randall & Scott (1995) observed an 8% reduction in the right hippocampus in Vietnam combat veterans with PTSD. The right hemisphere is believed to be involved in evaluating the emotional significance of incoming information and in regulating autonomic and hormonal responses to these stimuli (van der kolk, 1997).

Rauch, van der kolk, Fisler & Alpert (1996) carried out a positron emission tomography study with patients with PTSD. During exposure to narratives of their traumatic experiences, heightened activity in the right hemisphere was observed. The most active part of the brain was the amygdala. In contrast Broca's area (the part responsible for translating personal experience into communicable language) showed a decrease in oxygen during the exposure. The authors concluded that during activation of the traumatic memory, the brain is "having" its experience. The individual relives and experiences the sensory elements of the experience, however this physiological activity may prevent the experience being translated into communicable language.

This biological finding may offer support for the dual representation models of PTSD. Where it is believed that successful resolution of PTSD symptoms can occur when information is transferred from SAM to VAM memory (Brewin et al, 1995).

5.5.10 Integrative Cognitive Neuroscience Account of PTSD

Brewin (2001) proposes that a plausible model of PTSD should draw on both biological and psychological knowledge to develop an integrative account of the processes involved in the disorder. Again an integrative account does not move to far from Janet's belief that biologically based trauma responses result in fragmentation of mental cohesion, biological, cognitive and emotional dysfunction.

From the biological perspective he proposes that both the amygdala and hippocampus are key structures in understanding responses to threat, including fear

conditioning and the return of fear. As discussed earlier it is known that the acquisition of fear can be mediated by subcortical and cortical pathways. Projections from the hippocampus and from prefrontal cortex have the capacity to inhibit the activation of the amygdala (LeDoux,1995). However in unfamiliar contexts where there are no safety cues or in contexts associated with threat no inhibition takes place and the original fear response is reinstated (Brewin, 2001). He argues that return of fear may be in response to low-level perceptual features of the original learning situation, that has received little conscious processing and thus do not figure in verbally accessible memory (VAM).

Brewin's model infers that the hippocampal processing of fear-evoking information results in the laying down of integrated, coherent representations of conscious experience. These representations are located in the appropriate temporal and spatial context (Brewin, 2001). According to Brewin (2001) it is possible for information to reach the amygdala via a number of different routes, independently of the hippocampus. He points to the visual areas of the inferior temporal cortex, which are involved in the late stages of sensory processing, which project to the amygdala. The thalamo-amygdala route according to Brewin (2001) has a less sophisticated processing capacity and would be capable of transmitting lower-level sensory features. Therefore memories processed via these routes would not be available for deliberate recall, but would be accessed automatically by perceptual cues, similar to those held in the fear memory.

Incorporating recent findings of the effects on stress on the hippocampus and amygdala (Metcalf and Jacobs, 1998). Brewin (2001) proposes that the physiology of the hippocampus under acute stress mirrors the Yerkes-Dodson inverted-u shaped function for cognitive performance. Glucocorticoid exposure first increases activation of hippocampal neurons, however neuronal activation then declines, resulting in impaired hippocampal function due to increased stress. Declarative memory will be effected, with less evidence of binding of individual features into a coherent whole or location in a temporal and spatial context (Brewin, 2001; Metcalfe and Jacobs, 1998). The function of the amygdala however under stress is reported to be enhanced (Pitman, Shalev & Orr, 2000).

Brewin (2001) proposes that the mechanisms of these memory systems and the effects of stress upon them, provide a plausible neural substrate for the dual representation model proposed by Brewin et al (1996). He notes that in particular, this integrative model can account for flashbacks. He suggests that the features of flashbacks (automatically elicited under limited conscious control, stereotyped and unchanged even after many recall episodes and experienced in the present, without any associated temporal context) are suggestive of a non-hippocampally dependent form of memory (SAM).

Brewin (2001) suggests that flashbacks play a critical role in transferring information from the non-hippocampally dependent SAM memory system to the hippocampally based VAM system. It follows that by deliberately focussing attention on the content of the flashbacks, the sensory information can be effectively recoded (Brewin, 2001).

Rather than memories being coded without present and past discrimination, the hippocampus will locate the event in its appropriate context. This process however will be utilised using a limited-capacity system due to the transfer being undertaken by working memory. Therefore according to Brewin (2001) repeated flashbacks will be required to promote information transfer and amygdala inhibition. However also of importance is the individual's capacity to attend and process flashbacks, avoidance or distraction from these experiences will inhibit transfer of information.

Brewin's (2001) integrative model expands the cognitive conceptualisation of the Dual Representation model of PTSD discussed in the earlier section. Inclusion of neuroanatomical data which supports a distinction between hippocampally-dependent and non-hippocampally dependent forms of memory does appear to offer support for the separate memory systems identified in the Dual Representation model. This however still requires to be empirically investigated.

5.6 Treatment of PTSD

Van der Kolk, McFarlane and Hart (1996) propose that the aim of treatment with traumatised patients is to help them move from being "stuck" on the trauma, to being fully engaged in the present, and becoming capable of responding to current exigencies. Van der Hart, Brown and van der Kolk (1989) emphasise the following treatment phases:

- 1) Stabilisation including education and identification of feelings through verbalising somatic states.
- 2) Deconditioning of traumatic memories and responses

- 3) Restructuring of traumatic personal schemas
- 4) Reestablishment of secure social connections and interpersonal efficacy
- 5) Accumulation of restitutive emotional experiences

A number of treatments have been proposed for PTSD. Guidelines for treatment of PTSD (International Society of Traumatic Stress Studies (ISTSS) have recently been published (Foa, Keane and Friedman, 2000) in which a number of treatments have been evaluated for their efficacy.

The standard treatment for PTSD generally involves two elements, prolonged exposure (PE) (in vivo and imaginal) and cognitive restructuring including the modification of maladaptive beliefs about events, behaviours or symptoms associated with the trauma. The theoretical basis for implementing PE therapy is based on Lang's (1977) theory of fear (Foa and Kozak 1986). Eliciting the fear structure allows habituation and also constitutes an opportunity for corrective information to be integrated (Rothbaum & Foa, 1997). During PE sessions patients are asked to describe the event as if it was happening in the present and in the first person. Emphasis should be placed on engagement with the range of emotions experienced during the trauma (Bryant & Harvey, 2000). Foa and Riggs (1993) suggest that the repeated reliving in PE, generates a more organised memory record that can be readily integrated with existing schemata. Friedman (2000) also suggests that PE can abolish the conditioned emotional response evoked by traumatic stimuli, thus reducing the amount of avoidance behaviour associated with maintaining the symptoms.

Bryant and Harvey (2000) argue that in vivo PE has been poorly investigated in PTSD, with most studies including in vivo exposure secondary to imaginal exposure. A study by Richards, Lovell and Marks (1994) found that in vivo exposure was more effective than imaginal exposure in reducing avoidance symptoms.

Van der Hart and Spiegel (1993) however argue that probably the most important factor operating during PE (and it must be assumed through different treatment modalities) is the importance of the therapeutic alliance. They criticise behavioural therapists in their neglect to write about the intensely personal element in the psychotherapeutic alliance. They suggest that forming a new non-traumatic structure may arise from the fact the patient is able to confront the traumatic memory with a trusted therapist in a safe environment.

Rothbaum, Meadows, Resick and Foy (2000) (guidelines for treatment) found that the evidence for PE treatment was very compelling from a number of well-controlled treatment trials. In twelve studies all reported positive results. The studies involved a number of different traumatised groups including, Vietnam veterans, female assault victims and mixed variety of traumas. In the review of treatment guidelines, Rothbaum et al (2000) recommend that PE should be the first line of treatment, unless reasons exist for ruling it out.

Some studies have reported efficacy for systematic desensitisation (SD) (Wolpe, 1958), in which patients are exposed to fear eliciting stimuli (in vivo or imaginal) in

a state of relaxation. Frank and Stewart (1983) reported that SD was effective in reducing PTSD symptoms in rape victims, however there was no control group and PTSD measures were not used. Rothbaum et al (2000) found that studies assessed for the treatment guidelines, which involved SD were mostly methodologically flawed.

Although cognitive patterns in PTSD are considered important, according to Bryant and Harvey (2000) there has been little investigation of the role of cognitive therapy (CT) in facilitating trauma resolution. The difficulty in assessing if CT is an effective treatment for PTSD is due to studies, which integrate CT with PE (Resick & Schnicke, 1993). Tarrier and Humphreys (2000) allocated patients with PTSD to either a PE or CT group. CT was based on Beck and Emery (1985) and Resick and Schnicke (1993). CT was emotion focussed, eliciting patients beliefs about the meaning of the event and the attributions they made following the trauma. Maladaptive cognitions and patterns of emotions were identified and modified. PE was trauma focussed and aimed to produce habituation of emotional responses associated with the trauma. Exposure to the memory continued until emotional reactions decreased. It was found that patients in the PE group showed greater improvement (subjective ratings of symptoms) than the CT group, with the improvement in the PE group occurring after session eight. It is possible however that PE does lead to cognitive restructuring. Jaycox and Foa (1996) suggest that during exposure individuals learn that fear-based beliefs are not reality based. However Bryant and Harvey (2000) challenge this assumption as they argue that entrenched beliefs relating to guilt or anger may not necessarily be modified through

habituation. Only two CT controlled studies were evaluated for the ITSS guidelines for treatment, with the initial evidence supporting the use of CT.

Foa, Rothbaum, Riggs and Murdock (1991) conducted a study comparing PE (imaginal), stress inoculation training (SIT), supportive counselling and a waiting list control. They found that PE and SIT were significantly superior to supportive counselling on PTSD symptoms, but not on depression and anxiety. Marks, Lovell, Noshirvani, Livanou and Thrasher (1998) randomised patients with PTSD into four treatment groups, PE, cognitive restructuring, combined PE and cognitive restructuring and relaxation. Their results showed that all four groups improved significantly. However at three and six month follow-up PE, was slightly superior to cognitive restructuring and the combined group. Rothbaum et al (2000) concluded that combined approaches (CBT) have not resulted in significantly more improvements when compared to single treatments, such as PE. However recent Department of Health Guidelines (2000), based on systematically reviewed research suggests CBT as the therapy of choice for PTSD, including systematic desensitization , which as reported above has not been found to be conclusive due to methodologically, flawed research.

Another treatment purported to be effective in the treatment of PTSD is Eye Movement Desensitisation and Reprocessing (EMDR) (Shapiro, 1995). During EMDR it is proposed that saccadic eye movements reprogram brain function so that the emotional impact of a trauma can be resolved (Friedman, 2000). The patient is asked to imagine a painful, traumatic memory and an associated negative cognition.

The patient is then asked to articulate an incompatible positive cognition. The therapist then asks the patient to think about the traumatic memory while focussing on the rapid eye movements of the clinicians fingers. Following periods of 10-12 eye movements, the patient is asked to rate the strength of the traumatic memory and the belief in the positive outcome (Friedman, 2000). A number of positive results have been reported for the efficacy of EMDR (Shapiro, 1995; Wilson, Becker & Tinker, 1995; Rothbaum, 1997). However Bryant and Harvey (2000) report that a number of studies reporting successful outcome are limited due to the lack of blind assessments, reliance on self-report data and ambiguity about pre-treatment PTSD severity. Research also suggests that eye movements are not necessary for EMDR to work. Finger tapping (Bauman & Melnyk, 1994) has also been used and was found to be effective as eye movements (Friedman, 2000). According to Friedman (2000) the actual mechanisms by which EMDR works is poorly understood. Hyer and Brandsma (1997) suggest that EMDR works, due to:

- ◆ EMDR supports a belief that therapy will lead to positive growth
- ◆ Patients select the traumatic material, which they process in their own ways and at their own paces, this differs EMDR from PE which is a directive approach
- ◆ EMDR bypasses the intense interpersonal issues between patient and clinician that are emphasised in the transference reaction in psychoanalytic psychotherapy.

Brewin (2001) proposes that dual representation theory can offer an explanation for the possible mechanisms for EMDR. He argues that the real-time stimulus provided by the clinician's actions, impinge directly on the patient as they are attending to the traumatic image. This could function to encode a very distinctive attribute with a

new VAM representation. Trauma reminders would then lead to the rapid reinstatement of this memory in preference to the older representations (Brewin, 2001).

Bryant and Harvey (2000) argue that there is no evidence that EMDR provides any additional benefits relative to PE. The ISTSS guidelines recommend that additional studies be carried out that compare EMDR with other focussed PTSD treatments (Chemtob, Tolin, van der Kolk and Pitman, 2000).

Pharmacotherapy has also been employed as a treatment for PTSD. The rationale for drug therapy is based on the findings from the biological model of noradrenergic and serotonergic dysfunction. Selective Serotonin Reuptake Inhibitors (SSRIs) have been reported to be effective in the treatment of PTSD (ISTSS guidelines), (Friedman, Davidson, Mellman and Southwick, 2000). SSRIs have been found to be not only useful for PTSD symptomatology but have been found to be useful for co-morbid disorders, such as depression and panic disorder. Monoamine Oxidase Inhibitors (MAOIs) have been found to show a moderate improvement in trauma symptomatology (De Martino, Mollica and Wilk, 1995). However these drugs are limited in use due to serious consequences of non-compliance with dietary restrictions. Tricyclic antidepressants (TCAs) have not been found to be effective for treatment of PTSD (Southwick, Yehuda and Giller, 1994). The ISTSS guidelines recommend anxiolytics only for adjunctive time-limited treatment, especially sleep disturbance or global anxiety.

However there are a number of factors which may influence treatment outcome and treatment type. The ISTSS guidelines consider a number of factors such as comorbidity, suicidality, drug use and concurrent general medical conditions. Bryant and Harvey (2000) also propose treatment obstacles, especially related to PE that clinicians should be aware of, these include, anger, cultural issues and ongoing stressors.

5.7 PTSD Related to Medical and Dental Events

Mayou and Smith (1997) report that post-traumatic symptoms, immediate, short-term and long-term often present in general medical care. They argue that a number of factors may influence the development of traumatic symptoms in both medical and dental settings, e.g. investigations, severe acute illness and painful procedures. Shalev, Schreiber, Galai and Melmed (1993) reported four case studies involving, the emergence of PTSD after cardiac catheterization, diagnosis of a meningioma, cardiac arrest and a haemorrhaging artery. The authors argue that patients that have undergone procedures involving a feeling of intense inescapable distress, lack of control and perceived or actual threat to life may be at risk of developing PTSD. They suggest that clinicians should be aware that patients presenting with intrusive and distressing recollections of the event, avoidance of medical care and unexplained delay in recovery should be further assessed for trauma-related difficulties.

Studies have also found prevalence rates of PTSD amongst Myocardial Infarction (MI) survivors as between 8-16 per cent (Doefler, Pbert & DeCosimo 1994; Kutz, Shabati, Solomon, Neumann & David, 1994). Bennett and Brooke (1999) reported a

10 per cent prevalence rate for PTSD, twelve months following a MI. They found an association between the frequency of intrusive thoughts and PTSD symptoms. They also reported an association between the frequency of intrusive thoughts and awareness of the episode being an MI. Owen, Koutsakisi and Bennett (2001) reported that PTSD symptoms are relatively common following an MI, however most sufferers problems resolve. However, for a minority of those patients, their symptoms are prolonged and create significant difficulties. The authors argue that adjustments should be made in rehabilitation programmes to include screening for those at risk of PTSD following an MI.

PTSD and PTSD symptoms have also been found amongst patients following subarachnoid haemorrhage (Berry, 1998), uncontrollable pain (Schreiber and Galai-Gat 1993) cerebral vascular accident (Sembi, Tarrier, O'Neil, Burns and Faragher 1998) and following childbirth (Czarnocka and Slade, 2000). A number of studies however report a low uptake of participants (Bennett and Brooke 1999), or low rate of PTSD diagnosis (Sembi et al, 1998). For example six patients from sixty-one were found to have a PTSD diagnosis in Sembi et al's (1998) study. Eight patients from two hundred and sixty four in the Czarnocka and Slade (2000) study showed significant levels of PTSD symptoms. Whither this reflects a low prevalence of PTSD amongst medical and surgical disorders or difficulty in meeting the strict diagnostic criteria as proposed by Mc Millen et al (2000) is not known. For example, in Czarnocka and Slade's (2000) sample some patients did not meet all the three symptoms characteristic of PTSD, but presented with a number of symptoms in the PTSD sub-scales, 37 patients reported clinically significant frequency of intrusions,

72 clinically significant levels of hyperarousal and 19 avoidance and numbing symptoms.

Mayou and Smith (1997) also argue that dental phobics may present with trauma-related symptoms, as these patients are known to exhibit avoidance of the dental setting. Dental anxiety is also known to be frequently, based on past memories of distressing dental events or experiences of pain (Mazey & Mito, 1993). Muris et al (1998) reported that individuals with dental phobia reported higher levels of intrusive thinking regarding past dental experiences and attempts to suppress negative thoughts regarding dentist-related cognitions compared to a group of non-dental phobics. However it is not known if these memories present as post-traumatic symptomatology (frequent recurrent memories, suppression of memories and increased arousal). De Jongh, et al (1995c) and Locker et al (1999) clearly established a relationship between perceived trauma and dental anxiety (painful treatment or treatment associated with terror). However only one study as far as the author is aware has been carried out examining the relationship between dental anxiety and trauma-related symptoms.

De Jongh et al (manuscript submitted for publication) carried out a study to investigate the extent to which anxious dental patients displayed trauma-related symptomatology (intrusive memories and tendency to avoid these memories) associated with past dental events. The study compared two samples of patients, one group consisted of dentally anxious patients (n 37) and the other group were regular non-anxious dental patients (n 32).

De Jongh et al (manuscript submitted for publication) found that thirty-two patients in the patient group and twenty-four regular patients reported past unpleasant direct dental experiences. However the main difference between the groups appeared to be that the dentally anxious patients suffered intrusive memories of these experiences. Twenty-eight patients reported intrusive memories of these events when visiting a dentist. Half of the anxious patient group's IES scores were found to be indicative of a clinically significant level of trauma-related symptomatology, greater than 26 which has been reported to be a significant level of trauma-related symptomatology (Kleber, Brom & Defares, 1992). Six anxious patients were found to have IES scores above 44, which was the average score for individuals who had experienced a traumatic event in Horowitz et al's (1979) study (De Jongh et al, manuscript submitted for publication). Both intrusion and avoidance scores were found to be correlated. Dental anxiety and trauma-related symptomatology (intrusions and avoidance) were found to have a strong direct relationship. De Jongh et al (manuscript submitted for publication) concluded that post traumatic phenomenon are of considerable importance amongst this population, with regard to the understanding of the dynamics of anxiety about potentially aversive dental procedures and the influence that this has on avoidance behaviour, which may compromise the health and well-being of the patient.

Although the Impact of Event Scale (IES) is associated with scores on measures of PTSD (Foa, Riggs, Dancu & Rothbaum, 1993) it has no arousal measure and is not a diagnostic scale for PTSD. De Jongh et al (manuscript submitted for publication)

therefore were unable to conclude what proportion of their intrusive memory sample would have met a diagnosis of PTSD. They also only investigated direct dental experiences and did not consider the indirect pathways of fear acquisition, which has also been shown to be influential in dental fear acquisition (Milgrom et al, 1995; Townend et al, 2000). DSM-IV diagnosis for PTSD does include that the person may have witnessed a traumatic event as well as directly experiencing the event. It is possible therefore that the indirect pathway to dental fear may also be implicated in the dynamics of intrusive memories amongst this population.

Further De Jongh et al's (manuscript submitted for publication) research was carried out in a specialised centre for dentistry and psychotrauma. Therefore it is not known if their findings would generalise from a specialised centre to patients attending a National Health Service secondary care clinic, set up for undergraduate dental teaching.

At present in Scotland, a lack of data about dentally anxious patients and the lack of understanding, about the psychology of anxiety has been identified (National Dental Advisory Committee, 1998). Recently the General Dental Council and the British Dental Association have requested that the dental profession find alternatives to the current management of anxiety using General Anaesthesia. Undergraduate dental students are also taught assessment and behavioural management of dental anxiety. It is important therefore, that patients who may present with trauma-related symptomatology are properly identified.

If dentally anxious patients do present with PTSD or trauma-related symptomatology then recognition of these symptoms may lead to a better understanding of the fear beliefs of these patients, which would further contribute to more appropriate treatment interventions.

De Jongh, et al (1995c) found that individuals with dental anxiety presented with specific thought content resembling thoughts held by patients with other psychological difficulties. Individuals with dental anxiety were found to display a higher frequency of negative thoughts related to dentistry compared to a non-dentally anxious group. It is not known if the dentally anxious patients in de Jongh et al's (1995) study experienced intrusive memories of their past dental experiences. Therefore it is not known if patients who identify intrusive memories of past distressing dental experiences will differ in their frequency of thought content from patients who do not identify intrusive memories. Identifying negative beliefs held, by these patients would appear to be important for cognitive restructuring and modification of these beliefs.

As has also been reported from the literature, patients presenting with dental fears may also have other psychological difficulties (Aartman, 2000, Roy-Byrne et al, 1994). Again it is not known if patients presenting with intrusive memories of past dental experiences will present with greater psychological difficulties than patients without intrusive memories of past dental events.

The aims of this study are to

- 1) To investigate the association between intrusive memories of past distressing dental and or medical events and trauma-related symptomatology.
- 2) To investigate if a difference exists in dental anxiety level between participants reporting intrusive memories of past dental and or medical events and participants who do not report intrusive memories of such events.
- 3) To investigate if a difference exists in thought content related to dental care between participants reporting intrusive memories of past dental and or medical events and participants who do not report intrusive memories of such events.
- 4) To investigate if a difference exists in general psychopathology between participants reporting intrusive memories of past dental and or medical events and participants who do not report intrusive memories of such events.
- 5) To investigate the proportion of participants with intrusive memories who would meet DSM-IV criteria for PTSD.

Hypotheses:

- 1) Intrusive memories of past distressing dental /and or medical events will be associated with trauma-related symptomatology.
- 2) Participants who identify intrusive memories of past distressing dental and or medical events will present with higher dental anxiety levels as measured by the MDAS than those participants who do not identify such intrusive memories
- 3) Participants who identify intrusive memories of past distressing dental and or medical events will present with higher levels of frequency of thought content related to dental care as measured by the Dental Cognitions Checklist than those participants who do not identify such intrusive memories.
- 4) Participants who identify intrusive memories of past distressing dental and or medical events will present with higher levels of general psychopathology as measured by the Brief Symptom Inventory (global indices) than those participants who do not identify such intrusive memories.

Chapter 6: Study 2 Intrusive Memories and Trauma-Related Symptoms and the Differences between Individuals with and without Intrusive Memories on Measures of Dental Anxiety, General Psychopathology and Beliefs Related to Dental Care.

Method

6.1 Design: A correlational design was used to investigate the association between intrusive memories of past distressing dental and or medical experiences and trauma-related symptoms and a between subjects design was used to investigate the differences between individuals with and without intrusive memories on measures of general psychopathology and frequency of dentally related cognitions.

6.2 Sample: Adult patients who were attending the Dundee Dental Hospital Anxiety Clinic during the clinic period (October 2000- May 2001) were invited to participate in the study. In addition adult patients who had attended the Dundee Dental Anxiety Clinic, but who had been assessed during previous clinic periods were also contacted and invited to participate in the study. Patients were referred to the anxiety clinic from both hospital and community dental surgeons. Ethical approval was obtained for the study in November 2000 from Tayside Committee on Medical Research Ethics. Data collection commenced during December 2000 following this approval. Interviews and data collection were carried out by the author. Thirty-six participants were recruited for the study.

The number of appointments sent out for the October to May (2000-01) clinic, was twenty-four, eight patients did not attend for their first or second appointments and five cancelled and did not want a second appointment sent. Of the eleven patients left, nine agreed to take part in the study. Seventy-nine letters were sent out in January 2001 to patients who had attended the dental anxiety clinic but who had been assessed during previous assessment clinics. The number of positive returns was twenty-nine, five replied indicating they did not want to take part in the study. Nine letters were returned, due to wrong addresses. Thirty-nine patients (thirty-five females and four males) did not return the letters. In total there was a 36 per cent response rate. Of the twenty-four patients who agreed to take part in the study, one did not turn up for their first appointment, nor did they attend following a second appointment being arranged. Two were excluded due to not reaching the inclusion criteria.

Patients being seen at the IV sedation clinic (only one session was held during the Oct-May session) were also approached and invited to participate in the study. Five out of six patients did not attend for their appointments. One did turn up and agreed to take part in the study but later declined due to health reasons.

As the sample number was low in March 2001 the inclusion criteria were relaxed to include participants who reported a fear of the dentist but who were not attending the dental school anxiety clinic. Participants were recruited through opportunistic sampling. This involved giving information (written and oral) to participants, who heard about the study through discussion with the author. Participants were recruited

from hospital out-patients clinic staff (nursing and administration), community nursing staff and university lecturing staff. Four participants were recruited, however one was excluded due to not reaching the inclusion criteria. One patient was recruited from the Tayside Clinical Psychology Department. This patient had been referred due to severe dental anxiety, but was not referred to the dental school anxiety clinic. Three patients were referred to the author from the pain clinic at Ninewells Hospital, with dental anxiety. One patient was excluded due to not reaching the inclusion criteria.

Permission was also gained from the dental clinic co-ordinator to run one extra anxiety clinic assessment session (June 2001). Five patients were sent appointments to attend for this session. All five patients failed to attend.

Inclusion Criteria: Participants who were aged 16 years and over who have a MDAS score of 19 or greater. Participants were required to have sufficient English to complete the interview and questionnaires.

Exclusion Criteria: Participants who did not meet MDAS criteria for dental anxiety. Participants unable to complete the interview and questionnaires due to language difficulties.

6.3 Sample Size: the sample size is based upon data from de Jongh et al (manuscript submitted for publication). Using means and standard deviations from the Dental

Anxiety Scale from a group of dentally anxious patients with intrusive memories of past distressing dental experiences and a control group without dental anxiety or intrusive memories of past distressing dental experiences. A sample size of 20 per group would allow detection of an effect size of 0.8 at α .05 and a power of .80.

6.4 Measures:

The Modified Dental Anxiety Scale (MDAS) (Humpris, Morrison & Lindsay, 1995). See page forty-five for the description of this measure (used in study one). In the current study the Cronbach alpha coefficient was .48.

Dental Cognitions Questionnaire (DCQ) (de Jongh, Muris, Schoenmakers & Ter Horst, 1995). This is a self-report measure containing 38-items which assesses frequency and believability of negative cognitions related to dental treatment. It was reported that the DCQ appeared to possess sufficient internal consistency, Cronbach's alpha (frequency, 0.89 and believability 0.95) and high test-retest reliability (0.83).

This measure has not been validated for the British population. However at present a questionnaire assessing frequency of thoughts that individual's hold related to dental care is used by staff at the Dundee Dental School anxiety clinic. The Dental Cognitions Checklist (DCC) (Appendix 5) was devised by Milgrom et al (1985). This version contains 40 items. The individual is asked to indicate the degree to which each statement relates to their belief about dental care. There are an extra two blank spaces for the patient to write down any anxiety provoking or disturbing

thoughts that they might have related to dentistry. Thirty-five statements are related to negative consequences of treatment, with five statements being related to embarrassment about behaviour during treatment (no levels of agreement, regarding the categories in this measure has been undertaken). This version of the DCC has not been psychometrically evaluated but is used in the clinical setting to elicit primary anxiety focussed cognitions. Likert scoring is used for this measure (questions 1-40). The applied scoring was 0 = not at all, 1 = rarely, 2 = sometimes and 3 = often. Total scores were calculated for each subject, with higher scores indicating a greater frequency of negative beliefs regarding dental care.

The Impact of Event Scale (IES). (Horowitz, Wilner & Alvarez, 1979). This is a self-report instrument, which evaluates trauma-related intrusions and avoidance. Respondents are asked to rate each IES symptom item on a four-point scale marked 0 (not at all), 1 (rarely), 3 (sometimes), 5 (often). Seven items evaluate intrusion and eight evaluate avoidance. A scale score is computed by adding all 15 responses, thus the scale score can range from 0-75. The IES has been shown to discriminate between traumatised and nontraumatised groups (Bryant & Harvey, 1996). Horowitz et al (1979) demonstrated internal reliability of alpha 0.79 for the intrusion subscale and 0.82 for the avoidance subscale. A correlation of 0.42 was found between the intrusion and avoidance sub-scales, indicating that the two subsets are associated but do not measure identical dimensions. The IES was developed as a research tool and Briere (1998) suggests that it should be used only as a screen for the presence of non-arousal symptoms of PTSD. In the current study the Cronbach alpha coefficient was .60.

Structured Interview for PTSD (SIP) (Appendix 6) (Davidson, Smith & Kudler, 1989). The scale comprises 17 items reflective of the DSM-IV criteria for PTSD. Each item is rated on a 0-4 scale and represents a composite of frequency, severity and functional impairment (Davidson, Malik & Travers, 1997). There is a maximum score of 68. The SIP serves as a symptom severity instrument and as a diagnostic tool (Davidson et al, 1989). The diagnosis of PTSD is defined by DSM-IV criteria including the experience of a traumatic event and the reaction to that event (fear, helplessness or horror) plus:

- ◆ at least one item from category B/reexperiencing of the traumatic event with a SIPS score of at least 2
- ◆ at least three items from category C/avoidance of stimuli associated with the trauma and numbing of general responsiveness with a SIPS score of at least 2 (at least one item must be from the avoidance category and one must be from the numbing category)
- ◆ at least two items from category C/increased arousal with a SIPS score of at least 2.

Davidson et al (1997) demonstrated internal consistency of the scale, Cronbach alpha of 0.80, concurrent validity with regards to the Davidson Trauma Scale, the Pearson correlation coefficient was 0.67. In the current study the Cronbach alpha coefficient was .73.

Brief Symptom Inventory (BSI) (Derogatis, 1993). The BSI is a 53-item self-report symptom inventory which was designed to reflect the psychological symptoms of psychiatric and medical patients as well as community non-patient respondents. It is the brief form of the SCL-90-R. Each item of the BSI is rated on a five-point scale of distress (0-4) ranging from “not at all” (0) to “extremely” (4). The BSI is scored and profiled in terms of nine primary symptom dimensions and three global indices of distress (Derogatis, 1993). The primary symptom dimensions are: Somatization, Obsessive-Compulsive, Interpersonal Sensitivity, Depression, Anxiety, Hostility, Phobic Anxiety, Paranoid Ideation, Psychoticism. The three global indices provide a more flexible overall assessment of the patient’s psychopathological status. The three global indices are: Global Severity Index (GSI), Positive Symptom Total (PST), Positive Symptom Distress Index (PSDI). Internal consistency of the BSI is reported as .71 (Cronbachs alpha). Test-retest reliability has been reported between .68 (somatization) to .91 (phobic anxiety). In the current study the Cronbach alpha coefficient was .91.

Life-Events Checklist: this checklist forms part of the Clinician-Administered PTSD Scale (CAPS-1). This was used to assess the number of past life events that each participant may have experienced witnessed or learnt about.

The identification of distressing dental/medical incidents is based on de Jongh, Aartman & Brand (manuscript submitted for publication) four-item questionnaire. This was designed to assess traumatic incidents relating to dental treatment.

Participants are asked the following questions:

- 1) Have you ever experienced or been told about or seen someone experience a distressing event during dental or medical treatment that would explain your dental anxiety?
- 2) What is the worst dental or medical event you can remember experiencing, been told about or witnessing?
- 3) How many years ago did this happen?
- 4) Do memories of this incident come up when you visit a dentist?

6.5 Procedure: Adult patients who were attending the dental anxiety clinic were invited to participate in the study. Newly referred patients were approached by the author following their assessment (during the assessment session clinic with either the clinical psychologist/or dental surgeon) and given information about the study. An information sheet was provided (Appendix 7) describing the rationale of the study. Patients who received the information sheet were contacted one week later (as per ethical committee protocol) and those who agreed to take part in the study were given an appointment to attend at Ninewells Hospital and Medical School, Dundee.

Patients who were attending for treatment (but not attending the assessment clinic) were sent letters describing the rationale of the study. This group of participants were asked to indicate whether they agreed to take part in the study and if agreeable to note a point of contact (phone or further appointment letter). A SAE was included for patients to return this information.

Research interviews were carried out at Ninewells Hospital and Medical School, due to renovation of the dental anxiety clinic premises (dental assessment and treatment were carried out at another location within the dental hospital, however there was no suitable location for the research interview to take place). A £10.00 travel expense was paid to each participant.

The groups for this study were formed by the following procedure. Participants who attended for interview and who identified distressing experiences (direct/indirect) and who reported memories of the incident when visiting a dentist as identified by De Jongh et al's (manuscript submitted for publication) four part questionnaire, (question four) were asked to think about the worst event identified and the trauma questionnaires (IES & SIPS) were administered related to this event. They were also administered the MDAS, DCC and BSI.

Participants without intrusive memories as measured by De Jongh et al's (manuscript submitted for publication) four part questionnaire (question four) were administered the MDAS, DCC, and BSI the PTSD scales were omitted.

The time taken for each full interview (all measures) was found to be just over one hour and twenty minutes.

6.6 Analyses: Analysis was carried out using the Statistical Package for Social Sciences/ Windows (SPSS 10). Access to data was password protected. Exploratory data analysis was used to determine the level of normality relating to distribution of

measured variables. A Pearson-Moment Correlation was used to test for associations between variables and to test hypothesis 1. An independent-samples t-test was used to determine differences between groups and test hypothesis 2, 3 and 4.

Chapter 7: Results: Study 2 Intrusive Memories and Trauma-Related Symptoms and the differences between Individuals with and without Intrusive Memories on Measures of Dental Anxiety, General Psychopathology and Beliefs Related to Dental Care.

As for study 1 the data set was checked for errors prior to analysis. No out-of-range values were found for any of the variables. Exploratory data analysis was performed to provide information concerning the distribution of scores on continuous variables and to assess if the data met the assumptions for parametric or non-parametric statistical analysis.

7.1 Demographic Data:

7.1.1. Participants

Data was analysed from thirty-six participants, five males and thirty-one females. The mean age of the total sample was 39.17 years (SD 12.64, minimum 18- maximum 77 years). Thirty-five participants identified a distressing past dental event (no distressing medical procedures were identified) which they felt explained their present dental anxiety.

Thirty-two participants identified a direct conditioning experience and three participants identified an indirect conditioning experience. One participant could not recall any direct or indirect distressing experiences and reported that she had always been dentally anxious.

When asked to recall the worst distressing dental event, fourteen participants (12 participants from the dental school, one participant referred to the author from clinical psychology and one participant from the opportunistic sampling group) reported that memories of that event came into their mind when visiting a dentist. Three of the twelve participants from the dental anxiety clinic had attended during the October 2000-May 2001 session. Nine had been assessed prior to this session. Twelve participants of this group reported past direct conditioning experiences and two participants reported an indirect conditioning experience.

Of the fourteen participants in the intrusive memory group, eleven reported direct experiences which involved painful dental procedures. Two of this group reported that as well as painful experiences the dentist had been abrupt, and one reported that the dentist had held her down in the chair. The other directly conditioned participant reported an incident, which for reasons of confidentiality can not be recorded here (although the participant did agree that their data could be used in the study).

The two indirect participants in this group reported one family member becoming unwell following a dental procedure, which required hospitalisation (witnessed). The other participant reported the death of a family member following a dental procedure (transmission of information).

Twenty participants in the no intrusive memory group reported direct conditioning experiences, one reported an indirect experience and one could not recall a pathway of conditioning. The directly conditioned participants all reported incidents of painful

treatment. One recalled being told that they were just having a dental assessment then having a painful tooth extraction. Following this episode, mistrust of dentists developed. The indirect participant reported being told by a sibling that dental treatment was extremely painful, and from that point, the participant has experienced anticipatory anxiety regarding painful treatment.

Table 7. presents demographic details of the two groups (intrusive memories and no intrusive memories) for mean age, mean age at trauma and dental avoidance history.

Table 7. Mean age, age at trauma and dental avoidance history for the Intrusive memory and Non-Intrusive memory groups.

MEM		AGE	AGETRAUM	AVOIDHIS
Intrusive				
N (14)	Mean	40.57	14.07	13.36
	(SD)	(12.63)	(7.59)	(7.63)
Nonintrusive				
N (22)	Mean	38.27	15.86	15.00
	(SD)	(13.11)	(9.38)	(8.16)

An Independent-samples t-test was conducted to compare the ages of the two groups ($t(34)=.520$, $p=.606$, two-tailed) the age when trauma occurred ($t(34)=.269$, $p=.790$, two-tailed) and avoidance history ($t(34)=.600$, $p=.552$, two-tailed). No statistical differences were found between the two groups for either age, age when trauma occurred or years of avoidance.

For the intrusive memory group the mean number of years since their worst dental experience was 25 years (SD 15.34).

There were no differences in the number of life events that participants had experienced, witnessed or learnt about between the groups.

Data analysis was performed to assess whether the sample data conformed to a normal distribution. Visual inspection of the distribution of scores was carried out and a formal statistical analysis was performed. The results of the Kolmogorov-Smirnov statistic for the memory group are presented in table 8.

Table 8. Kolmogorov-Smirnov test of normality, Trauma measures

Measure	Df	Sig
IESSAV	14	0.53
IESINT	14	.176
IESTOT	14	.124
SIPAV	14	.150
SIPINT	14	.145
SIPHYP	14	.200
SIPTOT	14	.200

*IESAV Avoidance subscale of the IES *IESINT Intrusion subscale of the IES
 *IESTOT Total IES *SIPAV Avoidance subscale of the SIP *SIPINT Intrusion
 subscale of the SIP *SIPHYP Increased Arousal subscale of the SIP *SIPTOT Total
 SIP

The results of the Kolmogorov-Smirnov statistics for both groups is presented in
 table 9.

Table 9. Kolmogorov-Smirnov test of normality, General measures

MEM	Measures	df	Sig
Intrusive	MDAS	14	.200
	DCCTOT	14	.200
	BSIGLOB	14	.200
	PST	14	.125
	PSDI	14	.200
Nonintrusive	MDAS	22	.200
	DCCTOT	22	.111
	BSIGLOB	22	.070
	PST	22	.200
	PSDI	22	.095

*MDAS Modified Dental Anxiety Scale *DCC Dental Cognitions Checklist

* BSIGLOB Brief Symptom Inventory global score * PST Brief Symptom Inventory Positive Symptom Total * PSDI Brief Symptom Inventory Positive Symptom Distress Index

The results from the Kolmogorov-Smirnov statistic were found to be non-significant indicating that the assumption of normality has not been violated. Parametric tests were applied for the analysis.

A comparison was also made between the participants who took part in the study and those who did not return their letters. The five individuals who returned their letters but who did not agree to take part were also included in this group, as was the respondent who failed to turn up for both appointments after agreeing to take part in the study.

Table 10. Comparison of mean scores for age and dental anxiety level for responders and non-responders.

GROUP		MDAS	AGE
Ressample	Mean	22.26	39.33
N (36)	SD	(1.88)	(12.64)
Nonparticipants	Mean	21.56	34.36
N (45)	SD	(2.86)	(11.77)

- ressample (sample who took part in study two)
- nonparticipants (sample who either did not return their letters or who declined to take part in the study)

An independent-samples t-test was conducted to compare the ages ($t(79) = 1.208, p = .071$, two-tailed) and the level of dental anxiety ($t(79) = 1.208, p = .231$, two-tailed). No significant differences were found between the two groups.

Hypothesis 1:

Hypothesis one predicted that intrusive memories of past distressing dental and or medical events will be associated with trauma-related symptomatology. The mean scores for the trauma-related measures are presented in Table 11

Table 11. Mean scores for trauma measures, intrusive memory group (N14)

	IESINT	IESAV	ESTOT	SIPINT	SIPAV	SIPHYP	SIPTOT
Mean	19.50	17.71	37.21	7.21	4.79	5.21	15.86
SD	(9.09)	(8.29)	(14.75)	(4.19)	(3.51)	(3.14)	(10.17)

Correlation analysis was performed to describe the strength of the relationship between intrusive memories using the IES intrusive memory scale and the other measures of trauma-related symptoms. Prior to analysis scatterplots were generated to check for violation of the assumptions of linearity and homoscedasticity (appendix 8). Visual inspection of the scatterplots showed that these assumptions had not been violated.

Pearson product-moment correlation was carried out for the analysis. The strength of the relationship was based on Cohen's (1988) guidelines ($r = .10$ to $.29$ = small, $r = .30$ to $.49$ = medium, $r = .50$ to 1.0 = large).

Table 12. Pearson-Product – Moment Correlations between IES (intrusion scale) and IES (avoidance), SIPS (intrusion, avoidance, increased arousal and total SIP scale)

Measure	IES intrusion	Coefficient of determination $r^2 \times 100$
IES avoidance	.44 $p = .115$	19%
SIP intrusion	.50 $p = .065$	25%
SIP avoidance	.40 $p = .155$	16%
SIP increased arousal	.41 $p = .139$	16%
SIP total	.56 $p = .035^*$	31%

* $p < .05$ (two-tailed)

There was a medium positive correlation between intrusive memories (as measured by the IES intrusion subscale) and avoidance (as measured by the IES avoidance subscale), this was not found to be statistically significant. There was a large positive correlation between intrusive memories as measured by the IES and SIP intrusive memory scales, this was not found to be statistically significant. There was a medium positive correlation between intrusive memories (as measured by the IES intrusion subscale) and avoidance (as measured by the SIP avoidance subscale), this was not found to be statistically significant. There was a medium positive correlation

between intrusive memories (as measured by the IES intrusion subscale) and increased arousal (as measured by the SIP increased arousal subscale), this was not found to be statistically significant. There was a large positive correlation between intrusive memories (as measured by the IES intrusion subscale) and total SIP score. This was found to be statistically significant, with frequency of intrusive memories being associated with frequency and severity of intrusive memories, avoidance of stimuli associated with the trauma and increased arousal (as measured by the SIP).

The shared variance between intrusive memories (intrusive memory subscale of the IES) and avoidance (IES), intrusion (SIP), avoidance (SIP), increased arousal (SIP) and total SIP can all be accounted for by less than 50 per cent. This indicates that over 50 per cent of the variance in intrusive memories, avoidance, increased arousal and total SIP is due to other variables other than intrusive memories related to the trauma.

Hypothesis 2

Hypothesis two predicted that participants who identified intrusive memories of past distressing dental events will report higher levels of dental anxiety than those participants who did not identify such intrusive memories.

An independent-samples t-test was conducted to compare levels of dental anxiety between the two groups. Table 13 presents the mean scores for dental anxiety and Independent-samples t-test between the two groups.

Table13. Mean MDAS scores intrusive memory and non-intrusive memory group and Independent-samples t-test MDAS between the two groups.

Group	N	Mean (SD)	df	t	Sig (1-tailed)	95% CI	
						lower	upper
Intrusive	14	22.57 (2.21)	34	.959	.172	-.69	1.92
Nonintru	22	21.95 (1.65)					

* Nonintru (non-intrusive memory group)

There was no statistically significant difference found for level of dental anxiety between the two groups, ($t(34)=.959$, $p = .172$, one-tailed).

Hypothesis 3:

Hypothesis three predicted that participants who identify intrusive memories of past distressing dental and or medical events will present with higher levels of frequency of thought content related to dental care than those participants who do not identify such memories. Table 14 presents the mean DCC total for both groups and independent-samples t-test between the two groups.

Table 14. Mean DCC scores intrusive and non-intrusive memory groups and Independent-samples t-test DCC between the two groups

Group	N	Mean (SD)	df	t	Sig (1-tailed)	95% CI	
						lower	upper
Intrusive	14	57.93 (20.57)	34	.069	.473	-16.10	15.05
Nonintru	22	58.45 (23.48)					

* Nonintru (non-intrusive memory group)

There was no statistically significant difference found for frequency of thought content related to dental care between the two groups, ($t(34) = .069, p = .473$, one-tailed)

Hypothesis 4:

Hypothesis four predicted that participants who identify intrusive memories of past distressing dental and or medical events will present with higher levels of general psychopathology than those participants who do not identify such intrusive memories. An independent-samples t-test was conducted to compare levels of general psychopathology (BSIGLOB) between the two groups.

Table 15 presents the mean scores for BSIGLOB and independent-samples t-test between the two groups.

Table 15. Mean BSIGLOB scores intrusive and non-intrusive memory groups and Independent-samples t-test BSIGLOB between the two groups

Group	N	Mean (SD)	df	t	Sig (1-tailed)	95% CI	
						lower	upper
Intrusive	14	1.07 (.75)	34	.356	.362	-.39	.56
Nonintru	22	.99 (.64)					

* Nonintru (non-intrusive memory group)

There was no statistically significant difference found for global level of general psychopathology between the two groups, ($t(34) = .356, p = .362$, one-tailed)

An independent-samples t-test was conducted to compare levels of general psychopathology (BSIPST) between the two groups.

Table 16 presents the mean scores for BSIPST and independent-samples t-test between the two groups.

Table 16. Mean BSIPST scores intrusive and non-intrusive memory groups and Independent-samples t-test BSIPST between the two groups

Group	N	Mean (SD)	df	t	Sig (1-tailed)	95% CI	
						lower	upper
Intrusive	14	32.21 (9.07)	34	1.766	.073	-.77	11.02
Nonintru	22	27.09 (8.11)					

* Nonintru (non-intrusive memory group)

There was no statistical difference found for levels on the BSIPST between the two groups, ($t(34)=1.766$, $p=.073$, one-tailed)

An independent-samples t-test was conducted to compare levels of general psychopathology (BSIPSDI) between the two groups.

Table 17 presents the mean scores for BSIPSDI and independent-samples t-test between the two groups.

Table 17. Mean BSIPSDI scores intrusive and non-intrusive memory groups and Independent-samples t-test BSIPSDI between the two groups

Group	N	Mean (SD)	df	t	Sig (1-tailed)	95% CI	
						lower	upper
Intrusive	14	1.80 (.87)	34	.418	.339	-.44	.67
Nonintru	22	1.69 (.76)					

There was no statistical difference found for BSIPSDI scores between the two groups, ($t(34) = .418, p = .339$, one-tailed).

The fifth aim of this study was to investigate the proportion of participants presenting with intrusive memories who would meet DSM-IV criteria for PTSD. This was based on SIPS DSM-IV criteria. Only one participant from the intrusive memory group met the DSM-IV criteria, with symptoms lasting over a four-week period (more than 30 days). This participant was actively avoiding attending the dental anxiety clinic.

One participant reported increased arousal, intrusive memories and avoidance lasting for over two weeks. This participant was engaged in dental treatment at the time of the research interview, and although attended for treatment, reported that when away from the dental school, actively attempted to avoid thoughts or feelings regarding her planned treatment.

The remaining participants reported that their symptoms had been more frequent during the days approaching the interview.

Chapter 8: Discussion Study 2. Intrusive Memories and Trauma-Related Symptoms and the differences between Individuals with and without Intrusive Memories on Measures of Dental Anxiety, General Psychopathology and Beliefs Related to Dental Care

8.1 Summary of Research:

De Jongh et al (manuscript submitted for publication) reported that intrusive memories of past distressing dental experiences were associated with trauma-related symptomatology and level of dental anxiety. This was carried out in a specialised centre for dentistry and trauma, therefore it was unknown if these findings would generalise to other populations. It was also unknown how many patients presenting with intrusive memories would meet DSM-IV criteria for PTSD. De Jongh et al (1995a) also proposed that cognitive factors might influence, maintain or exacerbate dental anxiety. Further it is also recognised that individuals with dental anxiety may have additional psychological difficulties, which may lead to difficulties in treatment (Aartman, 2000).

It was not known if intrusive memories of past distressing dental and or medical experiences would influence degree of general psychopathology or thought content related to dental care.

Study two was conducted to investigate the association between intrusive memories of past distressing dental and or medical events and trauma-related symptomatology in participants with dental anxiety. The proportion of individuals meeting DSM-IV

diagnostic criteria for PTSD was also investigated. In addition a further investigation was carried out to assess if differences existed between participants identifying intrusive memories and those without intrusive memories on measures of dental anxiety, frequency of thought content related to dental care and general psychopathology.

8.2 Discussion of Research Findings:

Based on IES mean scores this study appears to show that participants who present with intrusive memories related to past distressing dental events do have significant trauma-related difficulties.

Ten of the participants were found to have mean IES scores above 26. According to De Jongh et al (manuscript submitted for publication) this is indicative of a clinically significant level of trauma-related symptomatology. Three of these participants had mean IES scores greater than 44. De Jongh et al (manuscript submitted for publication) reported that over half of their sample scored above the cut-off point of 26 on the IES. The findings from both studies suggest that participants who identify intrusive memories of past distressing dental experiences present with significant trauma-related distress where this is assessed by the IES. Briere (1998) however cautions using the IES as no more than a screening tool for the presence of non-arousal-related post-traumatic stress. Caution is required therefore for using it for diagnostic purposes.

One patient was identified with DSM-IV criteria for PTSD using SIPS DSM-IV criteria. This participant reported that the disturbance caused significant distress in both social and occupational functioning.

Results from the correlational analysis found a medium positive correlation between intrusive memories and avoidance (using both the IES and SIPS) however no statistical significance was found. A medium positive correlation was found between intrusive memories and increased arousal, no statistical significance was found. A large positive correlation was found between the IES intrusion subscale and the SIPS intrusion scale however no statistical significance was found. A large positive correlation was also found between the IES intrusion subscale and the total SIPS score, this was found to be statistically significant. This suggests a strong relationship between frequency of intrusive memories of past distressing dental events and frequency and severity of trauma-related symptoms as measured by a scale reflective of DSM-IV criteria. This supports the hypothesis that individuals reporting intrusive memories of past distressing dental events present with trauma-related symptoms. De Jongh et al (manuscript submitted for publication) found a large significant association between IES intrusions and IES avoidance. This was not supported by this study.

The longitudinal study of accident victims with PTSD suggests a picture of intrusive memories and heightened arousal many years following the trauma. The mean length of time from worst dental event to assessment in this study was 25 years, suggesting that trauma-related distress was still being identified in the intrusive memory group

many years following the event. McFarlane and Papay (1992) found that eight years after a disaster intrusive memories and symptoms of disordered arousal were still prominent. However failure to reach threshold for avoidance and estrangement represented the reasons as to why PTSD criteria were not met. It is possible this is reflected in this study where only one participant met DSM-IV diagnostic criteria. However the participants in the intrusive memory group did present with significant trauma-related symptoms (reflected by their mean IES scores and the strong association between IES intrusions and SIPS total score). This finding may offer support for the proposal by Stein et al (1997) that individuals may present with “partial PTSD” i.e. significant trauma-related distress without meeting full PTSD diagnosis.

It was reported by a number of participants that intrusive memories and trauma-related symptoms were triggered by the anticipation of discussing their past dental experiences. This is also similar to the finding by Lauth (1971). He originally reported that any reference to dentistry amongst a group of dental phobics produced vivid images of the traumatic experience.

Although a large positive correlation was found between intrusive memories, and total SIP score, the shared variance was found to be 31 per cent. Therefore more than half of the variance between intrusive memories, and SIPS total score appears to be due to other factors. Shalev (2001) argues that other factors may be involved in predicting PTSD especially post-trauma factors. Post and pre-trauma factors (social-support and further stressors) identified by Shalev (2001) are unknown in the

intrusive memory group in this study. It is also unknown if the intrusive memory group had experienced more distressing dental events than the non-intrusive memory group. The dental history of the participants prior to their worst identified event was not established in this study. This is a clear limitation, as previous trauma is known to be a risk factor for the development of PTSD and is also known to exacerbate reactions to current traumas.

There was no support for the hypotheses that intrusive memories would influence levels of dental anxiety. Although mean levels of dental anxiety were found to be higher in the intrusive memory group, there was no statistical difference found between the two groups, suggesting that the intrusive memory group did not differ in their levels of severity of dental anxiety. This does not support De Jongh et al's (manuscript submitted for publication) findings. The data from this study suggest that unless trauma-related symptoms are specifically assessed trauma-related difficulties may be missed if using level of dental anxiety as an indicator of distress. This has important implications for the assessment and treatment of individuals presenting with dental anxiety and trauma-related dental distress. If trauma-related symptoms are missed it may well preclude individuals from being given appropriate clinical treatment

A partial explanation for the differences obtained between this current study and that of De Jongh et al (manuscript submitted for publication) were differences in the measures used to assess dental anxiety. De Jongh et al (manuscript submitted for publication) used the CDAS for assessing dental anxiety. Humphris et al (1995)

have criticised the use of the CDAS due to the order of the items on the scale, which are believed to be confusing for the respondent. According to Humphris et al (1995) answers reflecting “slightly anxious” may be assigned moderately high scores, with the converse for moderate anxiety. The MDAS was used in this study in an attempt to remove this confusion plus it is also routinely used at the dental anxiety clinic. However of note was the low Cronbach coefficient alpha for this measure in this study. As this was a short scale (less than six items) Briggs and Cheek (1986) suggest that it may be more appropriate to assess the mean inter-item correlation for the items, with the optimal range of correlation of .2 to .4. The range of correlations for the MDAS in this study was found to be .0 to .2. Thus possibly limiting its use in this studies population and limiting any conclusions made from the results as to the influence on intrusive memories and levels of dental anxiety.

No statistical differences were found between the two groups for levels of frequency of thoughts regarding dental care. This suggests that the level of frequency of beliefs regarding negative consequences of treatment and social embarrassment related to dental care does not differ between the two groups. Muris et al (1998) found that a group of dental phobic patients actively tried harder to suppress negative thoughts regarding dentist-related cognitions and had higher levels of intrusive thinking regarding dental treatment compared to a group of non-phobic participants.

No statistical differences were found between the groups for levels of general psychopathology. McMillen et al (2000) found that avoidance and numbing PTSD symptoms were associated with psychiatric comorbidity. The participants in this

sample were found to endorse less avoidance and numbing symptoms of both measures therefore this might reflect a less general psychologically distressed intrusive memory group in this study.

The mean age of worst distressing dental event was found to differ between the two groups. Although there was no statistical difference between the groups, developmental stage at time of trauma is known to be an important factor for later traumatic stress and its sequelae (Yule et al, 1999). Chapman and Kirby-Turner (1999) suggest that appraisal of threat may be due to greater cognitive ability which may be associated with later adolescence. Contrary to the findings in this study where a mean younger age at time of worst experience was found. It is possible that experiencing a distressing dental event with less cognitive ability for appraisal may lead to more inappropriate levels of physiological arousal. Bryant et al (2000) and Shalev et al (1998) have reported higher levels of physiological arousal (higher heart-rates) amongst those who developed PTSD amongst adults, the processes involved amongst children and adolescents is less clear. It is possible that differences in physiological response to distressing events may be associated with stage of development. Perry (1994) has proposed that differences exist in response to trauma according to development level, with younger children dissociating at time of trauma compared to older children responding with heightened arousal.

Mean years of avoidance were not found to differentiate between the two groups although the non-intrusive memory group had a longer period of avoiding the dentist than the intrusive memory group. However avoidance can occur at both the cognitive

and behavioural levels including other activities that numb or distract. It is possible that assessing only one strategy of avoidance (behavioural) does not adequately target the number of potential avoidance strategies that may differ between these two populations.

Direct conditioning experiences were found to be the pathway recalled by thirty-two of the participants. Painful dental procedures were reportedly involved and identified in all the direct cases as the negative precipitant that triggered fear of the dentist. This finding is similar to previous research (Lautch, 1971, Locker et al, 1999). However identification of the “worst dental experience” was based on retrospective recall, which may be subject to memory and interpretation bias. A study by Kent (1989) found that patients with dental anxiety reported more pain three months following treatment than was reported directly after treatment. However Brewin, Andrews and Gotlib (1993) argue that claims about the unreliability of retrospective reports are exaggerated, citing evidence that retrospective accounts of onset experiences have been confirmed when evidence is sought from others.

Two participants in the intrusive memory group identified indirect experiences precipitating their dental anxiety. Although it is difficult to generalise from such a small number, this study has identified that participants recalling indirect experiences may also develop significant trauma-related difficulties. It is possible to argue that the indirect experiences identified by these two participants could be viewed as significant life-events, certainly fulfilling DSM-IV PTSD criteria A (1) and may not reflect Rachman’s (1968) proposal that indirect conditioning would be reflected by a

lesser strength of response than direct conditioning. However as previous research has shown (Ost and Hugdahl, 1981) and from the limited findings from study 1, indirect pathways of conditioning may also involve responses similar or greater than those acquired directly.

The actual numbers of participants in this study was low. Bennett and Brooke (1999) have also reported a low sample rate in their study investigating PTSD. It is possible that individuals who have more distressing symptoms may avoid a study, which is asking participants to describe previous dental experiences. There was no statistical difference between the participants who took part in the study and those who either did not respond, respond in the negative or who did not turn up for arranged appointments for either age or dental anxiety level. However as discussed above trauma distress related to dental anxiety may be missed if this is dependent on a measure of dental anxiety level alone.

8.3 Methodological Problems

There were methodological problems related to this study. The sample size was small and therefore was insufficient to achieve adequate statistical power, based on the sample size calculated from De Jongh et al's (manuscript submitted for publication) study. A retrospective power calculation was performed. A sample size of 60 per group would allow detection of an effect size of 0.5 at α .05 and a power of .80.

The correlational analysis was also calculated on a small sample. Significance of r is strongly influenced by the size of the sample. Therefore in a small sample moderate

correlation's may not reach statistical significance at .05 level. Pallant (2001) suggests that where sample size is < 30 then shared variance calculated by the coefficient of determination between the two variables should be the focus.

The MDAS Cronbach alpha coefficient was found to be .4 for this study. It is possible that this scale may be measuring something different to the scale as a whole. Further research is obviously required to evaluate this scale especially as it is widely used with this dentally anxious population.

The dental cognitions questionnaire (DCC) was used in this study to assess frequency of beliefs regarding dental care. This questionnaire has not been subjected to psychometric evaluation. No inter-rater agreement was carried out as to the items included in the DCC for this study. Therefore any conclusions drawn from the results of the DCC are severely limited. Neither reliability nor validity has been addressed regarding this scale. However as beliefs regarding dentistry are believed to have more explanatory power than a single measure of trait anxiety, it would be useful to subject this measure to psychometric evaluation and to apply it not just as a scoring measure (frequency) but also to evaluate the specific negative beliefs regarding dental care.

Due to low numbers inclusion criteria were relaxed. Therefore including participants from out-with the dental anxiety clinic may have led to a bias in the sample. Specifically, it can be argued that the use of opportunistic sampling may have introduced a respondent bias into the study. However only one participant from this

group identified intrusive memories and therefore it is likely that this did not have too great an effect on the final results. As the actual numbers of individuals with dental anxiety is unknown, it is also possible that participants included from the dental anxiety clinic are also a biased sample. As avoidance of dental care is known to be one of the main presenting behaviours involved in dental anxiety, then it is possible that the dental school sample represent a distinct group of dentally anxious individuals. Either those who are pre-contemplating involvement in the treatment process, or whose community dentists are unable to deliver appropriate management.

8.4 Study Limitations

In addition to the sample size and the methodological issues regarding study 2 a number of limitations have been identified.

Previous trauma is known to be a risk factor for reactions to current traumas. Although both groups were administered the Life Events checklist and no differences were found between the groups, there was no measure used for previous reactions to other traumas. It is unknown if participants in either group have trauma-related difficulties related to other non-dental experiences. This factor may have a consequence for any findings from this or any other similar study.

Briere (1998) has also reported underreporting of traumatic symptomatology. As only the participants who reported intrusive memories were assessed with trauma-related scales it is possible that some participants in the non-intrusive memory group

may have trauma-related difficulties but were not identified due to avoidance of reporting such symptoms.

De Silva and Marks (1999) argue that intrusive recollections can be thoughts or images or both, with the evidence that images are more common than thoughts. They point out that these two forms of intrusive recollections are often treated as one. They argue that accurate assessment is important because the nature of the cognitive experience may be important for treatment choice. The assessment use in this study did not differentiate between thoughts or images and used the term “memories”. This may have limited the responses for some participants and would not accurately differentiate the nature of the intrusions.

De Silva and Marks (1999) also argue that individuals may experience intrusive cognitions that are not recollections of the traumatic event itself but as a consequence of the trauma e.g. negative thoughts about the self, threat and danger and thoughts about the meaning of the event. These intrusive collections according to de Silva and Marks (1999) can be distressing for the individual. It is possible that these types of cognitions do operate in this group related to dental procedures, however this was not assessed.

8.5 Ethical Considerations

It is possible that participating in such a study may initiate discomfort and distress for the participants especially when being asked to recall distressing experiences. Parslow et al (2000) found that following an epidemiological survey of PTSD most

participants reported short-term distress but no evidence of long-term harm. All participants were fully briefed as to the rationale of the study (as per British Psychological Society Guidelines). Only one participant indicated that this was the first time that she had disclosed her past distressing experiences. She had not reported it in previous dental anxiety assessments. This participant was offered a referral to the clinical psychologist at the dental school, but she declined this offer.

8.6 Future Research

Despite the usefulness of the IES it is limited due to its exclusion of hyperarousal symptoms. Recently Weiss et al (1995) revised the IES, leading to a new scale IES-R that incorporates hyperarousal symptoms. The IES-R would be a useful instrument for future research in this area due to its inclusion of hyperarousal symptoms. If found to be a reliable measure of trauma-related symptoms in this population, it would be a useful and easy to administer measure, which would allow essential screening of this population.

It is presently unknown if these additional trauma-related difficulties will lead to obstacles in implementing therapy related to dental anxiety. It is known however that individuals with trauma-related symptoms can present with certain difficulties related to treatment. Richards and Lovell (1999) note that difficulty engaging with treatment is often found amongst individuals presenting with PTSD. Although only two studies have found evidence for trauma-related symptoms amongst dentally anxious individuals, future research should include treatment efficacy and outcome studies. At present there are no authoritative guidelines available to inform clinical decision

making in this area. The results of this study and that of de Jongh et al's suggest that the influence of trauma-related difficulties in this population require further elaboration.

At present it is not known what factors have contributed to some individuals presenting with trauma-related symptoms related to past distressing dental experiences. It would be useful to be able to predict which factors give rise to trauma-related symptoms amongst dentally anxious individuals. This would allow better utilisation of assessment and treatment methods. Both groups identified mainly painful past dental experiences as the precipitant leading to their dental anxiety. Pain however is a subjective process and experiences of pain, including reevaluation of the beliefs regarding pain are also subject to individual differences. Pain is also known to be affected by cognitive maturation (McGrath, 1995). It is not known if this would contribute to the age differences at distressing dental experience observed in this study or not. Assessing cognitions related to painful dental procedures may add to this debate.

It has been suggested in an earlier section that past traumatic experiences may have led to the group differences. Future research should address this issue to investigate if this has any influence on the development or maintenance of dental anxiety.

8.7 Conclusions

Dental anxiety has been implicated as a major barrier to optimal health care yet a lack of data regarding the psychology of dental anxiety has been identified in Scotland.

Direct conditioning experiences are believed to be the most helpful in explaining the acquisition of dental fears. However indirect pathways have also been investigated in the aetiology of the disorder. Rachman (1968) proposed that fears acquired by direct conditioning would lead to a greater strength of response than fears acquired indirectly. He also proposed (Rachman, 1977) that different modes of onset may provide data for appropriate treatment intervention. Results from study 1 although limited due to the nature of the data collection do not support Rachman's (1968) premise. Due to the methodological problems in study 1 any conclusions remain tentative, however the results appear to suggest that dental anxiety acquired via indirect pathways may lead to as great a response as those acquired directly. Therefore treatment interventions should focus on the individuals beliefs regarding their fears rather than primarily on route of fear acquisition.

Further to the lack of data regarding the dynamics of dental anxiety, this study has identified trauma-related symptomatology in individuals presenting with dental fears. This is the first study to identify the proportion of individuals with dental anxiety who would meet diagnosis for PTSD. The initial findings suggest that dentally anxious individuals do not suffer from PTSD but may present with intrusive memories of past dental experiences that still cause distress when exposed to dentally

related stimuli or reminders of their past distressing dental experiences. It is not presently known if the failure to meet diagnosis for PTSD amongst this population is due to the difficulty with PTSD diagnostic criteria or due to specific aspects of their trauma.

The results of this study will hopefully lead to further research and debate especially in the current climate of evidence-based practice. The findings of both this study and that of de Jongh et al's require to be replicated as the results suggest that specific management of individuals presenting with trauma-related symptoms may be required.

References

- Aartman, I. H. A., De Jongh, A., Makkes, P. C., & Hoogstraten, J. (1999). Treatment modalities in a dental fear clinic and the relation with general psychopathology and oral health variables. British Dental Journal, 186, 467-471.
- Aartman, I., H. A. (2000). Treating highly anxious dental patients in a dental fear clinic. Unpublished PhD thesis, Department of Social Dentistry and Dental Health Education of the Academic Centre for Dentistry, Amsterdam.
- Aitkin, R. C. B., Lister, J. A. & Main, C. J. (1981). Identification of features associated with flying phobias in aircrew, British Journal of Psychiatry, 139, 38-42.
- Alford, C. F. (1992). The Psychoanalytic Theory of Greek Tragedy, New Haven, CT: Yale University Press
- Alloy, L. B., & Tabachnik, N. (1984). Assessment of variation by humans and animals. The joint influence of prior expectations and current situational information. Psychological Review, 91, 112-149.
- Altman, D. G., & Bland, J. M (1995). The Normal Distribution, British Medical Journal, 310, 298
- APA (1984). Diagnostic and Statistical Manual of Mental Disorders (4th Edition), American Psychiatric Association Washington, DC.
- Armony, J. G. & LeDoux, J. E. (1997). How the brain processes emotional information. Annals of the New York Academy of Sciences, 821, 259-270.
- Arntz, A., Rauner, M., & van den Hout, M. A. (1995). "If I feel anxious, there must be danger": The fallacy of ex consequentia reasoning in inferring danger in anxiety disorders. Behaviour Research Therapy, 33, 917-925.
- Baddeley, A. (1997). Human Memory. Theory and Practice. Hove, UK.: Psychology Press,
- Bandura, A. (1969). The Principles of Behaviour Modification. New York: Holt, Rinehart & Winston,
- Bandura, A. (1977). Social learning Theory. New York: Prentice-Hall,
- Bandura, A. (1982). Self-efficacy mechanism in human agency. American Psychologist, 37, 122-147.
- Barlow, D.H. (1988). Anxiety and its disorders: The nature and treatment of anxiety and panic. New York: Guildford,

Barlow, D.H, Brown, T. A., & Craske, M. G. (1994). Definitions of panic attacks and panic disorder in the DSM-IV: Implications for research. Journal of Abnormal Psychology, 103, 553-564

Bauman, W., & Melnyk, W. T. (1994). A controlled comparison of eye movements and finger tapping in the treatment of test anxiety. Journal of Behaviour Therapy and Experimental Psychiatry, 25, 29-33.

Bechara, A., Tranel, D., Damasio, H., Adolphs, R., Rockland, C., & Damasio, A., R. (1995). Double Dissociation of Conditioning and Declarative Knowledge Relative to the Amygdala and Hippocampus in Humans. Science, 269, 1115-1118.

Beck, A.T (1976). Cognitive Therapy and the Emotional Disorders. New York: International Universities Press,

Beck, A. T., Emery, G. (1985). Anxiety disorders and phobias, New York: Basic Books,

Beck, A. T., Emery, G., & Greenberg, R. L. (1985). Anxiety Disorders and Phobias: A Cognitive Perspective. New York: Basic Books

Bennett, P., & Brooke, S. (1999). Intrusive memories, post-traumatic stress disorder and myocardial infarction. British Journal of Clinical Psychology, 38, 411-416.

Berggren, U., Carlsson, S. (1985). Usefulness of two psychometric scales in Swedish patients with severe dental anxiety. Community Dentistry and Oral Epidemiology, 13, 70-74.

Bernstein, D., Kleinknecht, R., Alexander, L. (1979). Antecedents of dental fear. Journal of Public Health Dentistry, 39, 113-124.

Berry, E. (1998). Post-traumatic stress disorder after subarachnoid haemorrhage. British Journal of Clinical Psychology, 37, 365-367.

Blank, A. S. (1993). The longitudinal course of posttraumatic stress disorder. In J. R. T. Davidson & E. B. Foa (eds) Posttraumatic Stress Disorder: DSM-IV and beyond (pp 3-22). Washington, DC: American Psychiatric Press,

Breaslau, N. Davis, G., Andreski, P. Peterson, E. (1991). Traumatic events and posttraumatic stress disorder in an urban population of young adults. Archives of General Psychiatry, 48, 216-222.

Breaslau, N., & Davis, G. C. (1992). Risk factors for PTSD related traumatic events: A prospective analysis. American Journal of Psychiatry, 152, 529-535.

Bremner, J. D., Randall, P., Scott, T. M., Bronen, R. A., Seibyl, J. P., Southwick, S. M., Delaney, R. C., McCarthy, G., Charnley, D. S., & Innis, R. B. (1995). MRI-based measures of hippocampal volume in patients with PTSD. American Journal of Psychiatry, 152, 973-981.

- Bremner, J. D., Vermetten, E., Southwick, S. M., Krystal, J. H., & Charney, D. S. (1998). In J.D. Bremner, & C. R. Marmar (eds) *Trauma, Memory, and Dissociation: An Integrative Formulation*. In Trauma, Memory and Dissociation, Washington DC: American Psychiatric Press,
- Breuer, J. & Freud, S. (1893). Studies on hysteria (1893-1895). In J Strachey (ed) The standard Edition of the Complete Psychological Works of Sigmund Freud, Vol 3, London: Hogarth Press
- Brewin, C. R., Dalgleish, T, & Joseph, S (1996). A dual representation theory of post traumatic stress disorder. Psychological Review, 103, 670-686.
- Brewin, C. R. (2001). 14 risk factors for post-traumatic stress disorder include childhood abuse and family psychiatric history. Evidence-Based Mental Health, 4, 61-62.
- Brewin, C. R (2001). A cognitive neuroscience account of posttraumatic stress disorder and its treatment. Behaviour Research and Therapy, 39, 373-393.
- Brewin, C. R., Andrews, B. & Gotlib, I. H. (1993). Psychopathology and early experience: A reappraisal of retrospective reports. Psychological Bulletin, 113, 82-98.
- Briere, J. (1998). Psychological Assessment of Adult Posttraumatic States. Washington, DC: American Psychological Association.
- Briere, J., & Conte, J. (1993). Self-reported amnesia for abuse in adults molested as children. Journal of Traumatic Stress, 6, 21-32.
- Briggs, S. R., & Cheek, J. M. (1986). The role of factor analysis in the development and evaluation of personality scales. Journal of Personality, 54, 106-148.
- British Psychological Society: Division of Clinical Psychology, Professional Practice Guidelines (1995).
- Brown G., & Harris, T. (1978). The Social Origins of Depression. London: Tavistok.
- Bryant, R. A., Harvey, A. G. (1996). Initial post-traumatic stress responses following motor vehicle accidents. Journal of Traumatic Stress, 9, 223-234.
- Bryant, R. A., Harvey, A. G, (1998). The relationship between acute stress disorder and posttraumatic stress disorder following motor vehicle accidents. Brain Injury, 13, 15-22.
- Bryant, R. A., Harvey, A. G. (2000). Acute Stress Disorder: A Handbook of Theory, Assessment, and Treatment. Washington, DC: American Psychological Association,

- Bryant, R. A., Harvey, A. G, Guthrie, R. M., & Moulds, M. L. (in press) A prospective study of psychophysiological arousal, acute stress disorder and posttraumatic stress disorder. Journal of Abnormal Psychology
- Bryman, A., & Cramer, D. (1995). Quantitative Data Analysis For Social Sciences. Revised Edition, London: Routledge.
- Chapman, H. R., Kirby-Turner, N. C. (1999). Dental Fear in Children-a proposed model. British Dental Journal, 187 (8), 1-7
- Chemtob, C. M, Tolin, D. F, van der Kolk, B., & Pitman, R. K. (2000). Eye Movement Desensitization and Reprocessing. Journal of Traumatic Stress, 13, 4, 539-589.
- Cohen, M.E. (1985). Dental anxiety and DMFS status: association with a US naval population versus differences between groups. Community Dentistry and Oral Epidemiology, 13, 75-78.
- Cohen, J. W. (1988). Statistical power analysis for the behavioural sciences (2nd edition). Hillsdale NJ: Lawrence Erlbaum Associates.
- Cohen, S.M., Fiske, J., Newton, J.T. (2000). Dental Phobics: how does dental anxiety affect their lives? British Dental Journal, 189, 385-390.
- Corah, N. L., Gale, E. N., & Illig, S. J. (1978). Assessment of a dental anxiety scale. Journal of the American Dental Association, 97, 816-819.
- Czarnocka, J., & Slade, P. (2000) Prevalence and predictors of post-traumatic stress symptoms following childbirth. British Journal of Clinical Psychology, 39, 35-51.
- Dalgleish, T. (1999). Cognitive Theories Of Post-Traumatic Stress Disorder, In W Yule, (ed) Post-Traumatic Stress Disorders: Concepts and Therapy. New York: John Wiley & Sons.
- Davey, G. C. L (1989). Dental phobias and anxieties: evidence for conditioning processes in the acquisition and modulation of a learned fear. Behaviour Research and Therapy, 27, 51-58
- Davey, G. C. L., McKenna, I. (1983). The effects of postconditioning reevaluation of CS1 and UCS following Pavlovian second-order electrodermal conditioning in humans. Quarterly Journal of Experimental Psychology, 35, 125-133.
- Davey, G. C. L. (1997). Phobias, A Handbook of Theory, Research and Treatment. New York: John Wiley & Sons.
- Davey, G. C. L. (1992). Classical conditioning and the acquisition of human fears and phobias: a review and synthesis of the literature. Advances in Behaviour Research & Therapy, 14, 29-66

Davey, G. C. L., & Dixon, A. (1996). The expectancy bias model of selective associations: the relationship of judgements of CS dangerousness, CS-UCS similarity and prior fear to a priori and a posteriori co-variation assessments. Behaviour Research and Therapy, 34, 235-252.

Davidson, J. R. T., Smith, R. D., & Kudler, H. S., (1989). Validity and reliability of the DSM-III criteria for posttraumatic stress disorder. Journal of Nervous Mental Disorders, 177, 336-341.

Davidson, J. R. T., & Fairbank, J. A., (1993). The Epidemiology of posttraumatic stress disorder. In J. R. T. Davidson & E. B. Foa (eds), Post-traumatic stress disorder: DSM-IV and beyond pp 147-169, Washington, DC: American Psychiatric Press.

Davidson, J. R. T., Malik M. A., & Travers, J. (1997). Structured Interview For PTSD (SIP): Psychometric Validation for DSM-IV Criteria. Depression and Anxiety, 5, 127-129.

DeMartino, R., Mollica, R. F & Wilk, V. (1995). Monoamine oxidase inhibitors in posttraumatic stress disorder. Journal of Nervous Mental Disorders, 183, 510-515.

De Jong, P.J., Merckelbach, H. & Arntz, A. (1995). Covariation bias in phobic women: The relationship between a priori expectancy, on-line expectancy, automatic responding, and a posteriori contingency judgement. Journal of Abnormal Psychology, 104, 55-62.

De Jongh, A, Muris, P., Ter Horst, G., Duyx, M. (1995a). Acquisition and maintenance of dental anxiety: the role of conditioning experience and cognitive factors. Behaviour Research and Therapy, 33, 205-210.

De Jongh, A., Muris, P., ter Zuuren, F., Schoenmakers, N., Makkes, P. (1995b). One-session cognitive treatment of dental phobia: preparing dental phobics for treatment by restructuring negative cognitions. Behaviour Research and Therapy, 33, 947-954.

De Jongh, A, Muris, P, Schoenmakers, N., & Ter Horst, G (1995c). Negative Cognitions of dental Phobics: Reliability and Validity of the Dental Cognitions Questionnaire, Behaviour Research and Therapy, 33, 507-515.

De Jongh, A., Bongaarts, G., Vermeule, I., Visser, K., De Vos, P., Makkes, P. (1998). Blood-injury-injection phobia and dental phobia. Behaviour Research and Therapy, 36, 971-982.

De Jongh, A, Aartman, I., & Bland, N. (2000) Trauma-related phenomena in individuals with dental anxiety. Manuscript submitted for publication.

Department of Health (2001). Treatment Choice in Psychological Therapies and Counselling, Evidence Based Clinical Practice Guideline. London

- Derogatis, L. R. (1993). BSI Brief Symptom Inventory. National Computer Systems, Inc Minneapolis.
- De Silva, P., & Marks, M. (1999). Intrusive Thinking in Post-Traumatic Stress Disorder. In W. Yule (ed). Post-Traumatic Stress Disorder: Concepts and Therapy. Chichester: John Wiley & Sons.
- Dickinson, A. (1980). Contemporary animal learning theory. Cambridge University Press, Cambridge.
- Dunmore, E., Clark, D. M., & Ehlers, A. (1997). Cognitive factors in persistent versus recovered posttraumatic stress disorder after physical or sexual assault: a pilot study. Behavioural and Cognitive Psychotherapy, 25, 147-159.
- Doerfler, L. A., Pbert, L., & DeCosimo, D. (1994). Symptoms of post-traumatic stress disorder following myocardial infarction and coronary artery bypass surgery. General Hospital Psychiatry, 16, 193-199.
- Dollar, J., & Miller, N. E. (1950). Personality & Psychotherapy: An analysis in terms of learning, thinking and culture. New York: Mc Graw Hill.
- Edelman, R.J. (1992). Anxiety. Theory, research and intervention in clinical and health psychology. Chicester: Wiley and Sons.
- Ehlers, A & Clark, D. M. (2000). A cognitive model of posttraumatic stress disorder. Behaviour Research and Therapy, 38, 319-345.
- Ellis, A (1962). Reason and Emotion in Psychotherapy. New York: Stuart.
- Emmelkamp, P. M. G. (1982). Phobic and Obsessive-Compulsive Disorders, new York: Plenum.
- Enneking, D., Milgrom, P., Weinstein, P., Getz., T. (1992). Treatment outcomes for specific subtypes of dental fear: Preliminary clinical findings. Special Care in Dentistry, Vol 12, 215-218.
- Eysenck, H. J. (1979). The conditioning model of neurosis. Behavioural and Brain Sciences, 2, 155-199.
- Eysenck, H. J (1987). Behaviour Therapy. In H.J. Eysenck & I Martin (eds). Theoretical foundations of behaviour therapy (pp3-34) New York: Plenum.
- Figley, C., (1978). Stress disorders among Vietnam veterans: Theory, research and treatment implications. New York: Brunner.
- Frank, E., & Stewart, B. D. (1983). Physical aggression: Treating the victim. In E. A. Bleckman (ed). Behaviour modification with women (pp 245-272). New York: Guilford Press.

- Friedman, M. J., Davidson, J. R. T., Mellman, A., & Southwick, S. M. (2000). Pharmacotherapy. Journal of Traumatic Stress, 13,4, 539-589.
- Foa, E.B., & Kozak., M. J. (1986). Emotional processing of fear: Exposure to corrective information. Psychological Bulletin, 99, 20-35.
- Foa, E. B., Rothbaum, B. O Riggs, D. S. & Murdock, G.B. (1991). Treatment of post-traumatic stress disorder in rape victims: Comparison between cognitive-behavioural procedures and counselling. Journal of Consulting and Clinical Psychology, 59, 715-723.
- Foa, E. B., Zinbarg, R & Rothbaum, B. O. (1992). Uncontrollability and unpredictability in posttraumatic stress disorder: An animal model. Psychological Bulletin, 112, 218-238.
- Foa, E. B., & Riggs, D. S. (1993). Posttraumatic stress disorder in rape victims. In J. Oldham, M. B. Riba., & A. Tasman (eds). American Psychiatric Press Review of Psychiatry. Vol. 12, pp, 273-303, Washington DC: American Psychiatric Press.
- Foa, E.B., Riggs, D. S., Dancu, C. V & Rothbaum, B. O. (1993). Reliability and validity of a brief instrument for assessing posttraumatic stress disorder. Journal of Traumatic Stress, 6, 459-473.
- Foa, E. B., Keane, T. M., & Freidman, M. J (2000). Guidelines for Treatment of PTSD. Journal of Traumatic Stress, Vol, 13, 4, 539-589.
- Forsyth, J & Eifert G.H. (1995). Systematic Alarms in Fear Conditioning: A Reappraisal of What Is Being Conditioned. Presentation at the Annual Conference of the Association for Advancement of Behaviour Therapy, Washington DC
- Forsyth, J, Eifert G.H & Thomson, R. N. (1996). Systematic alarms in fear conditioning II: An experimental methodology using 20% CO2 inhalation as a UCS. Behaviour Therapy, 27, 391-415.
- Forsyth, J., Daleiden, E. L., Chorpita, B. (2000). Response Primacy In Fear Conditioning Disentangling The Contributions of UCS VS UCR Intensity. The Psychological Record, 50, 17-33.
- Freeman, C., & Flitcroft, A. (2000) Traumatic Stress Handbook. Lothian Primary Care NHS Trust
- Freud, S. (1905). Three essays on the theory of sexuality, in J. Strachey, (ed). The Standard Edition of the Complete Psychological Works of Sigmund Freud, (1953) Vol 7. pp 125-243. London: Hogart Press
- Freud, S. (1973), The Dissection of the Psychical Personality, in J.Strachey & A. Richards (eds), New Introductory Lectures on Psychoanalysis, Penguin Books, London.

Friedman, M. J. (2000). Post Traumatic Stress Disorder: The Latest Assessment and Treatment Strategies. , Kansas City: Compact Clinicals

Fromm, E. (1965). Hypoanalysis: theory and two case excerpts. *Psychotherapy: theory, Research and Practice*, 127-133.

Hakeberg, M., Berggren, U., & Carlsson, S. G. (1990). A 10-year follow-up of patients treated for dental fear. *Scandinavian Journal of Dental Research*, 98, 53-59.

Hallam, R. S. & Rachman, S. (1976). Current status of aversion therapy. In M. Hersen, M. Eisler, M. & P.M. Miller, (eds). Progress in behaviour modification (vol 2), New York: Academic Press.

Helzer, J. K, Robins, L. N., & McEvoy. (1987). Post-traumatic stress disorder in the general population: Findings of the Epidemiological Catchment Area survey. *New England Journal of Medicine*, 230, 273-279.

Hensman, R, Guimaraes, F. S, Wang, M. Deakin, J.F.W. (1991). Effects of ritanserin on aversive classical conditioning in humans. *Psychopharmacology*, 104, 220-224.

Holen, A, (1993). The North sea oil rig disaster, In J. P. Wilson & B. Raphael (eds), International handbook of traumatic stress syndromes, pp 471-478. New York: Plenum.

Horowitz, M. J. (1973). Phase-orientated treatment of stress response syndromes. *American Journal of Psychotherapy*, 27, 506-515.

Horowitz, M. J., Wilner, N., & Alvarez, W. (1979). The Impact of Event Scale: A measure of subjective stress. *Psychosomatic Medicine*, 41, 209-218.

Horowitz, M. J. (1986). Stress Response Syndromes. New Jersey: Aronson.

Humphris, G.M., Morrison, T., & Lindsay, S.J. E. (1995). The Modified Dental Anxiety Scale: validation and United Kingdom norms. *Community Dental Health*, 12, 143-150.

Humphris, G.M, Freeman, R., Campbell, J., Tuutti, H., & D'souza. S. (2000). Further evidence for the reliability and validity of the Modified Dental Anxiety Scale. *International Dental Journal*, 50, 367-370.

Hyer, L. & Brandsma, J. M. (1997). EMDR minus eye movements equals good psychotherapy. *Journal of Traumatic Stress*, 10, 515-522.

Janet, P (1889). l'Automatisme Psychologique, Nouvelle edition. Paris, La Societe Pierre Janet

Janoff-Bulman, R. (1985). The aftermath of victimisation: Rebuilding shattered assumptions . In C. R Figley (ed), Trauma and its wake: The Study and Treatment of Post-Traumatic Stress Disorder. New York: Brunner/Mazel

Janoff-Bulman, R. (1992). Shattered Assumptions: Towards a New Psychology of Trauma, New York: The Free Press.

Jaycox, L. H & Foa, E. B. (1996). Obstacles in implementing exposure therapy for PTSD: Case discussions and practical solutions. Clinical Psychology and Psychotherapy, 3, 176-184.

Jeavons, S., Greenwood, K. M., & de L. Horne, D. (2000). Accident Cognitions and Subsequent Psychological Trauma. Journal of Traumatic Stress, 13, 359-367.

Johansson, P., & Berggren, U. (1992). Assessment of dental fear. A comparison of two psychometric instruments. Acta Odontol Scandinavia, 50, 43-49.

Joseph, S., Yule, W., Williams, R., & Hodgkinson, P. (1993). The Herald of Free Enterprise disaster: Measuring post-traumatic symptom thirty months on. British Journal of Clinical Psychology, 32, 327-332.

Kaakko, T., Coldwell, S. E., Getz, T., Milgrom, P., Roy-Byrne, P., Ramsay, D. S. (2000). Psychiatric diagnoses among self-referred dental injection phobics. Journal of Anxiety Disorders, 14, (3), 299-312.

Kardiner, A. (1941). The Traumatic Neuroses of War. New York: Hoeber.

Kaufman, E., Jastak, J. T. (1995). Sedation for outpatient dental procedures. Compendium, 16, 462-478.

Keane, T. M., Zimering, R. T., & Caddell, J. M. (1985). A behavioural formulation of posttraumatic stress disorder in the National Comorbidity Survey. The Behaviour Therapist, 8, 9-12.

Kent, G. (1987). Self-efficacious control over reported physiological, cognitive, and behavioural symptoms of dental anxiety. Behaviour Research and Therapy, 25, 341-347.

Kent, G., Rubin, G., Getz, T., & Humphries, G. (1996). The development of a scale to measure the social and psychological effects of severe anxiety: Social attributes of the Dental Anxiety Scale. Community Dentistry and Oral Epidemiology, 24, 394-397.

Kent, G. (1998). Dental Health. Dental Health, Comprehensive Clinical Psychology <http://www.ccp-online> 02/04/01

Kessler, R. C., Sonnega, A., Bromet, E., Hughes, M & Nelson, C. B. (1995). Posttraumatic stress disorder in the national comorbidity survey. Archives of General Psychiatry, 52, 1048-1060.

- Kleber, R. J., Brom, D., & Defares, P. B. (1992). Coping with Trauma, Theory, prevention and treatment. Lisse: Swets & Zeitlinger.
- Kleinknecht, R., Klepec, R & Alexander, L. (1973). Origins and characteristics of fear of dentistry. Journal of the American Dental Association, 86, 842-848.
- Kluver, H, & Bucy, P., C (1937). Preliminary analysis of functions of the temporal lobes in monkeys. Archives of Neurological Psychiatry, 42, 977-1000
- Kolb, L. C., & Multipassi., L. R. (1982). The conditioned emotional response: A subclass of chronic and delayed post-traumatic stress disorder. Psychiatric Annals, 12, 979-987.
- Kolb, L. C. (1987). Neurophysiological hypothesis explaining posttraumatic stress disorder. American Journal of Psychiatry. 144, 989-995.
- Koopman, C., Classen, C., & Spiegel, D. (1994). Predictors of posttraumatic stress symptoms among survivors of the Oakland/Berkeley, California, firestorm, American Journal of Psychiatry, 151, 888-894.
- Kosten, T. R., Mason, J. W., Giller, E. L., Ostroff, R. B., & Harkness, L (1987). Sustained urinary norepinephrine and epinephrine elevation in PTSD. Psychoneuroendocrinology, 12, 13-20.
- Kutz, I., Shabatai, H., Solomon, Z., Neumann, M., & David, D. (1994). Post-traumatic stress disorder in myocardial infarction patients: Prevalence study. Israeli Journal of Psychiatry and Related Sciences, 31, 48-56.
- Lang, P. J. (1977). Fear imagery: an information processing analysis. Behaviour Therapy, 8, 862-886.
- Lang, P. J (1979). A bio-informational theory of emotional imagery. Psychophysiology, 16, 495-512.
- Lautch, H. (1971). Dental phobia. British Journal of Psychiatry, 119, 151-158
- Lavy, E. H, van den Hout, M. A., & Arntz, A. (1993). Attentional bias and spider phobia. Conceptual and clinical issues. Behaviour Research and Therapy, 31, 17-24.
- LeDoux, J.E., Romanski, L., & Xagoraris, A., (1991). Indelibility of subcortical emotional memories. Journal of Cognitive Neuroscience, 1, 238-243.
- LeDoux, J.E. (1995). In search of an emotional system in the brain: leaping from fear to emotion and consciousness, in M. Gazzinga, (ed) The Cognitive Neurosciences, pp 1049-1061 Cambridge: MIT Press.
- Liddell, A., Locker, D. (1993). Dental anxiety in the elderly. Psychology and Health, 8, 175-183.

- Liddell, A. Gosse, V. (1998). Characteristics of early unpleasant dental experiences. Journal of Behaviour Therapy and Experimental Psychiatry, 29, 227-237.
- Lindsay, S., Jackson, C. (1993). Fear of routine dental treatment in adults: its nature and management. Psychology and Health, 8, 135-153.
- Litt, M. D., Nye, C., & Shafer, D. (1993). Coping with oral surgery by self-efficacy enhancement and perceptions of control. Journal of Dental Research, 72, 1237-1243.
- Locker, D., Shapiro, D., Liddell, A. (1996). Negative dental experiences and their relationship to dental anxiety. Community Dental Health, 13, 86-92.
- Locker, D., Shapiro, D., Liddell, A. (1997). Overlap between dental anxiety and blood-injury fears: Psychological characteristics and response to dental treatment. Behaviour Research and Therapy, 35, 583-590.
- Locker, D., Liddell, A., Dempster, L., & Shapiro, D. (1999). Age of onset of dental anxiety. Journal of Dental Research, 78 (3), 790-796.
- Locker, D., Liddell, A., Dempster, L., Shapiro, D. (1999). Age of Onset of Dental Anxiety. Journal of Dental Research, 78, (3), 790-796.
- Macleod, C., & Hagan, R. (1992). Individual differences in the selective processing of threatening information and emotional responses to a stressful life event. Behaviour Research and Therapy, 30, 151-161.
- Malloy, P., & Lewis, D. J. (1988). A laboratory demonstration of persistent human avoidance. Behaviour Therapy, 19, 229-241.
- Maren, S & Fanselow, M. S. (1996). The Amygdala and Fear Conditioning: Has the Nut Been Cracked. Neuron, Vol 16, 237-240
- Marks, I. M. (1987). Fears, phobias and rituals: Panic, anxiety and their disorders. New York: Oxford University Press.
- Marks, I. M., & Nesse, R. M. (1994). Fear and fitness: an evolutionary analysis of anxiety disorders. Ethology and Sociobiology, 15, 247-261.
- Marks, I. M., Lovell, K., Norshirvanie., H Livanou, M., & Thrasher., S. (1998). Treatment of posttraumatic stress disorder by exposure and /or cognitive restructuring: A controlled study. Archives of General Psychiatry, 55, 317-325.
- Marmar, C. R., Weiss, D. S., & Schlenger, W. E. (1994). Peritraumatic Dissociation and posttraumatic stress in male Vietnam theatre veterans. American Journal of Psychiatry, 151, 902-907.
- Masson, J. (1984) The assault on truth. New York: Farrar, Straus & Giroux,

- Mason, J. W., Giller, E. L. & Kosten, T. R. (1986). Urinary-free cortisol levels in post-traumatic stress disorder patients. Journal of Nervous and Mental Disease, 174, 145-159.
- Mayou, R. A & Smith, K. A. (1997). Post-traumatic symptoms following medical illness and treatment. Journal of Psychosomatic Research, 43, 121-123.
- Mazey, K. A., Mito, R. S. (1993). Multidisciplinary Treatment of Dental Phobia. Journal of the Californian Dental Association, 21 (3): 17-25.
- Menzies, R. G. & Clark, J. C. (1995). The Etiology of Phobias: A Nonassociative Account. Clinical Psychology Review, Vol 15, 23-48.
- Merckelbach, H., Ruiters, C. D., van den Hout, M. A., & Hoekstra, R. (1989). Conditioning experiences and phobias. Behaviour Research and Therapy, 6, 657-662
- Merckelbach, H., de Jong, P. J., Muris, P., & van den Hout, M. A. (1996). The Etiology of Specific Phobias: A Review. Clinical Psychology Review, 16, 337-361.
- Merckelbach, H. & de Jong, P. (1997). Evolutionary Models of Phobias. In G. C., L. Davey, (ed) Phobias a Handbook of Theory, Research and Treatment, New York: John Wiley & Sons.
- Metcalf, J., & Jacobs, W. J. (1998). Emotional memory: The effects of stress on "cool" and "hot" memory systems. In G. H. Bower (ed). The Psychology of learning and memory (Vol, 38) (pp 187-222). New York: Academic Press.
- Milgrom, P, Mancl, L., King, B & Weinstein, P. (1995). Origins of childhood dental fear. Behaviour Research and Therapy, 33, 313-319.
- Mc Farlane, A., & Papay, P. (1992). Multiple diagnoses in posttraumatic stress disorder in the victims of a natural disaster. Journal of Nervous and Mental Disease, 179 (5), 253-258
- Mc Farlane, A., Atchinson, M., Rafalowicz, E & Papay. P. (1994). Physical symptoms In posttraumatic stress disorder. Journal of Psychosomatic Research, 38, 715-726.
- Mc Farlane, A. C & de Girolamo, G. (1996). The Nature of Traumatic Stressors and the Epidemiology of Posttraumatic Reactions. Pp 129-154. In B., O. Van der Kolk, A. C. McFarlane, & L Weisaeth (eds) Traumatic Stress, The Effects of Overwhelming Experience on Mind, Body and Society, pp 303-327, New York: Guildford Press.

McFarlane, A. C., Atchinson, M., & Yehuda, R. (1997). The acute stress response following motor vehicle accidents and its relation to PTSD. In R. Yehuda, & A. C. McFarlane, (eds). Psychobiology of posttraumatic stress disorder (pp 433-436), New York: New York Academy of Sciences.

McGoldrick, P., & Durham, R. (2000). A systematic (Cochrane) review of psychotherapy-based treatments for dental anxiety.

McGoldrick, P., Levitt, J., De Jongh, A, Mason, A., & Evans, D. (manuscript submitted for publication). Referrals to a Secondary Care Dental Clinic for Anxious Adult Patients: Implications for Treatment.

McGrath, P. (1995). Aspects of pain in children and adolescents. Journal of Child Psychology and Psychiatry, 36, 717-731.

McMillen, J., C., North, C. S, & Smith, E. M. (2000). What Parts of PTSD Are Normal: Intrusion, Avoidance, or Arousal? Data from the Northridge, California, Earthquake. Journal of Traumatic Stress, 13, 57-73.

McNally, R. J., and Steketee, G. S (1985). The etiology and maintenance of severe animal phobias. Behaviour Research and Therapy, 23, 431-435.

McNally, R. J. & Foa, E. B. (1986) Preparedness and resistance to extinction to fear-relevant stimuli: A failure to replicate. Behaviour Research and Therapy, 24, 529-535.

McNally, R. J and Lukach, B. M. (1992). Are panic attacks traumatic stressors? American Journal of Psychiatry, 149, 431-435.

McNeil, D, W., Berryman, M.L. (1989). Components of dental fear in adults. Behaviour Research and Therapy, 27, 233-236.

Milgrom, P., Vignehsa, H., Weinstein, P. (1992). Adolescent dental fear and control: prevalence and theoretical implications. Behaviour, Research and Therapy, 29, 51-60.

Milgrom, P., Weinstein, P., Kleinknecht, R., Getz., T. (1995). Treating fearful dental patients: A patient management handbook. Seattle: University of Washington.

Minelka, S, & Cook, M. (1993). Mechanisms Involved in the Observational Conditioning of Fear. Journal of Experimental Psychology, 122, 23-38.

Moore, R. (1991). Dental fear treatment: comparison of a video training procedure and clinical rehearsals. Scandinavian Journal of Dental Research, 99, 229-235.

Moore, R., Brødsgaard, I., Birn, H. (1991). Manifestations, Acquisitions, and Diagnostic Categories of Dental Fear in a Self-Referred population. Behaviour Research and Therapy, 29, (1), 51-60.

Motulsky, H. (1995). Intuitive Biostatistics, New York, Oxford University Press.

Mowrer, O. H. (1939). A stimulus-response analysis of anxiety and its role as a reinforcing agent. Psychological Review, 46, 553-565.

Muris, P., De Jongh, A., Merckelbach, H., Postema, S., & Vet, M. (1998). Thought suppression in phobic and non-phobic dental patients. Anxiety, Stress & Coping, 11, 275-287.

Nemiah, J. (1989). Janet redivivus: the centenary of l' Automatismes Psychologique. American Journal of Psychiatry, 146, 1527-1529.

Nemiah, J. C. (1997). Early Concepts of Trauma, Dissociation, and the Unconscious: Their History and Current Implications, pp 1-26, In J.D Bremner & C.R Marmar, (eds) Trauma, Memory and Dissociation, Washington DC American Psychiatric Press.

Niehoff, D. L. & Kuhar, M. J. (1983). Benzodiazepine receptors: localization in rat amygdala. Journal of Neuroscience, 3, 2091-2097.

North, C. S., Smith, E. M., & Spitznagel, E. L. (1994). Posttraumatic stress disorder in survivors of a mass shooting. American Journal of Psychiatry, 15, 82-88.

Nuttall, N.M., Steele, J.G., Pine, C.M., White, D., Pitts, N. B. (2001). The impact of oral health on peoples in the UK in 1998. British Dental Journal, 190, (3), 121-126.

Ohman, A., Fredricson, M., Hugdahl, K., & Rimmo., P. (1976). The premise of equipotentiality in human classical stimuli for human autonomic responses. Journal of Abnormal Psychology, 84, 41-45.

Ollendick, T. H. & King, N. J. (1991). Origins of Childhood Fears: An Evaluation Of Rachman's Theory of Fear Acquisition. Behaviour Research and Therapy, 29, 117-123.

Ost, L. G. & Hugdahl, K. (1981). Acquisition of phobias and anxiety response patterns in clinical patients. Behaviour Therapy and Research, 23, 683-689.

Ost, L. G. & Hugdahl, K. (1985). Acquisition of Blood and Dental Phobia and Anxiety Response Patterns in Clinical Patients. Behaviour Research and Therapy, 23, 27-34

Ost, L. G. (1987). Age of onset of different phobias. Journal of Abnormal Psychology, 29, 223-229

- Owen, R. L., Koutsakis, S. & Bennett, P. D. (2001). Post-traumatic stress disorder as a sequel of acute myocardial infarction: an overlooked cause of psychosocial disability. Coronary Health Care, Vol 5, 9-15.
- Pallant, J. (2001). SPSS Survival Manual: A step by step guide to data analysis using SPSS. Buckingham: Open University Press.
- Parslow, R. A., Jorm, A. F., O'Toole, B. I., Marshall, R. P., & Grayson, D. A. (2000). Distress Experienced by Participants During an Epidemiological Survey of Posttraumatic Stress Disorder. Journal of Traumatic Stress, 13, 3, 465-472.
- Pavlov, I.P (1927). Conditioned reflexes. Oxford University Press, London.
- Penfield, W & Perot, P. (1963). The brain's record of auditory and visual experience: a final summary and discussion. Brain, 86, 595-696.
- Perry, B. D. (1994). Neurobiological sequelae of childhood trauma: post-traumatic stress disorder in children. In M. Murbey (ed). Catecholamines in PTSD: Emerging Concepts (pp 253-276). Washington DC American Psychiatric Press.
- Pitman, R., Orr. S. P., & Shalev, A. (1993). Once bitten, twice shy: Beyond the conditioning model of PTSD. Biological Psychiatry, 33, 145-146.
- Pitman, R., Shalev, A & Orr., S. P (2000). Posttraumatic stress disorder: Emotion, conditioning and memory. In M. S. Gazzaniga. The New Cognitive Neurosciences (pp 1133-1147), Cambridge, Mass: MIT Press
- Power, M. J & Dalgleish, T. (1997). Cognition and Emotion. From Order to Disorder. Hove UK: Psychology Press.
- Pribor, E. F., Yutzy, S. H., Dean, T. & Wetzel, R. D. (1993). Briquet's syndrome, dissociation and abuse. American Journal of Psychiatry, 150, 1507-1511.
- Rachman, S (1968) Phobias. Their nature and control. Springfield: Thomas.
- Rachman, S (1976). The passing of two-stage theory of fear and avoidance: fresh possibilities. Behaviour Research and Therapy, 14, 125-134.
- Rachman, S. (1977). The conditioning theory of fear acquisition: A critical examination. Behaviour Research and Therapy, 15, 375-387.
- Rachman, S (1990). Fear and Courage, second edition, New York: Freeman.
- Rachman, S. (1998). Anxiety. Clinical Psychology A Modular Course. Hove UK Psychology Press.

Rauch, S. L., van der Kolk, B. A., Fisler, R. E., Alpert, N. M., Orr, S. P., Savage, C. R., Fischman, A. J., Jenike, M. A., & Pitman, R. K. (1996). A symptom provocation study of post-traumatic stress disorder using Positron Emission Tomography and script-driven imagery. Archives of General Psychiatry, *53*, 380-387.

Rescorla, R. A. (1971). Variations in the effectiveness of reinforcement and nonreinforcement following prior inhibiting conditioning. Learning and Motivation, *2*, 113-123.

Rescorla, R. A. (1988). Pavlovian conditioning: It's not what you think it is. American Psychologist, *43*, 562-563.

Resick, P. A., & Schnicke, M. K. (1993). Cognitive processing therapy for rape victims: A treatment manual. London: Sage.

Rice, A. Liddell, A. (1998). Determinants of positive and negative attitudes towards dentistry. Journal of the Canadian Dental Association, *64*, 213-218.

Richards, D. A., Lovell, K. & Marks, I. M. (1994). Post-Traumatic Stress disorder: Evaluation of a Behavioural treatment program. Journal of Traumatic Stress, *7*, 669-680.

Richards, D. A., Lovell, K. (1999). Behavioural and Cognitive Behavioural Interventions In W. Yule, (ed) The Treatment of PTSD. In Adults, pp 239-266 in Post-Traumatic Stress Disorders: Concepts and Therapy. New York: John Wiley & Sons.

Roediger, H. L. (1990). Implicit memory: retention without remembering. American Psychologist, *45*, 1043-1056

Ross, R. J., Ball, W. & Cohen. M. E. (1989). Habituation of the startle response in post traumatic stress disorder. Journal of Neuropsychiatry, *1*, 305-307.

Rothbaum, B. O., & Foa, E. B. (1993). Subtypes of post-traumatic stress disorder and duration of symptoms. In J. R. T. Davidson & E. B. Foa (eds), Post-traumatic stress disorder: DSM-IV and beyond pp23-35, Washington, DC, American Psychiatric Press.

Rothbaum, B. O. (1997). A controlled study of eye movement desensitization and reprocessing in the treatment of posttraumatic stress disorder sexual assault victims. Bulletin of the Menninger Clinic, *61*, 1-18.

Rothbaum, B. O., Meadows, E. A., Resick, P. & Foy, D. W. (2000). Cognitive-Behavioural Therapy: Guidelines for Treatment of PTSD. Journal of Traumatic Stress, *Vol 13*, *4*, 539-589.

- Roy-Byrne, P., Milgrom, P., Khoon-Mei, K. M., Weinstein, P., Katon, W. (1994). Psychopathology and psychiatric diagnosis in subjects with dental phobia. Journal of Anxiety Disorders, 8, 19-31.
- Russell, C. & Davey, G. C. L. (1991). The effects of false response feedback on human "fear" conditioning. Behaviour Research and Therapy, 29, 191-196.
- Sanderson, R., Laverty, S., Campbell, D. (1963). Traumatically conditioned responses acquired during respiratory paralysis. Nature, 196, 1235-1236.
- Scaer, R. C. (2001). The Neurophysiology of Dissociation and chronic disease. Applied Psychophysiology and Biofeedback, 26 (1), 73-91.
- Schacter, D. L., Norman, K. A., & Koutstaal, W. (1997). The recovered memories debate: A cognitive neuroscience perspective. Oxford: Oxford University Press.
- Schreiber, S., & Galai-Gat, T. (1993). Uncontrollable pain following physical injury as the core-trauma in post-traumatic stress disorder. Pain, 54, 107-110.
- Schuurs, A. H. B., & Hoogstraten, J. (1993). Appraisal of dental anxiety and fear questionnaires: a review. Community Dentistry and Oral Epidemiology, 21, 329-339.
- Scott, M (2000). Assessment and Conceptualization. In M. J. Scott, & S. Palmer, (eds) Trauma and Post-traumatic Stress Disorder, pp 15-33, London: Cassell.
- Seligman, M. E. P. (1971). Phobias and preparedness. Behaviour Therapy, 2, 307-320.
- Seligman, M. E. P. & Hager J. (1972). Biological boundaries of learning. New York: Appleton Century Croft.
- Sembi, S., TARRIER, N., O'Neill, P., Burns, A., & Faragher, B (1998). Does Post-traumatic Stress Disorder Occur After Stroke: A Preliminary Study. International Journal of Geriatric Psychiatry, Vol 13, 315-322.
- Shalev, A. Y. (1992) Posttraumatic stress disorder among injured survivors of a terrorist attack: Predictive value of early intrusion and avoidance symptoms. Journal of Nervous and Mental Disease, 180, 505-509.
- Shalev, A. Y, Orr, S. P., Peri, T, Schreiber, S., & Pitman, R. K. (1992). Physiologic responses to loud noise tones in Israeli patients with post-traumatic stress disorder. Archives of General Psychiatry, 49, 870-875.
- Shalev, A. Y., Schrieber, S., Galai, T., & Melmed, R. N. (1993). Post-traumatic stress disorder following medical events. British Journal of Clinical Psychology, 32, 247-253.

Shalev, A. Y., Peri, T., Caneti, L., & Schreiber, S. (1996) predictors of PTSD in injured trauma survivors: A prospective study. American Journal of Psychiatry, 153, 219-225.

Shalev, A. Y., Sahar, T., Freedman, S., Peri, T., Glick, C., Brandes, D., Orr, S. P & Pitman, R. K. (1998). A Prospective Study of Heart Rate Response Following Trauma and the Subsequent Development of Posttraumatic Stress Disorder. Archives of General Psychiatry, Vol, 55, 553-559.

Shalev, A. Y. (2001) Commentary on Brewin, Evidence-Based Practice, 4, 62.

Shapiro, F. (1995). Eye movement desensitization and reprocessing: Basic principles, protocols and procedures. New York: Guilford Press.

Smith, E. M., North, C. S., McCool, R. E., & Shea, J.M. (1990). Acute postdisaster psychiatric disorders: Identification of persons at risk. American Journal of Psychiatry, 147, 202-206.

Southwick, S. M., Krystal, J. H., Morgan, C. A., Johnson, D., Nagy, B., Nicolaou, A., Henniger, G. R., & Charney, D. (1993). Abnormal noradrenergic function in post traumatic stress disorder. Archives of General Psychiatry, 50, 266-274.

Southwick, S. M., Yehuda, R & Giller, E. L. (1994). Use of tricyclics and monoamine oxidase inhibitors in the treatment of PTSD: A quantitative review. In M.M. Murburg, (ed).(pp 293-305). Catecholamine function in post-traumatic stress disorder Emerging concepts. Washington, DC: American Psychiatric Press.

Spiers, R. L., & Barsby, M. J. (1995). Hyperventilation in the Dental Chair. Dental Update, April, 95-98.

Stein, M. B., Walker, J. R., Hazen, A. L., & Forde, D. E. (1997). Full and partial post-traumatic stress disorder: findings from a community survey. American Journal of Psychiatry, 154, 1114-1119.

Stevenson, J., Batten, N., & Cherner, M. (1992). Fears and fearfulness in children and adolescents: a genetic analysis of twin data. Journal of Child Psychology and Psychiatry, 33, 977-985.

Sturgis, E. L., & Scott, R. (1984). Simple Phobia. In S.M. Turner, (ed) Behaviour theories and treatment of anxiety. New York: Plenum Press.

Stouthard, M., Hoogstraten, J (1987). Ratings of fears associated with twelve dental situations. Journal of Dental Research, 66, 1175-1178.

Stouthard, M., Hoogstraten, J. (1990). Prevalence of dental anxiety in the Netherlands. Community Dentistry and Oral Epidemiology, 18, 139-142.

Tarrier, N & Humphreys, L. (2000). Subjective Improvement in PTSD patients with treatment by imaginal exposure or cognitive therapy: Session by session changes. British Journal of Clinical Psychology, Vol 39, 27-35.

Ter Horst, G., De Wit, C. (1993). Review of behavioural research in dentistry, 1987-1992: dental anxiety, dentist-patient relationship, compliance and dental attendance. International Dental Journal, 43, 265-278.

Terr, L., C. (1991). Childhood Traumas: An Outline and Overview. American Journal of Psychiatry, 148, 10-20

Thrash, W., Marr, J., Box, T. (1982). Effects of continuous patient information in the dental environment. Journal of Dental Research, 61, 1063-1065

Thorpe, S. J. & Salkovskis, P. M. (1995). Phobic beliefs: Do cognitive factors play a role in specific phobias?. Behaviour Research and Therapy, 33, 805-816.

Tomarken, A. J., Mineka, S., & Cook. (1989). Fear-relevant selective associations and covariation bias. Journal of Abnormal Psychology, 98, 381-394.

Tomarken, A. J., Sutton, S. K., & Mineka, S. (1995). Fear-relevant illusionary correlations: What types of associations promote judgmental bias?. Journal of Abnormal Psychology, 104, 312-326.

Townend, E., Dimigen, G., & Fung, D. (2000). A clinical study of child dental anxiety. Behaviour Research and Therapy, 38, 31-46

True, W. R., Rice, J. Eisen, S. A., Heath, A. C., Goldberg, J., Lyons, M. J., & Nowak, J. (1993). A twin study of genetic and environmental contributions to liability for posttraumatic stress symptoms. Archives of General Psychiatry, 50, 257-264.

Van der Hart, O. V, Brown, P & van der Hart, B. A. (1989). Pierre Janet's treatment of posttraumatic stress.. Journal of Traumatic Stress, 5, 175-194.

Van der Hart, O. V., & Spiegel, D. (1993). Hypnotic assessment and treatment of trauma-induced psychosis: The early psychotherapy of H. Breukink and modern views. International Journal of Clinical and Experimental Hypnosis, 41, 191-209.

Van der Hart, O. V., Van der Kolk, B. A, Boon, S. (1996). Treatment of Dissociative Disorders, pp 253-283 in J.D. Bremner & C.R Marmar, (eds) Trauma, Memory and Dissociation, Washington DC: American Psychiatric Press.

Van der Kolk, B. A., & van der Hart, O. V. (1989). Pierre Janet and the Breakdown of Adaptation in Psychological Trauma. American Journal of Psychiatry, 146, 1530-1541.

Van der Kolk, B. A. (1994). The body keeps the score: Memory and the evolving psychobiology of PTSD. Harvard Review of Psychiatry, 1, 253-265.

Van der Kolk, B. A., & Fisler, R. (1995). Dissociation and the fragmentary nature of traumatic memories. Journal of Traumatic Stress, 8, 505-525.

Van der Kolk, B. A. (1996). Approaches to the Psychobiology of Posttraumatic Stress Disorder. in Psychiatry, in Traumatic Stress, in B. A. Van der Kolk, A. McFarlane, L. Weisaeth (eds) The Effects of Overwhelming Experience on Mind, Body and Society, pp 303-327, New York: Guildford Press.

Van der Kolk, B. A., McFarlane, A.C., & Van Der Hart, O. (1996). A general approach to Treatment of Posttraumatic stress disorder. in Traumatic Stress, in B. A. Van der Kolk, A. C. McFarlane, L. Weisaeth (eds) The Effects of Overwhelming Experience on Mind, Body and Society, pp 417-440, New York: Guildford Press.

Van der Kolk, B. A., & van der Hart, O V., & Marmar C. R. (1996). Dissociation and Information Processing in Posttraumatic Stress Disorder, in Psychiatry, in B. A. Van der Kolk, A. C. McFarlane, L. Weisaeth (eds). Traumatic Stress, in The Effects of Overwhelming Experience on Mind, Body and Society, pp 303-327, New York: Guildford Press

Van der Kolk, B. A., Weisaeth, L., & van der Hart, O. V. (1996). History of Trauma in Psychiatry, in Traumatic Stress, in B. A. Van der Kolk, A. C. McFarlane, L. Weisaeth (eds). The Effects of Overwhelming Experience on Mind, Body and Society, pp 47-77 New York: Guildford Press

Vassend, O. (1993). Anxiety, pain and discomfort associated with dental treatment. Behaviour Research and Therapy, 31, 659-666.

Walker, A, Cooper, I. (1998). Adult Dental Health Survey: Oral Health in the UK 1998. London Stationary Office

Wardle, J. (1983). Psychological management of anxiety and pain during dental treatment. Journal of Psychosomatic Research, 27, 399-402.

Watson, J.B., Raynor, R. (1920). Conditioned emotional reactions. Journal of Experimental Psychology, 3, 1-14.

Wegner, D. M. (1989). White bears and other unwanted thoughts: suppression, obsession and the psychology of mental control. New York: Viking.

Weiner, A. A. Sheehan, D. V (1990). Etiology of dental anxiety: Psychological trauma or CNS imbalance? General Dentistry, 38, 39-43.

Weiner, A. A., Forgione, A., Weiner, L. K., Hwang, J. (2000). Potential fear-provoking patient experiences during treatment. General Dentistry, 48, 466-471.

Weiss, D. S., Marmar, C. R., Metzler, T., & Ronfeldt, H. (1995). Predicting symptomatic distress in emergency services personnel. Journal of Consulting and Clinical Psychology, 63, 361-368.

Wilson, S. A., Becker, L. A., & Tinker, R. H. (1997). Fifteen-month follow-up of eye movement desensitization and reprocessing (EMDR) treatment of post-traumatic stress disorder and psychological trauma. Journal of Consulting and Clinical Psychology, 65, 1047-1056.

Wells, A. (1998) Cognitive Therapy of Anxiety Disorders: A Practice Manual and Conceptual Guide, New York: John Wiley & Sons.

Withers, R. D. & Deane, F. P. (1995). Origins of Common Fears: Effects of Severity, Anxiety Responses and Memories of Onset. Behaviour Therapy and Research, 33, 903-915.

Wolpe, J. (1958). Psychotherapy by reciprocal inhibition. Stanford: Stanford University Press.

Wong, M. R., & Cook, D. (1992). Shame and its contribution to PTSD. Journal of Traumatic Stress, 5, 557-562.

World Health Organisation, (1992). International Classification of Mental and Behavioural Disorders, Geneva.

Yehuda, R. Southwick, S. M., Mason, J. W., & Giller, E. L (1990). Interactions of the hypothalamic-pituitary-adrenal axis and the catecholaminergic system of the stress disoredr.. In E. L. Giller (ed). Biological assessment and treatment of PTSD, Washington DC: American Psychiatric Press.

Yehuda, R., Kahana, B., Southwick, S. M., & Giller, E. L. (1994). Depressive featires in Holocost survivors with posttraumatic stress disorder. Journal of Traumatic Stress, 7, 699-704.

Yehuda, R., & McFarlane, A., (1995). Conflict between current knowledge about posttraumatic stress disorder and its original conceptual basis. American Journal of Psychiatry, 152, 1705-1713.

Yule, W., Williams, R & Joseph, S. (1999). Post-Traumatic Stress Disorders In Adults, pp (1-24) in W. Yule (ed) Post-Traumatic Stress Disorders: Concepts and Therapy. New York: W. John Wiley & Sons.

APPENDIX 1-MODIFIED DENTAL ANXIETY SCALE

Name _____ Date _____

CAN YOU TELL US HOW ANXIOUS/FEARFUL YOU GET, IF AT ALL, WHEN YOU VISIT YOUR DENTIST?

Please indicate by inserting 'X' in the appropriate box

- 1. If you went to your Dentist for TREATMENT TOMORROW, how would you feel?**

Not Anxious Slightly Anxious Fairly Anxious Very Anxious Extremely Anxious

- 2. If you were sitting in the WAITING ROOM (waiting for your turn to see the dentist), how would you feel?**

Not Anxious Slightly Anxious Fairly Anxious Very Anxious Extremely Anxious

- 3. If you were about to have a TOOTH DRILLED, how would you feel?**

Not Anxious Slightly Anxious Fairly Anxious Very Anxious Extremely Anxious

- 4. If you were about to have your TEETH SCALED AND POLISHED, how would you feel?**

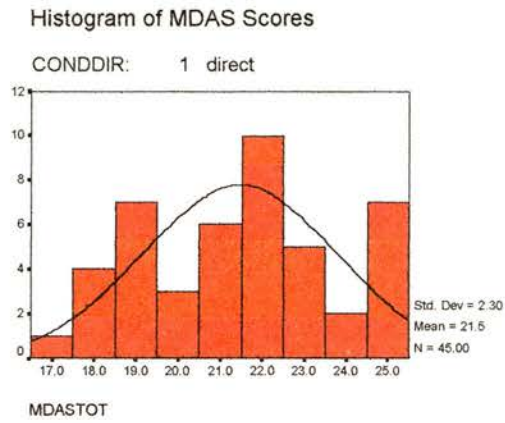
Not Anxious Slightly Anxious Fairly Anxious Very Anxious Extremely Anxious

- 5. If you were about to have a LOCAL ANAESTHETIC INJECTION in your gum, above a upper back tooth, how would you feel?**

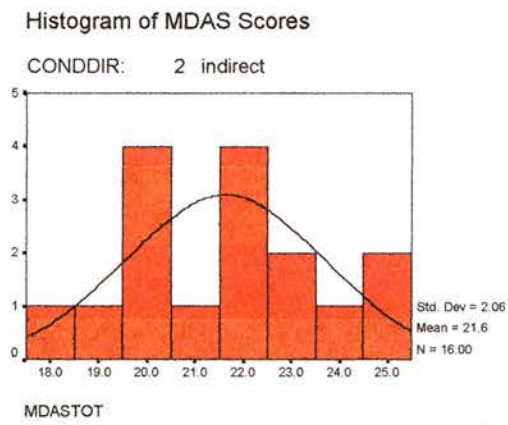
Not Anxious Slightly Anxious Fairly Anxious Very Anxious Extremely Anxious

APPENDIX 2-HISTOGRAMS MDAS STUDY 1

Histogram of MDAS Scores: Direct conditioning group

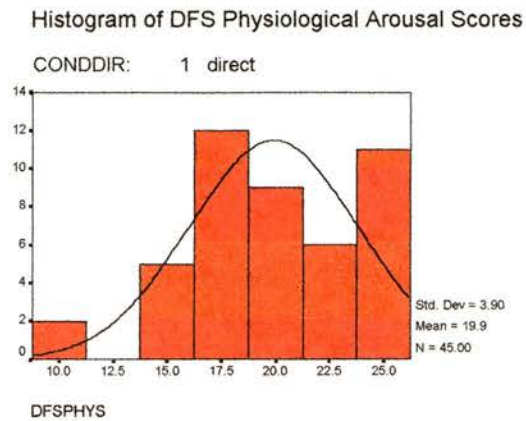


Histogram of MDAS Scores: Indirect conditioning group

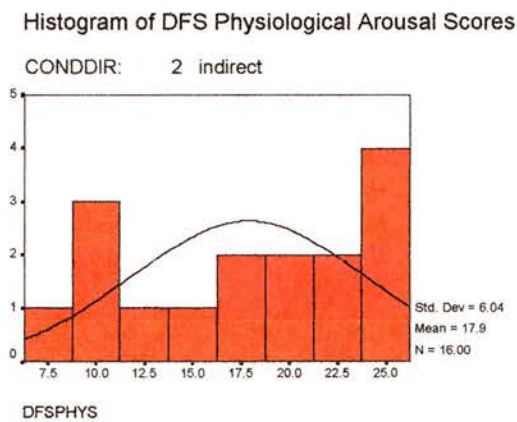


**APPENDIX 3-HISTOGRAMS DFS PHYSIOLOGICAL
AROUSAL STUDY 1**

Histogram of DFS Physiological Arousal Scores: Direct conditioning group

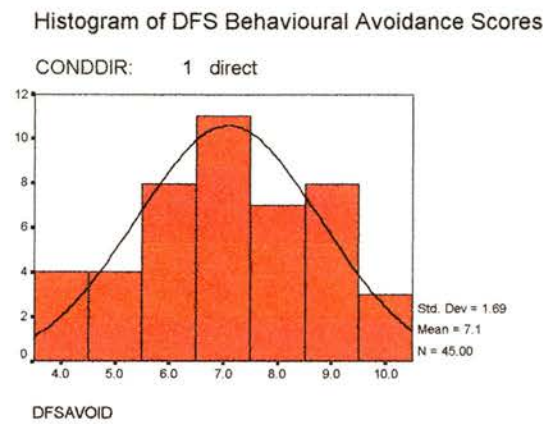


Histogram of DFS Physiological Arousal Scores: Indirect conditioning group

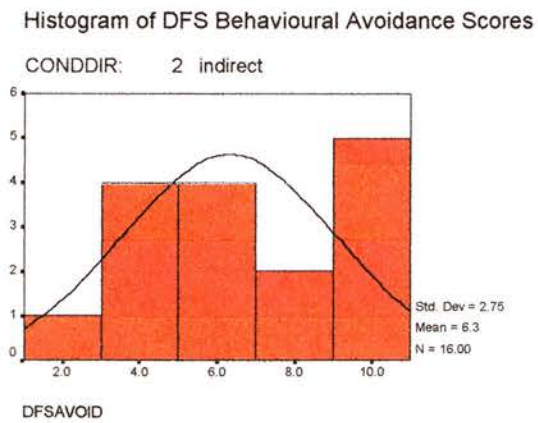


**APPENDIX 4-HISTOGRAMS DFS BEHAVIOURAL
AVOIDANCE STUDY 1**

Histogram of DFS Behavioural Avoidance Scores: Direct conditioning group



Histogram of DFS Behavioural Avoidance Scores: Indirect conditioning group



APPENDIX 5-DENTAL COGNITIONS CHECKLIST

Each item below describes a thought that some patients think to themselves about dental care. Please read each statement and indicate the degree to which it applies to you now. **I think that**

If statement does not apply fully to your situation, then just choose the closest answer	Don't Know	Not at all	Rarely	Sometimes	Often
1 the needle seems so long! Like it could stick into my eye, nose or brain.					
2 the needle might hit a nerve or something and damage it.					
3 nothing is as painful as a needle in my mouth.					
4 the needle might break off.					
5 medical needles are much smaller and less painful.					
6 I'm very hard to get numb.					
7 if my throat gets numb from an injection I won't be able to breathe or swallow.					
8 if I'm leaned back too far in the dental chair I get claustrophobic.					
9 when I'm in the dental chair I can't stop for a rest					
10 being in the dental chair can bring back bad memories from other events in my life.					
11 I can't breathe with instruments in my mouth.					
12 I can't swallow with instruments in my mouth					
13 I might get too much radiation from the x-rays.					
14 the mercury or other metals (or plastics) might be dangerous to my health.					
15 too much topical anaesthetic might make it so I could not breathe or swallow.					
16 I'm always waiting for the drill to hurt					
17 I'm fearful that the dentist might slip and injure me.					
18 I can't stand the sound of the drill.					

I think that	Don't Know	Not at all	Rarely	Sometimes	Often
19 I can't stand the sound of having my teeth cleaned (scraped).					
20 I can't stand that burning smell when they drill on teeth.					
21 I'm allergic to something - like the local anaesthetic agent, and it might harm me.					
22 impressions (moulds and models) make me feel like I can't swallow or breathe.					
23 impressions (moulds and models) make me feel like I will gag.					
24 x-rays make me gag.					
25 x-rays hurt.					
26 I will have lots of pain after treatment.					
27 they will find something terrible wrong with me.					
28 I might be so scared and I will do something embarrassing.					
29 it is so embarrassing to be fearful, I might not go ahead with treatment.					
30 I get anxious before a dental appointment.					
31 I am emotionally exhausted after an appointment.					
32 I am physically exhausted after an appointment.					
33 I can't stand the sight or taste of blood.					
34 they might drill too deep.					
35 the dentist is going to say I need a root canal.					

I think that	Don't Know	Not at all	Rarely	Sometimes	Often
37 the numbness will not go away.					
38 the dentist will think I'm foolish or childish.					
39 I feel so guilty about letting things go, I don't deserve treatment.					
40 I'm so fearful that I'm too much trouble to treat.					
Did we miss any? Please write down any anxiety provoking or disturbing thoughts you might have relating to dental care.					
41					
42					

APPENDIX 6-STRUCTURED INTERVIEW PTSD

Initials _____ ID# _____ Date _____ Visit # _____ Age _____ Sex _____ Race _____

**STRUCTURED INTERVIEW FOR
PTSD (SIP)**

Introduction [If this is the first encounter with subject]

I should like to ask about the difficulties or problems that caused you to come for help.

First would you please tell me your age: _____ Where do you live? _____

Are you employed? If yes: What is your job? _____

If no: When did you last work? _____

What did you do? _____ Why did you stop work? _____

With whom do you live? _____

Please tell me about your family, friends, and social activities.

Experience of Trauma

Did you ever experience, witness or have to confront an extremely stressful event which involved actual or threatened death or serious injury, or a threat to the physical integrity of yourself or others?

No _____ Yes _____

Did you react to the event(s) with intense fear, helplessness or horror?

No _____ Yes _____

How long were you in that situation? _____ What was the worst thing about it for you?

Define the event(s). Identify by numbers below. Narrative comment may be added.

Event

Age at Event

1 = Combat

2 = Rape

3 = Incest

4 = Other physical assault/attack

5 = Seeing someone killed or hurt

6 = Natural disaster

7 = Accident

8 = Complicated bereavement

9 = Threat of close call

10 = Life threatening illness

11 = Other (identify)

A4 Identify which event was the worst, and focus on this for the interview. _____

B. Reexperiencing the Traumatic Event

After it was over, did you find yourself persistently remembering or dreaming about the events over and over again for at least one month?

No _____ Yes _____

Did this happen even when you were not trying to remember?

No _____ Yes _____

Initials _____ ID# _____ Date _____ Visit # _____ Age _____ Sex _____ Race _____

STRUCTURED INTERVIEW FOR PTSD (SIP)

THE TIME PERIOD WILL USUALLY BE THE PAST WEEK. IT CAN BE ADJUSTED IF THE PURPOSE OF THE INTERVIEW IS FOR WORST EVER OF TO ASSESS DIAGNOSIS, FOR WHICH A 4 WEEK PERIOD OF SIMULTANEOUS SYMPTOMS IS REQUIRED.

A. Trauma _____

B1 Have you experienced painful images, thoughts or memories of the trauma which you could not get out of your mind even though you may have wanted to?

Have these been recurrent?

0 = not at all

1 = mild: rarely and/or not bothersome

2 = moderate: at least once a week and/or produces some distress

3 = severe: at least 4 times per week or moderately distressing

4 = extremely severe: daily or produces so much distress that patient cannot work or function socially

Rate past week _____

B2 Dreams

I would like to ask you about your dreams. Have you had repeated dreams of violence, injury, danger, combat, death or other theme related to trauma? Were these of actual scenes you were involved in? Do you recognize people in the dream? Are these dreams of the event? How frequent are these dreams? Do you wake up sweating or shouting? Trembling? Palpitations? Trouble breathing? Are the nightmares so bad that your spouse (partner) does not sleep in the same bed, or in the same room?

0 = no problems

1 = mild: infrequent or not disruptive

2 = moderate: at least once a week/somewhat distressing

3 = severe: at least four times a week/moderately distressing

4 = extremely severe: six to seven times a week/extremely distressing

Rate past week _____

B3 Acting or feeling as if event was currently happening

At times have you reacted to something as if you were back in the event? Has it seemed that the event was recurring or that you were living through it again? Did you have hallucinations of the event?

0 = not at all

1 = rarely/once a week

2 = sometimes/2 - 4 times a week

3 = often/5 - 6 times a week

4 = every day

Rate past week _____

Initials _____ ID# _____ Date _____ Visit # _____ Age _____ Sex _____ Race _____

B4 Psychological distress at exposure to reminders of event(s)

Do any of the symptoms occur or get worse if something reminds you of the stressful event? (Ask about TV programs, weather conditions, news, Veterans' Day, recent disaster involving loss of life, loss of good friends, being in places which remind person of the event). (Feel angry, sad, irritable, anxious, or frightened?)

- 0 = not at all
- 1 = a little bit: infrequent or of questionable significance
- 2 = somewhat
- 3 = significantly: several symptoms occur or one symptom with much distress
- 4 = marked: very distressing, may have activated an episode of the illness, resulting in hospitalization, different treatment, etc.

Rate past week _____

B5 Does exposure to an event that reminds you of, or resembles, the event cause you to have any physical response? (Sweating, trembling, heart racing, nausea, hyperventilating, dizziness etc.)

- 0 = not at all
- 1 = a little bit: infrequent or questionable
- 2 = somewhat: mildly distressing
- 3 = significantly: causes much distress
- 4 = marked: very distressing or has sought help from doctors because of the physical response (e.g., chest pain so severe that patient was sure he or she was having a heart attack)

Rate past week _____

C. Avoidance of Stimuli Associated with Trauma

C1 Have you tried to avoid thoughts or feelings about the trauma?

- 0 = no avoidance
- 1 = mild: of doubtful significance
- 2 = moderate: definite effort is made, but is able to function at work and socially
- 3 = severe: definite avoidance which affects life in some way (keeps moving from place to place/cannot work/works excessively/or episodic substance abuse because of need to avoid thoughts or feelings)
- 4 = very severe: dramatic effect on life

Rate past week _____

Initials _____ ID# _____ Date _____ Visit # _____ Age _____ Sex _____ Race _____

2 Avoidance of activities that arouse recollection of the event

Have you avoided places, people, conversations or activities that remind you of the event?

0 = no avoidance

1 = mild: of doubtful significance

2 = moderate: definite avoidance of situations

3 = severe: very uncomfortable and avoidance affects life in some way

4 = extremely severe: house-bound, cannot go out to shops and restaurants, major functional restrictions

Rate past week _____

3 Psychogenic amnesia

Is there an important part of the event that you cannot remember?

0 = no problem: remembers everything

1 = mild: remembers most details

2 = moderate: some difficulty remembering significant details

3 = severe: remembers only a few details

4 = very severe: claims total amnesia for the trauma

Rate past week _____

4 Loss of interest. Have you experienced less interest (pleasure) in things that you used to enjoy?

What things have you lost interest in? What do you still enjoy?

0 = no loss of interest

1 = one or two activities less pleasurable

2 = several activities less pleasurable

3 = most activities less pleasurable

4 = almost all activities less pleasurable

Rate past week _____

5 Detachment/estrangement

Do you have less to do with other people than you used to? Do you feel estranged from other people?

0 = no problem

1 = feel detached/estranged, but still has normal degree of contact with others

2 = sometimes avoids contact that you would normally participate in

3 = definitely and usually avoids people with whom would previously associate

4 = absolutely refuses or actively avoids all social contact

Rate past week _____

Initials _____ ID# _____ Date _____ Visit # _____ Age _____ Sex _____ Race _____

C6 Restricted range of affect

Can you have warm feelings/feel close to others? Do you feel numb?

0 = no problem

1 = mild: of questionable significance

2 = moderate: some difficulty expressing feelings

3 = severe: definite problems with expressing feelings

4 = very severe: have no feelings, feels numb most of the time

Rate past week _____

C7 Foreshortened future

What do you see happening in your future?

What do you visualize as you grow old? What are your expectations of the future?

0 = describes positive or realistic future

1 = mild: describes pessimistic outlook at times, but varies from day to day depending on events

2 = moderate: pessimistic much of the time

3 = severe: constantly pessimistic

4 = can see no future/views early death as likely (but without adequate medical basis)

Rate past week _____

D. Increased Arousal

D1 Sleep disturbance

We spoke earlier about nightmares. What about other aspects of sleeping? Have you had any trouble falling asleep? Do you wake in the middle of the night? Are you able to go back to sleep after waking?

0 = no loss of sleep

1 = mild: occasional difficulty but no more than two nights/week

2 = moderate: difficulty sleeping at least three nights/week

3 = severe: difficulty sleeping every night

4 = extremely severe: less than 3 hours sleep/night

Rate past week _____

Initials _____ ID# _____ Date _____ Visit # _____ Age _____ Sex _____ Race _____

D2 Have you been more irritable or more easily annoyed than usual?

How did you show your feelings? Have you had angry outbursts?

0 = not at all

1 = mild: occasional feelings of annoyance or anger which may go unnoticed by others

2 = moderate: increased feelings of annoyance, becomes snappy or argumentative (at least once every 2 weeks); others may have commented

3 = severe: almost constantly irritable or angry/often loses temper or has significant impairment in ability to relate to others as a result of this

4 = very severe: preoccupied with anger or feelings of retaliation, overtly aggressive or assaultive/marked impairment in function

Rate past week _____

D3 Impairment in concentration

Have you noticed any trouble concentrating? Is it hard to keep your mind on things?

Can you pay attention easily? What about reading or watching TV?

0 = no difficulty

1 = patient acknowledges slight problem

2 = patient describes difficulty

3 = interferes with daily activities, job, etc.

4 = constant problems, unable to do simple tasks

Rate past week _____

D4 Hypervigilance

Do you have to stay on guard? Are you watchful? Do you feel on edge? Do you have to sit with your back to the wall?

0 = no problem

1 = mild: occasional/not disruptive

2 = moderate: causes discomfort/feels on edge or watchful in some situations

3 = severe: causes discomfort/feels on edge or watchful in most situations

4 = very severe: causes extreme discomfort and alters life (feels constantly on guard/must keep back to wall/socially impaired because of feeling on edge)

Rate past week _____

Initials _____ ID# _____ Date _____ Visit # _____ Age _____ Sex _____ Race _____

D5 Startle

Do you startle easily? Do you have a tendency to jump? Is this a problem after unexpected noise, or if you hear or see something that reminds you of the original trauma?

0 = no problem

1 = mild: occasional/not disruptive

2 = moderate: causes definite discomfort or an exaggerated startle response at least every 2 weeks

3 = severe: happens more than once a week

4 = extremely severe: so bad that patient cannot function at work or socially

Rate past week _____

E. How long has this condition lasted?

E1 Did the symptoms which you have described last for at least four weeks? _____

E2 How many months after the trauma did these symptoms first develop? _____

E3 Age at the time symptoms began. _____

F. In the interviewer's judgment, and taking into account the subject responses, has the disturbance caused clinically significant distress or impairment in social, occupational or other important areas of functioning?

No _____ Yes _____

SCORESHEET: SIP

Initials _____	ID# _____	Date: _____	Visit # _____
B	1	_____	
	2	_____	
	3	_____	
	4	_____	
	5	_____	
	(Subtotal)	_____	
C	1	_____	
	2	_____	
	3	_____	
	4	_____	
	5	_____	
	6	_____	
	7	_____	
	(Subtotal)	_____	
D	1	_____	
	2	_____	
	3	_____	
	4	_____	
	5	_____	
	(Subtotal)	_____	
Total (B, C, D)		_____	

SCORE SHEET FOR STRUCTURED PTSD INTERVIEW

Total (past week or other designated period) score for all B, C, and D items.

_____ Total

Score No as 1, Yes as 2 to all answers below

DSM-IV Diagnosis

Traumatic event definitely present?

At least one item from category B with score of at least 2

At least three items from category C with score of at least 2 (at least one item must be from C 1-2 and one must be from C 3-7)

At least two items from category D 1-5 (each must score at least 2)

Diagnosis? _____

APPENDIX 7-PATIENT INFORMATION SHEET

Information sheet for participants

The Effects of Previous Dental Experiences on Current Dental Fears.

We invite you to participate in a research project. However, before you decide whether or not to participate, we need to be sure that you understand firstly why we are doing it, and secondly what it would involve if you agree to participate. We are therefore providing you with the following information. Read it carefully and be sure to ask any questions you have. If you do have any further questions or would like more information please do not hesitate to contact either Mandy Forbes or Dr. Pauline McGoldrick at the Department of Clinical Psychology, Ninewells Hospital (Tel 01382 425612) or Dundee Dental Hospital and School (Tel 01382 425760)

The Study

As someone who has been referred for help with dental treatment because you are worried about it, you have been chosen as a possible participant in this study. The aim of the study is to gain a better understanding of the effects of previous dental experiences and how these now relate to your current fears. It is hoped that any results from the research will help us have a greater understanding of the treatment needs of patients with dental anxiety.

What you would be asked to do

Participating in the study would involve you answering some questions regarding your past dental experiences. Then you would be asked to complete a number of short questionnaires. The research is being conducted at Dundee Dental Hospital and in total the appointment will take approximately 40 minutes to one hour. All information given to the researchers will be treated as confidential and will be

password protected however we are required to advise your General Practitioner of your involvement in the study.

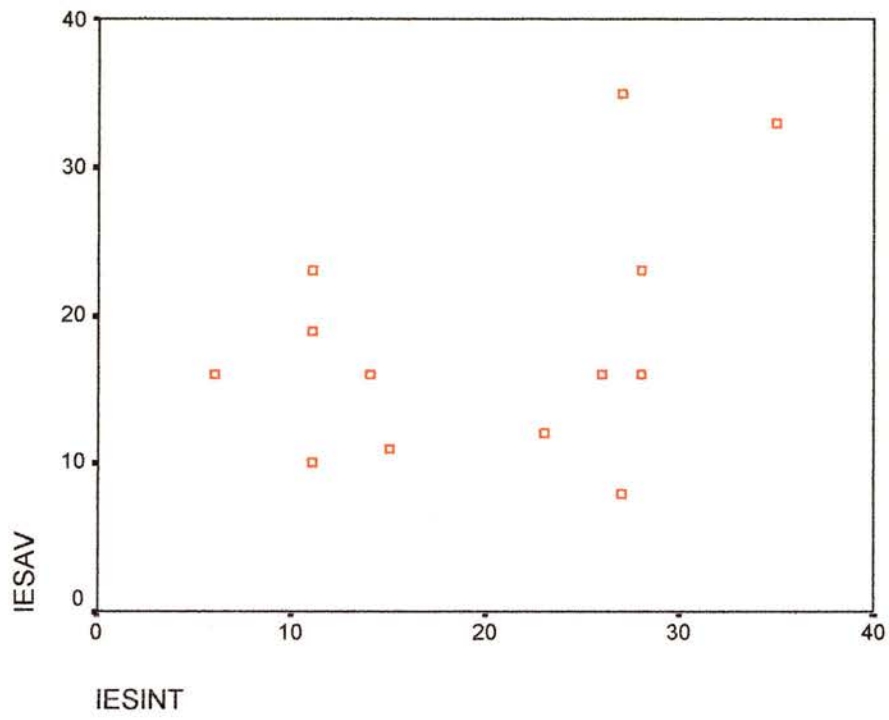
Participation in this study is entirely voluntary and you are free to refuse to take part or to withdraw from the study at any time without having to give a reason and without this affecting your future dental care or your relationship with the dental staff looking after you.

The Tayside Committee on Medical Research Ethics that has responsibility for scrutinising all proposals for medical research on humans in Tayside has examined the proposal and has raised no objections from the point of view of medical ethics. Monitors from the Tayside Medical Research Ethics Committee may examine your research records.

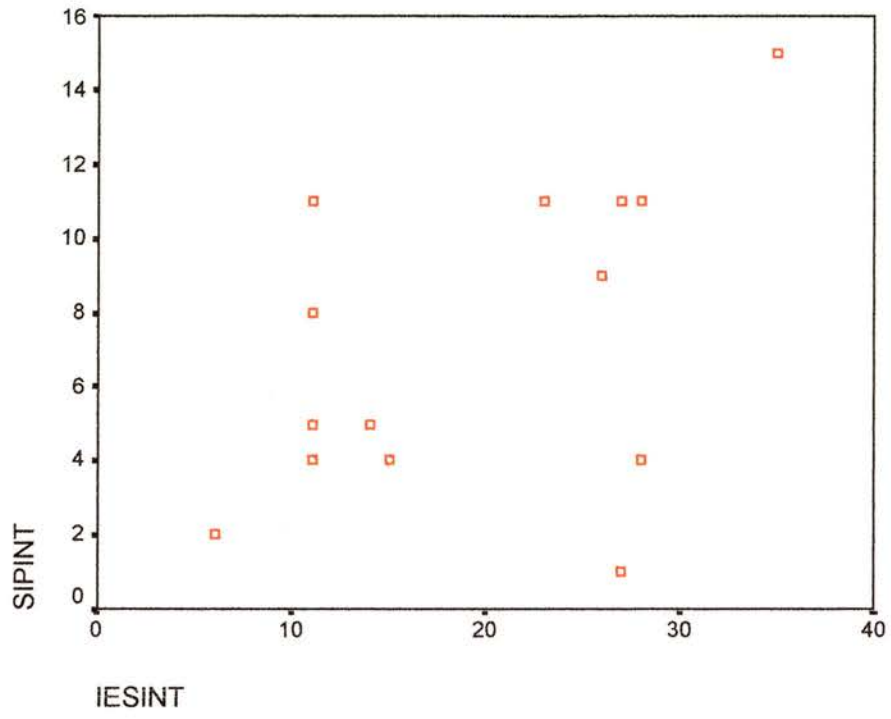
Mandy Forbes
Pauline McGoldrick

APPENDIX 8-SCATTERGRAMS STUDY 2

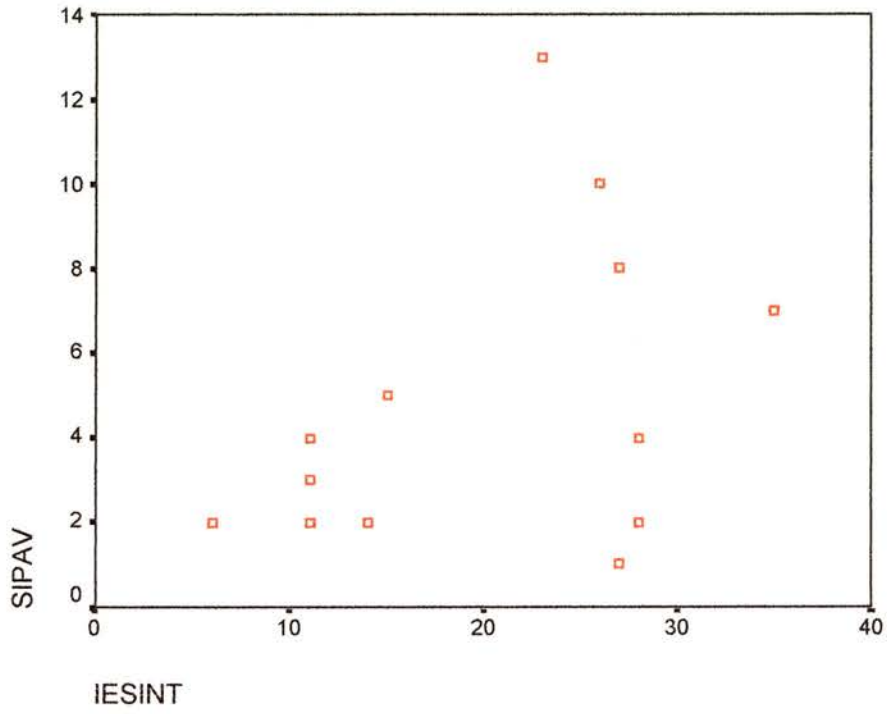
Scattergram, IES intrusions and avoidance



Scattergram IES intrusion and SIP intrusion



Scattergram IES intrusion and SIP avoidance



Scattergram IES intrusion and SIP Total score

