AUTONOMIC MECHANISMS IN SPIDER PHOBIA REACTIONS

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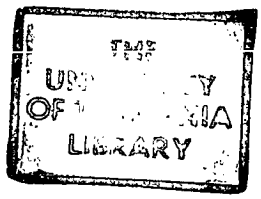

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This one's for you.

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A REVIEW OF THE LITERATURE

ABSTRACT

A strong fear of spiders is classified as a Specific Phobia, Animal Type (American Psychiatric Association, 1994). A number of theories have been suggested as to how this fear develops and how it is expressed in the physiological realm. This review addresses these issues. Systematic desensitisation has been the treatment of choice for phobias for some time. Issues concerning group administration of this procedure are discussed, as are a number of studies into physiological reflections of treatment success. Respiratory sinus arrhythmia (RSA) has been widely accepted as a measure of vagal tone in psychophysiological studies. This review outlines a current theory that explores the physiology of RSA and why it does not always respond in concert with heart rate. The potential implications of the RSA measure for psychophysiology and psychology as a whole are also discussed, as are a number of different variables which can affect the quantification of the RSA measure. The review concludes with an investigation into the possibility of using the RSA measure to specify the nature of the physiological mechanisms of specific phobia, both in terms of the strength of phobic response and also in terms of modifications due to a treatment program.

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The aim of this review is to explore the use of a psychophysiological measure, namely respiratory sinus arrhythmia, as an index of improvement in a clinical disorder, namely spider anxiety. The treatment of choice for phobia or strong fear is systematic desensitisation. This has been found to decrease autonomic nervous system responses to anxiety provoking stimuli, particularly heart rate and skin conductance responses. This arousal decrease has been assumed to be caused by reduced sympathetic tone. However, it could also reflect an increase in vagal control. The RSA index, when used as a measure sensitive to parasympathetic changes but independent of sympathetic alterations in arousal, may prove important in specifying the nature of the physiological mechanisms of fear and specific phobia.

SPIDER PHOBIA

A strong fear of spiders is classified by the Diagnostic and Statistical Manual of Mental Disorder, fourth edition, (DSM-IV) as a Specific Phobia, Animal Type (American Psychiatric Association, 1994). The essential feature of this disorder is a marked and persistent fear of clearly discernible, circumscribed objects or situations. Exposure to the phobic stimulus generally provokes an immediate anxiety response. The level of anxiety varies as a function of both the degree of proximity to the phobic stimulus (e.g., the fear intensifies as the spider approaches and decreases as it withdraws) and the degree to which escape from the stimulus is limited (e.g., the fear may be greater if the individual is near a spider in an enclosed space, such as a car). Adolescents and adults generally recognise that the degree of their fear is excessive or unreasonable, but this is not always the case with children. Age of onset for the animal subtype is generally during childhood and 75-90% of individuals with this type of Specific Phobia are female. An individual is only diagnosed with Specific Phobia if the avoidance, fear or anxious anticipation of encountering the phobic stimulus interferes significantly with daily routine, occupational or educational functioning or social life, or if the individual is markedly distressed about having the phobia (American Psychiatric Association, 1994).

The fear is experienced when the individual is either in the presence of, or is anticipating exposure to, the feared stimulus. The focus of this fear may be anticipated harm from some aspect of the object or situation (e.g., an individual may fear spiders because of concerns about being bitten) or may involve concerns about losing control, such as panicking or fainting on exposure to the feared object. As marked anticipatory anxiety occurs when the individual is confronted with the necessity of entering into the phobic situation, the phobic stimulus is generally avoided. Less commonly, the individual may force themselves to endure the situation, but it is then experienced with intense anxiety (American Psychiatric Association, 1994).

Cornelius and Averill (1983) reported the existence of a sex difference in the fear of spiders. In the presence of a live spider, women reported more subjective unpleasantness and tension and had higher heart rates than men. The women also displayed greater reluctance to be close to the spider. Analysis indicated that these differences were due to a genuine sex-linked difference in fear and were not merely an artefact of self-reports.

Although fears of circumscribed objects or situations are very common in the general population, especially amongst children, the degree of impairment or distress is rarely sufficient to warrant a diagnosis of Specific Phobia (American Psychiatric Association, 1994). The frequency of the different subtypes, which include Animal, Natural Environment, Blood-Injection-Injury and Situational, vary in adult clinical settings. The animal subtype is the least frequently presented. Several authors (e.g., Chapman, Fyer, Mannuzza, & Klein, 1993; Craske & Sipsas, 1992) have reported that individuals with specific phobias relatively rarely seek treatment, as interference with daily functioning is mostly limited, given the circumscribed nature of the phobia. Help seeking is not primarily associated with the severity of the illness. Rather, presentation in a clinical setting appears to be disproportionately associated with four main factors: 1) the presence of certain types of "functionally impairing" phobias, such as those involving commonly encountered stimuli (e.g., dogs or elevators), 2) the presence of more than one distinct phobia, 3) the presence of panic symptoms in the phobic situation, and 4) the relative absence of blood-injury fears (Chapman et al., 1993). Thus, specific phobias tend to present at clinical settings most often as

additional or secondary problems. It is partly because of this that many studies into anxiety choose to utilise a nonclinical, analogue subject sample, such as spider fearfuls (e.g., Craske & Sipsas, 1992; Hare, 1973; Robinson & Suinn, 1969).

Pathways to spider phobia

Historically, conditioning has been seen as the most important determining factor for the development of a phobia (Öst, 1985; Rachman, 1977). However, Rachman (1977) stated that while this theory had merit and some experimental and clinical support, it did not represent a satisfactory or comprehensive account of the development and maintenance of fear. It was thus acknowledged that there are at least three major pathways: conditioning, modelling or vicarious acquisition, and transmission via information or instruction.

A number of studies have investigated the pathways, or conditioning experiences, that lead to the development of spider phobia specifically (e.g., Merckelbach, Arntz, Arrindell, & De Jong, 1992; Merckelbach, Arntz, & De Jong, 1991). Such studies have tended to indicate that conditioning experiences are the most prominent factor, followed closely by modelling and then instruction / information. However, frequency of conditioning and modelling experiences failed to clearly differentiate spider phobic from non-fearful individuals; as both groups reported experiencing such situations. The only convincing difference between fearful and non-fearful individuals appeared to be instances of informational learning about spiders, with non-fearful individuals recalling a greater number of such instances. It may well be that it is the intensity of conditioning or modelling events, rather than their mere occurrence, that differentiates spider phobics from non-fearful individuals.

Phobic fear model

Phobic fear is held to comprise a set of loosely coupled components which include psychophysiological reactivity, verbal or cognitive reports of subjective anxiety and overt avoidance behaviour (Hugdahl, 1981). Each of these responds in a similar manner to the feared stimulus.

Alternatively, these three components may react differently and improve at different rates during treatment. The "Three-Systems-Model" of fear and emotion was originally proposed by Lang in 1968 (Hugdahl, 1981). It was based on the finding that, during automated desensitisation, some subjects showed rapid change in overt behaviour while continuing to label themselves as fearful, while others demonstrated a lessening of fear as measured by questionnaires but still experienced marked cardiovascular reactions. This phenomenon has been termed "desynchrony" (e.g., Abelson & Curtis, 1989; Hodgson & Rachman, 1974).

As these three components (psychophysiological reactivity, verbal/cognitive reports and overt avoidance behaviour) are seen as distinct behavioural systems and do not necessarily covary, it is important to record from all three during assessment procedures (Hugdahl, 1989).

The psychophysiology of specific phobia

Wolpe (1969) stated that, as a fear response, a phobic reaction should be associated with an increase in autonomic nervous system (ANS) arousal. Hugdahl (1989) summarised the findings concerning psychophysiological response patterns in specific phobia (see Table 1). There was found to be a general increase in both phasic and tonic electrodermal activity, reflecting increased sympathetic nervous system (SNS) activity. Also, an increase in both heart rate (HR) and blood pressure was found in phobic reactions, accompanied by an increase in peripheral resistance, or vasoconstriction. Skeletal muscle responses, usually recorded from the frontalis or forearm extensor muscles, involve either increased or decreased electromyograms. The electroencephalogram shows a reduction in alpha 8-13 Hz activity, coupled with reduced contingent negative variations and an increase in the P300-component amplitude of the event related potential to the fear relevant stimulus.

Prigatano and Johnson (1974) investigated ANS changes associated with a specific spider phobic reaction. Results indicated that spider phobics showed significantly faster HR, greater HR variability and vasoconstriction during exposure to spider slides as compared to non-spider phobics. It would generally be expected that reduced HR variance would occur in conjunction with increased HR (Porges, 1995). Phobics

also showed more frequent phasic skin responses, but not larger skin response amplitudes, to spider slides. Respiration rate and amplitude did not differ significantly between phobics and non-phobics. It was concluded that while general ANS arousal occurs during a spider phobic reaction, the demand characteristics of the phobic situation (such as the respective motor, cognitive and verbal requirements) may greatly influence which physiological responses are most affected.

Table 1: *Typical psychophysiological response patterns in simple phobic fears.*

Electrodermal system	- Increase in skin conductance response (SCR) amplitudes - Increase in frequency of non-specific skin conductance fluctuations (NSSCRs) - Increase in skin conductance levels (SCL), secondary to response increases - Large initial orienting responses, with retarded habituation
Cardiovascular system	- Increase in heart rate (HR) - Increase in systolic and diastolic blood pressure - Peripheral vasoconstriction -Phasic HR acceleration to repeated stimulation
Muscles	- Both muscle tension increase and decrease (frontalis, forearm extensors)
Electrocortical system	- Reduced alpha (8-13 Hz) activity - Reduced contingent negative variation (CNV) amplitude - Increased P300-component amplitude of the event-related potential (ERP)

Source: Hugdahl (1989). Simple Phobias. In G. Turpin (Ed.) *Handbook of Clinical Psychophysiology*. p. 300.

Investigation of the physiological component of the spider phobia reaction has primarily focused on cardiovascular and electrodermal responses. A number of studies have subsequently demonstrated that when spider phobics are presented with spider-related stimuli (e.g., photographic slides or live spiders), heart rate invariably increases to levels significantly higher than pre-stimulus levels (e.g., Cornelius & Averill, 1983; Craske & Sipsas, 1992; Hare, 1973; Hare & Blevings, 1975; Merckelbach et al., 1991) as does skin conductance (e.g., Hare, 1973; Hare & Blevings, 1975). Thus, most studies have specifically pointed to disturbances of the sympathetic branch of the ANS (Friedman, Thayer, Borkovec, Tyrrell, Johnson, & Columbo, 1993). Less commonly, diminished vagal tone has been implicated (e.g., George, Nutt, Walker, Porges, Adinoff, & Linnoila, 1989). One frequently used non-invasive method of assessing vagal tone is the quantification of respiratory sinus arrhythmia amplitude (Berntson, Cacioppo, Binkley, Uchino, Quigley, & Fieldstone, 1994).

RESPIRATORY SINUS ARRHYTHMIA

Reyes del Paso, Godoy, and Vila (1993) defined respiratory sinus arrhythmia (RSA) as "a phenomenon which consists of cyclic fluctuations in HR in close correspondence with respiratory phase" (p. 17). Put simply, during quiet breathing, HR accelerates with inspiration and decelerates with expiration. The phenomenon of RSA has been recognised as a measurable effect for many years; the first report of a connection between the phase of respiration and HR was attributed to Ludwig in 1847 (Melcher, 1976). At that time it was postulated that this effect was due to a change in the depolarisation frequency of the sinus node. Since this first documentation, explanations of the underlying physiology have undergone a great deal of change. Recent studies appear to largely agree that the physiological cause of RSA is central modulation of the baroreceptor reflex (e.g., Berntson, Cacioppo, & Quigley, 1993a; Kitney, 1986; Melcher, 1976; Selman, McDonald, Kitney, & Linkens, 1982). However, Porges (1995) has suggested a different emphasis.

The Polyvagal Theory

Physiological theory attributes both HR reduction (bradycardia) and increased RSA amplitude to direct vagal mechanisms (e.g., Berntson et al., 1994; Miller, 1994; Reyes del Paso et al., 1993; Riniolo, Doussard-Roosevelt, & Porges, 1994; Suess, Porges, & Plude, 1994). As a general rule, HR refers to the average of the heart rate level, while RSA refers to the range of variation in heart rate. However, although there are certain situations in which the two measures covary, such as during exercise or cholinergic blockade, there are other situations in which they appear to reflect independent sources of neural control.

During periods of stable respiratory parameters, and quite independent of method of quantification, data have been attained that demonstrate RSA and HR to respond differently. For example, Grossman and Kollai (1993) determined that individual differences in HR and RSA, monitored during resting conditions, provide independent contributions to measures of cardiac vagal tone derived from vagal blockade. The relationship between RSA and HR may also change both within and between individuals. Riniolo et al. (1994) observed that the relationship between RSA and HR varies with behavioural state; during states of drowsiness and sleep the correlation between the two is significantly lower than during alert states. Therefore, Porges (1995) summarised, at times RSA and HR appear to reflect the same physiological processes, whereas at other times they appear to reflect independent processes.

A number of arguments have been put forward to explain this discrepancy. Firstly, it has been argued that RSA and average HR may reflect different dimensions of vagal activity; with average HR reflecting tonic vagal influences and RSA reflecting phasic (Berntson et al., 1993a). Secondly, it has been suggested that the discrepancy may be caused by variations in respiratory parameters, whereby RSA is confounded by both respiratory rate and tidal volume and HR is not (Grossman, Karemaker, & Wieling, 1991). Alternatively, it has been hypothesised that differing quantification methods may form the basis for the discrepancy between RSA and HR (Byrne & Porges, 1993). Goldberger, Ahmed, Parker and Kadish (1994) argued that RSA may not always reliably measure parasympathetic tone because it decreases with baroreflex stimulation. Finally, it has been postulated that as average HR is influenced by a complex and dynamic interaction between sympathetic and vagal

systems it becomes difficult to extract a pure 'vagal tone' dimension (Berntson, Cacioppo, & Quigley, 1993b).

These arguments have generally utilised a definition of vagal tone as being determined by neural blockade, such that the functional effect of neural blockade on HR has been used as the criterion measure of vagal tone or parasympathetic control (e.g., Katona & Jih, 1975). RSA has long been recognised as a vagal phenomenon (e.g., Cacioppo, Uchino, & Berntson, 1994; Ekberg, 1983; Grossman, Stemmler, Karemaker, & Wieling, 1988; Grossman, Stemmler, & Meinhardt, 1990; Karemaker, 1986; Porges, 1986). HR, however, is determined by several sources, including vagal, sympathetic and mechanical factors (Porges, 1995). Therefore, the suitability of change in HR following neural blockade as an index of vagal tone may be legitimately challenged.

This apparent paradox between HR and RSA responses led Porges' (1995) to formulate the Polyvagal Theory which proposes that, in mammals, there are two anatomically based vagal response systems. The primary motor fibres of the vagus originate from two separate and definable nuclei in the medulla: the dorsal motor nucleus of the vagus (DMNX) and the nucleus ambiguus (NA). There are no apparent connections between these two nuclei. Most cells originating in the DMNX project to subdiaphragmatic structures, such as the stomach and intestines, while most cells in NA project to supradiaphragmatic structures, such as the larynx, pharynx and heart. It is hypothesised that such effects as bradycardia may be mediated by DMNX, while the suppression of HR variability, or reduced RSA amplitude, may be mediated by NA. Thus, the two commonly used measures of cardiac vagal tone, RSA and HR, may represent different mechanisms of vagal tone (Porges, 1995).

The Polyvagal Theory is based on a number of premises. The first of these states that neurogenic bradycardia and RSA are mediated by different branches of the vagus and therefore need not respond in concert (Porges, 1995). The second premise states that the neurogenic bradycardia associated with the orienting reflex is a relic of the reptilian brain and is mediated by DMNX. The third premise of the Polyvagal Theory states that withdrawal of cardiac vagal tone through NA-based mechanisms is a mammalian adaptation which facilitates the selection of novelty in the environment whilst maintaining metabolic output and continuous

social communication. The fourth premise states that the ability of NA to regulate special and general visceral efferents may be monitored by the amplitude of RSA. The fifth, and final, premise of Porges' (1995) Polyvagal theory states that emotion, as defined by changes in facial expression and vocalisations, will produce changes in RSA and bronchomotor tone mediated by NA.

Psychological implications of respiratory sinus arrhythmia

Recent applications of the RSA measure have included behavioural studies of attention, learning and cognitive effort, physiological studies of exercise, diurnal rhythms and central autonomic control and clinical studies of attentional dysfunctions and cardiovascular disease (Berntson et al., 1993a). Porges (1986) stated that RSA measures have been used to assess autonomic neuropathy in senility and diabetes research and as an indicator of developmental outcome of human neonates and that RSA amplitude is a very sensitive index of the level of anaesthesia during recovery from a surgical procedure. Uijtdehaage, Stern, & Koch (1992) indicated that RSA is also a good predictor of motion sickness.

Perhaps one of the most interesting uses of the RSA measure is as an indication of the effect of situational stressors on the cardiac parasympathetic response. Grossman and Svebak (1987) performed a study utilising RSA as an index of cardiac vagal responses to two active coping tasks: a video game with and without the threat of shock for inferior performance. The results indicated that parasympathetic withdrawal, as indexed by a reduction in RSA, could be an important contributor to exaggerated stress related cardiac reactivity. This withdrawal was associated with increased HR responses from rest to active coping conditions as well as between threat and no-threat conditions. Threat of shock was more effective in producing greater HR and sympathetic responses when the subject did not yet feel competent to perform the task. When a certain level of perceived competency had been achieved, more extreme reactions were not produced and HR responses appeared likely to be due to either parasympathetic withdrawal (the mechanism proposed by Grossman & Svebak (1987) to be responsible for psychophysiological reactions in panic attacks) or reciprocal diminution of vagal activity and sympathetic activation. Grossman and Svebak (1987) thus stressed the importance of vagal mechanisms and

sympathetic-parasympathetic interactions for stress-related cardiac reactivity.

Individual differences which may affect respiratory sinus arrhythmia

There are a number of different individual variables which may have an effect on the RSA measure; these include level of health and fitness, time of day, age and breathing rate.

One of the variables which can affect the RSA estimate is the level of health and fitness of the individual subject. Respiration, blood pressure and HR are all affected by this, especially in relation to any measurements taken after exercise (De Vries, 1980). Triffit (1991) suggested that it is plausible that fitness level may affect RSA amplitude and phase timing characteristics of RSA. Wilson and Archer (1994) found significant correlations for fitness with both age and sex, thus providing additional support for the importance of fitness as a predictor of RSA amplitude.

The time of day at which the RSA measure is sampled may also have an effect on the RSA amplitude. Stephens (1992) examined circadian variation in RSA amplitude, in a seated position, and discovered highly significant time of day effects, with a minimum at 0600 hours and peaks at both 1200 and 1800 hours for a six breaths per minute condition. At 12 breaths per minute a similar pattern was revealed. No time of day differences were found for inspiratory or expiratory phase lag, or under conditions of spontaneous breathing. Stephens (1992) thus concluded that time of day must be considered when measuring RSA or when investigating variables and tasks in which vagal tone is implicated. Hayano, Sakakibara, Yamada, Kamiya, Fujinami, Yokoyama, Watanabe, & Takata (1990) also examined diurnal variations in autonomic cardiac control. They discovered that, in the supine position, RSA was slightly but significantly greater in the morning than in the late afternoon and that it decreased markedly approximately thirty minutes after food intake. Under a standing condition, the RSA uniformly decreased to a level that was unaffected by time of day or food intake. This suggests that when measuring RSA, in either the supine or seated position, time of day should be recorded and kept constant in test-retest evaluations and should not be attempted within thirty minutes of food intake.

Another factor which may affect RSA amplitude is age. Wilson and Archer (1994) performed a study to determine what form the age-RSA effect takes, as previous studies had reached conflicting conclusions (e.g., Hellman & Stacy, 1976; Hirsch & Bishop, 1981). Subjects, both male and female and with no cardiopulmonary problems, ranged in age from sixteen to seventy-five years. Results supported the existence of an age-dependent linear degradation of the mechanisms producing RSA.

Perhaps one of the most important variables to affect RSA is respiration rate. Respiration rate (RR) has been demonstrated to have a strong negative relationship with RSA - as RR increases, RSA magnitude decreases (Grossman et al., 1991). However, this decrease in RSA does not necessarily reflect a decrease in the actual tonic level of cardiac vagal tone. Rather, it reflects an inspiratory-expiratory phasic change in vagal heart period. As it is the tonic cardiac vagal tone that is generally of interest to psychophysiological research it is thus recommended that respiratory parameters, such as RR, be controlled (Grossman et al., 1991).

Angelone and Coulter (1964) examined changes in both RSA amplitude and phase angle across a range of respiratory rates (1-40 cpm). Results demonstrated that HR is not necessarily in phase with respiration, but that the phase angle between HR fluctuations and respiration varies with frequency of breathing in a characteristic fashion. RSA amplitude varied from 3 to 25 cpm and phase angle between 0 and 300 degrees, with both phase angle and amplitude changing linearly as a function of RR. At a very low breathing rate the point of maximum expiration occurred at the same time as maximum HR - the expiration and HR were in phase. As breathing rate increased, the phase angle increased - such that HR began to "lag" breathing more and more. Peak RSA amplitude occurred at a rate of about 5-6 breaths per minute. This suggests that the optimal breathing rate for eliciting the classical pattern of RSA would be around 6-8 breaths per minute.

Increased RR has been found to be associated with intensified feelings of anxiety and fear (e.g., Clark & Hirschman, 1990; Ley, 1985; Svebak, Storfjell, & Dalen, 1982). In addition, Clark and Hirschman (1990), using a clinical population of alcohol-dependent inpatients scoring high in trait anxiety, found that the use of paced respiration (10 cpm) served to reduce self-rated tension, state anxiety and skin conductance levels. It was thus

concluded that respiratory pacing may serve as an easily learned self-control strategy and potentially useful therapeutic tool for dealing with anxiety (Clark & Hirschman, 1990).

SYSTEMATIC DESENSITISATION

The value of systematic desensitisation (SD) as a treatment for phobias has been well established (e.g., Carson, Butcher, & Coleman, 1988; Cormier & Cormier, 1991; Öst, 1978). The treatment is based on Wolpe's (1958) principle of reciprocal inhibition which states that "if a response antagonistic to anxiety can be made to occur in the presence of anxiety-evoking stimuli so that it is accompanied by a complete or partial suppression of the anxiety responses, the bond between these stimuli and the anxiety responses will be weakened" (p. 71). Put simply, it is impossible to be relaxed and anxious at the same time. If the sensation of relaxation is coupled with the image, or presence, of a feared object it should result in a decrease in subjective anxiety and its correlates.

When using SD, the phobic individual performs the response of relaxation in the presence of the stimulus whose anxiety arousing potency is to be eliminated (Lomont & Edwards, 1967). The relaxation response is said to suppress the anxiety response because it involves mainly parasympathetic processes, such as reductions in HR, blood pressure and RR, which act in an antagonistic way against the primarily sympathetic responses of the anxiety reaction (Sakakibara, Takeuchi, & Hayano, 1994).

Wolpe (1958) outlined the procedure for SD in the following way. Before the actual desensitisation process can begin an anxiety hierarchy must be formulated, which represents a list of stimulus situations to which the client reacts with graded amounts of anxiety. The client is then trained in a suitable relaxation technique, which may range from deep breathing to progressive muscle relaxation, depending on which is most appropriate for the individual. The weakest scenes from the hierarchy are then presented imaginally to the client, usually for between two to three seconds each to begin. Upon the raising of a finger, or any sign of bodily tension, presentation of the scene is stopped and relaxation begins.

The same scene is presented several times, usually with weaker reactions to each successive presentation, and then the therapy moves on to the next scene in the hierarchy. In this way, the client and therapist move through the hierarchy until the most anxiety-producing situation can be imagined with little or no apprehension.

Group administration

The increasing demand for psychological and psychiatric services creates a need for effective short-term therapeutic techniques. As a result, group techniques have grown in clinical stature. One of the most promising varieties of group therapy, as opposed to individualised therapy, is group administered SD (Lazarus, 1961). This technique employs a group hierarchy, which is composed of common elements extracted from individual client questionnaires or statements. Apart from this one deviation, the rest of the process follows the original SD plan as outlined by Wolpe (1958).

Robinson and Suinn (1969) studied twenty female spider fearful subjects who were given either group or individually administered SD following an exposure task. The results, upon conclusion of the therapy component, indicated no significant differences on a subjective fear questionnaire for individually versus group treated subjects. Using the direct behaviour measure, all subjects improved equally, whether they were treated in a group setting or individually. Thus, it can be concluded that group administration of SD is just as effective as individualised therapy.

Physiological reflections of treatment success

Psychophysiological analysis of the process of SD has revealed that fear signals are associated with an increase in autonomic arousal, while repeated presentation of the anxiety-provoking image is accompanied by a reduction in autonomic activity (Lang, Melamed, & Hart, 1970). The most consistently significant psychophysiological effects of desensitisation have been observed using skin resistance or cardiovascular responses, particularly HR (Mathews, 1971). Lacey (1967; in Mathews, 1971) suggested that HR decreases with the intake of external information from the environment, while concentration on internal, or

mental, events may lead to HR acceleration. The skin resistance response, however, was suggested to be more sensitive to the actual, as opposed to the imaginal, stimuli. Thus, HR may be a relatively sensitive indicator of response to phobic images while skin resistance may be more responsive to externally presented phobic stimuli. When considering these results it may be important to keep in mind the findings of Hugdahl (1981), in that the physiological, subjective and overt avoidance components may respond differently to treatment. Thus, a treatment program may be considered successful even when there is no obvious improvement in the physiological realm.

Levin, Cook and Lang (1982) examined the physiological response patterns of twenty-four clinically anxious clients during a series of tasks involving imagination of, and exposure to, their feared stimulus. HR, skin conductance level and several self-report measures of affective experience were recorded both before and after successful completion of exposure-based treatment. Levin et al. (1982) found a decline from pre-treatment levels in cardiac and skin conductance responses following treatment, during both imagery and exposure tasks. The authors thus concluded that psychophysiological assessment of emotional imagery can serve as a sensitive indicator of treatment progress.

In general, sympathetic stimulation increases the overall activity of the heart while parasympathetic stimulation decreases it (Guyton, 1986). Thus, HR can be determined by either or both of the sympathetic and parasympathetic nervous systems. Electrodermal phenomena are not only influenced by parts of the central nervous system but various cortical and subcortical regions are involved as well, forming a complex system, the role of which is not fully understood (Boucsein, 1992). Two different cerebral sources of electrodermal activity exist: a limbic-hypothalamic source, which is thermoregulatory and emotionally induced, and a premotor-basal ganglia source which prepares for specific motor actions. Part of the elicitation of such activity could be under parasympathetic control but debate persists on this point. Thus, while there have been studies into the physiological effects of SD, the question which remains is the extent to which these changes are sympathetic or parasympathetic in nature. The investigation of RSA as an indicator of treatment success may specify the extent of any vagal influences on this process.

CONCLUSION

In conclusion, a specific phobia is a persistent fear of a circumscribed stimulus, such as a spider. When individuals with such a phobia are exposed to their phobic stimulus they react with an immediate anxiety response. This has been shown to include an increase in ANS arousal, particularly in skin resistance and cardiovascular responses, such as HR. However, no reported studies have determined the precise autonomic process, or combination of processes, underlying these responses. A simultaneous investigation of both sympathetic and parasympathetic activity levels would enable a more comprehensive understanding of these underlying mechanisms.

One of the most frequently utilised measures of parasympathetic cardiac tone is RSA. Historically, this has been perceived to be due to central modulation of the baroreceptor reflex and has been regarded to have a close relationship with HR changes. However, a paradox has been found to exist as HR and RSA do not always covary. In an attempt to explain this paradox, Porges (1995) developed the Polyvagal Theory. This theory was explained in detail and its implications for RSA research were explored. A number of psychological and psychophysiological implications for the RSA measure have also been outlined, particularly the usefulness of RSA as an indicator for the effect of situational stressors on cardiac parasympathetic response. Additionally, a number of different individual variables which may influence the RSA measure have been discussed.

The utility of SD as a treatment of phobias or strong fears has been well established in the psychological literature and this has been summarised. Psychophysiological analysis of this procedure has revealed that it significantly reduced physiological activity, particularly within the ANS, in subjects anticipating a real-life phobic situation. However, it remains to be established whether these changes have their origin in the sympathetic or parasympathetic branch. It is thus likely that RSA, when used as a measure of vagal tone, may provide an effective indicator of severity of phobia and / also of treatment success based on physiological indices of arousal. As HR can be attributed to both sympathetic and parasympathetic activation, or a combination of the two, the utilisation of an RSA measure would specify the degree of direct vagal influence in

the process and thereby clarify the mechanism by which improvement occurs.

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AUTONOMIC MECHANISMS IN SPIDER PHOBIC REACTIONS

AN EMPIRICAL REPORT

ABSTRACT

The aim of this study was to investigate the physiological arousal response patterns of spider fearful individuals during exposure to a spider stimulus, both before and after completing a four week program of group administered systematic desensitisation, and to determine the extent of any vagal influences on these responses. Thirty-eight female participants (20 high spider fearful and 18 low spider fearful) were exposed to a spider at distances ranging from 310 cm to 25 cm. The physiological measures were respiratory sinus arrhythmia (RSA), mean heart rate, maximum and minimum heart rate and mean skin conductance level. Subjective ratings of stress, arousal and fear were also collected using visual analogue scales. The results indicated that when individuals are confronted with their feared stimulus they exhibit an initial increase in heart rate and skin conductance, coupled with a decrease in RSA. Following successful completion of the treatment program there was a reduction in heart rate and skin conductance and an increase in RSA, compared with pre-treatment levels. While most previous studies have focused on a sympathetic pathway to the physiological representation of fear, the major finding of this study was that vagal withdrawal was significantly implicated in the autonomic fear response.

A strong fear of spiders is classified by the Diagnostic and Statistical Manual of Mental Disorder, fourth edition, (DSM-IV) as a Specific Phobia, Animal Type (American Psychiatric Association, 1994). The essential feature of this disorder is a marked and persistent fear of clearly discernible, circumscribed objects or situations. Exposure to the phobic stimulus generally provokes an immediate anxiety response. The level of anxiety varies as a function of both the degree of proximity to the phobic stimulus and the degree to which escape from the stimulus is limited. Insight into the excessive or unreasonable nature of the fear tends to increase with age. Age of onset of the animal subtype is generally during childhood and 75-90% of individuals with this type of Specific Phobia are female. An individual is only diagnosed with Specific Phobia if the avoidance, fear or anxious anticipation of encountering the phobic stimulus interferes significantly with daily routine, occupational functioning or social life, or if the individual is markedly distressed about having the phobia (American Psychiatric Association, 1994).

Investigations into the origin of spider phobia have generally concluded that modelling, conditioning and informational learning all play an important role (e.g., Merckelbach, Arntz, Arrindell, & De Jong, 1992; Merckelbach, Arntz, & De Jong, 1991). It is possible, however, that it is the intensity of these conditioning and / or modelling events, rather than their mere occurrence, that differentiates phobics from non-fearful individuals. Additionally, it has been suggested that common animal fears may be mediated by a disgust reaction and that the acquisition process may reflect the transmission of disgust sensitivity in general (Davey, Forster, & Mayhew, 1993).

Investigation of the physiological component of the spider phobia reaction has primarily focused on cardiovascular and electrodermal responses. A number of studies have demonstrated that when spider phobics are presented with spider-related stimuli (e.g., photographic slides or live spiders), autonomic arousal occurs; as evidenced by heart rate (HR) increases to levels significantly higher than pre stimulus levels (e.g., Cornelius & Averill, 1983; Craske & Sipsas, 1992; Hare, 1973; Hare & Blevings, 1975; Merckelbach, de Jong, & Arntz, 1991; Prigatano & Johnson, 1974;) and similar increases in skin conductance (e.g., Hare, 1973; Hare & Blevings, 1975; Prigatano & Johnson, 1974).

The value of systematic desensitisation (SD) as a treatment for phobias has been well established (e.g., Carson, Butcher, & Coleman, 1988; Cormier & Cormier, 1991; Öst, 1978). The treatment is based on Wolpe's (1958) principle of reciprocal inhibition; in that if a response incompatible with anxiety can be induced in the presence of a feared stimulus, so that it leads to a complete or partial suppression of the anxiety, the bond between the stimuli and the anxiety response will be weakened.

Psychophysiological analysis of the process of SD has revealed that fear signals are associated with an increase in autonomic arousal, while repeated presentation of the anxiety-provoking image is accompanied by a reduction in autonomic activity (Lang, Melamed, & Hart, 1970). The most consistently significant psychophysiological effects of desensitisation have been observed using skin resistance or HR responses (Mathews, 1971). Lacey (1967; in Mathews, 1971) suggested that HR decreases with the intake of external information from the environment, while concentration on internal events may lead to HR acceleration. The skin resistance response, however, was suggested to be more sensitive to the actual, as opposed to the imaginal, stimuli. Thus, HR may be a relatively sensitive indicator of response to phobic images while skin resistance may be more responsive to externally presented phobic images.

Levin, Cook and Lang (1982) conducted a study investigating the physiological response patterns of anxious clients during exposure to their feared stimulus, both before and after successful completion of exposure-based treatment. They found a decline from pre-treatment levels in HR and skin conductance responses following treatment, during both imagery and exposure tasks. The authors thus concluded that psychophysiological assessment of emotional imagery can serve as a sensitive indicator of treatment progress.

Hugdahl (1981) described a "Three-Systems-Model" of fear, which allows for the three components of phobic fear (psychophysiological reactivity, subjective anxiety and overt avoidance behaviour) to improve at different rates during treatment. Thus, a treatment program can be interpreted as successful even if there is no large improvement in the physiological realm.

Traditionally, the psychophysiological literature has interpreted the tachycardia associated with anxiety to be a sign of beta-adrenergic hyperactivity, and thus largely sympathetic in origin (Kollai & Kollai, 1992). This has been despite the understanding that cardiac chronotropism is under the control of both the sympathetic and parasympathetic systems and that, in man, the parasympathetic system exerts the predominant influence on cardiac pacemaker function. In addition, studies examining the biochemical measures of sympathetic activity, such as plasma catecholamine levels, have yielded conflicting results; with some reporting increased and others reporting normal levels (George, Nutt, Walker, Porges, Adinoff, & Linnoila, 1989). This has led to exploration of the role of the parasympathetic nervous system in the autonomic arousal response and the suggestion that vagal withdrawal may contribute to the origin of panic. It has recently been elaborated that the tachycardia of anxiety disorders could be the result of either parasympathetic withdrawal, sympathetic activation or a combination of the two (e.g., Berntson, Cacioppo, & Quigley, 1993; Berntson, Cacioppo, Quigley, & Fabro, 1994).

To date, there have been no reported studies to investigate the precise autonomic process, or combination of processes, underlying the tachycardia seen in anxiety responses, such as a spider phobic reaction. A simultaneous assessment of both sympathetic and vagal cardiac activity levels would enable a more comprehensive understanding of these underlying mechanisms.

One frequently used non-invasive method of assessing vagal tone is the quantification of respiratory sinus arrhythmia (RSA) amplitude (Berntson, Cacioppo, Binkley, Uchino, Quigley, & Fieldstone, 1994). Reyes del Paso, Godoy, and Vila (1993) defined RSA as "a phenomenon which consists of cyclic fluctuations in HR in close correspondence with respiratory phase" (p. 17). Put simply, during quiet breathing, HR accelerates with inspiration and decelerates with expiration and RSA is a measure of this synchronised cardiac change. The non-invasive RSA measure correlates highly with more invasive estimates of vagal activity (e.g., Fouad, Tarazi, Ferrario, Fighaly, & Alicandri, 1984; Katona & Jih, 1975) and is more suitable for use in a laboratory setting. RSA has been demonstrated to vary in response to laboratory stressors (Grossman,

Stemmler, & Meinhardt, 1990) and to show a high degree of sensitivity to both psychological and behavioural variables (Berntson et al., 1993).

At present, most theories exploring the physiological basis for anxiety tend to favour a predominant role for either the autonomic nervous system or the central nervous system. Porges (1995) suggests that it is no longer appropriate to treat the two as functionally distinct, as the bidirectional connections between autonomic and central brain structures are becoming more apparent. Thus, a model is needed, based on the evolution of neural structures and the neural regulation of autonomic processes, to explain psychophysiological phenomena, such as emotion. In attempting to formulate such a model there is an obvious inconsistency between data and theory.

Physiological interpretation attributes HR decrease (bradycardia) and RSA amplitude increases to the direct effect of direct vagal mechanisms (e.g., Berntson, Cacioppo, Binkley, Uchino, Quigley, & Fieldstone, 1994; Miller, 1994; Reyes del Paso, Godoy, & Vila, 1993; Riniolo, Doussard-Roosevelt, & Porges, 1994; Suess, Porges, & Plude, 1994). In some situations, such as during exercise or cholinergic blockade, these two measures do covary. However, there are other situations in which HR and RSA appear to be independent and this creates a problem for the view that both responses are caused by the same mechanism (Porges, 1995).

A number of explanations have been suggested to explain this discrepancy. However Porges (1995) developed a new theory which states that the two responses are different mechanisms of vagal control.

Porges' (1995) Polyvagal Theory speculates that, in mammals, there are two anatomically distinct vagal response systems, based on two separate and definable nuclei in the medulla: the dorsal motor nucleus of the vagus (DMNX) and the nucleus ambiguus (NA). There do not appear to be any connections between these two nuclei. Most cells originating in the DMNX project to subdiaphragmatic structures, such as the stomach and intestines. In contrast, most cells in NA project to supradiaphragmatic structures, such as the larynx, pharynx and heart. Porges (1995) provides evidence to suggest that such effects as bradycardia may be mediated by DMNX while the suppression of HR variability, or

reduced RSA amplitude, may be mediated by NA. Thus, the two commonly used measures of cardiac vagal tone, RSA and HR, may represent different types of vagal tone (Porges, 1995).

The Polyvagal Theory is based on a number of premises. The major contribution of the theory is that there are two vagal systems, one mediated by DMNX and one by NA. Porges speculates that mammalian (and thus human), but not reptilian, brainstem organisation is characterised by a ventral vagal complex (including NA) related to processes associated with attention, motion, emotion and communication. The reptilian system is more largely characterised by DMNX action, which deals with unconscious reflexive vegetative functions. The other premises include the proposal that neurogenic bradycardia and RSA are mediated by different branches of the vagus and therefore need not respond in concert, that the ability of NA to regulate special and general visceral efferents may be monitored by the amplitude of RSA, and that emotion as defined by changes in facial expression and vocalisations will produce changes in RSA and bronchomotor tone mediated by NA (Porges, 1995). Given the strong theoretical relationships between lateralised autonomic and hemispheric function and between the neurons that control RSA and those controlling facial expression and vocalisation, research should be directed at evaluating the relationship between RSA and the primary emotions.

Based on the Polyvagal Theory, it would thus be expected that shifts in affective state would lead to parallel changes in RSA . Therefore, the elicitation of a negative primary emotion, such as fear, would result in a systematic withdrawal of vagal tone along NA to promote the fight-flight response. The theory would also acknowledge the importance of less conscious survival-oriented processes mediated by the dorsal vagal complex, which includes DMNX (Porges, 1995). In contrast, a shift to a more pleasant affective state would be associated with an increase in RSA.

Respiration rate (RR) has been demonstrated to have a strong negative relationship with RSA; as respiration rate increases, RSA magnitude decreases (Grossman, Karemaker, & Wieling, 1991). However, this resultant decrease does not necessarily reflect a decrease in the actual tonic level of cardiac vagal tone, but rather an inspiratory-expiratory

phasic change in vagal heart period. It is the tonic cardiac vagal tone that is generally of interest to psychophysiological researchers and it is thus recommended that respiratory parameters be controlled (Grossman et al., 1991). Wilson and Sharman (1995) demonstrated that slowing RR, by pacing respiration at slow frequencies, had the effect of maintaining high levels of cardiac vagal tone throughout a spider exposure task. Associated with this vagal tone maintenance was a reduction in HR minimum and HR maximum. In contrast, under naturalistic conditions, spider anxious individuals exhibited an increase in RR as the spider stimulus was moved closer, thus decreasing the utility of RSA as a tonic vagal tone indicator.

Increased RR has been found to be associated with intensified feelings of anxiety and fear (e.g., Clark & Hirschman, 1990; Ley, 1985; Svebak, Storfjell, & Dalen, 1982), while paced respiration has been demonstrated to lead to reductions in self-rated tension, state anxiety and skin conductance levels (Clark & Hirschman, 1990). Wilson and Sharman (1995), using spider anxious participants, demonstrated that highly anxious individuals are able to learn to pace their respiration at slow frequencies and that this reduction in RR is strongly associated with a decrease in the level of reported fear. On the basis of this finding, Wilson and Sharman (1995) postulated that paced respiration would substantially reduce the level of fear experienced by individuals undergoing therapy for spider phobia.

Given this background information, it was the aim of this experiment to investigate the physiological arousal response patterns of spider fearful individuals, during exposure to a spider stimulus, both before and after completion of a program of SD. It was hypothesised that, before treatment, if vagal withdrawal is involved a spider fearful individual, when confronted with a spider stimulus, would respond with a decrease in RSA and an increase in HR and skin conductance. This response pattern could be expected to be stronger in those individuals with a higher degree of spider anxiety than those with an average to high degree of spider fear. After successful completion of the treatment program, RSA should reflect any change due to the SD process; such that the participants could be expected to demonstrate an increase from pre-treatment levels of RSA accompanied with a decrease in the HR and skin conductance responses.

While several studies have investigated the physiological effects of SD, the question remains as to what extent these changes are sympathetic or parasympathetic in origin. The investigation of RSA as an indicator of treatment success may specify the extent of any vagal influences on this process. When combined with the Polyvagal Theory, this may permit the type and direction of any change(s) to be interpreted.

METHOD

Participants

Preselection procedures were conducted on the entire first year Psychology class at the University of Tasmania, using the Spider Phobia Questionnaire (SPQ) (Klorman, Weerts, Hastings, Melamed & Lang, 1974). Students who scored between 19 and 26 were classified as high spider fearfuls and those who scored between 12 and 14 were classified as low spider fearfuls. Female students who met these criteria were then approached and asked to participate. The study was restricted to females only due to the difference in fear reactions between males and females; females have been found to report more subjective unpleasantness and tension and to exhibit higher heart rates than males (Cornelius & Averill, 1983).

The participants were 38 volunteers, aged 17 to 38 years ($\bar{x}$ = 19.6 years, SD = 4.2), who agreed to participate for course credit. The high spider fearfuls (n = 20) had a mean SPQ score of 22.10 and the low spider fearfuls (n = 18) a mean of 13.00. All participants were in good physical health at the time of testing and were free of medications which might influence the investigation. Informed consent was obtained from each subject prior to the commencement of the first testing session (see Appendix A).

Apparatus

Fear apparatus . The behaviour avoidance test (BAT) consisted of a 3.5 m long track along which a huntsman spider (Helena Cancerides), mounted in a clear resin block and resting on a 45° angle in a raised sled, could advance. A pull cord was attached to the sled to enable the participant to advance or retreat the spider stimulus.

Physiological apparatus. A Macintosh IIci computer and MacLab 8 Physiological Data Acquisition System with the program Chart V3.4.1 was used to collect data. The MacLab was set up with four channels, which were all sampled at 100 Hz. Channel 1 displayed the electrocardiograph (ECG) signal, obtained via three miniature Gereonics Ag/AgCl electrodes placed on the participant's lower left and right rib cage, with an earth reference located on the left mastoid process. Electrodes were attached using standard laboratory procedures. Channel 2 displayed the cardiotachometer signal, which used the ECG signal from Channel 1 as raw data. A UFI Pneumotrace® respiration transducer was positioned around the upper chest of the participant at the level of the arm pits. The respiration transducer was connected to Channel 3 to display the signal. Skin conductance was measured using a Skin Conductance Bridge (Lykken & Venables, 1971) with a constant voltage of 0.5 V applied through 9 mm Ag/AgCl electrodes attached to the first and third fingers of the non-dominant hand. The Bridge output was applied to Channel 4 of the MacLab. The electrodes were filled with Electro-gel® (ECI International Inc.) electrolyte paste as the contact medium.

Respiration rate (RR) was controlled by a 4 channel interval generator. The interval generator provided two tones (1 KHz and 1.5 KHz) to signal the onset and duration of the inspiratory and expiratory periods. RR was set at 10 cycles per minute (cpm), with inspiratory and expiratory periods of 2.5 s and a 0.5 s pause between the two.

Psychological apparatus. Three visual analogue scales (VAS), 100 mm long, were presented to each participant (included in Appendix B). The left and right anchor points for the respective scales were: mental stress (calm - worried), physical arousal (active - sleepy) and subjective fear (no fear - extremely fearful). Participants indicated their position on each scale by drawing a line through the scale at the point that reflected their current level.

The Spider Phobia Questionnaire (SPQ), as developed by Klorman, Weerts, Hastings, Melamed and Lang (1974), was used as a measure of spider fear. It was utilised as both an initial screening device, to classify the high and low spider fearful participants, and as a measure of spider anxiety at each exposure session, to determine the effectiveness of the treatment programme. The Questionnaire of Mental Imagery (QMI), as

developed by Sheehan (1967), was used to determine each participant's ability to use mental imagery, as this is a possible confound when utilising systematic desensitisation. The State-Trait Anxiety Inventory (STAI) Form X, (Spielberger, Gorsuch, Lushene, Vagg and Jacobs, 1977), was used to assess pre-spider exposure anxiety levels for each participant at each session. A health and medical history checklist was also administered (see Appendix C) to screen participants for any current medications or existing medical conditions which might confound the physiological recordings.

Design

High and low spider fearfuls were randomly allocated to either an experimental or wait-list control group, providing four groups, with approximately ten participants in each. Wait-list control participants were required to attend a second spider exposure session, prior to undergoing the systematic desensitisation process, after an interval corresponding to the length of the treatment program (approximately four weeks). This controlled for the possible influence of test-retest effects, passage of time and seasonal changes and regression towards the mean.

A 2 x 2 x 6 mixed between and within groups design was employed. The between groups factor was spider anxiety, with two levels (high and low spider fear). The within groups factors were session (two levels: 1 and 2) and distance or proximity to the spider stimulus (six levels: baseline, 310 cm, 250 cm, 150 cm, 75 cm and 25 cm).

The dependent variables were RSA amplitude, skin conductance level (SCL), mean HR, VAS scores for stress, arousal and fear, SPQ scores and STAI scores. Two secondary dependent variables, maximum and minimum HR, were measured to assist in the interpretation of any RSA effects. The independent variables were spider exposure proximity and systematic desensitisation (SD) treatment.

Procedure

Physiological assessment . Participants were tested individually for each of the spider exposure sessions. All sessions complied with the following

format and were conducted in a sound attenuated room, light and temperature controlled (22-24°C). The experimenter and MacLab were in an adjacent room and communication was maintained with the participant via an intercom system.

Upon arrival in the laboratory, the experimental procedure was fully explained to the participant and informed consent was obtained. The electrodes for ECG / cardiotachometer and SCL were then applied and the respiration strain gauge was fitted, followed by a 15-minute adaptation period to allow adjustment to the laboratory environment. During this time, at each session, the participant was required to complete a STAI and SPQ. At the first spider exposure session, the participant was also required to complete a checklist concerning health and fitness and a QMI.

Once the questionnaires had been completed instructions were given for rate of breathing (10 cpm) and how to operate the BAT.

The interval generator was then turned on and the experimenter demonstrated paced breathing. The participants were left alone in the experimental room for five to ten minutes to practise paced breathing. Once paced breathing was established, with inspiration and expiration smooth and symmetrical and RR consistent at 10 cpm, baseline physiological responses were recorded for 60 seconds, without the spider stimulus in the room.

The experimenter then placed the spider stimulus in the sled at the furthest distance from the participant (310 cm). The participant was instructed to remain focused on the spider and concentrate on 'spider related' thoughts whilst continuing to breathe at 10 cpm. All physiological responses were recorded for 60 seconds. The participant then completed a VAS.

The experimenter asked the participant to move the spider sled up to the first marker (250 cm) and all physiological responses were recorded for a further 60 seconds, after which the participant again completed a VAS. The procedure was then repeated for the remaining three distances (150 cm, 75 cm and 25 cm). This procedure was followed for all subjects at the subsequent spider exposure session(s).

Treatment sessions. The treatment program was a group administered SD procedure conducted by the experimenter. Each of the four groups was randomly divided in half, leading to eight treatment groups of approximately five participants each. This was done primarily to avoid the difficulties in coordinating larger numbers of students with conflicting timetables. An anxiety hierarchy was developed for each treatment group, based both on common elements from the individual responses on the SPQ and other 'spider scenarios' developed during group discussion. The groups advanced through these hierarchies at the speed of the slowest member of each group.

In total, there were eight sessions of SD, two per week, of approximately 40 minutes duration each. In addition, the participants were required to practise the deep muscle relaxation task between each session, using a pre-recorded audio cassette tape. The first two sessions were devoted entirely to learning the deep muscle relaxation technique. Desensitisation with the anxiety hierarchy was used from the third to eighth sessions, with the first 10 minutes of these subsequent sessions spent attaining a deep level of relaxation, then moving on to the presentation of hierarchy scenes. It was stressed to the participants that if any scene proved upsetting or disturbing they were to indicate this by raising their right hand. When any participant signalled in this manner, the scene was withdrawn immediately and the group returned to their 'relaxing image'.

Data Reduction

Psychological measures. Stress, arousal and fear scores were measured from the VAS and expressed as percentages of full-scale score. High scores indicated high levels of stress, arousal and fear respectively. The SPQ, STAI and QMI were scored using standard scoring methods.

Physiological measures. For each measurement period a segment of physiological record showing HR and SCL recordings for 5 respiratory cycles was selected. Mean HR, average maximum HR, average minimum HR and mean SCL were calculated from the same period of respiratory cycles. RSA was the difference between average maximum HR and average minimum HR. RSA and HR were yielded in beats per minute (bpm) and SCL was yielded in micro Siemens (μS).

Data Analysis

For the purposes of these analyses, Vasey and Thayer's (1987) recommendation to use the Geisser-Greenhouse correction for repeated measures analysis of variance (ANOVA) was adopted. ANOVAs were conducted using the SPSS for Windows (1995) statistical package on a Macintosh IIci computer.

Initial ANOVAs were performed using six levels for the distance factor (2 x 2 x 6). However, using all six levels did not provide any further information than using only three (baseline, 310 cm [exposure 1/E1] and 25 cm [exposure 5/E5]). Thus, in all further analyses only three levels were used and this also reduced the repeated measures degrees of freedom.

Individual ANOVAs (2 x 2 x 3) were thus performed to calculate differences for RSA, mean HR, mean SCL, average maximum HR, average minimum HR, mental stress, physical arousal and fear for the two levels of spider fear (high and low) over the two sessions (pre- and post-treatment) at each of the three spider exposures (baseline, E1 and E5). The α level for all comparisons was set at $p = 0.05$. Statistical probability was reported as $< .05$, $< .01$ and $< .001$ (except where exact probabilities were precisely at these levels). Post hoc t-tests were conducted as required to account for significant differences in main effects or interactions. Bonferroni adjustment for the number of orthogonal comparisons were used to correct for Type 1 error. The maximally conservative Bonferroni adjustment was applied to all significant comparisons. Where the exact probability level did not reach the level of the Bonferroni alpha, it was interpreted as indicating that the comparison may not be valid and required verification through replication.

RESULTS

Preliminary Analyses

Pre-treatment differences

Independent group t-tests were performed between high and low spider fearfuls on age, QMI score and pre-treatment SPQ score to determine

whether any significant group differences existed on these variables. There were no differences for age ($t(38) = 0.58$, n.s.) or QMI score ($t(38) = 2.19$, n.s.). There was a significant difference for pre-treatment SPQ score ($t(38) = 18.40$, $p < .001$); with high spider fearfuls ($\bar{x} = 22.10$, $SD = 2.34$) recording significantly higher scores than low spider fearfuls ($\bar{x} = 13.00$, $SD = .77$). The mean pre-treatment score for the high spider fearful group is comparable to the pre-treatment mean score ($\bar{x} = 22.6$, $SD = 2.3$) reported by Fredrikson, Wik, Annas, Ericson and Stone-Elander (1995) for their phobic sample. These analyses indicate that the high and low spider fearful groups were equivalent on the control variables, while being highly different on the pre-existing independent variable of spider fearfulness.

Wait-list controls

Analyses of the two pre-treatment measures of the wait-list control group were also performed, using ANOVA. This was done to determine whether any significant differences existed between the two pre-treatment measures on the physiological variables. Means and standard deviations are shown in Table 1. Where significant differences were found, post hoc t-tests were performed to determine the location of the difference and the maximally conservative Bonferroni adjustment was then applied.

RSA. No significant differences were found for RSA.

Mean HR. The repeated measures ANOVA of pre-treatment baselines for mean HR revealed a significant exposure effect ($F(2, 36) = 4.89$, $p < .05$). Post hoc t-test comparisons showed that mean HR was significantly higher at E1 than baseline ($t(20) = -2.76$, $p < .05$) and at E5 than baseline ($t(20) = -2.61$, $p < .05$). When the Bonferroni α of .0167 (.05/3) was applied, the difference between mean HR at E5 and baseline was not significant, thus suggesting that this effect may merit replication. ANOVA revealed a significant subtype x exposure interaction ($F(2, 36) = 3.25$, $p < .05$). Post hoc t-test comparisons revealed that mean HR was significantly higher at E1 than baseline ($t(11) = -3.18$, $p = .01$) and at E5 than baseline ($t(11) = -3.33$, $p < .01$) for high spider fearfuls only. Both these differences failed to reach significance at the Bonferroni α of .0056 (.05/9) and may need to be replicated. ANOVA also revealed a

significant exposure x session interaction ($F(2, 36) = 4.34$, $p < .05$). Post hoc t-test comparisons showed a significant decrease in mean HR at E1 between the first and second pre-treatment baseline sessions ($t(20) = 2.19$, $p < .05$), a significant increase between baseline and E1 at the first pre-treatment session ($t(20) = -2.09$, $p=.05$), and at the second pre-treatment session ($t(20) = -2.97$, $p < .01$) and a significant increase in mean HR between baseline and E5 at the second pre-treatment session ($t(20) = -4.00$, $p =.001$). When the Bonferroni α of .0056 (.05/9) was applied, the only effect which remained robust was the difference between baseline and E5 at the second pre-treatment session, suggesting that the other effects may all merit replication.

Mean SCL. ANOVA revealed a significant exposure effect ($F(2, 36) = 6.46$, $p < .01$). Post hoc t-test comparisons showed a significant increase in mean SCL between baseline and E1 ($t(20) = -3.96$, $p = .001$) and between baseline and E5 ($t(20) = -2.96$, $p < .01$). When the Bonferroni α of .0167 (.05/3) was applied, both of these effects remained robust.

There were no significant main effects for session, or subtype x session interactions, for any measures and no indication of any general changes in arousal between high and low spider fearfuls as a function of the second pre-treatment session. The only significant difference that was indicated was that for mean HR at E1. This difference, however, did not survive the Bonferroni adjustment and thus warrants replication. Based on these findings, a decision was made to use the first exposure session as the baseline measure in all subsequent analyses. The experimental and wait-list control groups were thus collapsed to form single groups of high and low spider fearfuls.

Table 1: *Physiological measure means (and standard deviations) for high and low spider fearfuls for the three exposures across the two wait-list control pre-treatment sessions*

Measure	Exposure	Session 1	Session 2
High spider fearfuls			
RSA	Baseline	15.48 (5.81)	19.30 (6.54)
	E1	14.59 (5.10)	16.98 (6.42)
	E5	15.30 (5.47)	17.72 (5.67)
Mean HR	Baseline	75.54 (12.63)	74.02 (8.22)
	E1	81.07 (14.00)	78.00 (7.14)
	E5	80.38 (12.52)	79.85 (9.85)
Mean SCL	Baseline	8.78 (5.16)	7.12 (2.97)
	E1	11.34 (4.86)	8.64 (3.08)
	E5	11.42 (5.76)	9.16 (4.04)
Low spider fearfuls			
RSA	Baseline	18.72 (8.86)	19.61 (7.76)
	E1	16.39 (7.12)	17.49 (5.94)
	E5	18.47 (9.31)	18.34 (8.92)
Mean HR	Baseline	78.27 (11.16)	77.63 (8.31)
	E1	80.75 (11.05)	78.68 (8.53)
	E5	78.19 (9.52)	78.87 (7.55)
Mean SCL	Baseline	6.26 (4.50)	6.72 (2.70)
	E1	7.83 (4.56)	7.17 (3.29)
	E5	7.16 (4.16)	7.95 (3.64)

Experimental Analyses

Physiological measures

RSA . Means and standard deviations are shown in Table 2. ANOVA revealed a main effect for exposure ($F(2, 72) = 4.30$, $p < .05$) Post hoc t-test comparisons showed that RSA was significantly lower at E1 than baseline ($t(38) = -2.88$, $p < .01$) and at E1 than E5 ($t(38) = -2.25$, $p < .05$). When the Bonferroni α of .0167 (.05/3) was applied, the RSA difference between E1 and E5 was not significant. This suggests that this effect may need to be replicated. ANOVA also revealed a non-significant trend toward a subtype main effect ($F(1, 36) = 3.17$, $p = .083$), which indicated that high spider fearfuls tended to exhibit lower RSA than low spider fearfuls, as illustrated in Figure 1.

Table 2: *RSA means (and standard deviations) for high and low spider fearfuls for the three exposures across session*

	Session 1	Session 2
High spider fearfuls		
Baseline	15.48 (5.81)	14.68 (6.39)
E1	14.59 (5.10)	14.36 (5.94)
E5	15.30 (5.47)	15.07 (7.09)
Low spider fearfuls		
Baseline	18.72 (8.86)	19.26 (6.70)
E1	16.39 (7.12)	17.56 (5.47)
E5	18.47 (9.31)	19.27 (6.88)

ANOVA of the minimum and maximum HR data was performed to assist in the interpretation of the RSA exposure effect. This revealed a main effect for exposure for minimum HR ($F(2, 72) = 5.48$, $p < .01$). Post hoc t-test comparisons revealed that minimum HR was significantly

higher at E1 than baseline (t (38) = 3.74, p = .001) and at E1 than E5 (t (38) = 2.06, p < .05). The latter effect failed to reach significance after the Bonferroni α of .0167 (.05/3) was applied. This indicates that the minimum HR difference between E1 and E5 may merit replication.

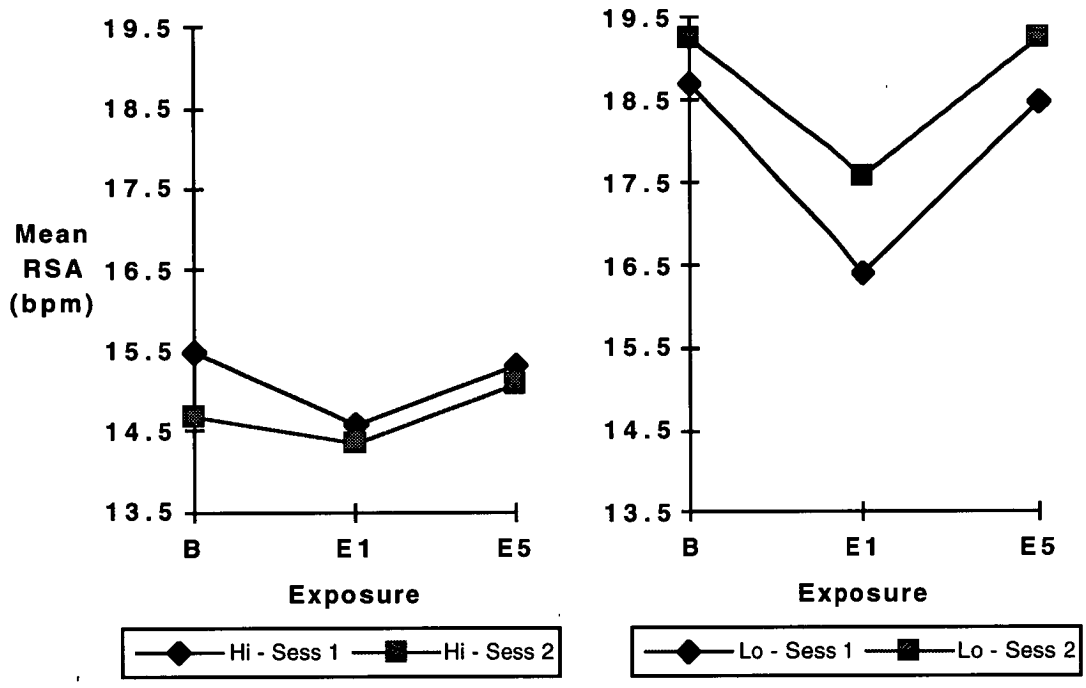

Figure 1: *Mean RSA means for high and low spider fearfuls for the three exposures across session*

Mean HR. Means and standard deviations are shown in Table 3. ANOVA revealed a main effect for exposure (F (2, 72) = 4.12, p < .05). Post hoc t-test comparisons showed that mean HR was significantly higher at E1 than baseline (t (38) = 3.22, p < .01) and at E5 than baseline (t (38) = 2.09, p < .05). When the Bonferroni α of .0167 (.05/3) was applied the latter effect did not reach significance, suggesting this effect may require replication. ANOVA also revealed a main effect for session (F (1, 36) = 4.23, P < .05). This effect is illustrated in Figure 2 and clearly shows the location of the session effect to lie in the decrease in mean HR at the second session for both groups.

ANOVA showed a significant subtype x exposure interaction (F (2, 72) = 3.34, p < .05). Post hoc t-test comparisons revealed a significant increase in mean HR between baseline and E1 (t (20) = 3.92, p = .001) and between

baseline and E5 ($t(20) = 3.17$, $p < .01$) for high spider fearfuls only. Both of these effects remained robust when the Bonferroni α of .0056 (.05/9) was applied.

Table 3: *Mean HR means (and standard deviations) for high and low spider fearfuls for the three exposures across session*

	Session 1	Session 2
High spider fearfuls		
Baseline	75.54 (12.63)	75.88 (9.22)
E1	81.07 (14.00)	76.79 (9.05)
E5	80.38 (12.52)	77.47 (8.95)
Low spider fearfuls		
Baseline	78.27 (11.16)	75.62 (7.39)
E1	80.75 (11.05)	74.69 (5.78)
E5	78.19 (9.52)	74.89 (6.66)

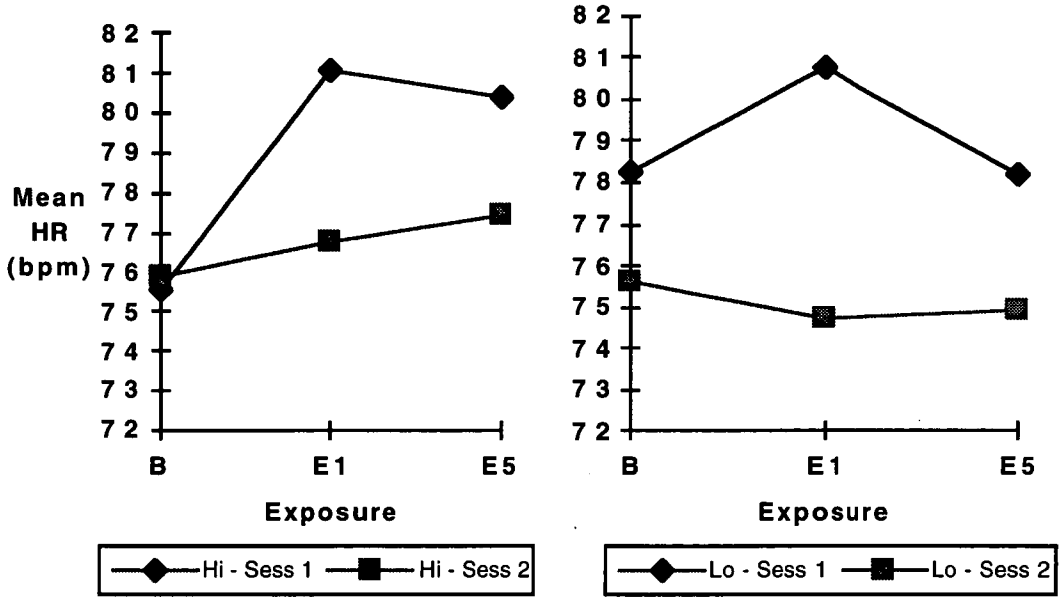

Figure 2: *Mean HR means for high and low spider fearfuls for the three exposures across session*

ANOVA also revealed a significant exposure x session interaction ($F(2, 72) = 7.64$, $p < .001$). Post hoc t-test comparisons showed a significant decrease at E1 between session 1 and 2 ($t(38) = 3.02$, $p < .01$), a significant increase in mean HR between baseline and E1 ($t(38) = -3.84$, $p < .001$) and between baseline and E5 ($t(38) = -2.81$, $p < .01$) at session 1. When the Bonferroni α of .0056 (.05/9) was applied, all effects except the latter remained robust. This indicates that the mean HR change from baseline to E5 at session 1 may merit replication.

Mean SCL. Means and standard deviations for SCL are shown in Table 4. ANOVA revealed a main effect for subtype ($F(1, 36) = 6.37$, $p < .05$). Figure 3 clearly shows that the subtype effect is based on the high spider fearfuls recording higher SCL responses than the low spider fearfuls. ANOVA also revealed a main effect for exposure ($F(2, 72) = 10.09$, $p < .001$). Post hoc t-test comparisons showed a significant increase in SCL between baseline and E1 ($t(38) = 4.13$, $p < 001$) and between baseline and E5 ($t(38) = 3.63$, $p = .001$). Both of these effects were robust when the Bonferroni α of .0167 (.05/3) was applied. ANOVA also revealed a main effect for session ($F(1, 36) = 20.58$, $p < .001$). Figure 3 clearly shows that the session effect is based on the decrease in SCL between session 1 and 2 for both high and low spider fearfuls.

ANOVA showed a significant exposure x session interaction ($F(2, 72) = 4.84$, $p < .05$). Post hoc t-test comparisons revealed a number of significant differences: a significant difference at E1 between session 1 and 2 ($t(38) = 5.10$, $p < .001$), at E5 between session 1 and 2 ($t(38) = 4.12$, $p < .001$) and an increase in SCL between baseline and E1 at session 1 ($t(38) = -4.41$, $p < .001$) . All of these effects remained robust when the Bonferroni α of .0056 (.05/9) was applied. However, a number of other effects failed to reach significance after the Bonferroni adjustment: the difference at baseline between session 1 and 2 ($t(38) = 2.85$, $p < .01$), an increase in SCL between baseline and E5 at session 1 ($t(38) = -2.78$, $p < .01$), between E1 and E5 at session 2 ($t(38) = -2.58$, $p < .05$) and between baseline and E5 at session 2 ($t(38) = -2.91$, $p < .01$). As these latter measures failed to survive the maximally conservative correction they may merit replication.

Table 4: *Mean SCL means (and standard deviations) for high and low spider fearfuls for the three exposures across session*

	Session 1	Session 2
High spider fearfuls		
Baseline	8.78 (5.16)	5.63 (2.00)
E1	11.34 (4.86)	5.61 (2.16)
E5	11.42 (5.76)	6.83 (2.91)
Low spider fearfuls		
Baseline	6.26 (4.50)	4.95 (2.03)
E1	7.83 (4.56)	5.16 (1.84)
E5	7.16 (4.16)	5.19 (1.81)

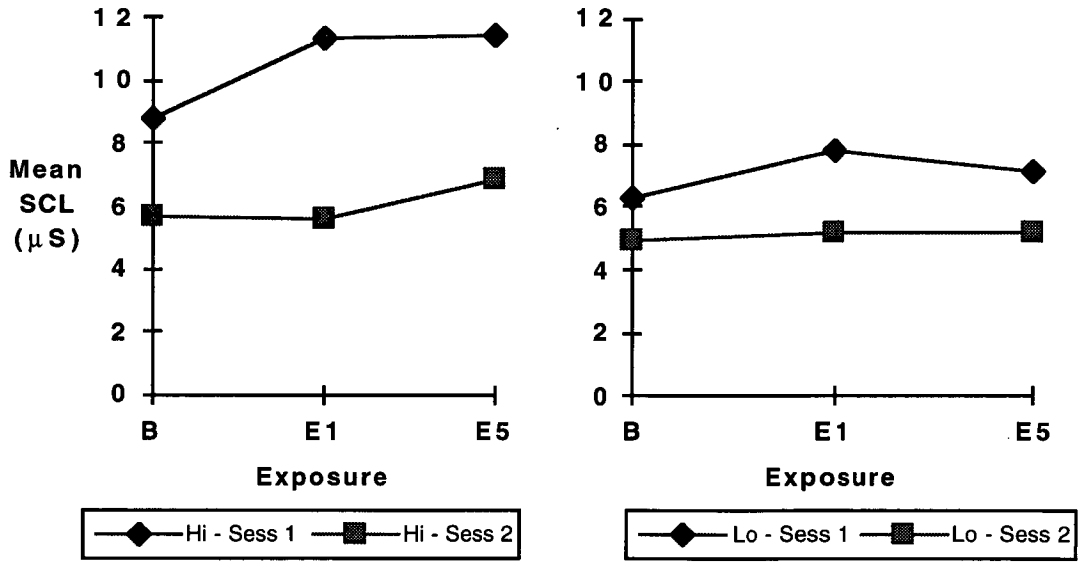

Figure 3: *Mean SCL means for high and low spider fearfuls for the three exposures across session*

Self-report measures

The results for the four specific measures of spider anxiety are presented in Table 5.

Table 5: *Self-report spider anxiety measure means (and standard deviations) for high and low spider fearfuls for the three exposures across session*

Measure	Exposure	Session 1	Session 2
High spider fearfuls			
SPQ		22.10 (2.34)	16.75 (5.29)
Stress	E1	31.05 (17.70)	15.10 (8.81)
	E5	59.65 (17.78)	32.05 (20.53)
Arousal	E1	51.70 (19.28)	41.30 (23.17)
	E5	65.65 (23.33)	59.20 (25.13)
Fear	E1	30.00 (16.01)	17.25 (11.26)
	E5	60.05 (17.57)	31.40 (23.30)
Low spider fearfuls			
SPQ		13.00 (.77)	10.78 (3.75)
Stress	E1	18.06 (16.91)	11.11 (8.46)
	E5	47.78 (26.33)	20.61 (13.57)
Arousal	E1	42.28 (30.61)	34.89 (25.04)
	E5	51.28 (32.78)	38.67 (19.42)
Fear	E1	17.11 (18.66)	10.17 (8.90)
	E5	46.17 (25.68)	23.50 (15.89)

Stress. ANOVA revealed a significant main effect for subtype ($F(1, 36) = 6.37$, $p < .05$). Figure 4 clearly demonstrates that the subtype effect is due to the high spider fearfuls reporting higher stress levels than the low

spider fearfuls. ANOVA also revealed a significant main effect for exposure ($F(1, 36) = 95.91$, $p < .001$). Figure 4 clearly illustrates that this effect is due to the increase in reported stress, for both high and low spider fearfuls, between E1 and E5. ANOVA also revealed a significant main effect for session ($F(1, 36) = 44.12$, $p < .001$), which Figure 4 shows to be due to a significant decrease in stress between session 1 and 2.

ANOVA revealed a significant exposure x session interaction ($F(1, 36) = 32.56$, $p < .001$). Post hoc t-test comparisons showed a number of significant differences: a significant difference at E1 between session 1 and 2 ($t(38) = 4.06$, $p < .001$) and at E5 between session 1 and 2 ($t(38) = 7.71$, $p < .001$), a significant difference between stress ratings at E1 and E5 at session 1 ($t(38) = -10.76$, $p < .001$) and at E1 and E5 at session 2 ($t(38) = -5.51$, $p < .001$). When the Bonferroni α of .0125 (.05/4) was applied all of these effects remained robust.

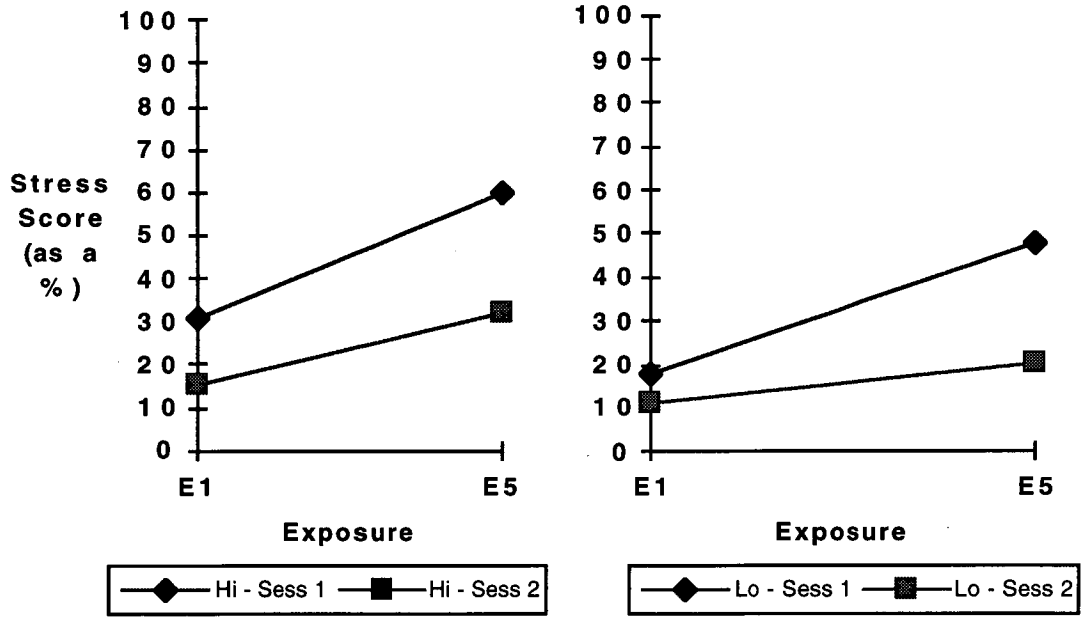

Figure 4: *Mean stress scores (expressed as a percentage) for high and low spider fearfuls at the two exposures across session*

Arousal. ANOVA revealed a significant main effect for subtype ($F(1, 36) = 5.38$, $p < .05$). Figure 5 clearly shows that this is due to the significantly higher arousal ratings of the high spider fearfuls. ANOVA also revealed

a significant main effect for exposure (F (1, 36) = 12.55, p < .001). Figure 5 demonstrates that this effect is due to the increase in reported arousal for both high and low spider fearfuls between E1 and E5.

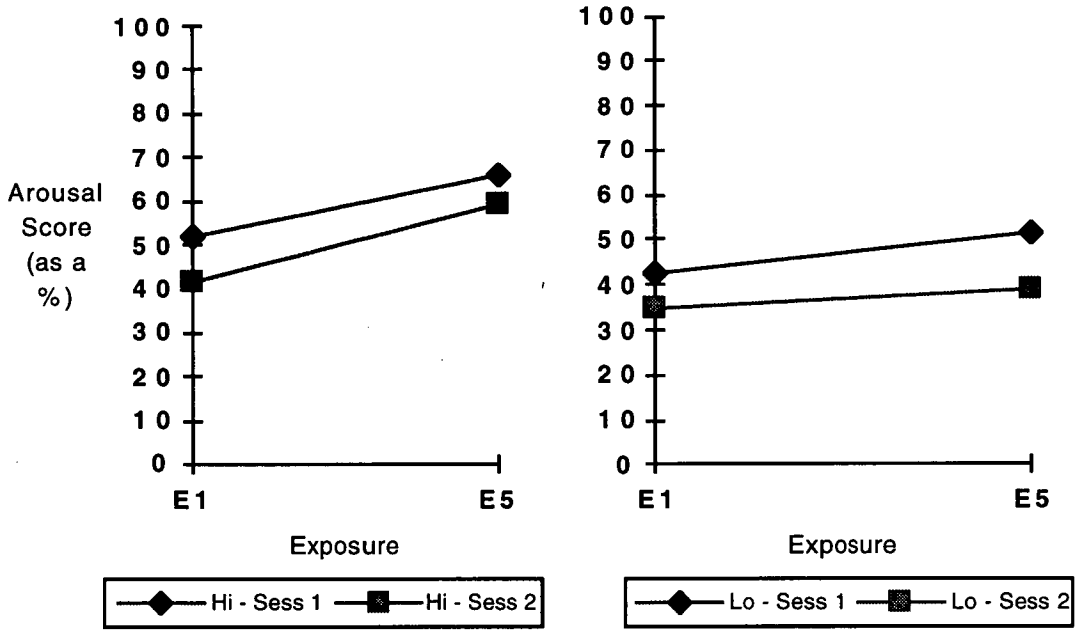

Figure 5: *Mean arousal scores (expressed as a percentage) for high and low spider fearfuls at the two exposures across session*

Fear. ANOVA revealed a significant main effect for subtype (F (1, 36) = 5.57, p < .05). Examination of Figure 6 shows that this effect is due to the higher fear ratings of the high spider fearfuls. ANOVA also revealed a significant main effect for exposure (F (1, 36) = 123.41, p < .001). Figure 6 clearly demonstrates that the exposure effect is due to the increase in reported fear between E1 and E5 for both high and low spider fearfuls. ANOVA also revealed a significant main effect for session (F (1, 36) = 38.23, p < .001). Figure 6 illustrates that this effect is due to the decrease in fear levels between session 1 and 2 for both high and low spider fearfuls.

ANOVA revealed a significant exposure x session interaction (F (1, 36) = 27.18, p < .001). Post hoc t-test comparisons showed a significant difference at E1 between session 1 and 2 (t (38) = 3.50, p = .001) and at E5 between session 1 and 2 (t (38) = 7.23, p < .001) and a significant increase

in reported fear between E1 and E5 at session 1 ($t(38) = -11.68$, $p < .001$) and between E1 and E5 at session 2 ($t(38) = -5.91$, $p < .001$). All of these effects remained robust when the conservative Bonferroni α of .0125 (.05/4) was applied.

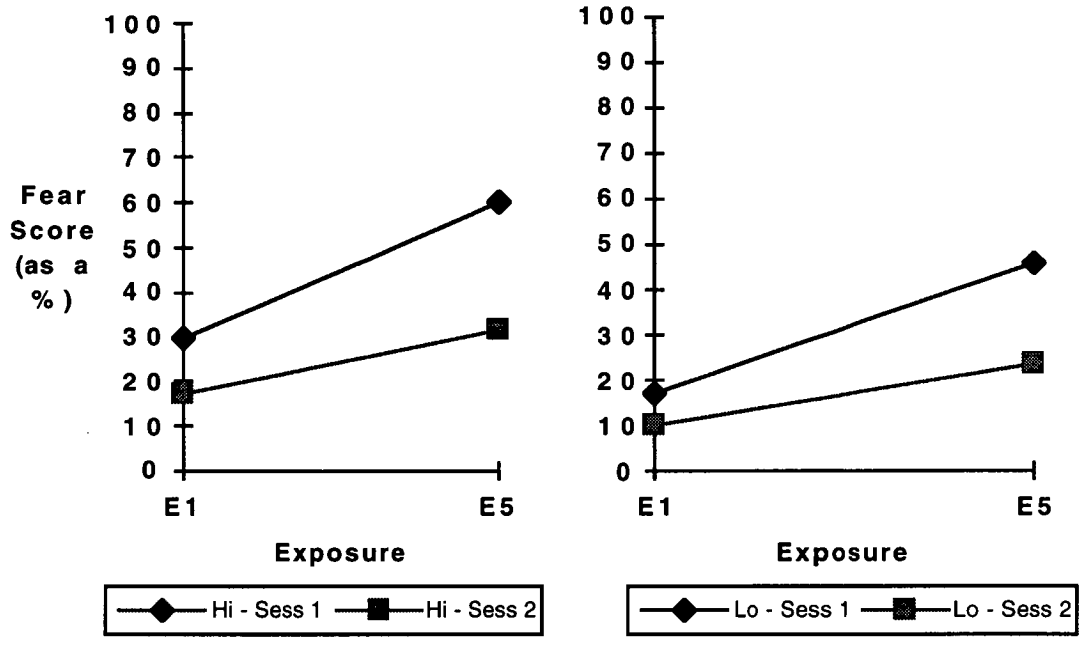

Figure 6: *Mean fear scores (expressed as a percentage) for high and low spider fearfuls at the two exposures across session*

DISCUSSION

The present experiment was designed to determine the physiological arousal response patterns of high and low spider fearful individuals, when confronted with a spider stimulus, both before and after a treatment program. An additional objective was to specify the extent of any vagal influences on the physiological effects of a SD program.

It was hypothesised that, if vagal withdrawal is involved, spider fearful individuals, particularly those who scored higher on the SPQ, would initially respond to the presentation of a spider stimulus with a decrease in RSA and an increase in HR and SCL. It was further hypothesised that,

after completing a treatment program, these individuals would respond to the presentation of the same spider stimulus with an increase in the pre-treatment levels of RSA and a decrease in HR and SCL responses.

Analysis of the results of this experiment revealed that participants did indeed exhibit a significant pre-treatment decrease in RSA from the baseline levels upon initial presentation of the spider stimulus, thus supporting the initial hypotheses and implicating a vagal involvement in the fear reaction. However, as the spider stimulus was moved closer toward the participant the RSA levels gradually began to increase back toward the baseline level. This gradual increase in RSA as the spider stimulus became closer may be explained in one of two ways. It is possible that there may have been an orienting / defensive reaction to the initial appearance of the spider stimulus (Sokolov, 1963), which was not elicited by later exposures. Alternatively, the difference reflected by higher RSA scores at later exposures, when compared with the higher VAS scores at the same exposures for stress, arousal and fear, may simply provide further support for the phenomenon of desynchrony within the 'Three-Systems-Model' of phobic fear (Hugdahl, 1981). This desynchrony could be due to experimental demand or subject expectancy of reduced fear or to a temporal lag between indications of improvement in the physiological and cognitive measures of anxiety. A long term follow-up, or re-assessment, could provide clarification of the processes involved in this desynchrony. The high spider fearful participants also demonstrated a tendency to exhibit generally lower RSA levels than their low spider fearful counterparts; also in support of the initial hypotheses. After completion of the treatment program, there was a non-significant trend toward an increase from the pre-treatment RSA levels for both high and low spider fearfuls; thus suggesting that RSA does index a reduction, or improvement, in the fear reaction.

Analyses also revealed a significant pre-treatment increase in average HR from baseline levels upon initial presentation of the spider stimulus, in support of the hypotheses. As the stimulus was moved closer the average HR levels decreased slightly, but still demonstrated a strong tendency to be higher than baseline levels. This suggests that the participants retained a fairly high degree of physiological arousal over the successive exposures. This initial pre-treatment increase in average HR scores upon introduction of the spider stimulus was significantly greater

in the high spider fearfuls than their low spider fearful counterparts, which also supported the initial hypotheses. After completion of the treatment program, there was a significant decrease in average HR for both high and low spider fearfuls, as predicted by the initial hypotheses. This decrease was greatest at the point of initial presentation of the spider stimulus, which suggests that the average HR was most sensitive to the initial decrease in fearful response.

In relation to SCL, analyses revealed a significant difference between the scores of the high and low spider fearfuls; with high spider fearfuls recording significantly higher SCL responses in support of the initial hypotheses. There was a significant increase in pre-treatment SCL between baseline and the first spider exposure, also predicted by the initial hypotheses. The SCL responses tended to increase slightly as the spider stimulus was moved closer for both high and low spider fearfuls; such that there was a significant increase between baseline levels and those at E5 but there was not a significant difference between E1 and E5. This suggests that the skin conductance response may have been slightly more sensitive than HR to the fearful response to the proximity of the spider stimulus, as postulated by Lacey (1967; in Mathews, 1971). After completion of the treatment program there was a significant decrease in SCL for both high and low spider fearfuls, supporting the hypotheses. This difference was greatest at both E1 and E5, as opposed to baseline levels which only demonstrated a trend to be higher in high spider fearfuls. This suggests that the SCL responses at the two spider exposure levels were particularly sensitive to treatment success, while the baseline responses did not differ quite as much. Therefore, anticipatory anxiety had not decreased as much as that evoked when the participants were actually confronted with the spider stimulus. There was also a significant increase in SCL between baseline and E5, which suggests that, even after treatment, the spider stimulus was still eliciting some degree of physiological arousal.

Analyses of the self-report measures (stress, arousal and fear) revealed a significant difference between high and low spider fearful participants on all three. The high spider fearfuls reported higher levels for all three measures, as could be expected. Significant increases were also revealed for all three measures between the initial spider presentation and the closest. This supports the physiological data, the participants became

more anxious as the spider stimulus was moved closer. After completion of the treatment program, both the stress and fear ratings demonstrated a significant decrease. The decrease in the stress ratings may have been due to some participants applying the relaxation techniques they had learnt to the anxiety producing situation. The decrease in fear ratings may have been due to perceived success of the treatment program. The fact that the difference between psychological arousal pre- and post-treatment showed only a tendency to decrease, may have been due to anticipatory anxiety at re-exposure to the spider stimulus and self-doubt as to their ability to cope.

The second major objective was to specify the extent of any vagal influences on the physiological effects of a treatment program. Based on the premises of the Polyvagal Theory (Porges, 1995), it appears likely that the vagal influences on the physiological effects of a SD program are mainly mediated by the nucleus ambiguus (NA). However, as the changes in both HR and SCL following completion of the treatment program showed greater significance than those for RSA, it is likely that sympathetic activation is also involved in the fear reaction response. A more precise index of sympathetic nervous system (SNS) arousal, such as plasma catecholamine levels, would allow the extent of any SNS activity to be more accurately specified, as opposed to activity within the dorsal motor nucleus of the vagus (DMNX).

When interpreting the greater change in HR and SCL, as opposed to RSA, it is important to note that the data were obtained under respiratory pacing control. Thus, the RSA increase discerned following treatment was over and above any 'normal' RR changes, as usually seen in the phobic / fearful reaction. If respiratory pacing was not applied, it could be expected that participants would have demonstrated an increase in RR as they became more anxious, or as the spider stimulus was moved closer (based on the findings of Wilson & Sharman, 1995). Under this condition, any associated RSA decreases upon presentation of the spider stimulus would have been expected to be even larger (Grossman et al., 1991). As respiratory control was applied in the present study, with the associated reduction in RR-related RSA decreases, and RSA changes were still observed, it can be concluded that RSA does represent quite a strong indicator of treatment success. Thus, there is support for the hypothesis

that there is vagal involvement in the physiological effects of a treatment program.

As the RSA responses were not as large as those reported by Wilson and Sharman (1995) without respiratory pacing, these results also support the use of respiratory training as part of a therapy program for phobic individuals (Barlow, 1988). This raises the interesting question that respiration control may be utilised by some individuals as a coping mechanism (i.e., some individuals may make a conscious effort to breathe more slowly and deeply when under environmental stress in an attempt to reduce their fear reaction). This may warrant further investigation.

Thus, these results support the findings of such earlier studies as Craske and Sipsas (1992), Hare and Blevings (1975), Merckelbach et al. (1991), and Prigatano and Johnson (1974) who all reported an increase in HR and SCL in spider phobics upon presentation of spider-related stimuli.

Lacey (1967; in Mathews, 1971) hypothesised that HR may be a more sensitive indicator of responses to phobic images, or internally presented stimuli, while SCL may be more sensitive to externally presented phobic stimuli. This experiment revealed a slightly more constant increase in SCL over the spider exposures than HR; which suggests that SCL may indeed be more sensitive to an externally presented spider stimulus.

The results of this investigation also support the findings of Levin et al. (1982), who reported a decline from pre-treatment levels in cardiac and skin conductance responses of anxious clients, when exposed to their feared stimulus, following treatment.

Porges' (1995) Polyvagal Theory was constituted of a number of different premises, one of which stated that emotion, such as fear, should produce a change in RSA as mediated by NA (Porges, 1995). This investigation did indeed find a fear-modulated RSA effect, in that RSA showed a significant decrease from baseline levels when the participants were initially confronted with a spider stimulus. The theory goes on to postulate that a shift to a more pleasant affective state, or a decrease in the negative emotion, would be associated with an increase in RSA. These results did demonstrate a tendency for RSA responses to increase

following the completion of the treatment program, thus supporting this hypothesis.

This was a clinical analogue study and the participants did not meet the criteria for a diagnosis of Specific Phobia (American Psychiatric Association, 1994). The utilisation of a student sample also created other difficulties, such as incomplete attendance for the total number of treatment sessions and poor motivation to practise the relaxation skill. These variables may well have affected the success of the SD program, as those participants who showed greater motivation to complete the demands of the study (as indicated by records kept for each participant in relation to completion of homework and session attendance) tended to exhibit greater improvement in their physiological and self-report measures following the treatment program than those who were less committed. This possible reduction in the overall success of the treatment program may have impacted on the changes in the physiological responses.

The results of this study also have implications for the manner in which phobic individuals are currently treated. If beta-adrenergic mechanisms are not as strongly involved with the tachycardia associated with anxiety as previously thought, as suggested by these results which tend to implicate a strong role for vagal withdrawal, it may not be appropriate to focus on these symptoms. It may be more appropriate to address the vagal withdrawal.

In summary, this experiment has revealed three main types of change - change related to the degree of spider anxiety, change related to the proximity of the spider stimulus, and change related to the completion of a treatment program.

All measures, both physiological and subjective, differentiated between the high and low spider fearful participants. High spider fearful individuals generally demonstrated higher levels of physiological arousal when confronted with the spider stimulus and this was accompanied by higher ratings of subjective distress.

Differences could also be differentiated due to the proximity of the spider stimulus. Both high and low spider fearfuls tended to respond more

strongly, at least within the physiological realm, when the spider was first introduced, as compared to the closer exposure distance. Participants demonstrated a significant increase in HR and skin conductance and a significant decrease in RSA levels. The HR and skin conductance levels then tended to reduce slightly as the spider was moved closer, while the RSA levels became higher. Subjective ratings of distress continued to increase as the spider was moved closer. Two alternative explanations of this desynchrony have been suggested.

Thirdly, changes were evident due to the completion of the treatment program. All measures, both physiological and subjective, reflected improvement when compared with pre-treatment levels after completion of the SD program.

This experiment also provided strong support for Porges' (1995) postulation that HR and RSA need not respond in concert to an environmental stressor. In this case, HR responded strongly, with a constant increase in response to proximity of the spider stimulus and a comparative decrease after administration of a treatment program, while RSA level initially responded with a decrease upon presentation of the spider stimulus and then gradually increased following treatment.

Perhaps the most important finding is that while most previous investigations have assumed a simple sympathetic nervous system involvement in the fear reaction, this study has significantly implicated vagal withdrawal.

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APPENDIX A

STATEMENT OF INFORMED CONSENT

STATEMENT OF INFORMED CONSENT

The experiment you have shown interest in being involved in, is investigating the use of physiological measures (heart rate, breathing and skin responses) to determine the level of success of a treatment program for spider anxiety. This experiment will advance our knowledge of what happens in the body of an individual with a fear of spiders, both before and after a treatment program, and should prove helpful in determining the best types of therapy for this kind of fear.

You will be asked to sit at one end of a table. Electrodes will be affixed to record the reactivity of your heart and skin and a belt will be placed around your chest to measure your breathing pattern. A dead spider, enclosed in a clear perspex box, will then be placed at the other end of a 3 metre runway. You will be asked to pull this towards you (as close as you feel you can without becoming too anxious), at the rate of about 20 cms every 30 seconds.

You may stop this procedure at any time if you feel you are becoming too anxious. Whilst it is the intention of this research for participants to attempt to bring the spider to a position at which they could touch the spider box, stop if you feel too tense or would prefer not to continue. If you decide not to continue, your choice will be respected and will not prejudice your participation in the therapy program to follow.

Upon completion of either one or two of these spider exposure sessions you will attend eight therapy sessions, of about 40 minutes duration each, spread over four weeks. Upon completion of the therapy program you will again attend this laboratory for a final spider exposure session following this same procedure.

If, at any stage during this program or in the future, you become in some way troubled as a result of your participation, please do not hesitate to contact Dr George Wilson on 20 2240, Lee Archer, through the Psychology Department on 20 2237 or the University Psychology Clinic on 20 2805.

"I have read the information above and any questions I have asked have been answered to my satisfaction. I agree to participate in this investigation and understand that I may withdraw at any time.

I agree that research data gathered for the study may be published provided I cannot be identified as a subject."

Signature of subject ..... Date .....

"I have explained this project and the implications of participation in it to this volunteer and I believe that the consent is informed and that she understands the implications of participation."

Signature of investigator ..... Date .....

APPENDIX B

VISUAL ANALOGUE SCALES

Please place a cross on the following scales to indicate how you feel AT THIS MOMENT.

CALM _____ WORRIED

ACTIVE_____ SLEEPY

NO EXTREMELY
FEAR _____ FEARFUL

APPENDIX C

HEALTH / MEDICAL HISTORY QUESTIONNAIRE

MEDICAL HISTORY AND CURRENT HEALTH QUESTIONNAIRE

NAME: ..... GENDER: .....

AGE: ..... CONTACT NUMBER: .....

DO YOU SUFFER, OR HAVE YOU SUFFERED IN THE PAST, FROM ANY CARDIAC COMPLAINTS? (For example, Palpitations or High Blood Pressure)

YES/NO

If YES, please give more details .....
.....
.....

DO YOU SUFFER, OR HAVE YOU SUFFERED IN THE PAST, FROM ANY RESPIRATORY COMPLAINTS? (For example, Asthma or Bronchitis)

YES/NO

If YES, please give more details .....
.....
.....

ARE YOU CURRENTLY ON ANY MEDICATIONS? YES/NO

If YES, please give more details .....
.....
.....

DO YOU SMOKE? YES/NO

If YES, how many packets on average would you smoke per week?.....

HOW MUCH TIME EACH WEEK WOULD YOU SPEND ON PHYSICAL ACTIVITY AND BRIEFLY DESCRIBE THE TYPE(S) OF ACTIVITY

.....

.....

ARE THERE ANY OTHER DETAILS WHICH MAY BE RELEVANT TO YOUR PARTICIPATION IN THIS STUDY?

.....

.....