An Evaluation of Functional Assessment and Biofeedback for the Treatment of Generalised Anxiety Disorder

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Award date: 2014
AN EVALUATION OF FUNCTIONAL ASSESSMENT AND BIOFEEDBACK FOR THE TREATMENT OF GENERALISED ANXIETY DISORDER
AN EVALUATION OF FUNCTIONAL ASSESSMENT AND BIOFEEDBACK FOR THE TREATMENT OF GENERALISED ANXIETY DISORDER

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A thesis submitted in partial fulfilment of the requirements of the degree of Doctorate of Counselling

Bond University

June, 2013
CANDIDATE’S DECLARATION

This thesis is submitted to Bond University in fulfilment of the partial requirements for the Degree of Doctorate of Counselling.

This thesis contains no material which has been accepted for the award of any other degree in any other University and, to the best of the candidate’s knowledge and belief, it contains no material previously published or written by another person except where due reference is made in the text of the project.

Signature: ........................................... Date: ..................................................
ABSTRACT

Anxiety Disorders, in particular Generalised Anxiety Disorder, have escalated in incidence over the past decade resulting not only in additional burden for primary health care systems but also debilitating impacts on those who suffer from this constellation of conditions. It is relevant to note that, because Anxiety Disorders commonly coexist with other mental disorders (e.g., depression) and physical illnesses (e.g., cancer) as well as maladaptive coping behaviours (e.g., excessive alcohol/drug consumption), their psychotherapeutic treatment is not always straightforward. Presence of co-existing conditions not only leads to complex presentation of symptomatology but also limits the effectiveness of generic or manualised treatment approaches. Those treatment approaches, which are common in the mental health field, have been shaped by conceptualisation of anxiety-based diagnoses as labels that indicate homogeneity in symptoms as well as equivalence in impacts of those symptoms across individual clients. Therefore, whilst diagnostic manuals such as the DSM have been essential to promoting accurate diagnosis of Anxiety Disorders, they do not provide a sound basis for the type of idiographic assessment that would facilitate individualised treatment planning. Therefore the issues of 1) how an idiographic assessment might be tailored to the investigation of anxiety and 2) how idiographic assessment data might be used to administer an intervention to remediate the somatic symptoms of anxiety require further examination.

This study examined the capacity of a Functional Assessment process tailored to the investigation of Generalised Anxiety Disorder to generate additional data to facilitate detailed understanding of anxiety behaviour (symptoms) and the impact of these behaviours on day-to-day functioning. This Functional Assessment process was also evaluated in relation to its capacity to determine the factors responsible for strengthening anxiety responses.
assessment process under investigation was applied individually to 11 adult participants with a diagnosis of Generalised Anxiety Disorder thus allowing for an $n = 1$ evaluation of individual cases as well as a comparative examination of group results. Findings suggested that the Functional Assessment succeeded in recording data which described behaviour-environment interactions in detail.

This study also evaluated the effects of a seven-session Biofeedback plus behavioural training intervention on anxiety level in the same group of 11 participants. The focus of this intervention was on anxiety reduction via greater participant awareness of physiological functions (i.e., Heart Rate and Respiratory Sinus Arrhythmia) and cumulative control over those functions. Change in anxiety level was established at pre-and-post intervention (i.e., self-report anxiety scale responses and Heart Rate plus Respiratory Sinus Arrhythmia measures) as well as during intervention (i.e., session-by-session measurement of Heart Rate and Respiratory Sinus Arrhythmia). The intervention was administered individually to each participant and results are reported in relation to $n = 1$ and group evaluations. Findings indicated that the intervention was effective in reducing anxiety level across all participants.
ACKNOWLEDGMENTS

I would like to thank my supervisor and mentor, Professor Vicki Bitsika, for her invaluable guidance, advice and encouragement. Without her assistance, patience, humour and persistent help this dissertation would not have been possible.

I extend my thanks to my other supervisor, Professor Christopher Sharpley, for his time, support and advice throughout this project. This research would not have seen the light of day without his feedback and inspiration.

I also wish to thank my family and friends for their support and encouragement.

Lastly I extend my gratitude to the 11 participants, who gave their time and commitment to this research project.
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CHAPTER 1

Introduction

Anxiety is ubiquitous in modern society and recent estimates suggest that most people will experience some form of anxiety at some stage of their lives (Greenberg et al., 1999). This escalation in anxiety has resulted in the need for effective and time-efficient procedures for accurate identification and targeted treatment of Anxiety Disorders. Simon, Ormel, Von Korff, and Barlow (1995) have argued for tailored treatments to be applied as close to anxiety onset as possible to minimise exacerbation of any co-existing mental health problems and use of maladaptive coping behaviours used by sufferers to reduce the emotional and somatic discomfort that is embedded in an Anxiety Disorder. This delivery of early and targeted interventions rest on accurate diagnosis of Anxiety Disorders, clear understanding of how this disorder manifests itself in day-to-day life and what constitutes effective intervention.

The DSM-IV-TR (APA, 2000) contains diagnostic criteria for eleven Anxiety Disorders with Generalised Anxiety Disorder (GAD) representing the most frequently made diagnosis among primary practitioners such as general practitioners and specialist clinicians (i.e., Psychiatrists and Clinical Psychologist). This disorder is particularly debilitating as it is associated with intense feelings of fear which, even if they initially relate to a specific event, pervade across most events and contexts in the person’s life (Beyond Blue, 2013). Therefore, on the bases of incidence and disruption to functioning, GAD is worthy of further and focused investigation. The DSM-IV-TR (APA, 2000) provides clear criteria to assist in the diagnosis of GAD from a labelling perspective but, if the challenge is delivery of targeted treatment to remediate specific difficulties, then the assessment process needs to extend beyond the use of this diagnostic manual. One framework for understanding the particular experiences and needs of people with the same diagnosis is Functional Assessment which has a strong history in the research literature as a process for collecting individualised data on
behaviour (symptoms) for the purpose of intervention planning (Bitsika, 2005). Despite application of Functional Assessment to diagnostic categories such as depression, Anorexia Nervosa, and school refusal, there have been very few uses of this process in Anxiety Disorders. Therefore, the question of what constitutes a Functional Assessment process tailored for GAD is worthy of investigation.

Psychotherapeutic treatment to assist with mental disorders has been shaped substantially by the push for use of evidence-based methods which have been submitted to evaluation in scientific research (Burns, Hoagwood & Mrazek, 1999). This push has had the distinct advantages of removing questionable treatments from use in the field and providing a uniform (via delivery of manualised treatments) method for treating disorders. However, manualised treatments have also been criticised for being label- rather than person-focused and generic in nature leading to lack of focus on individual experiences and life circumstances (Bitsika, 2003). Therefore, the need for targeted intervention (to the person and his/her life experiences) is increasing and one possible avenue for meeting this need is investigation of treatment that adhere to the criteria for being evidence-based and which take into account the factors which impact the individual’s particular experiences and circumstances.

Chapter 2 of this thesis discusses the defining features of GAD and differentiates this condition from the other Anxiety Disorder diagnoses in the DSM-IV-TR (APA, 2000). The major inter-disciplinary frameworks (i.e., psychological, functional analytic, and medical) for conceptualising anxiety are described and contrasted in order to illustrate practitioner differences in understanding anxiety impacts and developing treatment to remediate those impacts. This thesis is written from the perspective that anxiety-based disturbance is in large part attributable to somatic symptoms thus setting the foundation for treatment which directly
targets physiology. This thesis is also oriented towards trialling person-centred methods for assessing clients with GAD.

Chapter 3 of this thesis presents a framework for post-diagnosis, person-centred assessment of anxiety (i.e., Functional Assessment). Functional Assessment is discussed in relation to its evolution over the past 30 years, the key data-recording and data-interpretation methods it applies, and some possible reasons for why it has not been used to investigate anxiety. This chapter also presents a model, based on key findings from the literature, for adapting Functional Assessment to GAD and provides an outline for how this model was applied to investigate the experiences of the participant group for this study.

Chapter 4 of this thesis discusses Biofeedback Training as an evidence-based treatment which specifically targets the somatic symptoms of mental disorders. Biofeedback Training is reviewed from an historical perspective which suggests that its initial uses focused on physical conditions to discussion of contemporary applications designed to assist with reduction of somatic symptoms associated with mental disorders. The pitfalls of using of Biofeedback Training to treat poorly defined or ill-suited conditions (to this treatment modality) are addressed with reference to objective criteria for what constitutes evidence-based Biofeedback Training.

Chapter 5 of this thesis presents the procedures used to conduct a Functional Assessment of GAD and deliver a Biofeedback plus Behavioural Training intervention. The Functional Assessment provided the basis for idiographic investigation of GAD as a fear response, it also aimed to identify those antecedent factors associated with occurrences of the fear response and consequent events thought to strengthen that response and cause it to become pervasive. The Biofeedback plus Behavioural Training intervention consisted of seven individually-administered sessions which involved use of Heart Rate and Respiratory
Sinus Arrhythmia as the physiological indicators for anxiety. Those responses were used to provide participants with feedback on their capacity to create physiological change whilst engaging in a behavioural strategy.

Chapter 6 of this thesis discusses the findings obtained from the trial application of the Functional Assessment process for GAD and the implementation of the Biofeedback and Behavioural Training intervention. The major findings relating to the Functional Assessment process indicate that it did produce greater breadth and depth of information on anxiety experiences than would not be possible from a DSM-IV-TR (APA, 2000) diagnosis. That information was particularly relevant to understanding the factors which caused escalation in anxiety and the ways in which that aversive mood state had become an adaptive response. The evaluation of the Biofeedback and Behavioural Training intervention showed that participants were able to learn to control their physiological reactivity (i.e., Heart Rate and Respiratory Sinus Arrhythmia) to reduce somatic as well as cognitive/emotional anxiety. Findings suggest that participants were able to learn a number of behavioural strategies for reducing anxiety which they were able to apply to their day-to-day lives.

Chapter 7 of this thesis reviews the key findings in relation to the literature to comment on the possible contributions of the current study to the broader clinical field. The implications for incorporating Functional Assessment to person-centred investigation of anxiety in the Counselling context, as well as the relevance of Biofeedback Training to the treatment of the somatic and cognitive/emotional symptoms anxiety are also discussed.
CHAPTER 2

Description of Anxiety

Anxiety Disorder is the most common form of ill mental health in adults and children (ABS, 2009). The Australian Bureau of Statistics (ABS) figures obtained from a 2007 survey indicated that anxiety affected approximately 14% of all Australians aged between 16-85 years in the 12 months prior to that survey being conducted. Further, women were more likely to have experienced anxiety disorders than men (i.e., prevalence of 18% and 11% respectively). Data released by Beyond Blue (2013) indicate that Generalised Anxiety Disorder (GAD) is the most commonly-occurring diagnosis, and affects approximately 5 - 6 percent of Australians at some time in their lives (Beyond Blue, 2013). The American National Institute of Mental Health (NIMH) (2010) published figures showing a lifetime prevalence of GAD of 5.7 percent in the US population, with some variation in relation to gender (i.e., prevalence of 7.1% in women and 4.2% in men) and life stage (i.e., 4.3% in 18-29 year olds; 6.5% in 30-44 year olds; 7.6% in 45-59 year olds; 4.0% in 60+ year olds). Wittchen and Hoyer (2001), in their review of lifetime prevalence of GAD across countries, suggested that the prevalence of GAD can be as high as 21.7 percent (Iceland) and as low as 0.5 percent (Germany). Findings such as these clearly indicate that anxiety poses a substantial threat to adaptive functioning across the age span.

The most prominent subgroup of anxiety is GAD. A review of categorical descriptions of anxiety and its subtypes, from historical and more recent perspectives, is presented below to make explicit the diagnostic criteria which facilitate its identification and adverse impacts on adaptive functioning.
2.1 Categorical Descriptions of Anxiety

The term *anxiety* has a long history in the psychiatric field and was first described as a form of neurosis by William Cullen in 1769 (Knoff, 1970), being described as a mental disorder which caused sufferers to experience deterioration in behavioural functioning (within acceptable social norms) and no onset of delusions or hallucinations (Strachey, 1974). The broad Neurosis classification was also used in the first and second editions of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM) (titled respectively DSM-I [APA, 1952] and DSM-II [APA, 1968]). These editions of the DSM presented the following five distinct neurosis-based diagnoses: Phobic Neurosis, Anxiety Neurosis, Obsessive-Compulsive Disorder, Hysterical Neurosis, and Depressive Neurosis. The term *neurosis* was dropped in the DSM-III (APA, 1980) and subsequent DSM-III-R (APA, 1987) and replaced with *anxiety state* in recognition of the fact that anxiety was considered an adaptive response which became problematic only with increases in intensity and frequency of symptoms (Brown, O’Leary and Barlow, 2001).

Current categorical descriptions of anxiety, as described in the DSM-IV-TR (APA, 2000) and the International Classification of Diseases - Tenth Edition (ICD-10, 1993), agree that anxiety is best conceptualised as a *negative mood* state which is characterised by somatic symptoms of tension and apprehension about the future. These broad features are further refined in relation to anxiety subgroup (or diagnosis) and the following discussion will focus first on an elaboration of the diagnostic criteria for GAD, and second on a comparison of those criteria with the defining symptom clusters for the remaining anxiety subgroups listed in the current version of the DSM.

In broad terms, people affected by GAD report feeling extreme worry about life in its entirety. This worry, which might have initially occurred in response to a particular life event, generalises to numerous other situations that cause the person to feel intense anxiety with
minimal capacity to identify or explain any reasons for that anxiety (NIMH, 2010). The DSM-IV-TR (APA, 2000) presents six diagnostic criteria (A - F) to assist clinicians in the accurate identification of GAD (see Appendix A for GAD diagnostic criteria). Criterion A refers to specific symptoms (i.e., excessive worry and anxiety) plus temporal (i.e., presence of symptoms for greater than six months) and severity (i.e., symptoms associated with a number of life events) guidelines. Criterion B specifies that the reported worry will be outside of the person’s control. Criterion C presents six mostly somatic symptoms with the requirement that the person show evidence of three symptoms for more days than not during six months prior to diagnosis. Criterion D serves the purpose of emphasising that the worry and anxiety that are indicative of GAD must not be limited to the features of another Axis 1 Disorder. This criterion also lists eight such features plus the Axis 1 disorders those features derive from (i.e., Panic Disorder, Social Phobia, Obsessive Compulsive Disorder, Separation Anxiety Disorder, Anorexia Nervosa, Somatisation Disorder, Hypochondriasis, and Post-Traumatic Stress Disorder). Criterion E focuses on the negative impacts of GAD on functioning by making reference to anxiety symptoms that cause clinically significant impairment in social, occupational and other areas of day-to-day performance. Criterion F centres on differentiation between GAD and other physiological effects (e.g., medication), medical conditions (e.g., hyperthyroidism), and mental conditions (e.g., Psychotic Disorder) which might result in similar clinical presentation to GAD.

Accurate identification of mental disorder requires not only distinction between the target disorder and other diagnostic groups (e.g., GAD versus Psychotic Disorder) but also delineation between the target disorder and subgroups which fall within the same broad classification (e.g., GAD versus Social Phobia). Clarity on the ways in which the various anxiety subgroups differ is central not only to accurate diagnosis but also for determinations on the most appropriate therapeutic approaches. In light of this, the subsequent discussion
will examine the ways in which GAD differs from the codable anxiety disorders contained in the DSM-IV-TR (APA, 2000). It should be noted that the purpose of this discussion is to identify the key bases on which each codable anxiety disorder differs from GAD rather than in-depth presentation of diagnostic criteria and symptom manifestation (see Appendix B for summary of features for Anxiety Disorder as per DSM-IV-TR [2000] Diagnostic criteria).

Panic Disorder is characterised by the presence of repeated and unanticipated panic attacks plus intense concern (of at least one month in duration) about the possibility of another attack occurring, and the implications of losing control. Panic Disorder can present with or without Agoraphobia in which anxiety relates to being in situations where the avenues for escape or access to help, following an attack, are low (DSM-IV-TR [APA, 2000]). Differentiation between GAD and Panic Disorder centres on the absence of panic attacks in the former but, as with all anxiety disorders, substance use/abuse and medical conditions must be excluded as being the causes for panic attacks (DSM-IV-TR [APA, 2000]).

The defining feature of Specific Phobia is an intense and irrational fear arising from a particular object or situation which does not contain fear-inducing qualities. The reference to irrationality emphasises that the experience of fear is generally unjustified and, despite possible awareness that the fear-causing stimulus is indeed neutral and the fear-response is exaggerated, avoidance of that stimulus persists, causing disruption in day-to-day functioning (DSM-IV-TR [APA, 2000]). GAD can be distinguished from Specific Phobia because the symptoms of worry and anxiety in GAD are not linked to one specific event but generalised across multiple situations. Social Phobia is present when the person experiences severe and persistent fear in relation to one or more social or performance events which contain unfamiliar people or the potential for the person to be adversely judged by others. As is the case with Specific Phobia, a common behavioural outcome of Social Phobia is active
avoidance of and/or escape from the feared social event. Although GAD might include worry and anxiety arising from social events, these adverse states are associated with a broader range of situations and avoidance is not always the likeliest response (DSM-IV-TR [APA, 2000]).

Obsessive-Compulsive Disorder (OCD) features recurrent preoccupations (i.e., thoughts, impulses or images) and/or compulsive behaviour patterns accompanied by recognition that obsessions/compulsions are excessive. The time spent engaging in obsessions/compulsions and the disruption caused in day-to-day functioning result in substantial distress. GAD is not associated with intense fixations or repetitive behaviour patterns and this is the basis for differentiation from OCD (DSM-IV-TR [APA, 2000]). Post-Traumatic Stress Disorder is described as resulting from exposure to an identifiable extreme traumatic event. Key symptoms include re-experiencing (via thoughts and dreams) the event, persistent avoidance of antecedents associated with the event, and increased autonomic arousal when such antecedents are present. GAD does not involve prior exposure to a traumatic stressor nor does it prompt intense reactivity to, and avoidance of, antecedents in the environment (DSM-IV-TR [APA, 2000]). Acute Stress Disorder also results from exposure to a traumatic event and, in this instance, the person experiences a dissociative symptom such as depersonalisation or subjective sense of numbing either during or after the event occurs. The differentiation between Acute Stress Disorder and GAD is based on the absence of a traumatic event and dissociative symptomatology. GAD symptoms are also not associated with presence of a specified medical condition and this is the primary basis for differentiating GAD from Anxiety Disorder due to a Medical Condition. Similarly, GAD does not arise from the intake of an identifiable substance or medication, which is the case for Substance-Induced Anxiety Disorder.
2.2 Conceptualisations of Anxiety

Explicitly stated diagnostic criteria for mental disorders facilitate identification, interdisciplinary communication on client status, and decision-making regarding appropriate treatment. In particular, description of symptoms and the ways in which those symptoms cause disruption in day-to-day functioning enhances clinicians’ understanding of clients’ experiences and the challenges they face as a result of suffering from anxiety. However, and despite clearly articulated DSM diagnostic guidelines, the manner in which anxiety and its causes is conceptualised varies in relation to the theoretical perspective taken by researchers and clinicians in the field (Neef & Iwata, 1994; Jones & Friman, 1999). Therefore, a review of the major theoretical perspectives on anxiety will assist in establishing a clear basis for determining how anxiety is understood by those who treat it in their clinical practice.

2.2.1 Psychological Conceptualisation of Anxiety

Psychological descriptions of anxiety generally adhere to two themes: first, identifying symptom profiles to aid accurate diagnosis, and second, determining the capacity of those symptom profiles to cause significant disturbance in day-to-day social and occupational functioning. Therefore, DSM diagnostic criteria have been elaborated into specific covert (e.g., elevated heart rate and subjective unease) and overt (e.g., verbalised worrying and fidgeting) responses which, when present with sufficient intensity, indicate functional disturbance due to anxiety (Barlow & Durand, 2005). The decision regarding level of anxiety and associated disturbance to functioning is often based on norm-based comparisons or at the least clinician’s impression of behaviour which extends outside of the expected norm for stage of life (Bitsika, 2006). Psychologically-oriented definitions for anxiety are sometimes criticised due to their focus on only a limited range of the anxious person’s experiences. For instance, the Caltabiano and Sarafino (p. 95, 2008) definition which states that “.... anxiety is a vague feeling of unease or apprehension, a gloomy
anticipation of impending doom that often involves a relatively uncertain or unspecific threat ....” represents only the cognitive and emotional aspects of the anxiety experience. Similarly, restricting conceptualisations of anxiety to particular erroneous cognitive processes as is seen in the Good and Beitman (2000) hypothesis on the cause for anxiety (i.e., Anxiety equation = \[ \frac{\text{Overestimation of threat or danger}}{\text{Underestimation of coping resources}} \]) which suggests that anxiety results from cognitive appraisals which overestimate the level of situational threat plus underestimate the available resources to cope with that threat, do not allow for inclusive explanations regarding individuals’ experiences.

### 2.2.2 Medical Conceptualisation of Anxiety

The medical model of fear and anxiety originated from the evolutionary or primordial “fight or flight” mechanism and is focused on the more primitive areas of the brain leading to it being conceptualised as a direct physical process (Marks & Nesse, 1994). Physiological descriptions of anxiety are considered to emphasize and explain the somatic symptoms and underlying neurological pathways associated with onset of those symptoms. Hoehn-Saric and McLeod (2000) defined anxiety as a biological warning system that activates the sympathetic part of the autonomic nervous system via a neuroendocrine response (by activation and an escalation of epinephrine, norepinephrine, cortisol, growth hormone and prolactin). Activation of the sympathetic nervous system causes physiological hyperarousal (i.e., surge in heart rate, blood pressure, sweat gland activity, respiration and increased in gastrointestinal and bladder activity). More recent studies using neuroimaging and neuroendocrinological methods have illustrated the complexity of the anxiety response in humans with the interaction of cognitive processing of a stressful event and the activation of the biological fear responses (Hofmann & Dibartolo, 2010; Shin & Liberzon, 2010; Porto et al., 2005).
Therefore, from this theoretical perspective, anxiety is viewed primarily as a physiological disorder characterised by tension and overactivity of the autonomic nervous system (ANS). Further, it is the escalation in ANS activity which is at the basis of the cognitive (e.g., apprehension), emotional (e.g., worry), and behavioural (e.g., hyper-vigilance) symptoms used in psychological models for assessment to identify whether anxiety is present (Carlson, 2013; Sherwood, 2010; Tortora & Grabowski, 2000). As with psychological descriptions of anxiety, medical conceptualisations of this disorder have been appraised as being limited due to their focus on somatic symptomatology and neurological antecedents. The conceptualisation of anxiety as an adverse somatic experience which leads to emotional distress and behavioural disturbance will be elaborated in Chapter 4 of this thesis where the focus will be on employing physiological monitoring and biofeedback to measure and remediate anxiety.

2.2.3 Functional Analytic Conceptualisation of Anxiety

Functional Analytic Models for anxiety have received increasing attention in the clinical field due to their inclusion of all symptom modalities (i.e., somatic, cognitive, emotion, and behaviour) and their capacity to incorporate the relative impact of antecedents on the onset and development of this disorder (Miller, 1997; Barlow, 2002; Bitsika, 2005). These models are based on idiographic assessment methods characterised by collection of data samples which first, allow for individualised descriptions of symptoms and responses and then identify the antecedents which are most likely to cause escalation in anxiety and any associated deterioration in coping (Barlow, 2002; Friman, 2006). The association between antecedent factors and anxiety-based behaviours traditionally referred to as the stimulus-response link by Mowrer (1939) and Skinner (1953) is central to functional analytic explanations for persistent anxiety and has led to chain-of-events conceptualisations of this disorder. One such simplified chain-of-events might begin with a stimulus (e.g., spider) or
stimulus situation (e.g., public speaking) which is known to cause fear. The chain is initiated when the stimulus/stimulus situation is present, causing the onset of unpleasant somatic responses (arising from ANS arousal) and adverse cognitive/emotional responses, and leading the individual to engage in some form of escape or avoidance behaviour as the mechanism for reducing the elevated discomfort (i.e., the anxiety) being experienced (Barlow, 2002; Friman, 2006; Miltenberger, 2004). The Functional analytic model, with specific reference to its capacity for person-centred assessment of the contextual causes of anxiety, is submitted to in-depth examination in Chapter 3 of this thesis.

This thesis will apply the physiological and Functional Analytic Models for conceptualising GAD in order to gain an in-depth understanding of the disorder itself as well as the environmental variables which might be associated with its occurrence. Criterion C of the DSM-IV-TR (APA, 2000) specifies that the worry and anxiety which feature in GAD are associated with three of six symptoms: feeling restless or feeling keyed up or on the edge (somatic), being easily fatigued (somatic), having difficulty concentrating or mind going blank (cognitive), irritability (somatic), muscle tension (somatic), and sleep disturbances (somatic). Review of this symptom list indicates that the vast majority of indicators for GAD are somatic in nature, thus providing a basis for assessing anxiety in relation to its physiological manifestations. Additionally, research suggests (DSM-IV-TR [APA, 2000], Barlow & Durand, 2005; Sturmey, 2007) that the symptoms of GAD, whilst clearly linked to disordered functioning, can be more clearly defined when conceptualised as specific units of overt and covert behaviour and better understood as adaptive responses when linked to those variables which are responsible for the exacerbation of those symptoms. The Functional Analytic Model provides a sound basis for examining how particular situations in day-to-day life contribute to experiences of anxiety.
CHAPTER 3

Functional Assessment of Anxiety

Behaviourally-oriented methods which allow for individualised assessment of the day-to-day difficulties associated with mental illness are increasing in popularity in the Counselling field (Burns, Hoagwood & Mrazek, 1999). The intent and focus of these behavioural methods are best understood via comparison with traditional approaches to assessment. Traditional, or nomothetic, assessment processes centre on administration of standardised tests to determine the presence and severity of symptom clusters that are indicative of mental disorder. Review of client symptoms with an externally-derived standard, such as the diagnostic criteria of the DSM, becomes the basis for identifying whether a mental disorder exists (Bitsika, 2003). Nomothetic assessments are particularly suited to accurate diagnosis and, when applied to therapy, provide a label (e.g., Generalised Anxiety Disorder) which guides delivery of “manualised” treatments designed to reduce symptom severity. However, nomothetic approaches, whilst providing a robust foundation for accurate diagnosis, have received criticism as treatments because they do not adequately account for the variation in the experiences and life circumstances which exist between people with the same diagnosis.

In contrast, idiographic approaches collect in-depth information on the specific behaviours which arise from mental disorder and the range of variables which appear to be linked to occurrences of those behaviours (Sugai et. al., 1999). Of significance to this approach is examination of the ways in which behaviours assist the person to cope with demand in their day-to-day lives. This idea that the same behaviour (or symptom) which indicates the presence of a mental disorder can also become a coping method is central to understanding each person’s particular experiences. Therefore, idiographic treatment approaches, instead of focusing on symptom reduction, are designed to build positive coping
skills capable of generalising to the full range of situations which cause the person distress (Bitsika, 2005). Functional Assessment is an idiographic framework which has been submitted to extensive investigation and offers a validated basis for assessing and treating people with mental disorder. The discussion which follows describes Functional Assessment procedures and elaborates on the application of these to examination of GAD.

3.1. Evolution and Applications of Functional Assessment

The idea that “problem” behaviour might become purposeful over time was first introduced by B. F. Skinner (1953), who argued that behaviour did not occur in isolation but in response to particular external (in the environment) or ‘inner skin’ (within the person) variables. He is credited with introducing systematic processes for observation, recording, and analysis of the variables thought to encourage instances of problem behaviour (i.e., maintaining variables). The experimental work done by Bijou, Peterson and Ault (1968) focused on application of observational and data-recording techniques to highly challenging behaviour as it occurred in the natural environment. Their aim was to establish procedures for identifying the functions of challenging behaviour via examination of how it varied in the presence of particular antecedent and consequent variables in the environment. The research of Bijou, Peterson and Ault (1968) which centred on people with developmental disabilities, was crucial to shifting the assessment focus from labelling behaviour to understanding its causes and establishing the procedures which underpin Functional Assessment of behaviour.

Initial applications of Functional Assessment were limited to people with severe behavioural challenges and minimal communication skills often associated with an intellectual or developmental disability. These groups were often excluded from educational and other opportunities due to their highly aberrant behaviour and Functional Assessment provided one basis for understanding their needs and assisting them to learn alternative methods for interacting with their environment (Carr & Durand, 1985; Iwata et al, 1994).
Since its initial applications, Functional Assessment procedures have been sufficiently elaborated to allow for investigation of numerous client groups. Clinical researchers such as Miltenberger (2004) argued that, despite commonalities in the data-collection and data-interpretation procedures which characterise Functional Assessment, there now exist a range of frameworks which have been developed to tailored investigation of particular groups, contexts, and presenting problems. Sturmey (2007) described four models for Functional Assessment and these are briefly discussed below to demonstrate the range of investigations into behaviour-environment relationships which are possible.

Functional Assessment of *individual cases* is generally applied to understanding the purpose of problem behaviour which is highly aberrant, disruptive to functioning, and appears to be unpredictable to the observer. Due to these features, the behaviour is submitted to in-depth and multi-method investigation to maximise the chances of identifying covert antecedents and subtle and/or idiosyncratic reinforcers. Examples of this type of Functional Assessment include investigation into suicidal threats (Sturmey, 1995) hallucinations (Layng & Andronis, 1984), and self-injury (Iwata & Neef, 1994). Functional Assessment of a *process* has been applied to examining skill deficits or atypical responses in children with developmental disabilities and the focus is on tracking how development of a particular skill set (e.g., verbal communication) occurs in a normally-developed sample. These data are used to build a template for establishing the pre-requisite skills necessary for the process to occur effectively. Data on the ways in which the process occurs in individual children are compared against this template to identify any missing pre-requisite skills and adverse environmental factors which might interfere with effective development of the target process. Examples of this type of Functional Assessment include investigations into imitation (McCuller & Salzberg, 1982) and communication (Koegel, 2000). Ecological Functional Assessment refers to a data-collection and interpretation process designed to identify the maintaining variables
which might impact the behaviour of a group of people participating in the same system or ecology. Data-collection focuses on identifying multiple behaviour-environment interactions to describe how the system impacts on members of the group. Examples of this type of functional assessment include examination of how social reinforcement influences student behaviour in the classroom and identification of the variables thought to inhibit completion of academic tasks (Bitsika, 2008).

Of great significance to this thesis is the Functional Assessment of a diagnostic category in which a DSM diagnosis (e.g., Generalised Anxiety Disorder) or cluster of symptoms (e.g., anxiety escalation) that are possibly indicative of the presence of a mental disorder, become the focal point for the investigation. The majority of research has dealt with diagnostic categories which have been operationalized and investigated in relation to their specific behaviours (symptoms), plus the relationships between those specific behaviours and particular environmental variables (e.g., presence of anxiety-provoking stimulus) and inner-skin experiences (e.g., negative self-talk) (Bitsika & Sharpley, 2006). Data are analysed to identify significant antecedent-behaviour and behaviour-consequence links to assist in understanding how problem behaviour might be functional as well as problematic. This type of Functional Assessment has applied learning theory to understand firstly how disorder develops and secondly how it persists over time; examples include investigations into the onset of Anorexia Nervosa (Slade, 1982), school refusal (Kearney & Silverman, 1990), and depression (Dougher & Hackbert, 1994).

Contemporary definitions of Functional Assessment emphasise that this is not a standard process but instead a flexible set of procedures which are applied to identify the causes of problem behaviour (Cooper, Heron & Heward, 2007). Despite the advances in generating tailored methods for conducting investigations, the core features of Functional Assessment continue to include observation, data recording, and data interpretation (O’Neill
et al., 1997). There is also consistency in the targets for data-collection (i.e., antecedents-behaviour-consequences) and these will be defined and explained in the subsequent discussion.

3.2. Contemporary Functional Assessment Procedures

Various researchers (e.g., Cooper, Heron & Heward, 2007; Miltenberger, 2004; O’Neill et al, 1997) have proposed that a comprehensive Functional Assessment should involve the clinician in collecting data on three factors: behaviour of concern (i.e., target behaviour), pre-behaviour events thought to encourage occurrences of the behaviour (i.e., setting events, antecedents), and post-behaviour events considered to strengthen behaviour (i.e., consequences, functions). These data are examined to develop hypotheses as to the reasons why behaviour occurs; those hypotheses are then employed to assist people in learning alternative strategies for responding to demand.

The Functional Assessment process begins with an operationalised definition of the behaviour which is causing the person concern and disruption in his/her capacity for effective functioning. This definition, which provides clarity on the specific aspect of performance to be assessed, is broadly considered in relation to whether it is external and observable (i.e., overt behaviour) or internal or not able to be observed (i.e., covert behaviour). In addition to this, the definition must account for the topography of the behaviour by identifying whether it is an action, verbalisation, cognition, emotion, and/or physiological state (Miller, 1997). Carr (2000) argued that data-collection on ill-defined behaviour will not only lead to partial or misleading measurement of that behaviour but also erroneous identification of maintaining variables. In addition to developing an operationalised definition, the clinician is required to decide on the dimensions to be used in measuring instances of the target behaviour in the natural context. Target behaviour might be measured in relation to one or more of four dimensions including: frequency (i.e., number of occurrences of behaviour per unit time),
duration (i.e., length of time for which one instance of behaviour lasts), intensity (i.e., magnitude of effort or resources involved in exhibiting the behaviour), and latency (i.e., time period between presence of specific trigger event and onset of behaviour) (Miller, 1997). Cone (1997), and Crone and Horner (2000) have recommended that clinicians be parsimonious in their inclusion of measurement methods by limiting the number of dimensions they employ to investigate behaviour. Interestingly, contemporary Functional Assessments, especially those which investigate DSM diagnostic categories, incorporate global measures of the mental disorder as well as precise measures of target behaviours. For instance, depression might be measured in relation to severity of depressive mood as well as specific behaviours such as crying and negative self-statements. Similarly, GAD might be best investigated via measures of general anxiety plus focus on specific avoidant behaviours (Bitsika & Sharpley, 2006).

The Functional Assessment also collects information on those events or conditions which precede the behaviour and appear to encourage its occurrence. Those pre-behaviour events are described in relation to their temporal relationship to the behaviour. The term setting events refers to a range of factors which have either occurred well before the behaviour (e.g., migraine several days before escalation in anxiety) or represent a general aspect of the environment in which the behaviour occurs (e.g., high demand at work leading to intense feelings of self-doubt) (Bitsika, 2007). Sturmey (2007) has cautioned that, in cases where behaviour appears not to be associated with any identifiable event in the immediate environment, it is probable that it is under the control of setting events. This appears to be particularly relevant to behaviour patterns which are generalised in nature (e.g., GAD, PTSD) and research has increasingly focused on identification of classes of setting events that might be significant to different types of mental disorder as opposed to specific behaviours. In contrast to setting events, antecedents are more readily recorded as they immediately precede
the behaviour of concern. However, because behaviour is often under the influence of multiple antecedents, it is important to group related types of antecedents to facilitate data analysis and decisions regarding treatments involving antecedent manipulation strategies (Miltenberger, 2004). Bitsika (2007) recommended that antecedent events might be grouped according to whether they are task-based (e.g., reading a challenging email), object-based (e.g., diary), or interaction-based (e.g., speaking with a supervisor). Basic antecedent grouping methods such as this not only highlight any antecedent types that are significant to occurrences of the behaviour but also provide a sound basis for educating the person about the activities which potentially cause distress.

The primary goal of Functional Assessment is to identify those events which follow instances of the behaviour and cause it to be strengthened over time. It is believed that behaviour which continues to occur, often despite causing discomfort to the person who exhibits it, does so because it is purposeful and adaptive. Assessment of post-behaviour events requires the clinician to describe the reinforcement processes which strengthen behaviour and the functions which give it adaptive value. Sturmey, 2007 stated that four reinforcement processes should be reviewed to understand the mechanisms by which behaviour is strengthened: (i) social positive reinforcement, (ii) social negative reinforcement, (iii) automatic positive reinforcement, and (iv) automatic negative reinforcement. Social reinforcement is considered to occur when the behaviour reliably leads to some change in the social environment which is meaningful to the person. Social positive reinforcement involves the onset of a liked or valued social event (e.g., recognition, sympathy) and social negative reinforcement results in removal of a disliked or aversive social event (e.g., conflictual interaction, stressful work meeting) (Birchler, Weiss & Vincent, 1975). Automatic reinforcement occurs when the behaviour creates some form of internal change that is not influenced by any aspect of the social environment. This form of reinforcement is closely
linked to the concept of private events which is used to refer internal or ‘within skin’ factors which influence behaviour but cannot be detected by an observer. Automatic positive reinforcement involves the onset of a liked and valued internal change (e.g., satisfaction, pleasure), and automatic negative reinforcement occurs when an adverse internal state ceases (e.g., anxiety, sadness) (Bitsika & Sharpley, 2006). Mineka and Oehlberg (2008) proposed that clinicians should review consequent events in relation to all four reinforcement processes but also caution that, in the case of automatic reinforcement, identification depends on self-reports from the person regarding any changes in internal state following behaviour.

Behaviour-consequence data are also analysed to determine the purpose (or functions) of the behaviour in order to determine how the behaviour interacts with the environment to produce a desired outcome. Numerous studies (e.g., Dunlap et al., 1993; Horner, Sugai, Todd & Lewis-Palmer, 2000; O’Neill et al, 1997) have shown that problem behaviour is capable of serving one or more functions such as escape, avoidance, attention, access to a preferred activity/object, and sensory reinforcement. Recent research suggests that the behaviours (or symptoms) associated with mental disorders serve a number of functions and these must all be identified in the Functional Assessment process.

3.3. Functional Assessment Procedures for Anxiety

Dymond and Roche (2009) argued that high-incidence disorders such as anxiety are rarely submitted to idiographic investigation, and discuss two issues which have possibly contributed to the reluctance of behavioural researchers to advance Functional Assessment models specific to anxiety. The first issue relates to difficulties in generating operationalised and precise definitions for anxiety. The second issue centres on misinterpretation of the concept of ‘private events’ or ‘inner skin’ variables by radical behaviourists.

Sturmey (2007) pointed out that there are 12 anxiety subtypes (or diagnoses) listed in the DSM-IV-TR (APA, 2000) which contain symptom clusters described primarily on the
basis of the intensity and duration of symptoms with no clear definition of the anxiety disorder which produces those symptoms. Barlow (2002) proposed a remedy to this definitional problem by suggesting that anxiety be conceptualised as a fear response which involves awareness and thoughts of constant danger, an increase in autonomic nervous system arousal, plus onset of overt and covert behaviours to ensure avoidance and escape. This reference to fear response acknowledges that stimuli which evoke fear-like reactions are often generic in nature, anticipated but not actually present, and pose no danger to the person’s well-being (Barlow, 2002; Sturmey, 2007). This is in contrast with authentic fear which is generated in relation to specific, imminent, and hazardous events. Further complication to development of operational definitions of anxiety arises from clinical accounts which focus on ‘inner skin’ symptoms such as worry, nervousness and irritability rather than overt responses, which in radical behaviourism terms do not offer a sufficient basis for observation and accurate measurement of behaviour (Anderson, Hawkins & Scotti, 1997).

However, more recently, researchers have emphasised that even Skinner (1975) explicitly recognised the existence of private events in the treatment of anxiety and have argued persuasively for inclusion of those events in the assessment of anxiety disorder (Friman, Hayes & Wilson, 1998; Mineka & Zinbarg, 2006). Friman, Hayes and Wilson (1998) suggested that ‘inner skin’ events should be incorporated in behaviour analytic conceptualisations of anxiety disorders and argued strongly for inclusion of emotions as targets for investigation in the assessment process. Those researchers proposed that language-proficient humans are capable of forming connections between neutral stimuli and their responses with no direct training. Humans are also capable of communicating with clinicians about those connections to enhance understanding of their personal experiences of anxiety and the stimuli which cause it. Further, the distinguishing feature of anxiety disorders is their
occurrence as a result of derived rather direct relations between overt and private responses aimed at serving avoidant functions. Thus, those researchers recommended that anxiety is suited to Functional Assessment investigation.

Researchers (e.g., Dougher & Hackbert, 2000; Lovibond, Saunders, Weidemann, & Mitchell, 2008; Bronfenbrenner & Morris, 2006; Sturmey, 2007) worked towards the development of four general guidelines for idiographic assessment of anxiety. The first guideline suggests that private events, as they relate to possible explanations for antecedents, target behaviours, functions and reinforcement processes, should be included in the assessment of anxiety. The second guideline suggests that, since fear responses relate to generic and often anticipated stimuli, particular attention should be given to the investigation of setting events as variables which set the conditions for anxiety. The third guideline derives from research (e.g., Dymond and Roche, 2009) argues that there is a close connection between the fear response and avoidant behaviour and requires in-depth investigation of the mechanisms for avoidance in order to understand how it facilities reduction in anxiety. The fourth guideline relates to the operant research (e.g., Miltenberger, 2004; Skinner, 1975) which suggests that avoidance results in negative reinforcement and suggests that determination of reinforcement processes must centre on social negative as well as automatic negative reinforcement. Interestingly, Woods, Miltenberger and Carr (2006) alerted the clinician to a two-step reinforcement process which applies in the case of fear-based behaviour and is distinguished in temporal terms and reinforcement type. Those researchers suggested that the immediate consequence to fear responses results in automatic negative reinforcement as evidenced by feelings of relief and deceleration of physiological arousal when avoidance succeeds. (This process is referred to as primary reinforcement.) However, once separation from fear-evoking stimuli is secured, it is argued that the person will generally engage in an activity which produces social and/or automatic positive
reinforcement. (This process is referred to as secondary gain.) A practical illustration of this two-step reinforcement process might involve a person experiencing a fear response (e.g., increased heart rate, trembling, and feelings of panic) triggered by a fear-inducing stimulus (e.g., imminent dentist appointment) leading to cancellation of the appointment and feelings of relief (i.e., automatic negative reinforcement as the primary reinforcement process) and initiation of contact with a friend to discuss the fear responses (i.e., social positive reinforcement as the secondary gain).

3.4 Proposed Functional Assessment Framework for GAD

The current study employed Functional Assessment of GAD as the method for establishing an in-depth understanding of fear-based behaviour and identifying the key maintaining variables for that behaviour. The Functional Assessment Model applied in this study reflected the traditional data-collection and data-interpretation procedures for best-practice investigation into problem behaviour as well as the guidelines which refer specifically to investigation of anxiety. It was considered to reflect the key components necessary to conduct an idiographic assessment of a diagnostic group but, due to a paucity of research specifically dedicated to Anxiety Disorders, the intention was to apply this model in a trial manner and then to evaluate its capacity to provide meaningful data in relation to participant anxiety-based experiences as these occurred in day-to-day life (refer below for Functional Assessment data-recording steps employed in this study):

1. Define the fear response in relation to overt behaviour and ‘inner skin’ responses;
2. Measure fear response in relation to frequency, duration, intensity, and latency;
3. Identify key setting events and antecedents for the fear response;
4. Decide whether post-behaviour consequences result in positive reinforcement or negative reinforcement (social and/or automatic);
5. Identify the functions of the fear response.
3.5 **Functional Assessment Data-Collection and Data-Analysis Methods**

O’Neill, Horner, Albin, Sprague, Story and Newton (1997) stated that the Functional Assessment process is embedded in the use of observational and data-recording procedures designed to investigate a presenting problem and determine the range of antecedent and consequent events that contribute to and maintain that problem over time. Specific data-recording procedures can include standardized tests of ability, self-report scales, interviews, and direct observations in natural and/or simulated environments (O’Neill, 1997). Selection of data-recording procedures is undertaken in relation to factors such as client features (e.g., age, ability level, communication skills), presenting problem topography (e.g., self-injurious behaviour versus generalised feelings of worry and nervousness), treatment applications (e.g., one-to-one Counselling therapy, parent training, classroom intervention). Factors such as these are important in determining the strategies for data-recording and it falls to the clinician to design an individualised data-recording process tailored to the particular needs of individual clients and their presenting problems.

Kohlenberg and Tsai (1991), in their application of Functional Assessment principles, design of assessment and psychotherapeutic approaches suited to the Counselling context, recommended that data-recoding should be undertaken within a client-therapist relationship based on strong rapport, trust, and empathy. Further, it is suggested by those researchers that interviewing comprise the major strategy for data-recording because Counselling clients are capable of communication, self-reflection, and self-observation. The importance of interviewing as a data-recording process is supported by Hanely (2012), who argued that ‘open’ assessment methods such as semi-structured interviews hold most promise for gaining in-depth and client-relevant data regarding problem behaviour and its probable causes. Because the research (Carr, 1994, 2000; Sturmey, 1996) has clearly demonstrated that Functional Assessment requires a multi-source investigation of problem behaviour,
interviews are commonly paired with other data-recoding methods such as rating scales and direct observation. These methods are particularly suited to assessment of diagnostic categories and can provide a basis for reviewing key trends across data sources so as to ensure that any conclusions on client anxiety symptomatology are accurate.

Multi-source data are submitted to in-depth analysis and review in order for the clinician to understand the presenting problem (i.e., target behaviour, topography, and dimensions) and its maintaining variables (i.e., setting events, antecedents, functions). Most importantly, those data are used to generate a number of hypotheses regarding the purpose of target behaviour to further understand how particular behaviour which causes client distress might also be assisting him/her to adapt to and cope with demand. These hypotheses are thought to enhance the psychotherapeutic process by assisting the client to understand the adaptive aspects of their ‘problem’ and to form the foundation for individualised treatments aimed at creating client-focused (e.g., building coping behaviours to compete with and eventually replace concerning behaviours) and environment-focused changes (e.g., modifying antecedents shown to trigger use of concerning behaviours) (Kohlenberg & Tsai, 1991; Sturmey, 1996).

There is some confusion in the literature about the distinction between Functional Assessment and Functional Analysis, with these terms being used interchangeably in various studies. Mace (1994) and O’Neill et al. (1997) sought to clarify this confusion by proposing that a Functional Analysis of behaviour involves all the data-recording and data-interpretation procedures central to a Functional Assessment, plus the inclusion of a final process described as a systematic manipulation of consequent variables following instances of target behaviour in order to confirm the veracity of hypotheses in explaining the purpose of that behaviour. This confirmation, which required formal experimentation often conducted in a laboratory to minimize contamination of consequent variables, was historically devised for people with
developmental disabilities and is not suited to idiographic investigations of regularly-functioning adults.

This thesis was concerned with idiographic assessment of a group of adult participants with a diagnosed mental disorder (i.e., GAD) and applied multi-source assessment via semi-structured interviews and standardized anxiety scales within the context of the client-therapist relationship. Assessment data were submitted to an in-depth review to understand the particular ways in which GAD manifested itself and the impacts of that condition on the day-to-day functioning of the participant group. The results of the Functional Assessment for GAD are discussed in Chapter 6 of this thesis.
CHAPTER 4

What is Biofeedback?

Basmajian (1979) defined biofeedback (BFB) as a process whereby an organism (in this case, humans, although the biofeedback process is not restricted to human usage) can learn to modify physiological events in the body when these are displayed on a recording device, either as visual and/or auditory signals. That is, BFB is based upon learning principles of (i) exhibition of a specific physiological aspect of behaviour (e.g., heart rate) on some modality or replicate such as a pc screen, audio device, or other combination of audio and visual stimuli that accurately depict the physiological behaviour under scrutiny in a time-dependent manner so that moment-to-moment variations in the physiological behaviour are reflected in the audio-visual (A/V) depiction, (ii) awareness of that depiction by the person whose physiological function is being measured and depicted, and (iii) graduated self-control learning that enables the person to reliably alter their physiological function at will.

That is, the first stage of BFB requires that the person provides access to their particular physiological behaviour that is chosen for examination. While perhaps obvious, this stage is vital in terms of the overall process of BFB. Without voluntary provision of access to the measurement devices required to measure the physiological behaviour under scrutiny, the second and third stages are unlikely to follow. Therefore, as well as being a significant ethical issue in research and clinical treatment, voluntary access to the person’s physiology is a requirement of BFB and thus ensures that there is no uncertainty about the person’s ‘buy-in’ to the therapy and to their engagement in the therapy process via the Therapeutic Alliance. This is an important step in ensuring that, in clinical settings such as those described in this thesis; the client is an informed and willing part of the overall therapy process.
The second stage of BFB is open to some development and variation according to the sophistication (and budget) of the therapist or researcher. As will be described later in the Methods chapter, the equipment used in this thesis represented the most non-invasive available at the time of the research and so minimised the (perhaps daunting for some clients) challenge of being attached to electronic equipment. As will be described later, the BFB equipment used in this thesis presented a visual and auditory signal for clients to see and hear.

The third stage of BFB relies upon operant principles of learning in that the person undergoing BFB is aware of the A/V depiction of their physiological behaviour and then gradually also becomes aware that the signal (i.e., the A/V depiction) varies from time to time. This vital third step in BFB allows the person to experiment with other physiological behaviours that may influence the behaviour being depicted. In the present thesis, the behaviour being depicted is heart rate (HR) and this naturally varies with breathing as a function of the respiratory-sinus-arrhythmia changes which bring HR under the control of the Sympathetic Nervous System (SNS) when the person inhales and then under the control of the Parasympathetic Nervous System (PNS) when the person exhales. This SNS-PNS exchange over breathing cycles produces a gradual increase and decrease of HR during those breathing cycles. When training people to use HR BFB, it is awareness of this SNS-PNS variability in HR which is the first aspect of their learning. That is quickly followed by experimentation by the person receiving BFB so that they vary their breathing cycle (e.g., extend the exhale period) to see what effects this has upon the HR. Thus, the person is then effectively in control of some aspects of their HR and has achieved this by the BFB process.

Thus, the definition of BFB by Hayashino et.al, 2010 as a method helping individuals learn how to control various physiological processes such as muscle tension, blood pressure, breathing, heart rate, brainwave states, skin temperature and skin conductance measured by non-invasive sensors and providing visual feedback to the participant to consciously control
the physiological body reaction may be seen to rely upon the three stages of depiction, awareness and learning described above. Put in other terms, Kaushik (2007) defined BFB as a form of self-regulation in which individuals learn to control physiological responses by providing them with an information signal as sensory feedback about biological conditions of which they may not be ordinarily aware. Biofeedback also involves the application of operant conditioning to gain control of visceral, somatmotor or central nervous system activities.

4.1. **Biofeedback in Clinical Settings for Stress Hyperarousal**

When applied to persons with over-arousal as a clinical presenting problem, BFB can be used as a psychophysiological assessment tool used to teach those persons methods of reducing their arousal (Rice, Blanchard & Purcell, 1994). In such cases, BFB fits into the overall stress-reduction protocol in as steps 2 and 3 of the following three steps (Sharpley, Guidara & Lancaster, 1996).

1. Identifying the antecedent that causes the reactivity
2. Assisting the participant to recognise the physiological response to that antecedent
3. Teaching the participant new coping skills (ie, BFB) to reduce the reaction between the event and the physiological reactivity

The most common form in which BFB is provided (called its “modality”) in clinical settings is dictated by the particular physiological behaviour which is being monitored for change.

For example, if muscle tension is the target physiological response (e.g., for people with tension headaches), then **Electromyogram (EMG) Biofeedback** is used wherein electrodes or sensors are applied to the specific muscle group under examination and the electrical signal of those muscles is presented in A/V format, typically by a line graph on a screen or an increasing/decreasing tone that varies with the amount of electrical activity in the
muscle spindles with the muscle group being examined. For example, for a person who suffers from severe headaches as a result of prolonged tension of their neck muscles, BFB provides a depiction of the electrical activity in those neck muscles. If, as would be expected in such cases, the initial level of electrical activity in the neck muscles was high, then the client is set the task of trying to reduce that tension by (say) deep breathing, voluntary relaxation of the muscle group, or concentrating upon different mental imagery than that which is associated with the tension. In such cases, the use of purely auditory BFB can be efficacious due to the value of the client being able to close their eyes during this process.

Another commonly-used modality for BFB is **Electrodermal Activity (EDA) Biofeedback**, wherein the activity of sweat glands and the amount of perspiration on the skin can be indirectly assessed by sensors which measure the degree of resistance to a very small (in the region of 500 microvolts) electrical current that is passed between two electrodes on (say) adjoining fingers of one hand. The hand is often chosen in such cases because of the tendency of primates to sweat on their extremities during periods of SNS arousal instigated by environmental stress. However, while EDA is more effective as a measure of SNS activity than any other physiological index, it has the major limitation of being unsuitable for the third stage of BFB (i.e., self-control) due to the extended presence of sweat on the hands, which prevents the person who is receiving this BFB from learning how to reverse that sweat production as a means of controlling SNS arousal to stressors in the environment. This is an important limitation when dealing with clients who present with anxiety in the form of exaggerated SNS arousal. As will be explained below, HR feedback is more efficient for this process.

**Skin temperature (ST) Biofeedback** is another method for measuring SNS activity because blood flow to the extremities is reduced when SNS arousal occurs. This reduction in blood flow causes reductions in skin temperature, and so can be a reliable measure of anxiety.
However, as for EDA activity, there is an inherent limitation in using ST BFB for teaching clients to self-control anxiety because of the slow rate of reduction in skin temperature that occurs when SNS arousal occurs, and in skin temperature increase when relaxation occurs. This skin temperature change process can take several minutes, even as much as 15-20 mins, thus making it relatively difficult for clients to match the increase in skin temperature with their cognitive or behavioural attempts to reverse the decrease in skin temperature due to SNS arousal. However, it should be noted that, when clients do learn to control their skin temperature, it appears to be a profound form of self-control learning which they report to be highly efficacious for them.

**Electroencephalograph (EEG) Biofeedback** or **Neurofeedback** measures the frequency and relative amounts of brain electrical activity across various sites on the skull. Although not a facet of this thesis (and a very complicated procedure) put simply, EEG BFB is often used to train clients to induce alpha-wave activity (about 8 to 13 Hz) instead of beta-wave activity (13+ Hz) when they are experiencing the symptoms of cognitive stress arousal such as racing thoughts, mental confusion, and inability to make decisions. Alpha-wave activity is associated with a pleasant feeling of mental relaxation and occurs when a person is lightly drowsing or daydreaming.

As mentioned above, this thesis reports on the use of **Heart Rate (HR) Biofeedback** to assist clients to reduce their anxiety levels. During HR BFB, an electrode applied to a finger or earlobe monitors blood flow from the cardiac pulse as an indicator of heart rate (HR) and heart rate reactivity (HRR). HR is simply the reciprocal of the inter-beat interval between successive R-R spikes that are occasioned by electrical stimulation of the cardiac muscle by electrical signals from the brain that contact the heart at the sino-atrial node, a bundle of nerve fibres on the surface of the heart. Thus, instead of depicting the inter-beat intervals in milliseconds, the number of beats per minute is shown on a pc screen or by auditory methods.
HRR refers to the increase in HR following the onset of a stressor, and is usually calculated by comparing the mean HR during the minute immediately preceding the stressor with the mean HR during the minute immediately following the onset of the stressor (Sharpley & Gordon, 1999). HRR has been described by Sharpley, Scuderi and Heffernan (1989) as the predisposition of humans to react to the onset of a physical or psychological trigger by rises in heart rate which exceed those required by the motor responses triggered by the stressor. That is, HRR is most often used to refer to the ‘psychological’ aspect of an individual’s HR response to a stressful event.

4.2. Types of Disorders Suited for Treatment by Biofeedback

A method for evaluating the effectiveness of BFB for a range of disorders has been developed by the Association for Applied Psychophysiology based on research from Moss and Gunkelman (2002) and LaVague et.al (2002), and is presented in Table 1.

<table>
<thead>
<tr>
<th>Level</th>
<th>Description of efficacy level of biofeedback</th>
<th>Disorder</th>
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<tr>
<td>Level 1</td>
<td><em>Not empirically supported</em>: Supported only by anecdotal reports and/or case studies in non-peer reviewed venues.</td>
<td>Autism, Eating Disorders, Multiple Sclerosis, Spinal Cord Injury</td>
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<tr>
<td>Level 2</td>
<td><em>Possibly Efficacious</em>: At least one study of sufficient statistical power with well identified outcome measures, but lacking randomized assignment to a control condition internal to the study.</td>
<td>Asthma, Cancer and HIV, Effect on Immune Function, Cerebral Palsy, Chronic Obstructive Pulmonary Disease, Depressive Disorders, Diabetes Mellitus, Fibromyalgia, Foot Ulcers</td>
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Level 3  *Probably Efficacious*: Multiple observational studies, clinical studies, wait list controlled studies, and within subject and intrasubject replication studies that demonstrate efficacy.

Level 4  *Efficacious* a.) In a comparison with a no-treatment control group, alternative treatment group, or sham (placebo) control utilizing randomized assignment, the investigational treatment is shown to be statistically significantly superior to the control condition or the investigational treatment is equivalent to a treatment of established efficacy in a study with sufficient power to detect moderate differences, and
b.) The studies have been conducted with a population treated for a specific problem, for whom inclusion criteria are delineated in a reliable, operationally defined manner, and
c.) The study used valid and clearly specified outcome measures related to the problem being treated and
d.) The data are subjected to appropriate data analysis, and
e.) The diagnostic and treatment variables and procedures are clearly defined in a
manner that permits replication of the
study by independent researchers, and
f.) The superiority or equivalence of the
investigational treatment has been shown
in at least two independent research
settings.

Level 5  *Efficacious and specific:* The
investigational treatment has been shown
to be statistically superior to credible sham
therapy, pill, or alternative bona fide
treatment in at least two independent
research settings.

Using these criteria, Kuashik (2007) performed a meta-analysis on the effectiveness
of BFB for the treatment of a range of disorders and found that BFB is effective as a
treatment modality for anxiety, headaches, hypertension, coronary heart disease, epilepsy,
chronic pain, constipation, faecal incontinence, irritable bowel syndrome, bronchial asthma,
diabetes mellitus, cardiovascular rehabilitation and post-stroke rehabilitation. (see Appendix
C for level 5 efficacy studies for BFB and Appendix D for level 4 efficacy studies for BFB)

However, most studies reviewed in this thesis acknowledge that BFB is not always
effective when used as the only intervention, but that the efficacy rate of BFB biofeedback is
increased when BFB is combined with another treatment modality, such as various forms of
psychotherapy or relaxation therapy.

4.3.  **Biofeedback and Anxiety**

Yucha and Gilbert (2004) published an efficacy rating for the use of BFB as a
treatment for different disorders, and rated BFB for anxiety as ‘efficacious’. However,
although that review of the literature suggested that EEG was the most common form of BFB
treatment for anxiety disorder, it is noteworthy that, in those cases where EEG was applied as
a treatment for anxiety, it was delivered alone, as the sole treatment. However, as noted
above, BFB has been shown to be most effective when combined with other treatments. Further, behaviourally-oriented and psychosocial guidelines for the effective treatment of mental disorder (Ford & Kidd, 1998; Burns, Hoagwood & Mrazek, 1999; Kessler et al., 2005) recommend that multi-modal treatments are necessary for generalisation of skills learned in the clinic environment. Therefore, biofeedback should be part of a treatment package and not applied alone.

When deciding on which BFB modality to use with a specific disorder, it is crucial to consider the self-efficacy of clients in their capacity to create positive internal changes. For clients with anxiety, any delay or failure to create such changes can negatively impact on their treatment success. Therefore, in choosing the best treatment, the BFB modality should consider the influence of self-efficacy. However, EEG (due to requiring a longer learning time) might not be the best option for clients with intense anxiety and who require fairly fast treatment effects to become apparent. Therefore, in this study it was decided to use HR BFB as a means of training clients to become aware of their SNS-arousal states, how those states might be associated with specific anxiety-linked stressors, and thence to learn methods of reducing HR (and SNS arousal) that are generalisable to their usual working/living environments.
CHAPTER 5

Method

5.1 Participant Group

Participants were required to fulfil four criteria in order to be eligible for inclusion in this study. They were required to be a minimum age of 16 years, fulfil the DSM-IV-TR (APA, 2000) criteria for a formal diagnosis of Generalised Anxiety Disorder (GAD), and exhibit general good health (i.e., no symptoms of major cardiovascular, respiratory or endocrine illness for which they were receiving medical treatment). The fourth inclusion criterion required that participants not use any prescribed anxiolytic medication, autonomic nervous system-altering substances or illicit substances for the duration of their involvement in the study. All participants signed a disclaimer stating that they were not using medications of this type. Intake of stimulants (e.g., caffeine) and alcohol intake were monitored via verbal report to the researcher who recorded weekly caffeine and alcohol intake for each participant.

Twenty-one adults, comprising four academic staff, five general staff, and twelve student volunteers, underwent an initial screening interview to establish their eligibility for inclusion in this study (see Appendix E: Inclusion/Exclusion Criteria). Of the 21 initial respondents to an invitation to volunteer for the study, four were excluded due to the presence of an anxiety-based diagnosis other than GAD (i.e. Post-Traumatic Stress Disorder, Obsessive Compulsive Disorder, and Social Phobia), one was excluded due to presence of a physical illness (Myocardial Infarction), two were excluded as a consequence of using prescribed medication (i.e. Beta Blockers and Benzodiazepines) and two were excluded as a consequence of illicit drug use (i.e. Cannabis and Amphetamines). One initial respondent was excluded due to presence of a physical illness as well as using prescribed medication (i.e. Hyperthyroidism and Thyroxin). Therefore, the participants for this study were 11 adults who worked or studied at a private University on the Gold Coast in Queensland. Of these 11
participants, four (36%) were male and seven (64%) were female. They ranged in age from 18 to 57 years, with a mean age being 33.6 years (SD = 10.97). Ethnicity trends for the participants showed that the majority (i.e., seven) were Australians, two were Norwegians, one was Malaysian, and one Chinese. Below is a detailed description of each participant for this study.

5.1.1 Participant 1

Participant 1 was an 18 year old female studying an undergraduate degree in Psychology, and she reported symptoms which complied with the DSM-IV-TR (APA, 2000) criteria for a diagnosis of GAD. She reported being in good physical health and stated that she did not engage in stimulant or illicit drug use. The only medication she used during the duration of this study was paracetamol for headaches. This participant was single, did not have any dependants and lived alone. Participant 1 underwent counselling for approximately six months, five years prior to this study, to resolve personal difficulties pertaining to childhood sexual abuse. The psychotherapeutic interventions used to assist this participant included Cognitive Behaviour Therapy and Hypnosis. Participant 1 reported that she experienced moderate to severe anxiety “in all aspects of life”. She mentioned a strong history of anxiety-related conditions in the extended family. Her mother and two sisters were medically treated for anxiety-related disorders and her two sisters were also seeing therapists. Participant 1 reported that she did not undertake any form of exercise and did not compete in any sport.

5.1.2 Participant 2

Participant 2 was a 53 year old male senior academic working in the Faculty of Business at the University in which this study was undertaken. He complied with the diagnostic criteria for GAD as defined by the DSM-IV-TR (APA, 2000). He reported being in good health and stated that he did not engage in stimulant or illicit drug use. He had been
diagnosed with Hypercholesterolemia and treated with a cholesterol-lowering drug. He was married for 27 years and had four children, two male and two female, aged between 19 and 25 years. Participant 2 reported that his relationships with his wife and children were positive and supportive. Participant 2 reported that he had not undergone any previous medical treatment or psychological interventions for his anxiety. He had no family history of any anxiety-related conditions. He mentioned that, due to a very heavy workload and extra responsibilities, he had noticed an increase in his anxiety levels which had become progressively more severe during the two years prior to the study. Participant 2 reported that he worked seven days a week and his exercise regime consisted of a brisk walk for approximately two kilometres once a week.

5.1.3 Participant 3

Participant 3 was a 20 year old female student studying an undergraduate degree in Information Technology and she presented with the combination of symptoms necessary for a diagnosis of GAD as defined by the DSM-IV-TR (APA, 2000). She also suffered from Irritable Bowel Syndrome but was not on any medical treatment for this condition during the duration of the study. Participant 3 consumed one cup of instant coffee every morning and reported that she did not engage in illicit drug use. She was in a de facto relationship, sharing an apartment with her partner. She reported a stable, supportive relationship. Participant 3 reported that she had not undergone any previous medical treatment or psychological interventions for her anxiety. Participant 3 had a history of suffering from panic attacks and experienced the last attack approximately one year prior to the commencement of the study. She described herself as being “highly strung” with a low grade of anxiety present all the time and reported that her mother and grandmother both suffered from anxiety-related conditions. Her physical fitness schedule consisted of three to four gym sessions per week,
including weight training and aerobic exercise. The duration of each session was approximately one hour.

5.1.4 Participant 4

Participant 4 was a 57 year old male senior academic working in the Faculty of Information Technology who complied with the diagnostic criteria for GAD as defined by the DSM-IV-TR. (APA, 2000). He reported being in good physical health but being very sensitive to any stimulants including caffeine and therefore avoided coffee and other caffeine-based beverages. He was diagnosed with Hypertension four years prior to this study and was being treated with anti-hypertensive medication (not a β-receptor blocker that would have affected the autonomic nervous system). No other medication or illicit substances were consumed during the duration of the study. He had been married for 34 years and had seven children, three male and four female, aged between 22 and 32 years. He reported that the relationship has been stable without any major problems. Participant 4 reported that he had not undergone any previous medical treatment or psychological interventions for his anxiety. He reported that, due to a heavy workload, he developed anxiety-related symptoms which had become progressively more severe during the previous 16 months. His physical fitness schedule consisted of playing tennis most Saturdays for approximately 3 hours with no opportunity to partake in any other form of exercise.

5.1.5 Participant 5

Participant 5 was a 39 year old female junior academic in the Faculty of Humanities and Social Sciences who fulfilled the criteria for a diagnosis of GAD as defined by the DSM-IV-TR (APA, 2000). She suffered from mild Asthma but did not receive any medical treatment for this condition during the duration of the study. Participant 5 consumed three cups of coffee per day but did not report the use of any other medication or illicit substances during the duration of the study. She was in a defacto relationship and shared a house with
her partner; this participant reported that she did not have any children. Participant 5 reported that the relationship was unstable and that she and her partner had two trial separations during the previous year. She reported suffering from moderate anxiety-related symptoms for approximately the last 15 years. These symptoms had started to affect most aspects of her life two years prior to commencement of this study. Participant 5 reported that she had not undergone any previous medical treatment or psychological interventions for her anxiety. She mentioned a strong history of anxiety-related conditions in her extended family. Her father, mother, sister and brother were medically treated for anxiety-related disorders and, in addition to taking medication, her father and mother were seeing therapists. Participant 5 attended the gym on an irregular basis and did not partake in any other form of exercise.

5.1.6 Participant 6

Participant 6 was a 28 year old male student studying an undergraduate degree in Languages and he complied with the diagnostic criteria for GAD as defined by the DSM-IV-TR (APA, 2000). Participant 6 reported leading a healthy lifestyle and avoided all stimulants. He did not use any medication during the duration of the study and reported no illicit drug use. He was in a defacto relationship sharing a unit with his partner and had no dependants. He described their relationship as being stable. He described himself as being a very anxious and nervous child growing up in an unstable family with his parents going through a divorce when he was six years old. He had undergone medical treatment for persistent anxiety-related symptoms which consisted of Benzodiazepines but had ceased this medication due to addiction problems. Participant 6 had not undergone any previous psychological interventions. He suffered from severe anxiety-related problems on a daily basis. Participant 6 reported that he had no family history of any anxiety-related conditions and that he did not partake in any regular physical exercise.
5.1.7 Participant 7

Participant 7 was a 24 year old female student studying an undergraduate degree in Business Management. She complied with the diagnostic criteria for GAD as defined by the DSM-IV-TR (APA, 2000) and reported symptoms of gastro-intestinal discomfort but described her general health as being good. Participant 7 consumed one to two caffeine-containing soft drinks per day but reported no use of illicit substances or any other medication. She was single, did not have any dependants and lived alone. She reported suffering from anxiety from the age of 11 years. Participant 7 reported that she had not undergone any previous medical treatment or psychological interventions for her anxiety. She mentioned that both her father and sister suffered from anxiety-related disorders and that both were being medically treated in an effort to control their symptoms. Participant 7 reported that she swam four times per week for approximately 45 minutes and went to the gym three times per week doing cardiovascular exercises for 60 minutes per class.

5.1.8 Participant 8

Participant 8 was a 32 year old female working as an administrative staff person in the Faculty of Law who complied with the diagnostic criteria for a GAD as defined by the DSM-IV-TR (APA, 2000). She suffered from mild Asthma but was not receiving any medical treatment for this condition. She did not use any medication or illicit drugs during the duration of the study. Participant 8 reported drinking two cups of coffee per day and two glasses of red wine at night. She had been married for four years and had a five year old boy. She described her relationship as being happy and stable. Participant 8 described an unstable childhood with her parents going through a divorce when she was five years old and reported experiencing an incident of sexual abuse at the age of seven. She underwent counselling as a child but could not identify or describe the specific therapy used during these sessions. This participant reported that the current anxiety-related symptoms she experienced had been
triggered by a panic attack four years ago. There was no family history of any anxiety-related symptoms. Participant 8 did not partake in any regular physical activities or in any sports.

5.1.9 Participant 9

Participant 9 was a 23 year old female student studying an undergraduate degree in Information Technology who fulfilled the criteria for a diagnosis of GAD as defined by the DSM-IV-TR (APA, 2000). She reported being in good physical health and stated that she did not engage in stimulant or illicit drug use, stating that she refrained from drinking caffeinated and alcoholic drinks. The only medication she used during the duration of the study was aspirin for headaches. She had been married for two years and had two children, a four year old boy and a two year old girl. Participant 9 reported her relationship as stable and uneventful. Participant 9 described herself as having been a sensitive and anxious child and reported experiencing symptoms of anxiety throughout her life. She did not report receiving any previous psychological or medical interventions for her anxiety-related symptoms. There was no family history of any anxiety related symptoms. She reported living a healthy lifestyle which consisted of regular yoga sessions, meditation and a healthy diet.

5.1.10 Participant 10

Participant 10 was a 30 year old female working as a cleaner at Bond University and she complied with the diagnostic criteria for GAD as defined by the DSM-IV-TR (APA, 2000). She was HIV positive and had been receiving antiviral medical treatment for the last 18 months. Her general health was good and she was not receiving any other form of medical intervention during the duration of this study. Participant 10 stated that she did not engage in stimulant or illicit drug use. She consumed two cups of coffee per day. Participant 10 reported being divorced with two children, seven and ten year old girls. She reported that she had suffered from anxiety since early childhood but that her pre-existing anxiety-related symptoms worsened after she underwent drug rehabilitation five years prior to the study.
Participant 10 was not involved in any formal exercise programmes but reported being physically active due to her work.

5.1.11 Participant 11

Participant 11 was a 46 year old male student studying an undergraduate degree in Law and he reported experiencing symptoms which met the criteria for a diagnosis of GAD as defined by the DSM-IV-TR (APA, 2000). Participant 11 stated that he was in general good health but suffered from occasional headaches. He reported that he was not taking medication or engaging in illicit drug use but did report consuming one to two cups of coffee per day. This participant was divorced with two children, a boy aged 17 and a girl aged 19. Participant 11 reported being anxious for two years prior to this study but could not identify any reason for the onset of anxiety-related symptoms. He reported that he had not received access to any medical or psychological interventions to assist him in dealing with his growing anxiety. Participant 11 reported that he exercised reasonably regularly by taking two to three brisk beach walks per week.

5.2 Assessment Instruments

The ideographic assessment of participants included an initial intake interview to confirm the diagnosis of Generalised Anxiety Disorder according to DSM criteria. Once the presence of this anxiety disorder was established, the severity of anxiety and the profile of anxiety symptoms were measured via self-report using the Zung Self-Rating Anxiety Scale (1971). Finally, each participant completed a Functional Assessment interview in order to provide in-depth information on the particular ways in which anxiety manifested itself and the impacts it exerted on their day-to-day functioning. Below is a description of the assessment instruments employed in this assessment process.
5.2.1 Zung Self-Rating Anxiety Scale (Zung, 1971)

To quantify the severity of the participants’ anxiety-related symptoms, the Zung Self-Rating Anxiety Scale (SAS) was administered individually to each participant. The SAS gives standardised measures of physiological, cognitive and emotional symptoms of anxiety. The SAS is self-administered and consists of 20 items which are divided into physiological (muscle tremors, physical aches and pains, weakness, palpitations, shortness of breath, numbness, dizziness, nausea, fatigue, urinary frequency, sweating, flushing restlessness and insomnia), cognitive (mental disintegration) and emotional (nervousness, fear, panic, trepidation, and nightmares) symptoms of anxiety (Zung, 1971, 1980). The SAS is a brief and concise test that was used to make comparisons of participant’s anxiety status before and after treatment.

The SAS was completed by each participant for the period of one week prior to the interview. Each symptom on the SAS is rated on an intensity scale as “none or little of the time”, “some of the time”, “good part of the time”, or “most or all of the time”. Raw scores are derived from these ratings and may be converted to index scores (not necessary and not used in this study). High raw scores indicate severe anxiety and low raw scores indicate moderate to low levels of anxiety. Raw scores greater than 30 indicate clinically significant anxiety.

5.2.1.1 Validity

The SAS is based on the DSM-II and accounts for somatic, cognitive and affective symptoms of anxiety (Zung, 1971) and therefore the scale’s content validity (face validity) is high (Zung, 1980). The concurrent validity between the SAS and the Taylor Manifest Anxiety scale has been shown to be acceptable ($r = .30$, $r = .62$, $p < .01$; Zung, 1971, 1980) as well as between the SAS and the Hamilton Anxiety Scale ($r = .75$, $p < .01$; Zung, 1980, Sharpley, Bitsika & Efremidis, 1997). Construct validity was sound and the SAS
differentiated well, with a clear distinction between individuals with and without anxiety (Zung, 1980).

5.2.1.2 Reliability

Reliability for the SAS is clinically acceptable, ranging from 0.71 (split half: Zung, 1971) to 0.79 (coefficient alpha: Sharpley & Rogers, 1985) and 0.85 (coefficient alpha: Zung, 1980). Therefore, the SAS has adequate levels of internal consistency.

5.2.2 Diagnostic Criteria for Generalised Anxiety Disorder

The researcher developed a symptom checklist using the diagnostic criteria for Generalised Anxiety Disorder as presented in the fourth and text revised edition of the American Psychiatric Association’s Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR, 2000). This checklist contained the six diagnostic criteria for GAD and the six symptoms listed under criterion C. The DSM-IV-TR (APA, 2000) GAD checklist was administered at the end of the intake interview via the researcher asking each participant questions designed to investigate their experiences in relation to each diagnostic criterion. All participant responses were noted during this face-to-face interview and checked for accuracy by the researcher. A copy of the checklist appears in Appendix A.

5.2.3 Functional Assessment Interview Schedule

A formal interview schedule based upon Functional Assessment procedures, as described in Chapter 3, was developed and appears in Appendix F of this thesis. This face-to-face interview was designed to record participant self-reported data on demographic information (biographical details, family history and medical history), presenting problems and fear responses (i.e., anxiety-related symptomatology in the participant’s own words), setting events and antecedents for anxiety-based behaviours; as well as reinforcement processes for and functions served by these behaviours. The interview data were analysed in order to determine the variables which might predispose participants to engage in fear
responses in day-to-day life (see Appendix F for the Functional Assessment interview schedule).

5.3 Methods for Measuring Change in Participant Anxiety

This study was designed to use two procedures to measure change in self-reported somatic symptoms and, to a lesser extent, cognitive/emotional indicators of anxiety. First, a pre- and post-treatment procedure (involving administration of the SAS and physiological baseline) was used to compare level and intensity of anxiety symptoms before and after treatment application. Second, continuous measures of heart rate were taken during sessions III and VII (treatment phase) in order to establish a cumulative measure of any incremental changes in heart rate indicating a reduction in physiological reactivity (see Table 2 below for summary of measurement strategies). These HR data were also used to calculate Resting Heart Rate (RHR) and Respiratory Sinus Arrhythmia (RSA), which is defined as the difference between maximum HR when inhaling (HR comes under the control of the Sympathetic Nervous System [SNS] when inhaling) and the minimum HR when exhaling (when HR comes under the control of the Parasympathetic Nervous System [PNS]). Thus, RSA is depicted in beats per minute difference between maximum HR and minimum HR during a breathing cycle. RSA has been used as an index of SNS-PNS ‘balance’ in studies of psychological stress and anxiety (Dishman et al, 2000; Pancheri et al, 2002), and may be used as a method of providing immediate feedback to participants via the depiction of their HR on a computer screen. Such training procedures have shown their efficacy in helping people who are anxious to reduce their RHR and HR during stressful events in the clinic and workplace (Sharpley, 1994b).

The Functional Assessment process was used only at pre-treatment to conduct an idiographic investigation of anxiety.
Table 2:

Summary of Measurement Strategies

<table>
<thead>
<tr>
<th>Treatment Phase</th>
<th>Assessment Focus</th>
<th>Assessment Instrument</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-Treatment</td>
<td>Level and intensity of anxiety</td>
<td>Zung Self-Reported Anxiety Scale</td>
</tr>
<tr>
<td></td>
<td>Anxiety as a fear response and maintaining variables</td>
<td>Functional Assessment Interview</td>
</tr>
<tr>
<td></td>
<td>Somatic anxiety via heart rate</td>
<td>Bioview series IV, Zencor®</td>
</tr>
<tr>
<td>During Treatment</td>
<td>Graduated variation in anxiety</td>
<td>Bioview series IV, Zencor®</td>
</tr>
<tr>
<td>Post-Treatment</td>
<td>Level and intensity of anxiety</td>
<td>Zung Self-Report Anxiety Scale</td>
</tr>
<tr>
<td></td>
<td>Somatic anxiety via heart rate</td>
<td>Bioview series IV, Zencor®</td>
</tr>
</tbody>
</table>

5.4 Procedure

5.4.1 Recruitment

Participants were recruited from the researcher’s campus. An e-mail was send to all staff and students inviting their participation in this study (see Appendix G for e-mail to staff and students). A total of 21 staff members and students responded. After this initial interest was demonstrated, a receptionist at the Bond University Counselling Clinic recorded the potential participant’s contact details. Each potential participant was then contacted via telephone by the researcher in order to provide an explanation of the study’s aims, procedures and anticipated time commitments as outlined in the Explanatory Statement for the research project. Those people who expressed interest in participating in the study were invited to attend an intake interview to establish whether they met the inclusion criteria as specified below.

Criteria for inclusion were: having a diagnosis of GAD, being 16 years of age or older, having general good health, and not receiving treatment for any medical condition that might influence their neurological or cardiovascular responses. In addition, participants were asked not to use Autonomic Nervous System stimulants during the duration of the study.
On arrival at the clinic, the receptionist handed each potential participant an intake form (see Appendix H for the Intake Form). Each potential participant then met the researcher, and was taken to the interview and treatment room for the first intake interview.

The potential participants were informed that the first two sessions had no therapeutic intent. It was made clear to them that they were under no obligation to continue with this study and that they could terminate their participation at any time without any repercussions. An Explanatory Statement was handed to participants and explained during the initial stage of the intake interview (see Appendix I for the Explanatory Statement: Part 1). Due to the fact that all the participants were recruited from campus, and therefore could be identified at a future time by the researcher, the confidentiality clause outlined in the Explanatory Statement was emphasized. All aspects of the study were explained to potential participants and queries were answered.

If a potential participant decided to partake in this study, he/she was then handed a consent form (see Appendix J for the Consent Form). The consent form provided participants with a formal basis for confirming that they understood the explanations offered in relation to procedures, confidentiality, and voluntariness. All signed consent forms were collected and stored under lock and key for the duration of time specified by the Bond University Human Research Ethics Committee.

The participant was then familiarised with the setting and equipment. The treatment regime schedule was then explained in detail. If a participant decided to continue with this study, he/she was requested to make a follow-up appointment to commence the study.

5.4.2 Setting, Assessment and Treatment Phases, and Biofeedback Equipment

5.4.2.1 Setting

All sessions were conducted in the Counselling Clinic at the University by the researcher using the same clinic room to maintain consistency in environment. The clinic
room was soundproofed and approximately three by four meters in size. It contained three chairs and a desk with biofeedback equipment on the desk. One wall was covered by a bookcase and there were two prints on the other walls.

**5.4.2.2 Assessment and Treatment Phases**

This study consisted of two phases during which the researcher administered a number of assessment and treatment activities. Phase one (i.e., sessions 1 and 2) focused on assessment and involved administration of an intake interview, Zung Self-Rating Anxiety Scale (see Appendix K for Zung Self-rating Anxiety Scale), and Functional Assessment interview. Phase two (i.e., sessions 3-7) aimed to deliver a treatment designed to create reduction in somatic symptomatology and assist participants in learning basic skills for coping with anxiety as it occurred in their day-to-day life. The treatment was applied in a standard manner across all participants and included use of biofeedback equipment to deliver respiration-based HR BFB training via Respiratory Sinus Arrhythmia Feedback. The treatment also employed visualisation to rehearse non-anxious behaviour in anxiety-provoking situations, Progressive Muscle Relaxation to elicit reduction in muscle tension (see Appendix L for PMR script), and Systematic Desensitization to practice a competing response (to anxiety) in the presence of imaginal anxiety-provoking events presented to participants in hierarchical order from least to most anxiety inducing. Despite consistency in treatment strategies across participants, each strategy was tailored to the particular experiences and life circumstances of each participant. It is also relevant to note that participants were encouraged to apply their newly learned behavioural skills to deal with instances of anxiety as it occurred within the normal routine of their lives so as to assist in generalisation from the clinic to the natural environment. Table 3 below presents a summary of all assessment and treatment activities undertaken for the duration of the study and a more detailed description of those activities is contained in Appendix M of this thesis.
Table 3:

*Activities and Sessions for phases 1 and 2 of the Study*

<table>
<thead>
<tr>
<th>Session</th>
<th>Purpose</th>
<th>Activities</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Orientation of potential participants and detailed explanation of study procedures (session duration = 1 hour).</td>
<td>Complete intake interview to confirm presence of GAD as determined by the DSM-IV-TR. Administer Zung SAS. Provide participants with copy of the Explanatory Statement 1.</td>
</tr>
<tr>
<td>II</td>
<td>Idiographic assessment of anxiety in relation to investigation of fear response and its maintaining variables (session duration = 1 hour).</td>
<td>Final check on accuracy of researcher notes on anxiety symptomatology to confirm or reject presence of GAD. For participants who did not meet the inclusion criteria for the study, offer alternative counselling assistance. Provide participants with copy of the Explanatory Statement 2. Complete Functional Assessment interview for GAD.</td>
</tr>
<tr>
<td>III</td>
<td>Introduce process for biofeedback training (session duration = 45 minutes).</td>
<td>Establish baseline measure in Heart Rate and RSA. Initiate respiration-based training using an electrode and computer screen to assist participant monitoring of changes in heart rate. Use of verbal cues to present participant with anxiety-provoking event whilst prompting him/her to keep respiration even.</td>
</tr>
<tr>
<td>IV</td>
<td>Pair biofeedback training with visualisation technique (session duration = 45 minutes).</td>
<td>Review and retrain (if necessary) participant practice of techniques learned in session III. Introduce visualisation of anxiety-provoking event to shape distraction from salient features of that event. Engage in visualisation whilst</td>
</tr>
</tbody>
</table>
Pair biofeedback training with Progressive Muscle Relaxation (PMR) (session duration = 1 hour).

Review and retain (if necessary) participant practice of techniques learned in session IV.
Introduce PMR to assist participant capacity to discriminate between subjective feelings of muscle tension and muscle relaxation.
Engage in PMR whilst connected to an electrode for heart rate and viewing physiological changes on the computer screen.

Pair biofeedback training with exposure to tailored Systematic Desensitisation technique (session duration = 1 hour).

Review and retain (if necessary) participant practice of techniques learned in session V.
Expose participant to individually-created anxiety-inducing hierarchy containing events extracted from the Functional Assessment interview.
Use verbal instructions to present events from least to most distressing whilst cueing the participant to engage in PMR as a competing response to anxiety to re-establish calm.
Engage in SD whilst connected to an electrode for heart rate and viewing physiological changes on the computer screen.

Use biofeedback training in combination with all previously learned behavioural skill techniques (session duration = 1 hour).

Review participant proficiency in use of skills taught in sessions III to VI.
Instruct participant to engage in visualisation, PMR, and SD
whilst connected to an electrode for heart rate and viewing physiological changes on the computer screen.

5.4.3 Biofeedback Equipment

Heart Rate and Respiratory Sinus Arrhythmia (RSA) data were collected via BIOVIEW series IV (Zencor®). For pre-and post-treatment collected of RSA for statistical calculations and additional reliability surface electrocardiograph (ECG) electrodes (BlueSensor, Ambu) were applied to the participants’ left and right wrists after skin preparation of cleaning with methylated spirits. Data were downloaded via the HR VIEW program to a computer and analysed to produce statistical summaries. Training sessions used a less-invasive device for collection of heart rate via pulse (BIOVIEW IV), consisting of an earclip to the lobe of the participants’ left ear. Their pulse data were collected and displayed on the computer screen as an ongoing trace because it enables the client to move his/her hands without confounding the ECG signal that is collected when surface electrodes are applied to the participants’ wrists. Thus, this equipment and sensors enable relatively non-invasive collection of the effects of the SNS and PNS upon the participants’ heart rate and RSA.

Treatment was based upon the application of audio-visual feedback via a computer screen of the participants’ heart rate and RSA curve. This procedure has been previously validated in several studies. (e.g., Cowan, Kogan, Burr, Hendershot, & Buchanan, 1990; Reyes, del Paso, Godoy, & Vila, 1992, Sharpley, 1994a).
5.5 Aims of the Study

Therefore, and as indicated in previous chapters, the present study had the aims of:

1. Testing the applicability of Functional Analytic Assessment methods for understanding anxiety symptoms in a clinical sample;

2. Describing the outcomes of that application in terms of participants’ GAD symptomatology;

3. Using RSA-based BFB to reduce Resting Heart Rate;

4. Using RSA-BFB, Progressive Muscle Relaxation and Systematic Desensitization within imaginal anxiety-provoking events to reduce GAD symptomatology;

5. Use the Zung SAS as a method of assessing GAD symptomatology.

Due to the ethical restraints of providing only partial treatments to actual clinical cases, there was no attempt to distinguish the relative effects of each of the treatments listed under point 4.
CHAPTER 6

Results of Functional Assessment and HR-Control Biofeedback Training

6.1 Application of Functional Assessment to Eleven Cases of GAD

6.1.1 Functional Assessment Targets and Definitions

This chapter describes the application of Functional Assessment to eleven participants in order to gather in-depth information on the specific ways in which GAD impacted on their day-to-day functioning. As outlined in Chapter 3 of this thesis, there are very few studies which have applied Functional Assessment procedures to the investigation of anxiety disorders and the basis for this paucity is the pervasive difficulty in defining anxiety and further describing it in relation to specific and observable target behaviours. However, the literature does contain a number of suggestions which might be used to develop and trial a process for idiographic assessment of anxiety suitable to the Counselling context. These suggestions include reconceptualising anxiety as a fear response, incorporating covert or ‘inner-skin’ experiences as viable targets for assessment, exploring distal antecedent variables (i.e., setting events) as well as proximal antecedent variables, and ensuring that both primary reinforcement and secondary gain processes are assessed.

Therefore, the process designed to conduct a trial application of Functional Assessment for GAD and to describe the specific experiences relevant to each participant consisted of five procedures listed as: 1) defining the fear response by subdividing it into specific covert and overt response units; 2) measuring the fear response in relation to four dimensions of frequency, duration, intensity, and latency; 3) identifying the setting events and proximal antecedents thought to trigger the fear response; 4) establishing whether post-behaviour consequences result in positive reinforcement or negative reinforcement (social and/or automatic); and 5) determining the functions of the fear response.
In addition to explicitly stating data-recording targets, this chapter presents operationalised definitions for those targets in the sequence they appeared in the semi-structured Functional Assessment interview (see: Procedures, page 46 for a description of the interview). The first two data-recording targets (i.e., dimensions, specific anxiety behaviours) related to the fear response itself and aimed to provide information on its occurrence as well as its topography or appearance.

Target 1, referred to as Dimensions, gathered data on the possible frequency (i.e., number of fear responses in one day), duration (i.e., time period for each instance of fear response), intensity (i.e., severity of each fear response as rated on a ten-point scale ranging from 1 [minimum] to 10 [maximum]), and latency (i.e., time gap between onset of fear-evoking stimulus and start of fear response). It is relevant to note that, because there was no clear direction in the literature on the selection of dimensions particularly suited to measuring anxiety, it was decided that all four dimensions would be trialled. Target 2, referred to as Fear Response Units, examined the specific overt (i.e., physical and/or verbal) and covert (i.e., ‘inner skin’ somatic and cognitive/emotion) behaviours associated with the fear response. Target 3, referred to as Fear Response Triggers, gathered information on the setting events (i.e., distal antecedents) and antecedents (i.e., locations, interactions, and tasks) thought to contribute to instances of the fear response. Target 4, referred to as Fear Response Reinforcement Processes, aimed to identify the major reinforcement processes which appeared to strengthen the fear response. Those reinforcement processes were classified in relation to whether they were social (i.e., linked to the external environment) or automatic (i.e., linked to some change in internal state), positive (i.e., resulting in onset of a liked outcome) or negative (i.e., resulting in removal of an aversive outcome), and primary (i.e., occurring immediately following the fear response) or secondary (i.e., some gain occurring at some temporal distance from the fear response). Target 5, referred to as Functions, sought to
examine the purpose being served by the fear response in order to determine its adaptive value to participants. Possible functions of the fear response were listed as escape, avoidance, internal change – somatic, internal change – cognition/emotion, attention, and access to preferred activity (see Table 4 below for the structure used to record data during the Functional Assessment process).

Table 4:

*Functional Assessment Process for GAD as a Fear Response*

<table>
<thead>
<tr>
<th>Target</th>
<th>Factors Investigated</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Fear Response Dimensions</td>
<td>Frequency, duration, intensity, and latency</td>
</tr>
<tr>
<td>2. Fear Response Units</td>
<td>Overt and covert behaviours</td>
</tr>
<tr>
<td>3. Fear Response Triggers</td>
<td>Setting events and proximal antecedents</td>
</tr>
<tr>
<td>4. Fear Response Reinforcement Processes</td>
<td>Social positive and negative reinforcement, automatic social and negative reinforcement, primary reinforcement, and secondary gains</td>
</tr>
<tr>
<td>5. Fear Response Functions</td>
<td>Escape, avoidance, escape, avoidance, internal change–somatic, internal change–cognition/emotion, attention, and access to preferred activity</td>
</tr>
</tbody>
</table>

6.2 **Individual Functional Assessment Results**

The Functional Assessment data gathered via semi-structured interview for all eleven participants are presented in the tables 5 to 15 below. Column 1 for each table remains consistent with the table 4 above. The second column has been altered to Participant Self-Report and represents a summary of each participant’s verbal description of the ways in which each factor investigated applied to him/her.
6.2.1 Participant 1

This participant reported four overt symptoms (i.e. low grade tremor, stuttering, sweating, and insomnia) and six covert symptoms (exhaustion, neck and back pain, gastrointestinal disturbances, headache, and nausea) as being consistently characteristic of the fear responses she experienced. She was able to report on fear response dimension measures in relation to duration, frequency and magnitude (intensity) but could not comment on her day-to-day experiences of anxiety in relation to latency. The participant reported on a significant setting event that had contributed to her vulnerability to antecedents and onset of intense fear responses. She identified multiple functions for anxiety (see Table 5 below for assessment results).

Table 5:

*Participant 1: Functional Assessment Summary*

<table>
<thead>
<tr>
<th>Target</th>
<th>Participant Self-Report</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Fear Response Dimensions</td>
<td>Duration: 5-120 min.</td>
</tr>
<tr>
<td></td>
<td>Frequency: 4-5 times per day.</td>
</tr>
<tr>
<td></td>
<td>Intensity: 4-6 on a 10 point scale.</td>
</tr>
<tr>
<td>2. Fear Response Units</td>
<td><em>Overt behaviours</em>: Low grade tremor, stuttering, sweating and insomnia.</td>
</tr>
<tr>
<td></td>
<td><em>Covert behaviours</em>: Exhaustion, neck and back pain, gastro-intestinal disturbance, headache and nausea.</td>
</tr>
<tr>
<td>3. Fear Response Triggers</td>
<td><em>Setting events</em>: Fear of being alone with one male or being the only female in a group of males. Sexual abuse as a child.</td>
</tr>
<tr>
<td></td>
<td><em>Antecedents</em>: Being in close physical proximity to one or more males with or without direct interaction with them.</td>
</tr>
</tbody>
</table>
4. Fear Response Reinforcement Processes

Negative social reinforcement via removal of close physical proximity to male(s) (aversive social event).

Negative automatic reinforcement via reduction of intensity of fear response units (e.g., sweating, stuttering).

Secondary gain linked to positive social reinforcement via actively taking steps to gain access to social support.

Secondary gain linked to positive automatic reinforcement via onset of strong feelings of calm and relief once social support was secured.

5. Fear Response Functions

Escape by physical removal of self from social situations involving male presence.

Avoidance by avoiding the cognitive/emotional and somatic discomfort arising from male presence.

Internal change due to a reduction in anxiety intensity.

Attention from friends and family who were aware of history of sexual abuse and provided support and sympathy.

Access to preferred activity by seeking pleasurable interaction with a group of familiar people.

6.2.2 Participant 2

This participant reported three overt symptoms (i.e. tremor, irritability and insomnia) and four covert symptoms (dizziness, lack of concentration, severe muscle spasms of the neck and back and, headaches) as being consistently characteristic of the fear responses he experienced. He was able to report on fear response dimension measures in relation to duration and intensity but could not comment on his day-to-day experiences of anxiety in
relation to frequency and latency. The participant reported on a significant setting event that had contributed to his vulnerability to antecedents and onset of intense fear responses. He identified multiple functions for anxiety (see Table 6 below for assessment results)

Table 6:

*Participant 2: Functional Assessment Summary*

<table>
<thead>
<tr>
<th>Target</th>
<th>Participant Self-Report</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Fear Response Dimensions</td>
<td>Duration: 20-300 min.</td>
</tr>
<tr>
<td></td>
<td>Intensity: 7-8 on a 10 point scale.</td>
</tr>
<tr>
<td>2. Fear Response Units</td>
<td><em>Overt behaviours</em>: Insomnia, irritability and tremors.</td>
</tr>
<tr>
<td></td>
<td><em>Covert behaviours</em>: Dizziness, lack of concentration, severe muscle spasms in the neck and back and headaches.</td>
</tr>
<tr>
<td>3. Fear Response Triggers</td>
<td><em>Setting events</em>: Being promoted to a management position two years prior to participation in the study.</td>
</tr>
<tr>
<td></td>
<td><em>Antecedents</em>: Being at work, interacting with senior management and undertaking aversive tasks (i.e. preparing for meetings, dealing with “difficult” staff).</td>
</tr>
<tr>
<td>4. Fear Response Reinforcement</td>
<td><em>Negative social reinforcement</em> via removal of self from challenging interactions and difficult people plus discontinuing work tasks.</td>
</tr>
<tr>
<td>Processes</td>
<td><em>Negative automatic reinforcement</em> via reduction of intensity of fear response units (e.g., tremor, irritability).</td>
</tr>
<tr>
<td></td>
<td><em>Secondary gain linked to positive social reinforcement</em> via actively seeking social support in the form of debriefing and voicing concerns to wife and family.</td>
</tr>
<tr>
<td></td>
<td><em>Secondary gain linked to positive automatic reinforcement</em> via...</td>
</tr>
</tbody>
</table>
reinforcement via the onset of calm and relief.

5. Fear Response Functions

Escape by physical removal of self from the social stimuli of the work environment.

Avoidance by avoiding cognitive/emotional and somatic discomfort arising from work-related events known to include challenging interactions and/or difficult staff.

Internal change due to a reduction in anxiety intensity.

Access to preferred activity by seeking pleasurable interaction with family members.

6.2.3 Participant 3

This participant reported five overt symptoms (i.e. vomiting, dermatological problems, irritability, motor agitation, and insomnia) and two covert symptoms (gastro-intestinal discomfort, and nausea) as being consistently characteristic of the fear responses she experienced. She was able to report on fear response dimension measures in relation to duration, and intensity but could not comment on her day-to-day experiences of anxiety in relation to frequency and latency. The description of duration of fear response (e.g., “most of the day”) did not allow for determination of any variation due to the presence of any specific environmental conditions. The participant could not report on a significant setting event or antecedents that appeared to be associated with the onset of intense fear responses. She identified multiple functions for anxiety (see Table 7 below for assessment results).

Table 7:

Participant 3: Functional Assessment Summary
<table>
<thead>
<tr>
<th><strong>Target</strong></th>
<th><strong>Participant Self-Report</strong></th>
</tr>
</thead>
</table>
| 1. Fear Response Dimensions | Duration: Most of the day.  
Intensity: 8-9 on a 10 point scale. |
| 2. Fear Response Units | *Overt behaviours*: Vomiting, dermatological problems, irritability, motor agitation, and insomnia.  
*Covert behaviours*: Gastro-intestinal discomfort, and nausea. |
| 3. Fear Response Triggers | *Setting events*: Nil  
*Antecedents*: Nil |
| 4. Fear Response Reinforcement Processes | *Negative social reinforcement* via removal of self from the social environment once anxiety escalated but no information on specific aversive social events.  
*Negative automatic reinforcement* via reduction of intensity of fear response units (e.g., irritability, motor agitation).  
*Secondary gain links to negative social reinforcement*: Leaving aversive social environments such as work to drive home.  
*Positive automatic reinforcement* via the onset of calm and relief. |
| 5. Fear Response Functions | *Escape* by physical removal of self.  
*Avoidance* by avoiding cognitive/emotional and somatic discomfort arising from perceived anxiety provoking activity.  
*Internal change* due to a reduction in anxiety intensity.  
*Access to preferred activity* by going home to her “safe place”. |
6.2.4 Participant 4

This participant reported two overt symptoms (i.e. motor agitation, and insomnia) and four covert symptoms (neck and back pain, palpitations, headache, and nausea) as being consistently characteristic of the fear responses he experienced. He was able to report on fear response dimension measures in relation to duration, frequency and intensity but could not comment on his day-to-day experiences of anxiety in relation to latency. The participant reported on one setting event that had contributed significantly to his vulnerability to antecedents and onset of intense fear responses. He identified multiple functions for anxiety (see Table 8 below for assessment results).

Table 8:

Participant 4: Functional Assessment Summary

<table>
<thead>
<tr>
<th>Target</th>
<th>Participant Self-Report</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Fear Response Dimensions</td>
<td>Duration: 2 min – several hours.</td>
</tr>
<tr>
<td></td>
<td>Frequency: 4-5 time per day.</td>
</tr>
<tr>
<td></td>
<td>Intensity: 6-7 on a 10 point scale.</td>
</tr>
<tr>
<td>2. Fear Response Units</td>
<td>Overt behaviours: Motor agitation, and insomnia.</td>
</tr>
<tr>
<td></td>
<td>Covert behaviours: Neck and back pain, palpitations, headache, and nausea.</td>
</tr>
<tr>
<td>3. Fear Response Triggers</td>
<td>Setting events: Being at work.</td>
</tr>
<tr>
<td></td>
<td>Antecedents: Any work-related activity (i.e. report preparation, interactions with supervisors, and meeting preparation).</td>
</tr>
<tr>
<td>4. Fear Response Reinforcement</td>
<td>Negative social reinforcement via removal of an aversive social environment.</td>
</tr>
<tr>
<td>Processes</td>
<td>Negative automatic reinforcement via reduction of intensity of fear response units</td>
</tr>
</tbody>
</table>
(e.g., palpitations, nausea).

*Secondary gain linked to positive social reinforcement* and via actively seeking of social support.

*Positive automatic reinforcement* via the onset of calm and relief.

5. Fear Response Functions

*Escape* by physical removal of self from the work environment.

*Avoidance* by avoiding the cognitive/emotional and somatic discomfort arising from work-related activities.

*Internal change* due to a reduction in anxiety intensity.

*Attention* through his social support network which included family and friends who provided advice and sympathy.

*Access to preferred activity* by seeking pleasurable interaction with his family or going to the gym.

---

### 6.2.5 Participant 5

This participant reported five overt symptoms (i.e. tremor, difficulty speaking, Globus Hystericus, sweating, and insomnia) and two covert symptoms (fatigue and muscle tension) as being consistently characteristic of the fear responses she experienced. She was able to report on fear response dimension measures in relation to duration, frequency and intensity but could not comment on her day-to-day experiences of anxiety in relation to latency. The participant reported on significant setting events that had contributed to her vulnerability to antecedents and onset of intense fear responses. She identified multiple functions for anxiety (see Table 9 below for assessment results).

Table 9:
### Participant 5: Functional Assessment Summary

<table>
<thead>
<tr>
<th>Target</th>
<th>Participant Self-Report</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Fear Response Dimensions</td>
<td>Duration: 60-120 min.</td>
</tr>
<tr>
<td></td>
<td>Frequency: 1-2 time per day.</td>
</tr>
<tr>
<td></td>
<td>Intensity: 4-7 on a 10 point scale.</td>
</tr>
<tr>
<td>2. Fear Response Units</td>
<td><em>Overt behaviours</em>: Tremor, difficulty speaking, Globus Hystericus, sweating, and insomnia.</td>
</tr>
<tr>
<td></td>
<td><em>Covert behaviours</em>: Fatigue and muscle tension.</td>
</tr>
<tr>
<td>3. Fear Response Triggers</td>
<td><em>Setting events</em>: History of negative interactions with her partner, physical presence in the work environment, and growing up in a “highly-strung” family.</td>
</tr>
<tr>
<td></td>
<td><em>Antecedents</em>: Disagreement with a colleague, supervisor or her partner.</td>
</tr>
<tr>
<td>4. Fear Response Reinforcement</td>
<td><em>Negative social reinforcement</em> via removal of an aversive social event.</td>
</tr>
<tr>
<td>Processes</td>
<td><em>Negative automatic reinforcement</em> via reduction of intensity of fear responses (e.g., tremor, difficult speaking).</td>
</tr>
<tr>
<td></td>
<td><em>Secondary gain linked to negative social reinforcement</em>: Creating physical distance between self and potential sources of conflict and disagreement.</td>
</tr>
<tr>
<td></td>
<td><em>Positive automatic reinforcement</em> via the onset of calm and relief.</td>
</tr>
<tr>
<td>5. Fear Response Functions</td>
<td><em>Escape</em> by physical removal of self from conflictual social environments.</td>
</tr>
<tr>
<td></td>
<td><em>Avoidance</em> by avoiding the cognitive/emotional and somatic discomfort arising from interactions and disagreements with supervisors, colleagues and her partner.</td>
</tr>
</tbody>
</table>
Internal change due to a reduction in anxiety intensity.

Access to preferred activity by going for a walk on the beach.

6.2.6 Participant 6

This participant reported four overt symptoms (i.e. tremor, vomiting, motor agitation, and insomnia) and four covert symptoms (malaise, loss of concentration, muscle tension, and nausea) as being consistently characteristic of the fear responses he experienced. He was able to report on his fear response only in relation to the dimension of intensity and could not comment on his day-to-day experiences of anxiety in relation to frequency, duration and latency. The participant reported on a significant setting event that had contributed to his vulnerability to antecedents and onset of intense fear responses. He identified multiple functions for anxiety (see Table 10 below for assessment results).

Table 10:

Participant 6: Functional Assessment Summary

<table>
<thead>
<tr>
<th>Target</th>
<th>Participant Self-Report</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Fear Response Dimensions</td>
<td>Intensity: 8-9 on a 10 point scale.</td>
</tr>
<tr>
<td>2. Fear Response Units</td>
<td><em>Overt behaviours</em>: Tremor, vomiting, motor agitation, and insomnia.</td>
</tr>
<tr>
<td></td>
<td><em>Covert behaviours</em>: Malaise, loss of concentration, muscle tension, and nausea.</td>
</tr>
<tr>
<td>3. Fear Response Triggers</td>
<td><em>Setting events</em>: Parental divorce and parental arguments observed by the participant as a child.</td>
</tr>
<tr>
<td></td>
<td><em>Antecedents</em>: Any instance of conflict or disagreement with his partner.</td>
</tr>
<tr>
<td>4. Fear Response Reinforcement</td>
<td><em>Negative social reinforcement</em> via removal of</td>
</tr>
</tbody>
</table>
Processes

an aversive social event.

*Negative automatic reinforcement* via reduction of intensity of fear response units (e.g., tremor, loss of concentration).

*Positive automatic reinforcement* via the onset of calm and relief.

5. Fear Response Functions

*Escape* by physical removal of self from partner.

*Avoidance* by avoiding the cognitive/emotional and somatic discomfort arising from arguments or disagreements with his partner.

*Internal change* due to a reduction in anxiety intensity.

*Access to preferred activity* by taking his dog for a walk in the local dog park.

6.2.7 Participant 7

This participant reported five overt symptoms (i.e. vomiting, motor agitation, shortness of breath, tremors, and insomnia) and five covert symptoms (abdominal discomfort, palpitations, malaise, muscle tension, and nausea) as being consistently characteristic of the fear responses she experienced. She was able to report on fear response dimension measures in relation to duration, frequency and intensity but could not comment on her day-to-day experiences of anxiety in relation to latency. The participant reported on a significant setting event that had contributed to her vulnerability to antecedents and onset of intense fear responses. She identified multiple functions for anxiety (see Table 11 below for assessment results).

Table 11:

*Participant 7: Functional Assessment Summary*
<table>
<thead>
<tr>
<th>Target</th>
<th>Participant Self-Report</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Fear Response Dimensions</td>
<td>Duration: 15-20 min.</td>
</tr>
<tr>
<td></td>
<td>Frequency: 1-4 time per day.</td>
</tr>
<tr>
<td></td>
<td>Intensity: 6-9 on a 10 point scale.</td>
</tr>
<tr>
<td>2. Fear Response Units</td>
<td><em>Overt behaviours</em>: Vomiting, motor agitation, shortness of breath, tremors, and insomnia.</td>
</tr>
<tr>
<td></td>
<td><em>Covert behaviours</em>: Abdominal discomfort, palpitations, malaise, muscle tension, and nausea.</td>
</tr>
<tr>
<td>3. Fear Response Triggers</td>
<td><em>Setting events</em>: Enrolling in a university degree and undertaking any type of university related activity.</td>
</tr>
<tr>
<td></td>
<td><em>Antecedents</em>: Physical presence in the university environment, attending to any university-related activity, and interacting with fellow students or lecturers.</td>
</tr>
<tr>
<td>4. Fear Response Reinforcement Processes</td>
<td><em>Negative social reinforcement</em> via removal of an aversive social event.</td>
</tr>
<tr>
<td></td>
<td><em>Negative automatic reinforcement</em> via reduction of intensity of fear response units (e.g., shortness of breath, tremors).</td>
</tr>
<tr>
<td></td>
<td><em>Secondary gain linked to positive social reinforcement</em> via actively seeking social support.</td>
</tr>
<tr>
<td></td>
<td><em>Positive automatic reinforcement</em> via the onset of calm and relief.</td>
</tr>
<tr>
<td>5. Fear Response Functions</td>
<td><em>Escape</em> by physical removal of self from the university environment.</td>
</tr>
<tr>
<td></td>
<td><em>Avoidance</em> by avoiding the cognitive/emotional and somatic discomfort arising from being involved in any university related activity.</td>
</tr>
<tr>
<td></td>
<td><em>Internal change</em> due to a reduction in anxiety intensity.</td>
</tr>
</tbody>
</table>
Attention arising from close contact and support from her social support network.

Access to preferred activity by organising pleasurable interaction with a familiar person.

6.2.8 Participant 8

This participant reported five overt symptoms (i.e. shortness of breath, occasional hyperventilation, blushing, tremors, and insomnia) and four covert symptoms (palpitations, malaise, muscle tension, and nausea) as being consistently characteristic of the fear responses she experienced. She was able to report on fear response dimension measures in relation to duration and intensity but could not comment on her day-to-day experiences of anxiety in relation to frequency and latency. The participant reported on a significant setting event that had contributed to her vulnerability to antecedents and onset of intense fear responses. She identified multiple functions for anxiety (see Table 12 below for assessment results).

Table 12:

Participant 8: Functional Assessment Summary

<table>
<thead>
<tr>
<th>Target</th>
<th>Participant Self-Report</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Fear Response Dimensions</td>
<td>Duration: 10-180 min. Intensity: 6-7 on a 10 point scale.</td>
</tr>
<tr>
<td>2. Fear Response Units</td>
<td>Overt behaviours: Shortness of breath, occasional hyperventilation, blushing, tremors, and insomnia</td>
</tr>
<tr>
<td></td>
<td>Covert behaviours: Palpitations, malaise, muscle tension, and nausea.</td>
</tr>
<tr>
<td>3. Fear Response Triggers</td>
<td>Setting events: Sexual abuse as a child.</td>
</tr>
<tr>
<td></td>
<td>Antecedents: Being alone in the home and</td>
</tr>
</tbody>
</table>
4. Fear Response Reinforcement Processes

Negative automatic reinforcement via reduction of intensity of fear response units (hyperventilation, nausea).

Secondary gain linked to positive social reinforcement via actively seeking of social support.

Secondary gain linked to positive automatic reinforcement via the onset of calm and relief.

5. Fear Response Functions

Escape by physical removal of self from isolated locations.

Avoidance by avoiding the cognitive/emotional and somatic discomfort arising from being alone.

Internal change due to a reduction in anxiety intensity.

Access to preferred activity by seeking physical presence of and pleasurable interactions with familiar and trusted people.

6.2.9 Participant 9

This participant reported five overt symptoms (i.e. tremor, blushing, shortness of breath, irritability, and insomnia) and four covert symptoms (severe headaches, palpitations, dizziness, and indigestion) as being consistently characteristic of the fear responses she experienced. She was able to report on fear response dimension measures in relation to duration, frequency and intensity but could not comment on her day-to-day experiences of anxiety in relation to latency. The participant reported on a significant setting event that had contributed to her vulnerability to antecedents and onset of intense fear responses. She identified multiple functions for anxiety (see Table 13 below for assessment results).
Table 13:

**Participant 9: Functional Assessment Summary**

<table>
<thead>
<tr>
<th>Target</th>
<th>Participant Self-Report</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Fear Response Dimensions</td>
<td>Duration: 20-140 min.</td>
</tr>
<tr>
<td></td>
<td>Frequency: 10 times per week.</td>
</tr>
<tr>
<td></td>
<td>Intensity: 4-7 on a 10 point scale.</td>
</tr>
<tr>
<td>2. Fear Response Units</td>
<td><em>Overt behaviours</em>: Tremor, blushing, shortness of breath, irritability, and insomnia.</td>
</tr>
<tr>
<td></td>
<td><em>Covert behaviours</em>: Severe headaches, palpitations, dizziness, and indigestion.</td>
</tr>
<tr>
<td>3. Fear Response Triggers</td>
<td><em>Setting events</em>: History of high expectations for academic achievement from family and enrolling in a university degree.</td>
</tr>
<tr>
<td></td>
<td><em>Antecedents</em>: Physical presence in the university environment and attending to any university related activity.</td>
</tr>
<tr>
<td></td>
<td><em>Negative automatic reinforcement</em> via reduction of intensity of fear response units (e.g., dizziness, palpitations).</td>
</tr>
<tr>
<td></td>
<td><em>Secondary gain linked to positive social reinforcement</em> via actively seeking social support.</td>
</tr>
<tr>
<td></td>
<td><em>Positive automatic reinforcement</em> via the onset of calm and relief.</td>
</tr>
<tr>
<td>5. Fear Response Functions</td>
<td><em>Escape</em> by physical removal of self from the university environment.</td>
</tr>
<tr>
<td></td>
<td><em>Avoidance</em> by avoiding the cognitive/emotional and somatic discomfort arising from any university related activity.</td>
</tr>
<tr>
<td></td>
<td><em>Internal change</em> due to a reduction in anxiety</td>
</tr>
</tbody>
</table>
intensity.

Attention from speaking with and gaining support and sympathy from long-term friends.

Access to preferred activity by initiating and engaging in a pleasurable interaction with a familiar person.

6.2.10 Participant 10

This participant reported five overt symptoms (i.e. tremor, shortness of breath, blushing, motor agitation, and insomnia) and three covert symptoms (palpitations, indigestion, and headaches) as being consistently characteristic of the fear responses she experienced. She was able to report on fear response dimension measures in relation to duration, frequency and intensity but could not comment on her day-to-day experiences of anxiety in relation to latency. The participant reported on a significant setting event that had contributed to her vulnerability to the onset of intense fear responses but could not describe any distinct antecedents which might have influenced her behaviour. She identified multiple functions for anxiety (see Table 14 below for assessment results).

Table 14:

Participant 10: Functional Assessment Summary

<table>
<thead>
<tr>
<th>Target</th>
<th>Participant Self-Report</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Fear Response Dimensions</td>
<td>Duration: 5-360 min.</td>
</tr>
<tr>
<td></td>
<td>Frequency: 2-3 times per day.</td>
</tr>
<tr>
<td></td>
<td>Intensity: 3-9 on a 10 point scale.</td>
</tr>
<tr>
<td>2. Fear Response Units</td>
<td>Overt behaviours: Tremor, shortness of breath, blushing, motor agitation, and insomnia.</td>
</tr>
</tbody>
</table>
Covert behaviours: Palpitations, indigestion, and headaches.

3. Fear Response Triggers

Setting events: Prior drug addiction and her HIV diagnosis.

Antecedents: None reported

4. Fear Response Reinforcement Processes

Negative social reinforcement via removal of an aversive social event when she has to interact with strangers or work colleagues.

Negative automatic reinforcement via reduction of intensity of fear response units (e.g., tremor, palpitations).

Secondary gain linked to positive social reinforcement via actively seeking social support.

Secondary gain linked to positive automatic reinforcement via the onset of calm and relief.

5. Fear Response Functions

Escape by physical removal of self from interactions with strangers or colleagues.

Avoidance by avoiding the cognitive/emotional and somatic discomfort arising from a social or public event.

Internal change due to a reduction in anxiety intensity.

Attention gained from the support and sympathy offered by family members.

Access to preferred activity by seeking pleasurable interaction with her family.

6.2.11 Participant 11

This participant reported three overt symptoms (i.e. motor agitation, tremors, and insomnia) and five covert symptoms (nausea, indigestion, malaise, loss of concentration and, muscle tension) as being consistently characteristic of the fear responses he experienced. He
was able to report on fear response dimension measures in relation to duration, frequency and intensity but could not comment on his day-to-day experiences of anxiety in relation to latency. The participant reported on a significant setting event that had contributed significantly to his vulnerability to antecedents and onset of intense fear responses. He identified multiple functions for anxiety (see Table 15 below for assessment results).

Table 15:

*Participant 11: Functional Assessment Summary*

<table>
<thead>
<tr>
<th>Target</th>
<th>Participant Self-Report</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Fear Response Dimensions</td>
<td>Duration: 10-15 min.</td>
</tr>
<tr>
<td></td>
<td>Frequency: 2-3 times per day.</td>
</tr>
<tr>
<td></td>
<td>Intensity: 6-8 on a 10 point scale.</td>
</tr>
<tr>
<td>2. Fear Response Units</td>
<td><em>Overt behaviours</em>: Motor agitation, tremors, and insomnia.</td>
</tr>
<tr>
<td></td>
<td><em>Overt behaviours</em>: Nausea, indigestion, malaise, loss of concentration and, muscle tension.</td>
</tr>
<tr>
<td>3. Fear Response Triggers</td>
<td><em>Setting events</em>: Enrolling for a university degree, being physically present in the university environment or involved in any university related activity.</td>
</tr>
<tr>
<td></td>
<td><em>Antecedents</em>: Being physically present in the classroom and interacting with lecturers and fellow students.</td>
</tr>
<tr>
<td>4. Fear Response Reinforcement Processes</td>
<td><em>Negative social reinforcement</em> via removal from the university environment.</td>
</tr>
<tr>
<td></td>
<td><em>Negative automatic reinforcement</em> via reduction of intensity of fear responses.</td>
</tr>
<tr>
<td></td>
<td><em>Secondary gain linked to positive social reinforcement</em> via actively seeking social</td>
</tr>
</tbody>
</table>
support.

Secondary gain linked to positive automatic reinforcement via the onset of calm and relief.

5. Fear Response Functions

Escape by physical removal of self from all tasks and social stimuli present in the university environment.

Avoidance by avoiding the cognitive and emotional discomfort arising from being at university.

Internal change due to a reduction in anxiety intensity.

Attention from his social support network.

Access to preferred activity by seeking pleasurable interaction with a familiar person.

6.3 Group Functional Assessment Results

Table 16

Group Functional Assessment Results
<table>
<thead>
<tr>
<th>Target</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
<th>11</th>
<th>Percent</th>
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<tbody>
<tr>
<td>Fear Response Dimensions</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Duration (min/episode)</td>
<td>5 - 120</td>
<td>20 - 300</td>
<td>Most ≥1/day</td>
<td>2 - &gt; hours</td>
<td>60 - 120</td>
<td>2 - &gt; hours</td>
<td>15 - 20</td>
<td>10 - 100</td>
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</tr>
<tr>
<td>Frequency (episodes/day)</td>
<td>4 - 6</td>
<td>ND*</td>
<td>ND</td>
<td>ND</td>
<td>4 - 5</td>
<td>1 - 2</td>
<td>ND</td>
<td>1 - 4</td>
<td>ND</td>
<td>1 - 2</td>
<td>ND</td>
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</tr>
<tr>
<td>Latency (S-R time lapse in min)</td>
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<td>7 - 8</td>
<td>8 - 9</td>
<td>6 - 7</td>
<td>6 - 7</td>
<td>4 - 7</td>
<td>8 - 9</td>
<td>6 - 9</td>
<td>6 - 7</td>
<td>4 - 7</td>
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<td>6 - 8</td>
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<td>Fatigue</td>
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<td>GT Disturbance</td>
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<td>GT2 Disturbance</td>
<td>GT3 Disturbance</td>
<td>GT4 Disturbance</td>
<td>GT5 Disturbance</td>
<td>GT6 Disturbance</td>
<td>GT7 Disturbance</td>
<td>GT8 Disturbance</td>
<td>GT9 Disturbance</td>
<td>GT10 Disturbance</td>
<td>GT11 Disturbance</td>
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<tr>
<td>Poor Concentration</td>
<td>Poor Concentration</td>
<td>Poor Concentration</td>
<td>Poor Concentration</td>
<td>Poor Concentration</td>
<td>Poor Concentration</td>
<td>Poor Concentration</td>
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<tr>
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<td>Shortness of breath</td>
<td>Shortness of breath</td>
<td>Shortness of breath</td>
<td>Shortness of breath</td>
<td>Shortness of breath</td>
<td>Shortness of breath</td>
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<td>Present</td>
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<td>NR</td>
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<td>Present</td>
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<td>Present</td>
<td>Present</td>
<td>Present</td>
<td>Present</td>
<td>Present</td>
<td>Present</td>
<td>Present</td>
<td>63.63%</td>
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<td>Fear Response</td>
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<td></td>
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<tr>
<td>Primary Automatic Negative</td>
<td>Primary Automatic Negative</td>
<td>Primary Automatic Negative</td>
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<td>Primary Automatic Negative</td>
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<td>Primary Automatic Negative</td>
<td>Primary Automatic Negative</td>
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</tr>
<tr>
<td>Fear Response Functions</td>
<td>Escape</td>
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<td>Escape</td>
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<td>Escape</td>
<td>Escape</td>
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</tr>
<tr>
<td>Avoidance</td>
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<td>Avoidance</td>
<td>Avoidance</td>
<td>100.0%</td>
</tr>
<tr>
<td>Internal change-affective</td>
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<td>Internal change-affective</td>
<td>Internal change-affective</td>
<td>Internal change-affective</td>
<td>Internal change-affective</td>
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</tr>
<tr>
<td>GHS³</td>
<td>GHS³</td>
<td>GHS³</td>
<td>GHS³</td>
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<td>ND*</td>
<td>ND*</td>
<td>ND*</td>
<td>ND*</td>
<td>ND*</td>
<td>ND*</td>
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<td>ND*</td>
<td>ND*</td>
<td>ND*</td>
<td>100.0%</td>
</tr>
<tr>
<td>GT²</td>
<td>Gastrointestinal disturbance</td>
<td>Gastrointestinal disturbance</td>
<td>Gastrointestinal disturbance</td>
<td>Gastrointestinal disturbance</td>
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<td>GHS³</td>
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<td>Global Hysteric</td>
<td>Global Hysteric</td>
<td>Global Hysteric</td>
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<td>Global Hysteric</td>
<td>Global Hysteric</td>
<td>Global Hysteric</td>
<td>100.0%</td>
</tr>
</tbody>
</table>

*ND*: Not defined as the participant not being able to provide data on the Functional Assessment target during interview.
The Functional Assessment of GAD was intended to assist in determination of the specific experiences of individual participants in order to account for the variation in symptomatology that is not possible by simply allocating a diagnostic label. It was also intended that the Functional Assessment data be reviewed in relation to depth of self-reported descriptions and similarities across the group of adults who participated in this study. This procedure was used to identify those assessment targets most likely to be of relevance to idiographic investigation of this diagnostic category. The group review of data also aimed to detect any assessment targets that did not succeed in eliciting information on participant responses because they were possibly not suited to their experiences. Each Functional Assessment target is discussed below in relation to group trends.

The findings for fear response dimensions (assessment target 1) indicated that participants were most able to describe occurrences of anxiety in relation to duration (i.e., minutes taken in each instance of fear response) and intensity (i.e., rating of anxiety magnitude on a 10-point scale). The most detailed and readily delivered descriptions were elicited in relation to intensity. In contrast, participants experienced difficulty in commenting on the frequency (i.e., number of fear responses per day) with which fear responses occurred and, at best, provided generalised responses such as “most of the day” or “the majority of time”. Interestingly, researcher queries on latency (i.e., stimulus—anxiety onset time gap) were consistent in their incapacity to gather information. Even participants who were proficient in recognising the antecedents which caused them to become anxiety, reported that they were unable to describe their responses in relation to latency.

The findings for fear response units (assessment target 2) suggested that participants reported a range of covert behaviours with most commonality in experiences of muscle tension and back pain (73 percent), gastrointestinal disturbance and palpitations (55 percent), and headaches (54 percent). All participants described experiencing a number of overt
behaviours in addition to covert or ‘inner skin’ responses with similarities in insomnia (100 percent), tremor (82 percent), and motor agitation (55 percent).

The findings for fear response triggers (assessment target 3) showed that all (100 percent) participants were able to describe setting events which they believed increased their vulnerability to anxiety-provoking stimuli in the environment. Just over half of the participant group (63 percent) were also able to discuss an antecedent known to cause an escalation in fear response when present in the environment.

The findings for fear response reinforcement process (assessment target 4) indicated that all four primary reinforcement processes (i.e., social positive/negative reinforcement and automatic positive/negative reinforcement) were reported to be relevant to strengthening fear responses and possibly sustaining them over time. It also appears that participant fear responses had become multi-functional, leading to numerous functions with avoidance, escape, and internal change—somatic more likely to follow instances of this response. Attention and access to preferred activity functions were mostly reported to occur some time after occurrence of the fear response as secondary gains.

6.4 Pre- and Post-Treatment Biofeedback Training Results

6.4.1 Group Pre- and Post-Treatment Biofeedback Training Results

**Raw data:** Table 17 presents the raw data from each participant for the pre- and post-training observations. There were no significant differences between male and female data from all these observations, allowing both genders’ data to be collapsed for further analysis. Table 18 shows the mean and standard deviations for each measure for pre- versus post-intervention for all measures.
Table 17

**Raw Data for all Participants for all Measures**

<table>
<thead>
<tr>
<th>Participant</th>
<th>Gender</th>
<th>SAS Pre test Raw Score</th>
<th>SAS Post test Raw Score</th>
<th>RHR Pre Int BPM</th>
<th>RHR Post Int BPM</th>
<th>Post</th>
<th>RSA Pre Int</th>
<th>RSA Post Int</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>F</td>
<td>46</td>
<td>35</td>
<td>75</td>
<td>64</td>
<td>321.9</td>
<td>363.1</td>
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<tr>
<td>2</td>
<td>M</td>
<td>55</td>
<td>37</td>
<td>83</td>
<td>64</td>
<td>234.9</td>
<td>907.7</td>
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</tr>
<tr>
<td>3</td>
<td>F</td>
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<td>4</td>
<td>M</td>
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<td>919.8</td>
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<tr>
<td>5</td>
<td>F</td>
<td>60</td>
<td>42</td>
<td>118</td>
<td>85</td>
<td>89.1</td>
<td>134.8</td>
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</tr>
<tr>
<td>6</td>
<td>M</td>
<td>57</td>
<td>47</td>
<td>111</td>
<td>96</td>
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<td>7</td>
<td>F</td>
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<td>53</td>
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<td>81.0</td>
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<td>8</td>
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<td>96</td>
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<tr>
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<td>M</td>
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<td>33</td>
<td>139</td>
<td>90</td>
<td>766.2</td>
<td>1005.8</td>
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</tr>
</tbody>
</table>

Table 18

**Means and Standard Deviation for Pre- and Post-Intervention for all Measures**

<table>
<thead>
<tr>
<th>Measure</th>
<th>Gender</th>
<th>Mean</th>
<th>Standard Deviation</th>
<th>N</th>
</tr>
</thead>
<tbody>
<tr>
<td>SAS Pre-score</td>
<td>Male</td>
<td>55.2500</td>
<td>2.36291</td>
<td>4</td>
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<td></td>
<td>Female</td>
<td>57.2857</td>
<td>5.25085</td>
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</tr>
<tr>
<td>SAS Post-score</td>
<td>Male</td>
<td>38.7500</td>
<td>5.90903</td>
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</tr>
<tr>
<td></td>
<td>Female</td>
<td>40.4286</td>
<td>6.67975</td>
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</tr>
<tr>
<td>RHR Pre-score</td>
<td>Male</td>
<td>1.1425</td>
<td>23.76798</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>1.0557</td>
<td>15.91495</td>
<td>7</td>
</tr>
<tr>
<td>RHR Post-score</td>
<td>Male</td>
<td>79.7500</td>
<td>15.62850</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>80.7143</td>
<td>12.43268</td>
<td>7</td>
</tr>
<tr>
<td>RSA Pre-score</td>
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<td>2.8705</td>
<td>329.00043</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>1.5691</td>
<td>131.40101</td>
<td>7</td>
</tr>
<tr>
<td>RSA Post-score</td>
<td>Male</td>
<td>1.0744</td>
<td>263.47808</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>6.3180</td>
<td>320.95478</td>
<td>7</td>
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</tbody>
</table>

The mean SAS pre-score was 55.66 (SD = 5.17), median = 57.00, ranging from 46 to 62/80. The 5% trimmed mean was 55.85, only -0.19 below the sample mean, indicating negligible effects on the mean score from outliers in the sample. Skewness was negative (-1.11), suggesting a clustering at the upper end of the possible range of scores, to be expected within
a clinical sample. Kurtosis was 0.365, indicating a relatively flat distribution, confirmed by examination of the histogram. Inspection of the Boxplot revealed no outliers (i.e., more than 1.5 box lengths from the edge of the box), and all scores were valid, but relatively high SAS scores. Using Zung’s cut-off score of 36, all of the sample was experiencing “clinically significant” anxiety (Zung, 1971). The Kolmogorov-Smirnov statistic was nonsignificant, indicating that the distribution was normal. The Normal Q-Q Plot was almost a completely straight line, and the Detrended Normal Q-Q Plot showed that most points collected around the zero line. Thus, the SAS pre-score data may be accepted as satisfying the requirements of normality for further analyses.

The mean SAS post-score was 39.81 (SD = 6.16), ranging from 33 to 53. The 5% trimmed mean was 39.46, only 0.35 lower than the sample mean and again indicating negligible effects from outliers. Skewness was positive, indicative of a clustering of scores to the lower end of the SAS range, and suggestive of an intervention effect when compared with the SAS pre-score data in the preceding paragraph. Kurtosis was again minor (0.596), indicative of a relatively flat distribution, confirmed by inspection of the histogram. There were no outliers and all SAS scores were valid, although comparatively lower than those collected pre-intervention. Zung’s cut-off score for clinically significant anxiety showed that seven participants had scores above 36, a reduction of 36.4% from the pre-intervention SAS data. The Kolmogorov-Smirnov statistic was again nonsignificant, the Normal Q-Q Plot was almost a completely straight line, and the Detrended Normal Q-Q Plot showed that most points collected around the zero line. Thus, the SAS post-score data may be accepted as satisfying the requirements of normality for further analyses. Normality analysis of all other measures shown in Table 17 indicated that all scores from the variables shown there (i.e., RHR pre- and post-intervention, RSA pre- and post-intervention) were also normal for the purposes of further data analysis (all Kolmogorov-Smirnov statistics were non-significant).
Of most interest are the changes in dependent variable scores that appear in Table 17 and Table 18. MANOVA on SAS, RHR and RSA data from the pre- to post-intervention observations indicated a significant main effect ($F(3,18) = 21.889, p < .001$, Wilks’ Lambda, partial eta squared = .785) for time of observation. As might be expected from the mean scores shown in Table 2, there were also significant univariate effects for SAS ($F(1,21) = 53.774, p < .001$, partial eta squared = .729), RHR ($F = 17.459, p < .001$, partial eta squared = .466) and RSA ($F = 21.208, p < .001$, partial eta squared = .515). These SAS and RHR results indicate decreases in anxiety and resting heart rate following intervention, plus an increase in RSA amplitude, and together show evidence of a significant and powerful intervention effect on these dependent variables in terms of the group’s data. Individual Pre and post treatment RSA changes can be seen in the figure below.

Figure 1. Individual pre- and post- treatment RSA (msec) changes.
and post intervention RHR changes and the individual participant results for pre- and post SAS raw score changes.

Figure 2. Pre- and post- intervention RHR (BPM) changes.

![Graph showing RHR changes](image)

Figure 3. Individual participant results for pre- and post- SAS raw score changes:

![Graph showing SAS raw score changes](image)
6.5 Individual Pre- and Post-Treatment Biofeedback Training Results

6.5.1 Participant 1

Participant 1 was aged 18 with self-reported symptoms of anxiety including insomnia, tremor, and constant feelings of exhaustion. The pre- versus post-intervention difference in SAS score for this participant was 11 (i.e., pre-intervention SAS score = 46 and post intervention SAS score = 45). This SAS score difference reflected an improved sleeping pattern, overall increase in energy and the disappearance of her tremor. The participant’s pre-intervention RHR was 75 beats per minute and 64 beats per minute at post-intervention. This indicated a reduction in sympathetic nervous system activity. A pre- versus post-intervention difference was also found for RSA (i.e., pre-intervention measure = 321.9 msec and post-intervention measure = 363.1 msec). Overall, the findings obtained for Participant 1 show a decrease on all three measures of anxiety symptomatology and anxiety reactivity.

6.5.2 Participant 2

Participant 2 was aged 53 with self-reported symptoms of anxiety including insomnia, irritability, and headaches. The pre- versus post-intervention difference in SAS score for this participant was 18 (i.e., pre-intervention SAS score = 55 and post intervention SAS score = 37). This SAS score difference reflected an improved sleeping pattern, the participant being less irritable and a reduction in frequency and intensity of his headaches. The participant’s pre-intervention RHR was 83 beats per minute and 64 beats per minute at post-intervention. This indicated a reduction in sympathetic nervous system activity. A pre- versus post-intervention difference was also found for RSA (i.e., pre-intervention measure = 234.9 msec and post-intervention measure = 907.7 msec). Overall, the findings obtained for Participant 2 show a decrease on all three measures of anxiety symptomatology and anxiety reactivity.
6.5.3 Participant 3

Participant 3 was aged 20 with self-reported symptoms of anxiety including insomnia, irritability, and motor agitation. The pre- versus post-intervention difference in SAS score for this participant was 14 (i.e., pre-intervention SAS score = 57 and post intervention SAS score = 43). This SAS score difference reflected an improved sleeping pattern, the participant being less irritable and a decrease in motor agitation. The participant’s pre-intervention RHR was 120 beats per minute and 86 beats per minute at post-intervention. This indicated a reduction in sympathetic nervous system activity. A pre- versus post-intervention difference was also found for RSA (i.e., pre-intervention measure = 147.3 msec and post-intervention measure = 620.8 msec). Overall, the findings obtained for Participant 3 show a decrease on all three measures of anxiety symptomatology and anxiety reactivity.

6.5.4 Participant 4

Participant 4 was aged 57 with self-reported symptoms of anxiety including insomnia, muscle tension in his neck and back and motor agitation. The pre- versus post-intervention difference in SAS score for this participant was 14 (i.e., pre-intervention SAS score = 52 and post intervention SAS score = 38). This SAS score difference reflected an improved sleeping pattern and a decrease in motor agitation and muscle tension. The participant’s pre-intervention RHR was 124 beats per minute and 69 beats per minute at post-intervention. This indicated a reduction in sympathetic nervous system activity. A pre- versus post-intervention difference was also found for RSA (i.e., pre-intervention measure = 98.9 msec and post-intervention measure = 919.8 msec). Overall, the findings obtained for Participant 4 show a decrease on all three measures of anxiety symptomatology and anxiety reactivity.
6.5.5 Participant 5

Participant 5 was aged 39 with self-reported symptoms of anxiety including insomnia, globus hystericus and sweating. The pre- versus post-intervention difference in SAS score for this participant was 18 (i.e., pre-intervention SAS score = 60 and post intervention SAS score = 42). This SAS score difference reflected an improved sleeping pattern and the disappearance of her globus hystericus and a reduction in sweating. The participant’s pre-intervention RHR was 118 beats per minute and 85 beats per minute at post-intervention. This indicated a reduction in sympathetic nervous system activity. A pre- versus post-intervention difference was also found for RSA (i.e., pre-intervention measure = 89.1 msec and post-intervention measure = 134.8 msec). Overall, the findings obtained for Participant 5 show a decrease on all three measures of anxiety symptomatology and anxiety reactivity.

6.5.6 Participant 6

Participant 6 was aged 28 with self-reported symptoms of anxiety including insomnia, loss of concentration and motor agitation. The pre- versus post-intervention difference in SAS score for this participant was 10 (i.e., pre-intervention SAS score = 57 and post intervention SAS score = 47). This SAS score difference reflected an improved sleeping pattern, improved concentration and a decrease in muscle tension. The participant’s pre-intervention RHR was 111 beats per minute and 96 beats per minute at post-intervention. This indicated a reduction in sympathetic nervous system activity. A pre- versus post-intervention difference was also found for RSA (i.e., pre-intervention measure = 48.5 msec and post-intervention measure = 1464.1 msec). Overall, the findings obtained for Participant 6 show a decrease on all three measures of anxiety symptomatology and anxiety reactivity.
6.5.7 Participant 7

Participant 7 was aged 24 with self-reported symptoms of anxiety including insomnia, vomiting and motor agitation. The pre- versus post-intervention difference in SAS score for this participant was 5 (i.e., pre-intervention SAS score = 58 and post intervention SAS score = 53). This SAS score difference reflected an improved sleeping pattern and a decrease in motor agitation with a reduction in the frequency of vomiting. The participant’s pre-intervention RHR was 116 beats per minute and 101 beats per minute at post-intervention. This indicated a reduction in sympathetic nervous system activity. A pre- versus post-intervention difference was also found for RSA (i.e., pre-intervention measure = 81.0 msec and post-intervention measure = 698.8 msec). Overall, the findings obtained for Participant 7 show a decrease on all three measures of anxiety symptomatology and anxiety reactivity.

6.5.8 Participant 8

Participant 8 was aged 32 with self-reported symptoms of anxiety including insomnia, hyperventilation and blushing. The pre- versus post-intervention difference in SAS score for this participant was 27 (i.e., pre-intervention SAS score = 62 and post intervention SAS score = 35). This SAS score difference reflected an improved sleeping pattern, no episodes of hyperventilation and a reduction in the frequency of blushing episodes. The participant’s pre-intervention RHR was 96 beats per minute and 67 beats per minute at post-intervention. This indicated a reduction in sympathetic nervous system activity. A pre- versus post-intervention difference was also found for RSA (i.e., pre-intervention measure = 48.7 msec and post-intervention measure = 926.3 msec). Overall, the findings obtained for Participant 8 show a decrease on all three measures of anxiety symptomatology and anxiety reactivity.
6.5.9 Participant 9

Participant 9 was aged 23 with self-reported symptoms of anxiety including insomnia, severe headaches and motor agitation. The pre- versus post-intervention difference in SAS score for this participant was 17 (i.e., pre-intervention SAS score = 58 and post intervention SAS score = 41). This SAS score difference reflected an improved sleeping pattern and a decrease in motor agitation and a reduction in frequency and intensity of headaches. The participant’s pre-intervention RHR was 104 beats per minute and 80 beats per minute at post-intervention. This indicated a reduction in sympathetic nervous system activity. A pre- versus post-intervention difference was also found for RSA (i.e., pre-intervention measure = 47.8 msec and post-intervention measure = 593.1 msec). Overall, the findings obtained for Participant 9 show a decrease on all three measures of anxiety symptomatology and anxiety reactivity.

6.5.10 Participant 10

Participant 10 was aged 30 with self-reported symptoms of anxiety including insomnia, shortness of breath and motor agitation. The pre- versus post-intervention difference in SAS score for this participant was 26 (i.e., pre-intervention SAS score = 60 and post intervention SAS score = 34). This SAS score difference reflected an improved sleeping pattern and breathing and a reduction in motor agitation. The participant’s pre-intervention RHR was 110 beats per minute and 82 beats per minute at post-intervention. This indicated a reduction in sympathetic nervous system activity. A pre- versus post-intervention difference was also found for RSA (i.e., pre-intervention measure = 362.6 msec and post-intervention measure = 1085.7 msec). Overall, the findings obtained for Participant 10 show a decrease on all three measures of anxiety symptomatology and anxiety reactivity.
6.5.11 Participant 11

Participant 11 was aged 46 with self-reported symptoms of anxiety including insomnia, palpitations and motor agitation. The pre- versus post-intervention difference in SAS score for this participant was 24 (i.e., pre-intervention SAS score = 57 and post intervention SAS score = 33). This SAS score difference reflected an improved sleeping pattern and a decrease in palpitations and motor agitation. The participant’s pre-intervention RHR was 139 beats per minute and 90 beats per minute at post-intervention. This indicated a reduction in sympathetic nervous system activity. A pre- versus post-intervention difference was also found for RSA (i.e., pre-intervention measure = 766.2 msec and post-intervention measure = 1005.8 msec). Overall, the findings obtained for Participant 11 show a decrease on all three measures of anxiety symptomatology and anxiety reactivity.
CHAPTER 7

Discussion and Conclusion

7.1 Summary of Results

This research study aimed to evaluate the effectiveness of Functional Assessment as a method of understanding GAD symptoms among a sample of clinical cases, and to test for the effects of a combined treatment protocol that included RSA-BFB, Progressive Muscle Relaxation and Systematic Desensitization with imaginal anxiety-provoking events. As mentioned above, for ethical reasons, no attempt was made to distinguish the relative effectiveness of the training procedures, and so the conclusions drawn here are restricted to the effectiveness of the combined treatments.

However, prior to considering the effectiveness of the combined treatments for GAD, the applicability of Functional Assessment procedures as a means of understanding how GAD symptoms functioned for the participants in this study was clearly demonstrated. The diagnosis provided for each of the 11 cases under section 6.2 and the interpretation of group results of the Functional Assessment protocol presented under section 6.3 clearly display the applicability of this process with the kinds of GAD symptomatology presented by these 11 cases. Although not previously demonstrated in such detail, it is realistic to conclude from these data that Functional Assessment models and processes may be profitably applied to GAD within counselling settings.

The second major aim of this project was to evaluate the effectiveness of a combined RSA-based BFB, Progressive Muscle Relaxation and Systematic Desensitization with imaginal anxiety-provoking events treatment protocol for GAD, and to evaluate that outcome via participant self-reports of their symptomatology, their resting heart rate data, and their
scores on the Zung SAS. As shown in sections 6.3 and 6.4, the successful group data were reflected in consistent success across each of the 11 participants, verifying the overall outcome in ways that may sometimes be obscured when only group results are considered. That is, as well as showing success in the usual way via statistical and visual analysis of the group means on the dependent variables (Zung SAS, RHR), the fact that all 11 cases showed consistent reductions in both of these DVs provides an additional level of confirmation for the combined training that was provided to these participants. As may be seen from the results chapter prior to this discussion, all participants showed clinically significant reductions in their Zung SAS scores, reductions in RHR and increases in RSA, and also reported the reduction or disappearance of those major symptoms of GAD which had most troubled them before treatment. As well as consistency across the 11 participants in the study, the overall group results reported via MANOVA of the principal dependent variables clearly indicate that, at the combined level as well as the individual participant level, the treatment was successful in meeting its aims of reducing GAD symptoms. As well as these overall reductions in GAD-related symptoms, all participants also reported their satisfaction with the treatment provided to them, and commented that they would recommend the treatment to others, thus providing a final indication of the success of the treatments being tested in this research study.

7.2 Participant Learning

Counselling and allied therapies are often focussed upon the alleviation of some issue or problem which the client presents, which has been reported above. However, in this study, it is also worthwhile commenting upon the incidental learning that participants underwent. That is, each participant learnt how to observe and control their own HR and RSA, and thereby gained a sense of self-control and confidence which, although not measured as a defined outcome of the study, was voluntarily commented upon by almost all participants. As
has been noted in many clinical settings and with many disorders, the increase in participants’ self-efficacy in regard to their ability to control some of their (previously uncontrollable) physiological responses to stress (i.e., HR, RSA) is a key aspect of generalising the kinds of learning that occurs within clinical settings to everyday life environments. Such increases in self-efficacy over what are most commonly referred to as ‘uncontrollable’ parameters of physiological systems can provide a major boost to the self-esteem of people who (like those in this study) have an established history of self-perceived inadequacy in regard to controlling their own somatic responses to environmental stressors and threats.

7.3 Implications for Counselling Practice

It is worthwhile commenting on another aspect of the outcomes of this study - that of the use of the Functional Assessment and BFB procedures in everyday Counselling settings. As mentioned during the introductory chapters, relatively few attempts have been reported in which Functional Assessment has been applied to the diagnosis and treatment of GAD. Although the present setting (i.e., a research study conducted in a university clinic) may have a greater degree of supervisory and clinical ability than is present in relatively isolated settings, the ease with which the Functional Assessment and BFB protocols were mastered and applied to participants by the therapist argues that they could also be similarly applied by other counselling professionals in the field. Although that process requires some further learning by practitioners, the consistency of participant outcomes in terms of RHR, RSA, SAS and related symptoms of GAD argues for the consideration of these techniques in everyday counselling practice for anxiety. Finally, it is of interest that this study did not focus upon previous life histories, use more complex counselling and psychotherapeutic techniques, nor spend a great deal of time with the participants. Although there is no attempt being made here to suggest that those processes are not valuable and required with some clients who
present with GAD, it is worthwhile commenting that they were not evidently necessary with these 11 participants.

7.4 Limitations of the Study and Suggestions for Further Research

As a clinically-focussed study, this project suffers from the same limitations on generalisation that apply to any such studies. That is, there is no attempt made here to claim that these results compare with those from much larger studies that are group-based, and extension of these findings to such a comparison would enable greater generalisability to be established. Similarly, only a single therapist was used in this study, and therefore it was not possible to definitively claim that the positive outcomes were not influenced by ‘personal’ variables surrounding the therapeutic relationship established by that therapist with the 11 participants. Use of multiple therapists in future studies would allow testing of the ‘therapist’ effects. Although GAD is a very widespread disorder, it is not the only psychological disorder that is amenable to either Functional Assessment and/or BFB, and inclusion of other disorders (e.g., PTSD, Phobia, Depression) that are common within the non-hospitalised mental illness range could add to the data reported here. Finally, as explained earlier, the separation of the three major treatments (BFB, Relaxation Training, Systematic Desensitization) was not undertaken for ethical reasons. It is not justifiable to provide less-than-optimal treatment to people who seek treatment for their disorder, and therefore the issue of comparing these three aspects of the overall combined treatment package was not possible here.

7.5 Conclusions

This study resulted in a consistent and clinically significant reduction in the symptoms of GAD across all 11 participants, plus reductions in elevated RHR and improvement in RSA, two established indicators of SNS hyperarousal. Because the results of the Functional
Assessment and HR training were so uniform across all participants, and because these therapeutic techniques are within the ability of professionally-trained counsellors, this study might be used as an argument for including such therapeutic techniques in courses which train counsellors and related personnel who aspire to assist people to deal with one of the most common and debilitating of non-hospitalised mental health problems. Generalised Anxiety Disorder that arises from environmental and historical events is common in many people and can contribute an unnecessary disease burden upon those who suffer from it, those who care from them, and the wider society that loses the talents of people who suffer from GAD. This study has demonstrated one method of alleviating that suffering in the counselling setting.
REFERENCES


APPENDIX A

Diagnostic Criteria for a Generalised Anxiety Disorder - DSM-IV-TR

A. Excessive anxiety and worry (apprehensive expectation), occurring more days than not for at least 6 months, about a number of events or activities (such as work or school performance).

B. The person finds it difficult to control the worry.

C. The anxiety and worry are associated with three (or more) of the following six symptoms (with at least some symptoms present for more days than not for the past 6 months). NOTE: Only one item is required in children.

1. Restlessness or feeling keyed up or on the edge.
2. Being easily fatigued.
3. Difficulty concentrating or mind going blank.
4. Irritability.
5. Muscle tension.
6. Sleep disturbances.

D. The focus of the anxiety and worry is not confined to features of an Axis I disorder:

The anxiety or worry is not about having a Panic Attack (as in Panic Disorder)
Being embarrassed in public (as Social Phobia)
Being contaminated (as in Obsessive-Compulsive Disorder)
Being away from home or close relatives (as in Separation Anxiety Disorder)
Gaining weight (as in Anorexia Nervosa)
Having multiple physical complaints (as in Somatisation Disorder)
Having a serious illness (as in Hypochondriasis)
The anxiety and worry do not occur exclusively during Post-Traumatic Stress Disorder.

E. The anxiety, worry or physical symptoms cause clinically significant distress or impairment in social, occupational or other important areas of functioning.

F. The disturbance is not due to the direct physiological effects of:
A substance (e.g. a drug of abuse, a medication)
A general medical condition (e.g. hyperthyroidism)
Does not occur exclusively during a Mood Disorder, a Psychotic Disorder or a Pervasive Developmental Disorder.
### APPENDIX B

**Summary of Features for Anxiety Disorders**

**Classification DSM-IV-TR**

<table>
<thead>
<tr>
<th>Anxiety Disorders</th>
<th>Psychological symptoms</th>
<th>Physiological Symptoms</th>
<th>Other</th>
</tr>
</thead>
<tbody>
<tr>
<td>Panic Disorder without Agoraphobia</td>
<td>Derealisation, Depersonalisation</td>
<td>Palpitations, Sweating, Trembling, Shortness of Breath, Feeling of Choking, Chest Pains, Nausea and Abdominal Distress, Dizziness, Paraesthesia, Chills and Hot Flashes</td>
<td>Must be recurrent</td>
</tr>
<tr>
<td></td>
<td>Fear of Losing Control</td>
<td></td>
<td>Not caused by medical conditions or substance use /abuse</td>
</tr>
<tr>
<td></td>
<td>Fear of Dying</td>
<td></td>
<td>Not caused by any other mental disorder</td>
</tr>
<tr>
<td>Panic Disorder with Agoraphobia</td>
<td>As for Panic Disorder without Agoraphobia</td>
<td>As for Panic Disorder without Agoraphobia</td>
<td>Must be recurrent</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Not caused by medical conditions or substance use /abuse</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Not caused by any other mental disorder</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Anxiety about being in places or situations from which escape might be difficult or in which help may not be available</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Specific situations are avoided or severe distress/panic attack</td>
</tr>
<tr>
<td>Agoraphobia without a History of Panic Disorder</td>
<td>No symptoms present to diagnose a Panic Disorder</td>
<td>No symptoms present to diagnose a Panic Disorder</td>
<td>Fear of developing panic-like symptoms</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Not caused by medical conditions or substance use /abuse</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Not caused by any other mental disorder</td>
</tr>
<tr>
<td>Specific Phobia</td>
<td>As for Panic Disorder without Agoraphobia</td>
<td>As for Panic Disorder without Agoraphobia</td>
<td>Marked and persistent fear that is excessive or unreasonable, cued by the presence or anticipation of a specific object or situation</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>The person recognise the fear is excessive or unreasonable</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>The phobic situation is avoided or intense anxiety is present when faced with situations</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>The avoidance significantly interferes with their normal routine</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Not caused by medical conditions or substance use /abuse</td>
</tr>
<tr>
<td>Disorder</td>
<td>As for Panic Disorder without Agoraphobia</td>
<td>As for Panic Disorder without Agoraphobia</td>
<td>Not caused by any other mental disorder</td>
</tr>
<tr>
<td>--------------------------------</td>
<td>------------------------------------------</td>
<td>------------------------------------------</td>
<td>---------------------------------------</td>
</tr>
<tr>
<td>Social Phobia</td>
<td></td>
<td></td>
<td>Marked and persistent fear of one or more social or performance situations Exposure to the situation provokes anxiety The person recognise the fear is excessive or unreasonable The situation is avoided The avoidance significantly interferes with the persons normal routine Not caused by medical conditions or substance use /abuse Not caused by any other mental disorder</td>
</tr>
<tr>
<td>Obsessive-Compulsive Disorder</td>
<td></td>
<td></td>
<td>Recurrent obsessions/compulsions The person recognises that the obsessions/ compulsions are excessive The obsessions/compulsions caused marked distress and interferes with the person’s normal routine Not caused by medical conditions or substance use /abuse Not caused by any other mental disorder</td>
</tr>
<tr>
<td>Post-Traumatic Stress Disorder</td>
<td>Recurrent intrusive thoughts Recurrent distressing dreams Intense psychological distress Difficulty concentrating</td>
<td>Physiological reactivity Persistent symptoms of insomnia, irritability or anger Hypervigilance Exaggerated startle response</td>
<td>The person has been exposed to a traumatic event which threatened death or serious injury The person’s response involves intense fear, helplessness or horror Acting and feeling as if the traumatic event was reoccurring Persistent avoidance of stimuli associated with the event The symptoms significantly interfere with the person’s normal routine</td>
</tr>
<tr>
<td>Acute stress Disorder</td>
<td>Subjective sense of numbing Detachment or absence of emotional responsiveness Reduction of awareness of surroundings Derealisation, depersonalisation Dissociative amnesia</td>
<td>Increased arousal Difficulty sleeping Irritability Hypervigilance Exaggerated startle response Motor restlessness</td>
<td>The person re-experience the traumatic event with recurrent images, thoughts, dreams, illusions, flashback episodes or a sense of reliving the experience Marked avoidance of stimuli that arouse recollection to the trauma</td>
</tr>
</tbody>
</table>
| Generalised Anxiety Disorder | Difficulty in controlling worry  
Easily fatigued  
Difficulty in concentrating | Restlessness  
Irritability  
Muscle tension  
Sleep Disturbances; insomnia, restless sleep | Excessive anxiety and worry  
The worry or anxiety significantly interfere with the person’s normal routine  
Not caused by medical conditions or substance use /abuse  
Not caused by any other mental disorder |
| --- | --- | --- | --- |
| Anxiety Disorder due to a Medical Condition | As for Panic Disorder without Agoraphobia | As for Panic Disorder without Agoraphobia | History, physical or laboratory findings of a medical condition  
The disturbance does not occur exclusively during the course of delirium  
The disturbance significantly interferes with the person’s normal routine |
| Substance-Induced Anxiety Disorder | As for Panic Disorder without Agoraphobia | As for Panic Disorder without Agoraphobia | History, physical or laboratory findings of intoxication or withdrawal or medication use etiology related to the disturbance  
The disturbance does not occur exclusively during the course of delirium  
The disturbance significantly interferes with the person’s normal routine |
| Anxiety Disorder NOS | As for Panic Disorder without Agoraphobia | As for Panic Disorder without Agoraphobia | Mixed anxiety-depressive disorder  
Clinical significant social phobia due to a general medical or mental disorder  
Situations where the disturbance is severe enough but the individual fails to report enough symptoms for a specific anxiety disorder |
### APPENDIX C

**Level 5 Efficacy for Biofeedback**

<table>
<thead>
<tr>
<th>Author/s</th>
<th>Year</th>
<th>Participants</th>
<th>Diagnosis</th>
<th>Modalities</th>
<th>Treatment outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Susset, J. G., Galea, G., &amp; Read, L.</td>
<td>1990</td>
<td>Women from Veterans Administration - Brown University Medical School Rhode Island 15 participants</td>
<td>Urinary incontinence</td>
<td>Intravaginal probe biofeedback</td>
<td>Significant improvement proven 12 women had 100% subjective and objective improvement. 2 women had 65-75% improvement. 1 woman failed to respond.</td>
</tr>
</tbody>
</table>
## APPENDIX D

### Level 4 Efficacy for Biofeedback

<table>
<thead>
<tr>
<th>Author/s</th>
<th>Year</th>
<th>Participants</th>
<th>Diagnosis</th>
<th>Modalities</th>
<th>Treatment outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Garrett B.L., &amp; Silver M.P.</td>
<td>1976</td>
<td>Enrolled college students 50 divided in 5 groups</td>
<td>Anxiety disorder</td>
<td>Randomised control study  1. Alpha enhancement  2. EMG voltage reduction  3. Alpha enhancement + EMG voltage reduction  4. Relaxation training  5. No treatment</td>
<td>Only participants in the 3 feedback groups reported significant reduction in test anxiety suggesting that improvements were not due to passage of time or non-specific factors.</td>
</tr>
<tr>
<td>Rice K.M., Blanchard E. B., &amp; Purcell M.</td>
<td>1993</td>
<td>Students and staff from SUNY-Albany and community members 45 participants</td>
<td>GAD</td>
<td>1. Frontal EMG Biofeedback  2. Alpha Enhancement Biofeedback  3. Alpha suppressant biofeedback  4. Psuedomeditation</td>
<td>The EEG alpha increase biofeedback group showed significant reduction in heart rate The EMG biofeedback groups showed significant clinical improvements in STAI-Trait scores.</td>
</tr>
<tr>
<td>Vanathy S., Sharma P. S. V. N., &amp; Kumar K. B.</td>
<td>1998</td>
<td>Community members 18 participants</td>
<td>GAD</td>
<td>1. Waiting list  2. Alpha enhancement/ Beta suppressant protocol  3. Theta enhancement/ Beta suppressant protocol</td>
<td>Improvement in both treatment groups relative to the untreated control group.</td>
</tr>
<tr>
<td>Hitanshu A., Maman P., &amp; Jaspal S.S.</td>
<td>2008</td>
<td>Community members 45 participants</td>
<td>GAD</td>
<td>1. EMG biofeedback  2. EEG biofeedback  3. Control group</td>
<td>Significant reduction in both treatment groups relative to the untreated control group EMG group was the most effective treatment.</td>
</tr>
<tr>
<td>Romano, J.L.</td>
<td>1978</td>
<td>Students from the College of Education 40 participants</td>
<td>Anxiety disorder</td>
<td>1. EMG Assisted Systematic Desensitization  2. EMG Relaxation Training  3. Automated Systematic Desensitization</td>
<td>No significant difference between treatment efficacy in all 3 groups.</td>
</tr>
<tr>
<td>Author(s)</td>
<td>Year</td>
<td>Study Details</td>
<td>Participants</td>
<td>Interventions</td>
<td>Outcomes</td>
</tr>
<tr>
<td>-----------</td>
<td>------</td>
<td>---------------</td>
<td>--------------</td>
<td>---------------</td>
<td>----------</td>
</tr>
<tr>
<td>Bradley R. T., McCraty R., Atkinson M., Tomasino D., Daugherty A., &amp; Arguelles L.</td>
<td>2010</td>
<td>10th grade students from Two schools in Northern Carolina</td>
<td>136 participants</td>
<td>Healthy individuals but exposed to stressors causing anxiety</td>
<td>1. TestEdge program with Psychophysiological Feedback 2. Control group</td>
</tr>
<tr>
<td>Hendriques G., Keffer S., Abrahamson C., &amp; Horst S.J.</td>
<td>2011</td>
<td>First year Psychology Students</td>
<td>28 participants</td>
<td>GAD</td>
<td>HRV</td>
</tr>
<tr>
<td>Masafi S., Rezaei O., &amp; Ahadi H.</td>
<td>2011</td>
<td>Women from Emam Khomeimi Hospital</td>
<td>14 participants</td>
<td>Anxiety Disorder</td>
<td>1. Biofeedback training group 2. Control group</td>
</tr>
<tr>
<td>Nakao M.</td>
<td>2000</td>
<td>Harvard University students</td>
<td>38 participants</td>
<td>1. White-coat hypertension 2. Essential hypertension 3. Control group</td>
<td>Biofeedback Training Biofeedback Training No treatment</td>
</tr>
<tr>
<td>Thompson L., &amp; Thompson M.</td>
<td>2005</td>
<td>Meta-Analysis</td>
<td>ADHD</td>
<td>Neurofeedback (EEG biofeedback)</td>
<td>Significant improvement in listening skills, memory, focus and following through on completing tasks.</td>
</tr>
<tr>
<td>Authors</td>
<td>Year</td>
<td>Study Type</td>
<td>Participants</td>
<td>Conditions</td>
<td>Treatments / Methods</td>
</tr>
<tr>
<td>-------------------------</td>
<td>------</td>
<td>---------------------</td>
<td>---------------------------------------</td>
<td>------------------------------------------------</td>
<td>--------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Schwartz M.S. &amp; Andrasik F.</td>
<td>2003</td>
<td>Meta-Analysis</td>
<td>Various types of headaches</td>
<td>All biofeedback modalities</td>
<td>Effective treatment for muscle tension headaches, psychogenic headaches, vascular headaches, migraines, combination headaches and mixed headaches.</td>
</tr>
<tr>
<td>Andrasik F.</td>
<td>2003</td>
<td>Comparative study</td>
<td>Various types of headaches</td>
<td>Biofeedback training, Relaxation training, CBT</td>
<td>Efficacy proven in Chronic daily high intensity headaches, refractory headaches, cluster headaches, chronic tension headaches, post-traumatic headaches, psychiatric co morbidity, and chronic pain traps.</td>
</tr>
<tr>
<td>Wahbeh H., &amp; Oken B.S.</td>
<td>2012</td>
<td>Community members from Portland Veterans Administration Medical Centre 86 participants</td>
<td>59 with PTSD, 27 without PTSD</td>
<td>EEG Feedback, HRV, Respiration rate</td>
<td>PTSD group had more statistically significant treatment changes than non PTSD group.</td>
</tr>
<tr>
<td>Bornas X., Riera del Amo A., Tortella-Feliu M., &amp; Llabres J</td>
<td>2012</td>
<td>Community members 45 participants</td>
<td>Flight phobia</td>
<td>HRV</td>
<td>Treatment study reports high or acceptable rate of success.</td>
</tr>
</tbody>
</table>
# APPENDIX E

Inclusion/Exclusion Criteria

<table>
<thead>
<tr>
<th>Participant</th>
<th>Age</th>
<th>Gender</th>
<th>Class</th>
<th>Diagnosis of GAD</th>
<th>Health Exclusions</th>
<th>Medication</th>
<th>Illicit Drug Use</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>18</td>
<td>F</td>
<td>Student</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>2</td>
<td>53</td>
<td>M</td>
<td>Lecturer</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>3</td>
<td>20</td>
<td>F</td>
<td>Student</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>4</td>
<td>57</td>
<td>M</td>
<td>Lecturer</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>5</td>
<td>39</td>
<td>F</td>
<td>Lecturer</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>6</td>
<td>28</td>
<td>M</td>
<td>Student</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>7</td>
<td>24</td>
<td>F</td>
<td>Student</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>8</td>
<td>32</td>
<td>F</td>
<td>General Staff</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>9</td>
<td>23</td>
<td>F</td>
<td>Student</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>10</td>
<td>30</td>
<td>F</td>
<td>General Staff</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>11</td>
<td>46</td>
<td>M</td>
<td>Student</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>12</td>
<td>23</td>
<td>F</td>
<td>Student</td>
<td>PTSD</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>13</td>
<td>26</td>
<td>M</td>
<td>General Staff</td>
<td>Social Phobia</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>14</td>
<td>29</td>
<td>F</td>
<td>General Staff</td>
<td>Yes</td>
<td>No</td>
<td>Beta blockers</td>
<td>No</td>
</tr>
<tr>
<td>15</td>
<td>20</td>
<td>F</td>
<td>Student</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>Cannabis</td>
</tr>
<tr>
<td>16</td>
<td>45</td>
<td>F</td>
<td>Lecturer</td>
<td>Yes</td>
<td>Myocardial infarction</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>17</td>
<td>32</td>
<td>F</td>
<td>General Staff</td>
<td>Yes</td>
<td>Hyperthyroidism</td>
<td>Thyroxin</td>
<td>No</td>
</tr>
<tr>
<td>18</td>
<td>19</td>
<td>M</td>
<td>Student</td>
<td>OCD</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>19</td>
<td>21</td>
<td>F</td>
<td>Student</td>
<td>PTSD</td>
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<td>No</td>
<td>No</td>
</tr>
<tr>
<td>20</td>
<td>20</td>
<td>M</td>
<td>Student</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>Amphetamines</td>
</tr>
<tr>
<td>21</td>
<td>20</td>
<td>F</td>
<td>Student</td>
<td>Yes</td>
<td>Yes</td>
<td>Benzodiazepines</td>
<td>No</td>
</tr>
</tbody>
</table>
APPENDIX F

BOND UNIVERSITY

Interview Schedule

The interview will be adapted to the individual client’s needs. The following questions are included in order to obtain data for the Functional Analysis.

1. Client’s biographical details:
   Name: ____________________________________________________________
   Date of birth: ______________________________________________________
   Sex: ______________________________________________________________
   Address: __________________________________________________________
   ________________________________________________________________
   ________________________________________________________________
   ________________________________________________________________
   Phone: ___________________________________________________________
   Occupation: _______________________________________________________  

2. Family history:
   Married, de-facto, single, widowed, divorced or separated: ________________
   Number of children: ________________________________________________

3. Medical history:
   Existing medical condition(s): _________________________________________
   Current medication(s): ______________________________________________
   Previous medical condition(s): _________________________________________
   Previous psychological condition(s): ________________________________
   Family history of medical/psychological condition(s): __________________


4. History of professional assistance:
_________________________________________________________________
_________________________________________________________________
_________________________________________________________________

5. Presenting problem:
(In client's own words)
_________________________________________________________________
_________________________________________________________________
_________________________________________________________________
_________________________________________________________________

6. Behavioural difficulties as expressed by the client:
_________________________________________________________________
_________________________________________________________________
_________________________________________________________________
_________________________________________________________________

7. Length of time the behaviour has been a concern to or limiting factors in the client's life:
_________________________________________________________________

8. Possible maintaining conditions:
Location or setting in which the behaviour occurs:
_________________________________________________________________

Times at which the behaviour occurs:
_________________________________________________________________

Activity undertaken when the behaviour occurs:
_________________________________________________________________

People present when the behaviour occurs:
_________________________________________________________________
9. **Form analysis:**
Dimensions of the anxiety related behaviour:

- **Duration:**

- **Frequency:**

- **Latency:**

- **Magnitude:**

10. **Functional assessment:**
Setting events:

Proximal antecedents:

- **Setting:**

- **Interactions:**

- **Tasks:**

11. **Post-behaviour maintaining variables:**
Immediate consequences:

- **Positive reinforcements:**

- **Negative reinforcements:**

Delayed consequences:

- **Positive reinforcement:**

- **Negative reinforcement:**
12. Functions for GAD
Are any of the following factors present?

<table>
<thead>
<tr>
<th>Function Label</th>
<th>Participant Self-Report Experience</th>
</tr>
</thead>
<tbody>
<tr>
<td>Escape</td>
<td></td>
</tr>
<tr>
<td>Sensory reinforcement</td>
<td></td>
</tr>
<tr>
<td>Avoidance of social context</td>
<td></td>
</tr>
<tr>
<td>Attention</td>
<td></td>
</tr>
<tr>
<td>Change to reactions of others</td>
<td></td>
</tr>
<tr>
<td>Access to preferred Activity</td>
<td></td>
</tr>
<tr>
<td>Internal change</td>
<td></td>
</tr>
<tr>
<td>Internal state - psychological change</td>
<td></td>
</tr>
</tbody>
</table>

Diagnostic Criteria for a Generalised Anxiety Disorder - DSM-IV-TR.

A. Excessive anxiety and worry occurring more days than not for at least 6 months, about a number of events or activities (such as work or school performance).

   **Criterion A**

<table>
<thead>
<tr>
<th>YES</th>
<th>NO</th>
</tr>
</thead>
</table>

B. The person finds it difficult to control the worry.

   **Criterion B**

<table>
<thead>
<tr>
<th>YES</th>
<th>NO</th>
</tr>
</thead>
</table>

C. The anxiety and worry are associated with three (or more) of the following six symptoms (with at least some symptoms present for more days than not for the past 6 months).

   7. Restlessness or feeling keyed up or on the edge.   YES   NO
   8. Being easily fatigued.                             YES   NO
   9. Difficulty concentrating or mind going blank.     YES   NO
   10. Irritability.                                     YES   NO
   11. Muscle tension.                                   YES   NO
   12. Sleep disturbances.                               YES   NO

   **Criterion C**

<table>
<thead>
<tr>
<th>YES</th>
<th>NO</th>
</tr>
</thead>
</table>
D. The focus of the anxiety and worry is not confined to features of an Axis I disorder:

The anxiety or worry is not about having a Panic Attack (as in Panic Disorder)  

| YES | NO |

Being embarrassed in public (as Social Phobia)  

| YES | NO |

Being contaminated (as in Obsessive-Compulsive Disorder)  

| YES | NO |

Being away from home or close relatives (as in Separation Anxiety Disorder)  

| YES | NO |

Gaining weight (as in Anorexia Nervosa)  

| YES | NO |

Having multiple physical complaints (as in Somatisation Disorder)  

| YES | NO |

Having a serious illness (as in Hypochondriasis)  

| YES | NO |

The anxiety and worry do not occur exclusively during Post-Traumatic Stress Disorder.  

| YES | NO |

**Criterion D**  

YES  NO

E. The anxiety, worry or physical symptoms cause clinically significant distress or impairment in social, occupational or other important areas of functioning.

**Criterion E**  

YES  NO

F. The disturbance is not due to the direct physiological effects of:

A substance (e.g. a drug of abuse, a medication)  

| YES | NO |

A general medical condition (e.g. hyperthyroidism)  

| YES | NO |

Does not occur exclusively during a Mood Disorder, a Psychotic Disorder or a Pervasive Developmental Disorder.  

| YES | NO |

**Criterion F**  

YES  NO

**Does the client meet the criteria for a Generalised Anxiety Disorder?**  

YES  NO
APPENDIX G

Problems with anxiety in daily life?

Participate in this new project!

The research will be conducted by Dr Phillip Fourie at the School of Health Science at Bond University. All sessions are held during office hours.

This study will help me to analyse and understand anxiety and anxiety-related behaviour and to find more effective treatment for clients suffering from general anxiety.

The study consists of two stages:

**Stage one:** One or two one-hour interview session(s) at the School of Health Sciences, Bond University. This will be an interview to collect data regarding anxiety and anxiety-related symptoms.

**Stage two:** Four to five one-hour treatment sessions for anxiety-related problems.

During stage one data will be collected (This stage has no therapeutic intent) and treatment will only start in stage two of this study.

If you are interested please call 07-5595 4171 or return my e-mail: pfourie@staff.bond.edu.au and leave your name and contact number and I will get back to you with details.
Intake Form

The interview will be adapted to the individual client’s needs. The following questions are included in order to obtain data for the Functional Analysis.

1. **Client’s biographical details:**

   Name: ________________________________________________________________
   
   Date of birth: __________________________________________________________
   
   Sex: __________________________________________________________________
   
   Address: ______________________________________________________________
   
  OMB: __________________________________________________________________
   
   Phone: __________________________________________________________________

   Occupation: _____________________________________________________________

2. **Family history:**

   Married, de-facto, single, widowed, divorced or separated:

   Number of children: ______________________________________________________
3. Medical history:

Existing medical condition(s):

Current medication(s):

Previous medical condition(s):

Previous psychological condition(s):

Family history of medical/psychological condition(s):

4. History of professional assistance:


5. Presenting problem:


I acknowledge that all information on this form will be treated as confidential information at all times.

Signed: ______________________              Dated: ______________________
Explanatory Statement
Stage 1

School of Health Science
Dear Sir/Madam

I am conducting a study, “An evaluation of Functional Assessment and Biofeedback for the Treatment of Generalised Anxiety Disorder”, project number: R0224, to help me understand anxiety and anxiety-related behaviour and to find more effective treatment for clients suffering from anxiety.

The first stage of this study would involve one or two formal interview(s) of approximately one hour each. These interviews will be conducted at the School of Health Sciences, Bond University. During these interviews, information will be gathered in relation to the reason(s) that caused the anxiety and the duration of the anxiety-related symptoms and the specific behaviour that developed because of the anxiety. Please note that Stage 1 has no therapeutic intent. The information from this interview would be analysed for the second stage of the study.

The research is conducted for a thesis for a Doctorate of Counseling, under the supervision of Professor Chris Sharples and Associate Professor Vicki Bitsika.

The collected data is treated with the strictest confidentiality and will be secured in locked premises after completion of the project. Only researchers taking part in the study will have access to the data. Your name will be on the data collected in stage one but the final report contains summarized findings and individual results will remain anonymous.

Your participation would be highly appreciated. However, you are under no obligation to do so. If at any stage during the course of the study you no longer wish to participate, you are free to withdraw consent.
Please sign and date the attached form if you agree to participate.

If any problems occur during the research, the Complaints Officer, Jodie Maguire can be contacted at Bond University, telephone: 07-5595 4001.

If you require any further information, do not hesitate to contact me on 5595 4171.

Yours truly,

Phillip Fourie
Consent Form

Research Title: An evaluation of Functional Assessment and Biofeedback for the Treatment of Generalised Anxiety Disorder.
Project number: R0244

Principal investigator: Professor Chris Sharpley.
Associate investigator: Doctor Phillip Fourie.

I, ___________________________ have read the information provided and understand the (Full Name) requirements of this study.

I agree to participate under the following terms:

1. I have read the explanatory information associated with this consent form and agree to requirements contained therein.
2. I am free to withdraw from this study at any time, without any repercussions.
3. My anonymity will be maintained in any publication.
4. Any data I provide will be confidential and access will be restricted to the researchers.

Name: ___________________________________ (please print)

Signature: ___________________________ Date: ______________
## APPENDIX K

### Zung Anxiety Scale

<table>
<thead>
<tr>
<th>Question</th>
<th>None or a little of the time</th>
<th>Some of the time</th>
<th>Good part of the time</th>
<th>Most or all of the time</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. I feel more nervous and anxious than usual.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. I feel afraid for no reason at all.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. I get upset easily or feel panicky.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. I feel like I am falling apart and going to pieces.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. I feel that everything is all right and nothing bad will happen.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. My arms and legs shake and tremble.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7. I am bothered by headaches, neck and back pains.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8. I feel weak and get tired easily.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9. I feel calm and can sit still easily.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10. I can feel my heart beating fast.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>11. I am bothered by dizzy spells.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>12. I have fainting spells or feel like it.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>13. I can breathe in and out easily.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>14. I get feelings of numbness and tingling in my fingers and toes.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>15. I am bothered by stomach aches or indigestion.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>16. I have to empty my bladder often.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>17. My hands are usually dry and warm.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>18. My face gets hot and blushes.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>19. I fall asleep easily and get a good night’s rest.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>20. I have nightmares.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
APPENDIX L

Progressive Muscle Relaxation (PMR)

PMR can be done by physically contracting the muscles/muscle groups or by focussing on or visualizing the specific muscle/muscle group and then to relax it.

The idea is to override the autonomic nervous system (sympathetic part) and voluntary taking control of muscle tension/tonus.

Be careful! If any part of your body is injured or has a weak area, only do visualization of that specific part.

Always remember to start with diaphragmatic breathing for a couple of minutes (as demonstrated and practiced during the session) and then do the PMR.
I prefer to start as far away from the central nervous system (brain) as possible.

Please follow the instructions below:

1. Toes – flex and extend your toes.
2. Arch the bridge of your feet and relax.
3. Ankles - flex and extend your ankles.
4. Contract your calf muscles and relax.
5. Contract and relax your upper legs.
6. Pull your pelvis in and relax.
7. Clench your gluteus muscle (back side) and relax.
8. Pull your abdomen in and relax.
9. Arch your lower back and relax (only if you do not have any back problems!)
10. Contract and relax your pectoral muscles (chest).
11. Form fists with your hands and relax.
12. Flex and extend your wrists.
13. Turn your forearms to one side then to the other (pronation and supination).
14. Flex and relax your biceps (front of upper arm).
15. Extend and relax you triceps (back of upper arm).
16. Pull your shoulders backwards.
17. Pull your shoulders forwards.
18. Pull your shoulders upwards. (towards your ears).
19. Put your chin on your chest (if you do not suffer from neck problems!)
20. Move your head to the left and relax.
21. Move your head to the right and relax.
22. Press your lips together and relax.
23. Close your eyes tight and relax.
24. Just relax and focus on breathing.

Remember to focus regularly on diaphragmatic breathing!
Do these exercises very slowly – approx. 20-30 minutes.
# APPENDIX M

**Treatment Regime by Sessions**

<table>
<thead>
<tr>
<th>Session</th>
<th>Purpose</th>
<th>Activity</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Orientation of participants and detailed explanation of study procedures.</td>
<td>Potential participants were greeted at the clinic by the receptionist. The receptionist provided each participant with a hard copy of the Intake Form and requested that (s)he complete it (Appendix H). The researcher introduced himself to the potential participant once the Intake Form was completed. The potential participant was taken to the consultation room begin the process of orienting him/her the study objectives and requirements. The inclusion criteria for the study were explained to the potential participant and the researcher checked that (s)he was willing to proceed with the session on the understanding that, if inclusion criteria were not met, the person would not be able to take part in the study. The two stages of the study were explained to the potential participant. The Explanatory Statement for Stage I was handed to him/her and the researcher requested that (s)he read the statement in his presence in order to gain clarification (if needed) and to re-state willingness to participate in the study after having reviewed study requirements a second time (Appendix I). After the potential participant read the Explanatory Statement for Stage I, (s)he was asked to sign the Consent form to confirm his/her intention to continue with the study (Appendix J). The Zung SAS was administered to the participant as part of the Functional Assessment and as a pre intervention measure for anxiety severity. An Intake Interview was conducted to gather biographical information and explore for the possible presence of GAD as</td>
</tr>
</tbody>
</table>
determined by the DSM-IV-TR diagnostic criteria (Appendix A).
The potential participant was asked to make a follow-up appointment the following week.
Session duration = 1 hour.

<table>
<thead>
<tr>
<th>II</th>
<th>Idiographic assessment of anxiety in relation to investigation of fear response and its maintaining variables.</th>
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<tbody>
<tr>
<td></td>
<td>The Intake Interview was continued to collect further information that allowed for a final check on the accuracy of researcher notes on anxiety symptomatology. This procedure was followed to confirm or reject the presence of GAD. This interview was also used to conduct a functional assessment of each participant’s anxiety-based behaviours and the impacts of those on day-to-day functioning (Appendix F). If the participant met the inclusion criteria for this study, (s)he was invited to continue with Stage 2. If the potential participant did not meet the criteria for this study, (s)he was offered alternative counselling by the researcher or (s)he was given 2-3 other counsellor’s contact details and a referral letter. The Explanatory Form, Stage II was handed and explained to the participant (Appendix N). The participant was asked to make a follow-up appointment the following week. Session Duration: 1 hour.</td>
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</table>

The Functional Assessment data were analysed between sessions II and III to determine fear response topography, setting and antecedent events which acted as triggers for the fear response, reinforcement processes which strengthened the fear response, and the functions which made the fear response adaptive.

<table>
<thead>
<tr>
<th>III</th>
<th>Introduce biofeedback training.</th>
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<td></td>
<td>The researcher familiarised each participant with the Biofeedback equipment (Bioview series IV, Zencor®) by explaining and demonstrating its functions and the locations of placement of electrodes. Participants were also shown, on a computer screen, examples of the RSA and Heart Rate graphs generated by collecting physiological data from them in this preliminary trial.</td>
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</tbody>
</table>
The researcher initiated the first Biofeedback session (i.e., baseline data collection [Heart Rate and RSA]) whilst discussing an anxiety-provoking incident with the participant. Descriptions of the anxiety-provoking incident were obtained from the Functional Assessment data.

Biofeedback Session Duration = 10 minutes.

The researcher explained the respiration-based training process via Respiratory Sinus Arrhythmia Feedback (RSAFB) to the participant.

The participant was familiarised with the type of feedback (s)he would be receiving during RSABF by viewing the computer screen on which his/her heart rate plots were shown. The researcher also demonstrated how he/she could increase the amplitude of his/her Respiratory Sinus Arrhythmia (RSA) by performing diaphragmatic breathing via an audio response.

The participant was asked to practice respiration-based training at home for 10 minutes twice daily over a one week period to assist him/her in becoming accustomed to diaphragmatic breathing.

The participant was asked to make a follow-up appointment the following week.

Session Duration = 45 minutes

| IV | Biofeedback training plus Visualisation. | Brief Biofeedback training of approximately 10 minutes was done to review the RSAFB while doing diaphragmatic breathing. If needed, the breathing technique was refined by explaining the procedure for a second time and practising the procedure until the participant mastered the technique. Visualisation was utilised in two ways. First, visualisation of an anxiety-provoking event with the instruction to the participant to focus on its neutral features was used to shape distraction from the threatening features of that event. Second, visualisation of a calm environment in which the participant felt secure. The participant was shown (via plots on the computer screen) |
how the two aspects (i.e., anxiety-provoking event *versus* calm environment) of this visualisation technique influenced his/her RSA amplitude.

The participant was asked to practice both the respiration-based training and the visualisation techniques at home, for 20 minutes per day for one week.

The participant was asked to make a follow-up appointment the next week.

**Session Duration = 1 hour.**

| V | Pair biofeedback training plus Progressive Muscle Relaxation (PMR). | Brief Biofeedback training of approximately 15 minutes was employed to review RSAFB while doing diaphragmatic breathing and the visualisation technique. If needed the techniques were refined via additional explanation and demonstration.

PMR was introduced as the basis for assisting the participant to discriminate between subjective feelings of muscle tension and muscle relaxation (Appendix L).

While the participant was guided through the PMR process in a stepwise manner, (s)he was also instructed to view how this technique was affecting his/her RSA amplitude on the computer screen. Audio cues were also used to assist in building participant focus on reactivity changes.

The participant was asked to make a follow-up appointment in two weeks.

**Session Duration = 1 hour.**

| VI | Pair biofeedback training plus exposure to tailored Systematic Desensitisation. | Biofeedback training of approximately 20 minutes was conducted to review RSAFB while doing diaphragmatic breathing, visualisation technique and PMR. If needed, these techniques were refined via active practice and explanation.

Systemic Desensitization was applied to assist participants in learning a non-anxious response to compete with and eventually replace anxious behaviour. The competing response, primarily focused on creating positive internal change, was |
shaped and reinforced over sessions via introduction of a participant-specific anxiety hierarchy with data obtained from the Functional Assessment. In order to ensure consistency across all participants, the anxiety hierarchy contained consistent features which included: only five-seven anxiety provoking events, a SUDS rating system which extended across 1 – 100 units, emphasis of the key aversive features of each anxiety-provoking event, and termination of hierarchy exposure at the anxiety-provoking event for which successful anxiety reduction was achieved.

The participant was verbally guided through the anxiety hierarchy and instructed to apply session III–V techniques to minimise any escalation in anxiety. (S)he received feedback via audio and RSAFB plots shown on the computer screen. The participant was then instructed to apply those techniques (s)he believed to be most effective in controlling his/her able RSA amplitude and therefore anxiety level in response to imagining a highly aversive anxiety-provoking event(s).

The participant was instructed to practice learned techniques at home, at least once a day for 30 minutes per session. The participant was asked to make a follow-up appointment in two weeks.

Session Duration = 1 hour.

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<tr>
<th>VII</th>
<th>Biofeedback training plus all learned technique</th>
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<td>Biofeedback training of approximately 30 minutes was conducted to review the participant’s capacity to apply the techniques learned during sessions III – VI. The participant was instructed to apply those techniques during exposure to a highly anxiety-provoking event to exert control over his/her RSA and heart rate and therefore anxiety. Audio and visual RSAFB was provided. The participant was asked to complete the Zung Anxiety Scale as a post intervention measure of anxiety level (Appendix K). Session Duration = 1 hour.</td>
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</table>
School of Health Science  
Dear Sir/Madam

I am conducting a study, “An evaluation of Functional Analytic Therapy for Generalised Anxiety Disorder”, project number: R0224, to help me understand anxiety and anxiety-related behaviour and to find more effective treatment for clients suffering from anxiety.

The second stage of my study consists of 4-5 one-hour counselling sessions. These sessions will take place at the Institute for Health Sciences at Bond University. During these sessions, I will use the information gained from the first stage of the study to develop a treatment plan that will be aimed to deal with the anxiety you experience and to help you to develop alternative coping skills to deal with this. This therapy will consist of relaxation therapy, biofeedback and respiration-based training, depending on your needs.

The research is conducted for a thesis for a Doctorate of Counseling, under the supervision of Professor Chris Sharpley.

The collected data is treated with the strictest confidentiality and will be secured in locked premises after completion of the project. Only researchers taking part in the study will have access to the data. Your name will be on your personal file but the final report contains summarized findings and individual results will remain anonymous.

Your participation would be highly appreciated. However, you are under no obligation to do so. If at any stage during the course of the study you no longer wish to participate, you are free to withdraw consent. Please sign and date the attached form if you agree to participate.

If any problems occur during the research, the Complaints Officer, Jodie Maguire can be contacted at Bond University, telephone: 07-5595 4001.

If you require any further information, do not hesitate to contact me on 5595 4171.

Yours truly,

Phillip Fourie