UNIVERSITY OF SOUTHAMPTON
FACULTY OF SOCIAL SCIENCE
PSYCHOLOGY

A BEHAVIOURAL APPROACH TO THE MANAGEMENT OF THE SYMPTOMS OF RAYNAUD'S DISEASE AND PHENOMENON

A THESIS SUBMITTED FOR THE DEGREE OF DOCTOR OF PHILOSOPHY

BY

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ABSTRACT

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A BEHAVIOURAL APPROACH TO THE MANAGEMENT OF THE SYMPTOMS OF RAYNAUD'S DISEASE AND PHENOMENON

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Raynaud's Disease, a condition affecting peripheral blood flow, is characterised by bilateral vasospasm of the affected area. Symptoms include pain, numbness and characteristic blanching (whiteness), cyanosis (blueness) and hyperaemia (redness) of the skin of the hands and feet. Symptoms are commonly precipitated by exposure to cold, although also implicated are a range of other triggers including emotional upset and physical pressure at the site of symptom manifestation. Raynaud's Disease differs from a related condition termed Raynaud's Phenomenon in that although both share the symptoms of vasospasm, symptoms of the former have no known aetiology, whereas the latter's symptoms are associated with a range of underlying physiological abnormalities such as Scleroderma.

This thesis addresses the questions of cause and effect in Raynaud's symptomatology (the former theoretically; the latter through questionnaire and interview), describes medical and behavioural approaches to treatment, and investigates the effects of one behavioural approach to the management of symptoms.

The subjective effects of Raynaud's are often ignored in the literature; therefore, pilot interviews with 4 patients were used in the design of a larger scale (79 participants) questionnaire based description of Raynaud's through the eyes of the sufferer. Results described three areas of interest: symptoms; the onset of Raynaud's and 'vasospastic' attacks; and the subjective effects of symptoms. Amongst other details, results suggested that for many sufferers the term attack is inappropriate as symptoms are continuous.

The treatment investigation consisted of a longitudinal, controlled comparison of the efficacy of Autogenic Training and Applied Relaxation with or without supplementary EMG or Temperature Biofeedback in 30 Raynaud's trainees and 10 Raynaud's controls. A cognitive model of biofeedback was adopted such that rather than being viewed as a treatment in its own right, EMG or temperature biofeedback signals 'fed back' to participants provided an insight into the effects on skin temperature control of the taught relaxation technique. 6 or 0 (control participants) training sessions were provided, and results evaluated across 3 sets of pre-, post- and follow-up assessments. The results indicated that the treated participants reported subjective improvement at post-treatment and follow-up assessment; however, objective evidence of the efficacy of treatment - as assessed through laboratory tests of voluntary control of finger skin temperature and cold stress, and ambulatory monitoring tests of finger skin temperature away from the laboratory - was collected only at the post-treatment assessment. The requirement to practice the relaxation exercises is noted.

The results of both investigations are discussed in relation to participant expectation in symptom onset, maintenance and treatment, the cause of Raynaud's attacks, and suggested methodological improvements in the future design of such investigations.

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For Mum: 29.3.34-20.1.97. Always in my thoughts.

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Chapter 1

INTRODUCTION

1.1 INTRODUCTION

Raynaud's Disease is a disorder of the peripheral vascular system. It is characterised by episodic "Vasospastic attacks" ie. periods of decreased peripheral blood flow to certain areas of the body - most commonly the hands and feet. Attacks occur as a result of spasm of the peripheral blood vessels following exposure to low ambient temperatures; however, some sufferers also report an association between their symptoms and emotional "stress".

There are two diagnostic categories of Raynaud's: Primary Raynaud's and Secondary Raynaud's. In both cases, the immediate symptoms include decreased dextrous sensitivity, severe pain, and a sequence of bilateral skin colour changes; however, in the latter case, further symptoms may be associated with some underlying physiological abnormality. Indeed, Primary and Secondary Raynaud's are differentiated on the basis of the presence or absence of identified probable organic cause. In the long term, an erratic blood supply to the skin results in physical changes over time. Consequently, ulcers are a frequent manifestation of severe symptoms, and occasionally gangrene will occur which may result in amputation.

The cause(s) of Raynaud's symptoms are yet to be identified. It is not entirely surprising therefore, that medical research is still searching for a cure. However, a range of palliative medical, and surgical procedures are on offer to the sufferer, and in recent years, behavioural means of symptom management have been explored. Although a series of papers claiming to evaluate the effects of such treatments have been published, methodological flaws, as outlined in the forthcoming chapters, at best limit the drawing of conclusions as to their strengths.

Therefore, the purpose of this research is to address the efficacy of the Behavioural approach to the management of Raynaud's symptoms.

The aims of this introductory chapter, and the two following chapters, are essentially to provide the reader with the necessary background to promote understanding of the condition, and hence, the theories behind current treatments. Initially, the structure and function of the peripheral vascular system will be described in detail, and a more in depth description of Primary and Secondary Raynaud's will be provided, before moving on to discuss the ongoing debate as to the probable cause(s) of the condition. The second and third chapters will address the question of precipitators of Raynaud's attacks.

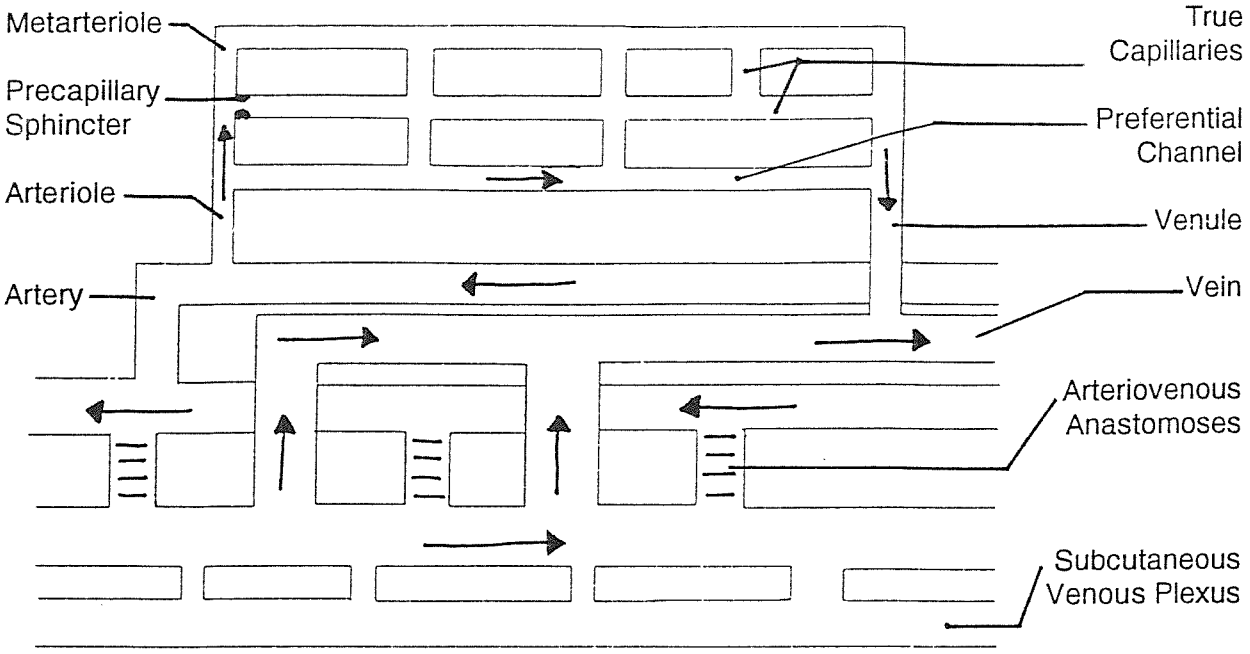
1.2 THE PERIPHERAL VASCULAR SYSTEM

The function of the peripheral vascular system

Generally, the volume of blood supplied to different organs of the body alters as a function of need. Thus, blood flow to the skeletal muscles increases during vigorous exercise to meet an increase in nutritive needs. However, the blood supply to the skin is not regulated purely according to its metabolic needs; indeed, 90% of blood flow through the peripheral tissues is involved in thermoregulation of the body. As the volume of blood in the body is constant, an increase in the metabolic needs of a particular organ requires a reduction in blood flow in other areas. For reasons of survival, blood tends to be diverted away from the skin toward the central organs of the body at times of need; thereby minimising heat loss to the environment and maintaining the core temperature of the body. This diversion of blood away from the skin is possible given the structure of the peripheral vascular system.

The structure of the peripheral vascular system

The peripheral vascular system consists of the network of blood vessels that transport blood to and from the skin. This network is illustrated in figure 1.1, and explained below.



------> Direction of blood flow

Fig 1.1: The layout of the peripheral blood vessels

Blood is carried away from the heart by the muscular arteries, which branch into arterioles and metarterioles before reaching a network of capillaries in the skin. Blood flow through the capillary beds is slow; thus enabling nutrient and gaseous exchange. The flow into the capillaries is controlled by the constriction and dilation of the muscular arterial walls, and small Precapillary sphincter muscles. From the capillaries, blood flows into the venules and finally the veins; however, a few large capillaries called preferential channels by-pass the main capillary bed and course directly into the venule. Areas of the skin frequently exposed to the environment (eg. the hands, feet, lips, nose, and ears) are served by arteriovenous anastomoses (or shunt vessels) and venous plexuses (or storage vessels) which serve in the thermoregulation of the skin. At cold or comfortable ambient temperatures, the shunt vessels are almost closed; therefore, blood flows directly through the vascular system. However, at high ambient temperatures, the shunt vessels dilate. This allows large quantities of warm blood to flow into the storage vessels under the skin thereby cooling the blood as heat evaporates to the surrounding air. At low ambient temperatures, arteriolar constriction limits blood flow through the skin, so that potential heat loss to the environment is reduced. The characteristic Raynaud's attacks are associated with such

arteriolar constriction. If we are to begin to understand why attacks occur, we need a basic understanding of the factors involved in the control of peripheral blood flow.

The control of peripheral blood flow

Blood flow is determined by cardiac output and vascular resistance (ie. the diameter and pressure) within the blood vessels. High cardiac output, and low vascular resistance will support high levels of peripheral flow. In contrast, any action to reduce intra-vascular pressure will reduce blood flow in the vessel. Therefore, blood vessels will close completely if the internal pressure is lower than that of the wall of the vessel; moreover, if the vessel diameter falls below a particular size, the blood cells will be unable to pass through the vessel such that blood flow is prevented. However, three main factors are implicated in the control of peripheral flow: namely local tissue requirements (local control), Nervous control and Humoral control.

Local control of peripheral blood flow

Local control is determined by factors in the immediate environment of the blood vessels. Local control may occur in the short or long term. In the short term, the metabolic needs of the tissues, and the pressure inside the vessels will determine blood flow. High levels of metabolic waste (vasodilator substances) - or low levels of nutrients - in surrounding tissues will have a vasodilatory effect on the peripheral blood vessels. Thus, following a period of tissue blood deprivation, metabolic waste such as CO_2, lactic acid and histamine will build up such that once circulation is regained, blood flow to the affected area(s) will be as much as five times the "normal" volume. This process, known as "Reactive Hyperaemia", prevents long term nutritional loss to the tissues in that it repays the oxygen and nutrient debt incurred during the period of deprivation. The precapillary sphincter is assumed to be involved as the number of open precapillary sphincters generally remain proportional to the metabolic needs of the tissues. As to the role of the pressure of the peripheral vasculature, high arteriolar pressure will force tissue fluid through the capillary wall to the surrounding tissues thereby reducing overall blood volume and decreasing pressure in the arteriole (a phenomenon known as the capillary fluid shift).

Long term local control, in contrast, will occur when the long term metabolic needs of the tissue change. Thus, the blood supply (and associated tissue vascularity) will gradually increase in line with a sustained, long term increase in the metabolic needs of the tissues.

Nervous control of peripheral blood flow

Nervous regulation differs from its local counterpart in terms of both speed and span of effect ie. nervous (and humoral) control precipitate effects rapidly, and exert control of large areas of the circulatory system simultaneously. Peripheral blood flow is under the control of the Autonomic Nervous system. As illustrated in figure 1.2 below, there are two systems of nervous control within the human body: the autonomic nervous system (ANS) and the central nervous system (CNS).

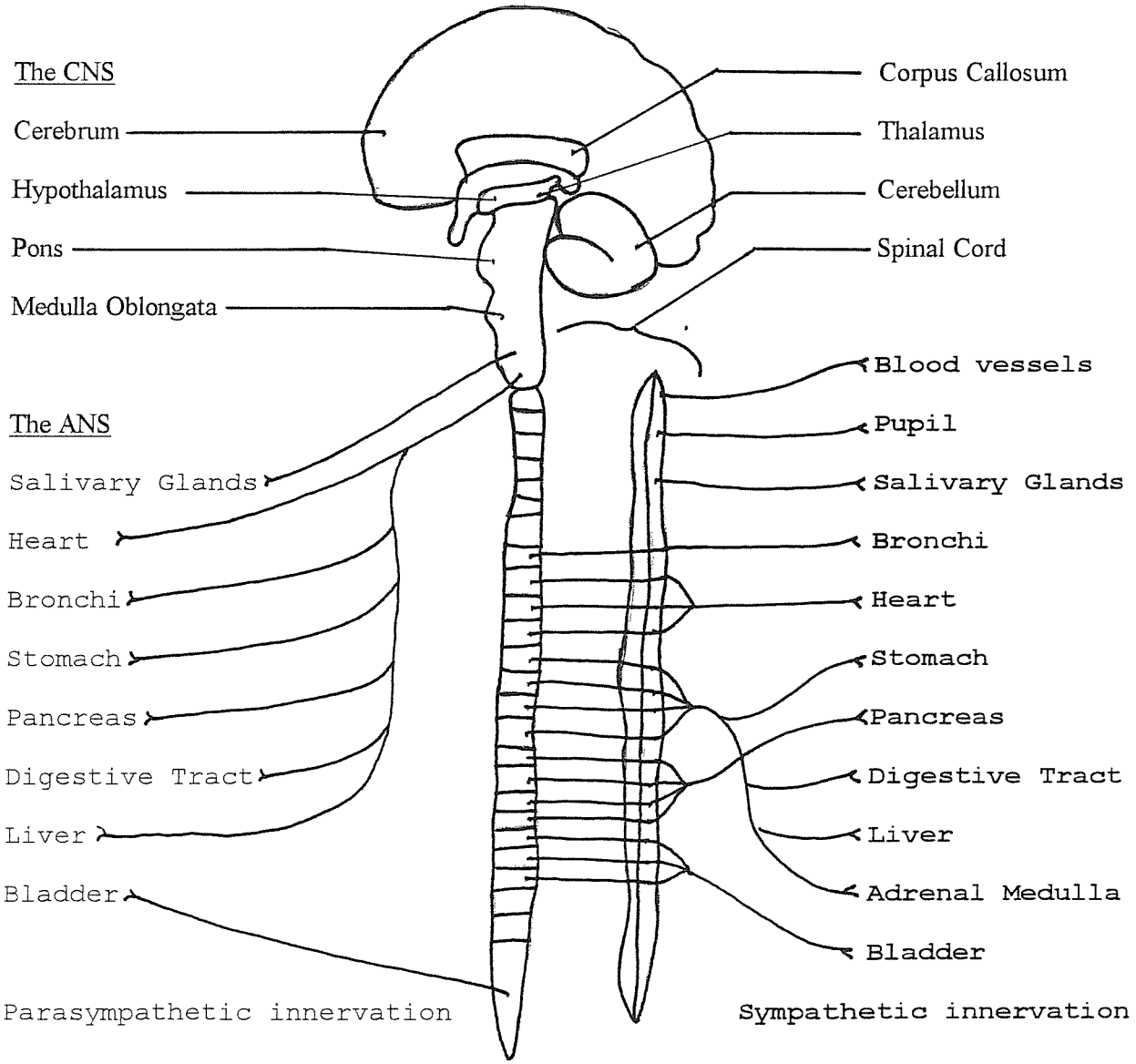


Fig 1.2: The systems of the body controlled by the branches of the Autonomic Nervous system

The CNS, which consists of the brain and spinal cord, governs activities under voluntary control such as movement of the skeletal muscles, and cognitive processes. The ANS, however, regulates involuntary processes such as heart rate and breathing. There are two branches of the ANS: the Sympathetic nervous system and the Parasympathetic nervous system. For the most part, the branches have opposing effects. For example, increased sympathetic innervation tends to increase metabolic processes such as heart rate. In contrast, increased parasympathetic innervation is associated with a decrease in such processes. The physiological functions of the two systems are listed in table 1.1 below.

Area of the body	**Effect of increased Sympathetic nervous activity**	**Effect of increased Para-sympathetic nervous activity**
The Pupil	Dilation	Constriction
The Salivary glands	Inhibits secretion	Stimulates secretion
Heart rate	Increases	Decreases
The Bronchi	Dilation	Constriction
Peristalsis/Secretion in the digestive tract	Inhibits	Promotes
Sphincters of the digestive tract	Constriction	Relaxation
Bladder	Inhibits contraction	Contraction
Adrenal gland	Secretion of adrenaline and Noradrenaline	Local release of ACH
Bile duct	Conversion of glycogen to bile	Release of bile
External Genitalia	None	Vasodilation of erectile tissue
Blood Vessels	*Constriction*	*No effect*
Sweat glands	Increases production of sweat	No effect

Table 1.1: The effect of the sympathetic and parasympathetic branches of the ANS on the body

As is evident from the table and figure above, peripheral blood flow to the skin is not under the control of the parasympathetic nervous system. Indeed, in the main, the peripheral blood vessels are entirely sympathetically innervated (the exceptions being the capillaries, precapillary sphincters and the bulk of the metarterioles which are influenced by the local humoral environment.) However, as illustrated in figure 1.3 below, there are two levels of control of sympathetic innervation to the peripheral blood vessels - namely the vasomotor centre and the higher brain centres such as the anterior temporal lobe, the temporal cortex and the hypothalamus.

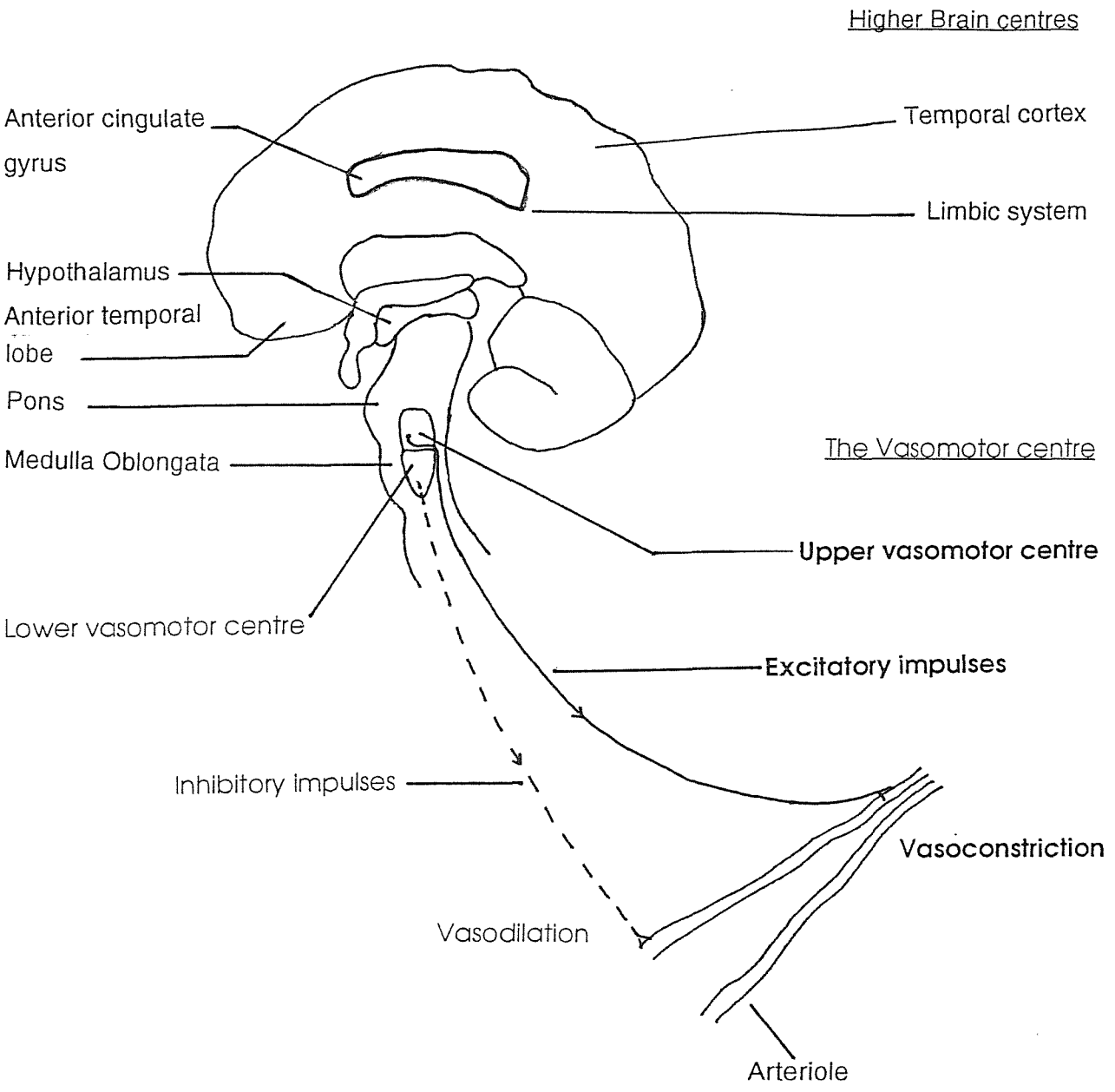


Fig 1.3: The control of sympathetic innervation to the peripheral blood vessels

The Vasomotor Centre is located in the brain stem: the upper part in the pons; the lower in the medulla. In addition to being structurally separate, the two parts of the vasomotor centre are also functionally distinct. The upper centre continually fires nerve impulses; thus maintaining a partial state of contraction (vasomotor tone) in the blood vessels at all times. However, the upper vasomotor centre also fires excitatory impulses which increase contraction of the peripheral vessels still further to bring about a state of vasoconstriction. In contrast, the action of the lower vasomotor centre is to transmit inhibitory impulses to decrease vasomotor tone, and consequently result in arteriolar dilation.

The higher brain centres such as the anterior temporal lobe, the orbital areas of the temporal cortex, the anterior cingulate gyrus, the limbic system, and the hypothalamus may all influence peripheral blood flow. For example, the preoptic region of the anterior hypothalamus acts as the body's temperature control centre. The hypothalamus receives information about body temperature from thermal receptors in the skin, and directly by monitoring the temperature of hypothalamic blood flow. A feedback mechanism is involved in that if the body temperature is higher than normal (core body temperature 36-37.5^{0}C; extremities 32-35^{0}C), the vasomotor centre will transmit inhibitory impulses to the peripheral blood vessels resulting in vasodilation and the evaporation of heat from the surface of the skin. Similarly, cooling of the body causes vasoconstriction, which minimises the amount of body heat lost to the environment.

Humoral control of peripheral blood flow

Humoral control is related to nervous control in that sympathetic innervation acts on the adrenal medullae, causing the secretion of the neurotransmitter Norepinephrine which elicits vasoconstriction of the peripheral blood vessels (among others). A number of other hormones and circulatory substances such as angiotensin, bradykinin, vasopressin, serotonin and prostaglandins may be implicated in the control of the circulatory system; however, as they are generally not involved directly in thermoregulation, they will not be discussed in depth.

Further factors involved in the control and influence of body temperature include increased sweating, decreased liver metabolism, a low ambient temperature, low humidity, and wind chill factors. Within limits, human skin can be exposed to extreme cold without adverse effects on tissue vitality. Raynaud's sufferers differ from non-sufferers in that they will experience the effects of exposure to extreme cold at relatively "normal" temperatures; therefore, complete vasoconstriction is a common occurrence which over time may put the tissues of the sufferer at risk.

1.3 RAYNAUD'S DISEASE AND PHENOMENON

The first description of Raynaud's Disease appeared in a paper by the French physician Maurice Raynaud in 1862[1]. The article described the symptoms of 25 patients suffering from a disorder of the peripheral vascular system defined as *'...a neurosis characterised by enormous exaggeration of the excitomotor energy of grey parts of the spinal cord which control vasomotor innervation.'* Later reviews of Raynaud's original work suggested that the patients described were not an homogeneous group (Hutchinson, 1901). Two classifications of the condition were identified: Raynaud's Disease and Raynaud's Phenomenon. The symptomatology of Raynaud's Phenomenon appears to be associated with some underlying pathological abnormality; in contrast, Raynaud's Disease has no identified organic cause. The two categories of Raynaud's have not been clearly isolated for purposes of definition owing to disagreement as to the correct terminology to employ when referring to the disorders. Indeed, there are a number of synonymous "Raynaud's labels". Raynaud's Disease is often referred to as 'Idiopathic' or 'Primary' Raynaud's. Raynaud's Phenomenon may be termed 'Raynaud's Syndrome', 'Secondary Raynaud's', or 'Secondary Raynaud's Disease'. In addition, the terms 'Disease', 'Phenomenon', and 'Syndrome' are used interchangeably to refer to Raynaud's disorders as a whole. (Belch, 1989). For purposes of clarity, only the terms 'Raynaud's Disease' and Raynaud's Phenomenon' will be used in this and the remaining chapters.

Research into the condition(s) has been ongoing for the last 100 years. A useful definition of Raynaud's conditions as a whole has evolved during this time: *'...a functional disorder of the peripheral vascular system in which the patient suffers from painful episodes of vasoconstriction in the hands and sometimes the feet. During an attack, the skin blanches and is cold to the touch. Attacks are usually precipitated either by exposure to cold, such as touching a cold object, or by emotional upset.'* (Blanchard, 1979). However, if the reader is to gain a clearer picture, further description of the disorder is required.

[1] Translation: Raynaud M. (1888) New Research on the nature and treatment of local asphyxia of the extremities. (T. Barlow, Trans) London: The New Sydenham Society

The symptoms of Raynaud's Disease and Phenomenon

Vasospastic attacks (or vasospasms) are the symptoms characteristic of both Raynaud's Disease and Raynaud's Phenomenon. A Vasospastic attack is commonly described as a sequence of three[2] bilateral colour changes in the skin in response to some stimulus (commonly cold ambient temperatures). The first stage of the attack is whitening of the skin following constriction, or spasm, of the peripheral arterioles. Blood flow to the extremities is thus reduced, resulting in blanching of skin as the white subcutaneous connective tissue is no longer masked by the flow of blood. A decrease in dextral sensitivity, or numbness, accompanies this stage of the attack. The blood trapped in the capillaries rapidly becomes deoxygenated, starving the surrounding tissue of oxygen and turning the skin blue. This cyanotic phase of the attack is painful, and may last for several hours. The final, most painful phase of the attack is the red hyperaemic phase, or rubor, in which circulation is regained. Circulation does not return to normal until the huge oxygen debt incurred is repaid. Therefore, large quantities of blood circulate as a result of excessive vasodilation.

Symptoms most commonly affect the fingers, hands and feet, although the ears, nose and other peripheral areas may be involved. However, sufferers of Raynaud's Phenomenon experience further symptoms associated with some underlying physiological abnormality such that they may experience involvement of the oesophagus (Belch et al, 1989), the lungs (Fahey et al, 1984), the tongue (Da Cunha Bang et al, 1985, Vagn Neilsen, 1984), or other areas of the body. Table 1.2 details conditions associated with Raynaud's Phenomenon.

It has been suggested that the symptoms of Raynaud's Disease are also associated with other disorders. However, this association is likely to be coincidental given the vasospastic nature of the conditions. O'Keeffe et al (1992) investigated the prevalence of migraine in 93 Raynaud's Disease patients and 93 age and sex matched controls. The results indicate a higher incidence of migraine in the Raynaud's sample than in the non-symptomatic controls. Indeed, such findings led Kaiser (1992) to suggest that Raynaud's Disease and migraine are simply manifestations of the same disorder.

[2] Two colour changes are now considered sufficient for diagnostic purposes (Belch 1994).

Category of Raynaud's-related disorder	Common disorders
Connective Tissue Diseases	Scleroderma Mixed connective tissue disease CREST syndrome Systemic Lupus Erythmatosus Rheumatoid Arthritis Dermatomyositis and Polymyositis Pansystemic Sclerosis Hepatitis B (antigen induced)
Peripheral Vascular disorders	Arteriosclerosis Embolic Disease Venous Thrombosis Thromboangiitis Obliterans
Blood Abnormalities	Protein and Cellular disorders Anaemias
Occupational Disorders	Vibration injury (Vibration White Finger) Cold Injury Vinyl chloride work Ammunition (nitrate) work
Drug associations	Ergot/antimigraine compounds Beta Blockers Cytotoxics Bromocriptine Sulphasalazine Birth Control pills
Miscellaneous	Thyroid disease Cold agglutinins Cryoglobulinaemia Chronic renal failure Neoplasms Neurological disorders (Inflammatory) Vasculitides

Table 1.2: Disorders commonly associated with the symptoms of Raynaud's Phenomenon

The diagnosis of Raynaud's Disease and Phenomenon

Given the problems associated with the labelling of Raynaud's disorders, one might rightly assume some associated difficulties in terms of diagnosis. Allen and Brown (1932) proposed a medically accepted set of diagnostic criteria for Raynaud's Disease which are still used today. "Raynaud's" is diagnosed when a patient presents with at least a two year history of episodic bilateral colour changes in the skin that are precipitated by cold or emotion. The diagnosis of Raynaud's Disease further

requires the absence of severe gangrene, and of systemic disease that might account for attacks[3]. In general practice, three methods of diagnosis are used: a colour chart of skin colour changes supplemented with a simple questionnaire; a more in depth questionnaire derived from clinical consensus of the symptoms of Raynaud's; and a standard description of symptoms (again based on clinical consensus) from which GPs form an opinion. In a comparative study, Brennan et al (1993) asked 6 clinicians to diagnose 30 patients using the three systems. They suggest that the only reliable system for the diagnosis of Raynaud's is individual opinion based on a standard description of the disorder.

However, the situation is complicated further by the suggestion that Raynaud's Disease may be an initial manifestation of the symptoms of Raynaud's Phenomenon (Cruz et al, 1988; Kallenberg, 1992); indeed, Lee et al (1993) suggest that the presence of ACA in the blood is not always indicative of connective tissue disease (although one might assume that the presence of ACA could indicate the later development of some underlying condition).

For diagnostic purposes therefore, Raynaud's Disease should perhaps be classified as "Raynaud's that is not yet (and may not become) Raynaud's Phenomenon" - perhaps not the most helpful of diagnostic criteria. To clarify matters somewhat, Veale et al (1993) list a series of clinical features that might predict the later development of some systemic illness characteristic of Raynaud's Phenomenon. These include thin, leathery skin, pitting scars on the finger pads, finger ulceration, the occurrence of Raynaud's attacks throughout the year, initial symptom onset during childhood or in late(r) life, chilblains in adulthood, and vasospasms initially affecting only one or two fingers of each hand.

To reiterate, therefore, the diagnosis of Raynaud's Disease requires a two year history of vasospastic attacks, together with the absence of both obvious involvement of underlying conditions, and the suspicion that such conditions may later develop.

[3] Commonly, clinical features of associated disorders, Anticentromere antibodies (ACA) in the blood, and abnormal nailfold capillary patterns are felt to be indicative of the presence of systemic disease.

The prevalence of Raynaud's Disease and Phenomenon

It has been estimated that response of the peripheral vascular system to cold is abnormal for up to twenty percent of the population if one includes sufferers of Raynaud's Disease, Raynaud's Phenomenon, and cold sensitive individuals (Lewis, 1949). The figure drops to 10% if one examines only those seeking medical advice for their condition (Challenor, 1989). Indeed, surveys of symptom prevalence are notoriously contradictory (owing to the difficulties of labelling and diagnosis outlined above): some claim that Raynaud's is more common in the female population than the male (Spittel, 1972 suggests a 15:1 ratio); others report no such gender differences (eg. Porter et al, 1976). However, trends across a number of surveys support the notion that Raynaud's is, at least in part, related to climate. Maricq et al (1976) report a low incidence (1.9%) of those seeking medical advice in the warm conditions of South Carolina. Similarly, Riera et al (1993) report an overall prevalence of 3.7% (3.2% for men; 4.7% for women) in their "healthy" Spanish population of approximately 1500 men and women. In contrast, both Heslop et al (1983) and Leppert et al (1987) report a far higher incidence (13% and 15% respectively) in the cooler climes of northern Europe.

Initial symptom onset usually occurs before the 40th birthday (Allen and Brown, 1932; Heslop et al, 1983); however, it is rare in children unless associated with some underlying disorder (Emery and Schaller, 1977). As to family history, there is, at present, no evidence to suggest differences in the incidence of Raynaud's in terms of whether relatives are similarly diagnosed.

The Raynaud's and Scleroderma Association

Financial support for the first two years of this project was provided by The Raynaud's Association - a self-help group set up in 1982 by Anne Mawdsley. The initial aims were to put sufferers of Raynaud's Disease and Phenomenon in contact with each other for emotional and practical support as well as to increase general awareness of the condition. In the 13 years since it was formed, the Association, which has since become "The Raynaud's and Scleroderma Association[4]", has raised over 2 million

[4] The Raynaud's and Scleroderma Association: 112 Crewe Road, Alsager, Cheshire. ST7 2JA (01270) 872776 Charity Reg. No. 326306

pounds for research into Raynaud's and associated conditions, and through Newsletters and information leaflets, has kept both its members and the medical profession up to date with current research and treatments.

1.4 THE CAUSAL MECHANISM(S) OF RAYNAUD'S DISEASE AND PHENOMENON

The mechanisms of the vascular change associated with Raynaud's symptoms are readily understood; however, what actually causes the symptoms, remains to be determined. Two theories are currently under debate: Raynaud's suggestion of Hyperactivity of the sympathetic nervous system (HSNS); and Lewis' (1928) "Local Fault" at the site of symptom manifestation.

Hyperactivity of the Sympathetic Nervous System: (Raynaud, 1862)

Raynaud's explanation for the occurrence of "Vasospastic attacks" rests with the established role of the SNS in the control of peripheral blood flow. To recapitulate, an increase in sympathetic excitatory innervation to the blood vessels will promote vasoconstriction of the peripheral blood vessels. In Raynaud's sufferers, this response is claimed to be *exaggerated* owing to SNS hyper-responsiveness (HSNS) to cold or cool stimuli. Consequently, the sufferer will experience periods of intense vasoconstriction in response to relatively "normal" ambient conditions.

A Local fault at the site of symptom manifestation (Lewis, 1928)

An opposing theory (Lewis, 1928) suggests that Vasospastic attacks are the result of a "local fault" at the site of symptom manifestation. He claimed that symptoms could be induced (through local cooling) in anaesthetized and sympathetically denervated fingers which both challenges the mediating role of the SNS in the elicitation of attacks, and suggests some "fault" at the point of cooling - a local fault.

HSNS or local hypersensitivity as the cause of Raynaud's attacks ?

Since before the second world war, conclusive evidence of Raynaud's attacks being mediated by central (HSNS) or peripheral (local fault) mechanisms has eluded the academic world. That said, a series of articles have been published in favour of both theories.

Support for Lewis' suggestion of a Local fault falls into two camps: namely, research that provides dismissive evidence of central mediation of attacks; and hard evidence as to the nature of the alleged local "impairment". If Raynaud's attacks are centrally mediated, one might expect some measurable index of increased sympathetic activity during attacks. Yet, there appears to be no evidence of increased sympathetic activity in Raynaud's volunteers as compared with controls. Fagius and Blumberg (1985) failed to isolate abnormal sympathetic increase to the hands during attacks, despite the employment of microelectrode recordings of sympathetic nervous activity. Similarly, Downey and Frewin (1973) found no increased reflex sympathetic vasoconstriction to cold in Raynaud's sufferers. A related point concerns the unsatisfactory nature of the sympathectomy[5] as treatment for Raynaud's symptoms: symptoms tend to recur despite loss of sympathetic innervation to the affected area.

Quite clearly, though loosely suggesting a lack of SNS involvement in the elicitation of Raynaud's attacks, the evidence above fails to implicate an alternative local fault explanation. However, in a series of studies based on Lewis' original work, Freedman and co-workers have induced both vasospasm (Freedman et al, 1989a), and vasodilation (Freedman et al, 1987) in nerve blocked individuals thereby suggesting that Raynaud's attacks are not centrally mediated, but rather associted with some local fault. Further, a number of researchers have sought to identify the "site" of the fault. The arteriovenous anastomoses (or shunt vessels) were rejected by Miller and Walder (1972) on the basis that there were no differences between Raynaud's sufferers and controls in the effects of local cooling on finger blood flow compared with capillary flow. As the normal dilatory action of the shunt vessels reduces capillary flow, these vessels (and the connection between the capillaries and shunt vessels) could not be implicated in the elicitation of vasospastic attacks. The endothelium of the

[5] Sympathectomy (see chapter 4): a surgical procedure, used extensively in the past as treatment for Raynaud's, in which SNS innervation to the hands and/or feet is severed.

peripheral blood vessels was nominated by Naidu (1992) as his initial studies suggest lower levels of EDRF secretion (endothelium derived relaxing factor - a vasodilator) in Raynaud's patients than non-symptomatic controls. Indeed work by Bedarida et al (1993), and Zamora et al (1990) further support the association between vasospasm and an impairment in the endothelium-dependent dilation of the peripheral blood vessels.

Earlier work by Cohen and Coffman (1981) isolated the presence of a beta-adrenergic mechanism that modulates the effects of local vasoconstricting agents. Freedman et al (1989c) relate this mechanism to the elicitation of Raynaud's attacks. The roles of beta-adrenergic mechanisms and EDRF are not entirely incompatible in that both point to the peripheral vascular endothelium as a possible site of some local fault in the circulatory system of Raynaud's sufferers.

Although a possible "local fault" site has been isolated, several published articles appear to support Raynaud's original HSNS hypothesis. Anecdotally, a number of points suggest some involvement of sympathetic innervation in the elicitation of vasospastic attacks. The induction of vasospasm through local cooling has, as outlined above, been cited as evidence of peripheral mediation of attacks. However, Raynaud's sufferers claim that laboratory induced "vasospasm" is very unlike that of a characteristic Raynaud's vasopastic attack in that the former involves only isolated cooling, whereas during attacks, even if symptoms are only visible in the hands, the whole body feels cold (personal communication). Therefore, although local factors may well be involved in local cooling, this does not prove that a local fault is the cause of Raynaud's vasospastic attacks. Along similar lines, a number of points can not be clarified by a local fault explanation: Why are Raynaud's symptoms bilateral in nature ? How might a local fault account for the alleged role of emotional stress in the elicitation of attacks ? Moreover, the effects of cervical sympathectomy (cutting innervation to the hands) may be unsatisfactory, but those of lumbar sympathectomy (for treatment of symptoms in the feet) are highly acceptable (Coffman and Davies, 1975). Further, researchers investigating peripheral flow in denervated volunteers, fail to account for the role of the SNS in the release of circulating adrenal

Norepinephrine; thus, even if direct sympathetic innervation is lost through the action of anaesthetic[6], discharge of the SNS will cause indirect vasoconstriction of the peripheral vessels.

On a more empirical note, Day and Klingman (1939) discovered that the skin of sufferers of acrocyanosis[7] warms and reddens during sleep. As sleep is associated with a decrease in sympathetic nervous activity, this must suggest involvement of the SNS. Similarly, Peacock (1957) reported "normal" hand blood flow in Raynaud's patients following total body warming (which elicits sympathetic release), and following sympathetic blockade (Peacock, 1959). Further, Mittleman and Wolff (1939) claim that temperature drops in the extremities do not occur if the sympathetic nerve supply to the extremities has been interrupted. However, the vasodilatory role of the SNS is not disputed. Indeed, pro "local fault" researchers such as Freedman et al (1987) report that laboratory induced vasodilation is not attenuated by sympathetic nerve blockade. The area of controversy lies in the role of the SNS in the mediation of vasoconstrictive responses. A series of papers concerned with the role of the SNS in vasospasm associated with vibration induced injury, favour SNS mediation: Olsen et al (1987) applied vibration to one hand and produced vasospasm in the contralateral hand; Olsen et al (1988, 1989) inhibited vibration-induced vasospasm by proximal nerve blockade. In terms of Primary symptoms, Jamieson et al (1971) report enhanced reflex sympathetic vasoconstriction to local cooling following ice application to the neck[8] in Raynaud's sufferers but not controls. It is difficult to see how such a finding could be attributed to structural abnormalities of the digital vessels. Englehart (1990) reports equivalent finger systolic pressure in non-symptomatic controls and nerve blocked Raynaud's sufferers during tests of body and finger cooling. Without the nerve block, a reduction in blood pressure associated with vasospasm would have been expected. Lastly, Cooke et al (1990) describe reduced blood flow in the contralateral hand of non-symptomatic volunteers during local hand cooling, which must be consistent with sympathetic involvement of peripheral vasoconstriction.

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[6] - though generally not through the action of sympathectomy (see Chapter 4).

[7] Acrocyanosis: a Secondary Raynaud's condition characterised by continuous (rather than the usual intermittent) symptoms.

[8] Vasoconstriction induced through local cooling and ice application to the neck is more akin to vasospastic attack than vasospasm induced purely through cooling because in the former case, the whole body is cooled.

As the evidence above suggests, both central and peripheral mediation remain viable causes of Raynaud's attacks. (It is, of course, not without grounds that both may have a role). The two theories clearly share common ground in that both encompass the idea that some area of the circulatory/nervous system is hypersensitive to the cold. In terms of the Local Fault theory, explanations for local hypersensitivity are offered (eg. low levels of EDRF etc.); however, equivalent reasons for over-sensitivity of the SNS eg. Social learning theory (Bandura, 1972), operant conditioning, expectancy learning, are poorly explored. Though a worthy avenue of investigation, an in depth exploration of these areas falls outside the scope of this thesis.

It is clearly evident that further research is required to differentiate between (or at least clearly define) the relative roles of central and peripheral mechanisms in the onset of Raynaud's symptoms. That aside, whatever the causal mechanism(s), the factors that trigger vasospastic attacks are still under debate. Cold is a universally accepted trigger; however, a number of other "catalysts" have been suggested. The second chapter looks at these in more depth when it asks "What precipitates Raynaud's attacks ?"

Chapter 2 WHAT PRECIPITATES RAYNAUD'S ATTACKS ?

2.1 INTRODUCTION

As noted in the previous chapter, a number of triggers have been cited as eliciting Raynaud's attacks. Most commonly, and indeed universally, cold weather is mentioned; however, it is also claimed that vasospasm may be associated with localised physical pressure, and with "emotional stress". Furthermore, a precipitating role has been suggested for hormones and other chemicals such as prescribed drugs and the effects of smoking. This chapter examines more closely the documented evidence in support of such claims, and attempts to describe the action of the alleged precipitators in the elicitation of attacks. Moreover, anecdotal and empirical evidence of involvement of the triggers in the onset of the condition as a whole will also be explored.

2.2 COLD AS A TRIGGER OF RAYNAUD'S ATTACKS

The action of cold in the elicitation of Vasospastic attacks

An explanation of how any precipitator triggers vasospasm must touch on the inherent mechanism of Vasospastic attacks. As described in the previous chapter, two mechanisms (Hypersensitivity of the Sympathetic Nervous System, and some "Local Fault" at the site of symptom manifestation) have been forwarded. Current evidence favours both accounts; therefore, the role of cold in the elicitation of attacks will be addressed firstly in terms of sympathetic innervation and secondly in regard to local factors.

The body's global thermoregulatory processes, as outlined in chapter 1, clearly account for the role of cold ambient conditions in the elicitation of vasospasm in terms of the combined roles of the temperature receptors, the hypothalamus, and the vasomotor centre on the SNS, and the resulting (direct and indirect) vasoconstriction of the peripheral vessels. In contrast, local mediation would be explained in terms of some local vasoconstrictor reflex.

Evidence of the role of cold in the elicitation of Vasospastic attacks

As cold is universally accepted as a trigger of Raynaud's symptoms, and a number of the papers cited in the previous chapter have been shown to employ local or total body cooling to promote vasospasm, only a brief overview of documented evidence of the role of cold in the onset of Raynaud's symptoms will be presented here. Several research groups in the 1970's (Lottenbach, 1971, Jamieson et al, 1971, and Cohen and Coffman, 1971) report a heightened vasoconstrictive response to cold in Raynaud's patients compared with non-symptomatic controls. Moreover Olive (1990) refers to a 42 year old sufferer of Raynaud's Disease who experienced vasospasm and a frostbite injury at the point of contact with the spray button of an aerosol can. The area of frostbite injury exactly matched that, in size and shape, of the button; and indeed, although not reaching freezing temperatures, the button was confirmed, through laboratory testing, to be cold enough to precipitate vasospasm. However, it appears that the symptoms of Secondary conditions, though generally associated with cold, are not necessarily triggered by cold. Dormandy and Evans (1989) cite a 42 year old male sufferer of Vibration White Finger whose Raynaud's-like symptoms were eased, rather than precipitated, by the cold. Indeed, he would put his hands in the fridge to curtail vasospasm. The authors could offer no explanation for this apparent anomaly - though given the large number of Raynaud's associated disorders, it should perhaps come as no surprise that a few might differ in cause.

2.3 PHYSICAL PRESSURE AS A TRIGGER OF RAYNAUD'S ATTACKS

The action of physical pressure in vasospasm

The effect of localised pressure on the skin - as might occur when carrying a heavy bag by the handles - is to create an obstruction to both arterial flow and venous return from the pressure site. Depending on the degree of pressure on the skin, the resultant flow may be completely occluded, or merely reduced. In the non-symptomatic hand, on removal of the cause of pressure, normal blood flow will resume following a period of reactive hyperaemia. In the Raynaud's hand however, the return to normal flow is

slowed (particularly in those with secondary damage to the vessels) as once an attack has been elicited, it may take minutes or hours for normal circulation to resume.

2.4 "STRESS" AS A TRIGGER OF RAYNAUD'S ATTACKS

What is "Stress" ?

Since the last century, "stress" has been regarded as a causal factor in the onset of disease. A "stressful life style" is one of the many readily accepted explanations of ill health and premature death. Studies claim to show adverse effects of stress in patients with coronary heart disease, asthma, and even diabetes mellitus (as cited in Bradley and Cox, 1983). Consequently, stress reduction techniques (eg. stress management courses, meditation classes, and calming tonics) have fallen into favour as a means to the "stress-free lifestyle" associated with good health.

The concept of stress was initially introduced by Cannon (1914) in his investigation of the fight or flight response to danger (ie. the physiological preparation of the body for action). On the basis of Cannon's work, a number of rather complex models of the relationship between stress and disease have been published in the psychological and medical literature. For example, Hans Selye (1946) proposed the General Adaptation Syndrome (GAS) model of how stress might lead to ill-health. The GAS model involves three stages. The first, termed the "alarm reaction", consists of two separate phases: "the shock phase", during which the body's resistance to a stressor is lowered; and the period of "counter shock" when the body's defense mechanisms are elicited. The second "stage of resistance" describes the period during which the defense/resistance mechanisms continue to be active until they are unable to further counter the effects of the stressor. This cues the third stage, in which the adaptive resources collapse, leading to disease and possibly death. Consequently, the model proposes that it is the body's inability to cope with the stressor, that causes the onset of disease. However, the stressors employed by Selye in the empirical work on which the model is based, were mainly physical (eg. water or sleep deprivation) such that the model does little to describe the effects of "psychological stressors". Moreover, the GAS model proposes that the effect(s) of a stressor will be alike in all exposed to that

stressor. Although not unreasonable in terms of physiological stressors, such a proposition does not bode well within the realm of psychological stressors in that what is "stressful" to one person, may not be so to others; and indeed, factors that an individual considers "stressful" may alter with a change in context. Lazarus (1966) suggested that the effect(s) of stressors depend on the individuals' view of the stressor ie. an event or process is only stressful if so appraised by the individual. Thus it is the individuals' inability to cope with an identified stressor that preempts ill-health and disease.

Such models of "stress" have formed the foundations of a number of popular theories of stress and disease. For example, the idea that individuals may be predisposed to the effects of certain stressors is entertained in Lachman's (1972) "Weak Link" theory. In brief, genetic or traumatic influences are claimed to predispose an organ system to biological vulnerability during stress; and thus respond in a stereotypic way to particular stressors. Consequently, the idea of "stress" as the root of ill-health has gained in popularity. Moreover, given its ambiguous nature, the term congenially covers the cracks in our understanding of the processes of disease. It is a concept that we inherently understand. If the cause(s) of a disorder are unknown, both patient and doctor will be content with a "stress-related" explanation; a tired, overworked business woman will put her headaches down to stress. Yet, the term "stress" is not clearly defined. The dictionary definition - "*difficult circumstances, mental or physical distress caused by these*[9]". - beautifully demonstrates the circular nature of the concept of stress. It is both stimulus (stressor) and response (stress); it is an umbrella term that tells us nothing about the underlying mechanisms of the "stress response"; indeed, "*The inclusive label 'stress' contributes little to an analysis of the mechanisms that may underline or determine the organism's response.*" (Ader, 1990). Further, the measurement of "stress" is somewhat problematic in that it relies entirely on self-report, and the measurement of physiological variables claimed to be associated with the stress response eg. changes in heart rate, breathing, and levels of circulating corticosteroids in the blood. In terms of self report, the lack of an acceptable definition drastically reduces the reliability of data. As to the physiological correlates of stress, these may also be associated with increased physical activity, or a

[9] The Oxford Paperback Dictionary: Oxford University Press, 1987

number of other processes; indeed, proponents of the term "stress" fail to take into account that physiological responses are complex and multiple.

Thus, in spite of growing opposition to the universal association of "stress" and disease, it remains a popular concept both within the lay and the academic communities. Indeed, the term "stress" has become an inherent part of the definition of Raynaud's Disease, and a number of published articles have addressed its role in the onset of symptoms. For purposes of completeness, the "Raynaud's Disease and Stress" literature will be reviewed below.

However, as "stress" has not been adequately defined, the researcher may attach any definition to all or part of the concept as fits her or his current thinking. Stress is described as both an emotional and physical entity. In terms of Raynaud's symptoms, "emotional stress" is inappropriate; however, as a physical entity, "stress" may be considered as "physical tension", which, as described below, is associated with Raynaud's symptoms.

The action of physical tension in the elicitation of Raynaud's symptoms

The role of physical tension in the onset and maintenence of Raynaud's symptoms rests with the established role of the SNS in the control of peripheral blood flow. Physical tension is associated with an increase in sympathetic nervous activity; a reduction in physical tension with a decrease. Thus, an increase in physical tension - as commonly occurs in those exposed to cold ambient conditions (ie. when bracing onself against the cold) - may have some eliciting role in the onset of symptoms. Moreover, once a Raynaud's attack has started (whatever the trigger), the (unconscious) anticipation of the pain to be experienced during the hyperaemic phase, is likely to increase physical tension such that the effects of the trigger will be amplified, and so the attack prolonged.

A review of the role of "stress" in the elicitation of Raynaud's symptoms

As noted above, stress has become somewhat of an outmoded concept; however, the initial popularity of the idea has lead to a number of papers that manipulate stress variables in Raynaud's research. As such, this chapter would be incomplete without some mention of the five decades of work that attempt to associate stress and Raynaud's.

The earliest study to assess the role of emotion in the elicitation of Raynaud's attacks was that of Mittleman and Wolff (1939) in which they claimed to demonstrate that " ... *a low environmental temperature in itself was not sufficient to precipitate pain and cyanosis....whereas emotional stress under the same physical circumstances was followed by distressing symptoms*". (P291). Indeed, they recorded 200 instances of a drop in digital skin temperature under emotionally stressful situations (discussing difficulties in individual life situations, performing mental arithmetic, or reading horrifying literature) at both cool (20 C) and warm (26 C) ambient conditions. However, later studies eg. Freedman et al (1982), failed to replicate the findings.

Emotional stress and finger temperature in non-symptomatic volunteers

A number of papers have investigated the role of emotional stress on the finger skin temperature of non-symptomatic volunteers. Baker and Taylor (1954) reported skin temperature increases in 52 male volunteers during periods of emotional stress (induced by way of a jumping electric spark). In contrast, however, Boudewyns (1976) claimed that digital skin temperature increases from baseline during relaxation, but decreases during periods of stress (as induced by electric shock). Similarly, Crawford et al (1977) demonstrated that "cognitively induced anxiety" (through the discussion of anxiety provoking topics) induces a drop in digital temperature from baseline levels, whereas such decreases are not shown by those discussing pleasant topics or watching a neutral film. However, a range of methodological flaws (eg. failure to permit sufficient adaptation time to the environmental conditions prior to the start of the experiment, and failure to account for the effects of participant movement on digital temperature) somewhat undermine the results of the above studies.

Emotional stress and finger temperature in Primary and Secondary Raynaud's

In the 50's, Graham and co-workers claimed an association between decreased digital temperature and specific emotions (Grace and Graham, 1952; Graham, 1955; Graham et al, 1958). Hostility, anxiety and depression were induced (through hypnosis in the latter case) in non-symptomatic volunteers, and those suffering from disorders thought to be associated with stress (such as Raynaud's, eczema, and asthma). Seemingly, the periods of attitude induction correlated with a decrease in digital skin temperature. However, Peters and Stern (1971), using participants screened for hypnotic susceptibility, though noting the expected physiological changes during hypnosis, could establish no relationship between specific attitudes and those physiological changes. Further, Hugdahl et al (1984) failed to demonstrate vasoconstricting effects of mental stress (threatened electric shock) in Raynaud's patients, beyond those elicited by a cold ambient temperature of 0 C. This is despite such differences being demonstrated in non-symptomatic controls. However, one might assume some floor effect in the former case as Raynaud's patients vasoconstrict both faster and at higher temperatures than non-symptomatic volunteers. Consequently, any effect of stress would be masked by the effects of the ambient temperature during the experiment.

In contrast, Fagerstrom et al (1982) demonstrated a role of cognitive processing in the precipitation of vasospastic attacks. Raynaud's Disease patients, told that they would be exposed to cold ambient temperatures associated with the elicitation of Raynaud's attacks, failed to experience vasospasm on actual exposure; thereby suggesting that cold stress alone is insufficient to elicit vasospastic attacks - cognitive intervention of some description must be implicated. Freedman and Ianni (1983a) correlated subjective reports of stress ratings with finger temperature during an ambulatory monitoring exercise[10], revealing an association between vasospastic attacks and subjective reports of emotional stress in up to a third of the Raynaud's Disease volunteers. Moreover, Freedman and Ianni (1985a) further investigated the effects of "Raynaud's-specific stress" on symptoms. Raynaud's and non-symptomatic volunteers watched short films depicting either Raynaud's-specific stress (loss of keys and gloves in the snow), general stress, or no stress, and were then instructed to imagine briefly the scene portrayed in the

[10] Ambulatory monitoring: measuring physiological correlates (such as finger skin temperature) over an extended period, away from the laboratory situation (see Chapter 6).

film. Results suggested that temperature decreases occurred only in the Raynaud's volunteers and only in response to the Raynaud's specific stress. The authors suggest therefore, that Raynaud's patients have learned to vasoconstrict in response to conditions that they associate with a decrease in finger temperature - in brief, that vasospasms are a conditioned response.

Conflicting effects of the role of stress might be explained in terms of both individual differences in response to stressors, and the context of the session. Indeed, a number of papers underline this very point. Halperin, Cohen and Coffman (1983) claim that mental stress actually increases finger blood flow in female Raynaud's patients. However, Cooke et al (1990) suggests that male (non-symptomatic) volunteers vasodilate under cold and stress conditions; whereas, vasoconstriction is the response in female volunteers. Martinez et al (1992) claim that controls and Primary Raynaud's patients generally vasoconstrict in response to mental stress (mental arithmetic), yet Secondary Raynaud's volunteers vasodilate. Moreover, the work of Elam and Wallin (1987), and Oberle et al (1988) suggests mental stress (mental arithmetic or stressful conversation) causes vasoconstriction in warm volunteers (skin temperature approximately 30 C), and vasodilation in those who are cold (25 C or below).

Therefore, empirical research investigating the role of "stress" in the onset of Raynaud's symptoms is perhaps a redundant venture owing to the difficulties in defining stressors, and consequently, in accounting for the vast number of confounding variables circulating in the area of stress research. That said, as demonstrated in chapter 3, a proportion of Raynaud's volunteers continue to implicate a role of stress (under various guises) in the onset of both their symptoms and the condition as a whole.

2.5 HORMONAL AND CHEMICAL FACTORS AS TRIGGERS OF VASOSPASTIC ATTACKS

The action of hormones in the elicitation of vasospastic attacks

As previously noted, the control of peripheral blood flow is influenced by a number of circulating hormones such as norepinephrine, vasopressin and angiotensin (the latter two through involvement in

renal mechanisms); moreover, the sex hormones oestrogen and progesterone are also thought to influence peripheral blood flow. The main vasoconstrictive action of hormones is to increase the calcium ion concentration in the blood, and thus, stimulate contraction of the smooth muscular wall of the peripheral vessels; however, a slight decrease in hydrogen ion concentration will have a similar effect. In terms of the role of hormones in the elicitation of vasospastic attacks, one might assume a degree of hypersensitivity of the blood vessels of Raynaud's sufferers to the concentration of circulating calcium ions.

The action of other chemicals in the elicitation of vasospastic attacks

A range of chemicals have been associated with the precipitation of Raynaud's symptoms. As described in chapter 1, nitrates, vinyl chloride, ergot, and birth control pills are related to the Secondary disorder. Moreover, chemicals such as nicotine and carbon monoxide aggravate symptoms; indeed, Raynaud's sufferers are advised not to smoke.

Chemical action is mediated through arterial and cardiopulmonary chemoreceptors located in the carotid and aortic bodies, and the heart and lungs respectively. The mechanisms by which chemoreceptors are influenced by levels of circulating chemicals in the blood appears to be poorly understood; however, it is known that the arterial chemoreceptors are sensitive to changes in PH and blood gases; whereas the cardiopulmonary chemoreceptors respond to nicotine and other chemicals. Again, one must assume oversensitivity to the effects of environmental chemicals in the case of the Raynaud's sufferer.

Evidence of the effects of circulating hormones and chemicals in the onset of symptoms

The effects of circulating hormones such as norepinephrine, angiotensin and vasopressin have already been touched on above; therefore, evidence relating Raynaud's symptoms with hormonal action will be

confined to the sex hormones. Similarly, given the large range of chemicals that might influence peripheral blood flow, the overview of chemical effects will be restricted to that of cigarette smoking.

Jarrett (1976) reports a decrease in Raynaud's symptoms in three cases following the cessation of the combined contraceptive pill. Further, in one case, symptoms improved on switching to the mini pill (progesterone only); thereby implicating oestrogen in the elicitation of symptoms. Moreover, the role of oestrogen was further supported by Lafferty et al (1985) who reported equivalent finger blood flow in Raynaud's patients as in 10 pre-ovulatory (late post menstruum) non-symptomatic volunteers. Subjective evidence of a correlation between oestrogen levels and Raynaud's attacks is less easily attained. Indeed, Bartelink et al (1992) measured symptoms in relation to the menstrual cycle in a large scale retrospective questionnaire supplemented by a smaller, 3 month symptom diary exercise (frequency, severity and duration of symptoms). Less than 20% of the respondents associated their symptoms with the menstrual cycle, and attacks were shown to last longer during the pre-menstruum and menstruation ie. during the periods associated with low levels of both oestrogen and progesterone. However, it appears that for analysis purposes, cycle duration was assumed to be uniform (ie. ovulation was assumed to occur on day 14 in volunteers whose menstrual cycle ranged between 26 and 32 days). Therefore, the study can do little to dispute the role of oestrogen in the elicitation of Raynaud's attacks.

The role of chemicals in the precipitation of Raynaud's attacks are seemingly less vehemently disputed. Smoking is particularly frowned upon in sufferers of Raynaud's symptoms. However, Goodfield et al's (1990) investigation of the effects of cigarette smoking in Raynaud's and non-symptomatic volunteers indicated that the smoking of a single cigarette was associated with a reduction in finger blood flow only in regular smokers - whether the smoker suffers from Raynaud's or not. That said, the study does imply a relationship between regular smoking and finger blood flow. Indeed, Bocanegra et al (1980) report a case of an association between Raynaud's symptoms and passive smoking, and Coffman (1967) claims that the passive inhalation of nicotine decreases cutaneous blood flow through the stimulation of the SNS. Consequently, Raynaud's sufferers would be advised to avoid both smoking and, where possible, those who smoke.

2.6 THE ROLE OF VASOSPASTIC TRIGGERS IN THE INITIAL ONSET OF RAYNAUD'S

As a number of precipitators have been shown to be associated with Raynaud's symptoms, it surely follows that such precipitators might be involved in the initial onset of Raynaud's as a whole (ie. rather than just in the onset of isolated Raynaud's attacks). However, given the necessary retrospective and subjective nature of such an avenue of investigation, a causal relationship between symptom onset and specific triggers would be impossible to prove. That said, anecdotal and subjective report does associate certain precipitators with the initial onset of symptoms: the effects of cold injury, vibration (Vibration White Finger) and chemical factors have been clearly documented in relation to Secondary Raynaud's; general anaesthetic and pregnancy occasionally occur prior to initial symptoms, although pregnancy may also be associated with symptom improvement (both Bartelink et al, 1992); and anecdotal evidence links trauma with the onset of symptoms.

The next chapter addresses the question of precipitators in more detail through the analysis of a subjective account of Raynaud's symptoms and associated factors.

Chapter 3 SUBJECTIVE ACCOUNTS OF RAYNAUD'S

3.1 INTRODUCTION

Precipitators other than cold may be implicated in the onset of Raynaud's, and in the elicitation of individual attacks. However, few articles provide a clear subjective account of the triggers of Raynaud's symptoms, of the nature of those symptoms, nor of how they might shape the life of the sufferer. The purpose of this chapter therefore, is to build up a picture of a typical Raynaud's sufferer - including symptom details, age of onset and diagnosis, the initial and general triggers of attacks, and how, if at all, Raynaud's symptoms impose on the life of the sufferer. Pilot work involved a small number of semi-structured interviews. On the basis of the interviews, a questionnaire was constructed to assess the role that Raynaud's plays in the life of the sufferer.

3.2 PILOT WORK: SEMI-STRUCTURED INTERVIEWS

Method

Participants

10 Raynaud's patients, randomly chosen from a list of 111 referred to Southampton General Hospital since 1985, were approached by letter with regard to interest in discussing their experience of Raynaud's. Four agreed (2 male, 2 female). Interviews were conducted in September 1992.

Design

A semi-structured interview. The list of questions used to guide the interviewer may be found in Appendix 1.1.

Procedure

The interviewees were offered a choice of location for the interview: the Department of Psychology (University of Southampton); their home; or their own place of work. Three interviews were conducted in the Department of Psychology, and one at the volunteer's place of work. With the consent of the participants, each interview was recorded on audio tape, and later transcribed for analysis. The interview transcripts may be found in Appendix 1.2.

Results

Given the small number of participants, in depth analysis of the interview data was inappropriate; however, a brief content analysis was performed from which 4 main categories of interest emerged: namely, symptoms, details of the onset of the condition and of Raynaud's attacks, the effects of symptoms, and the treatments offered to and used by the sufferer. Demographic details are presented in table 3.1, and each of the emergent categories discussed below.

Participant	HC	JM	AP	RP
Diagnosis	Raynaud's Phenomenon	Raynaud's Phenomenon: Scleroderma	Unsure[11]	Raynaud's Disease
Gender	Female	Female	Male	Male
Age (1992)	41 years	24 years	43 years	66 years
Occupation	Service industry	Sales	Office worker	Retired
Marital status	Married	single	Married	Married
Children	Yes	No	Yes	Yes
Smoker ?	No	No	No	No

Table 3.1: Demographic details of the interviewees

1. Symptoms

As the reader will recall, the textbook description of Raynaud's principally involves three bilateral colour changes of the skin of the hands and feet (with or without involvement in other areas).

[11] The unsure category is included in line with the previously mentioned confusion in terms of diagnostic terminology.

Generally, this description correlated well with subjective report; however, one Raynaud's Phenomenon participant described symptom involvement only on the right hand side of the body; moreover, she described feelings of nausea and fatigue during her attacks. As to the sensation of Raynaud's attacks, descriptions included "pain", "very much like a burning sensation", "constant pain and numbness", and "dead hands". Further, symptom descriptions include reported inability to straighten the fingers owing to the tightness of the skin, the mention of general body temperature fluctuations ('freezing cold' one minute, and sweating the next), and the presence, in some sufferers of continuous symptoms rather than discrete Raynaud's "attacks"; indeed, symptoms were described as lasting "hours, days or weeks...", or "until warm".

2. Onset of the condition and individual attacks

It is claimed that symptom onset generally occurs before the fortieth birthday, or in late(r) life. Again, this sits well with the interview data in that the onset ages of the two Raynaud's Phenomenon and the Raynaud's Disease participants were 23, 35 and 52 years respectively. However, the undiagnosed participant described symptom onset in childhood. This is generally considered rare unless the symptoms experienced are disguising some underlying condition.

Retrospective information as to what preceded the initial occurrence of symptoms was mentioned by only the Raynaud's Phenomenon participants. In one case, Raynaud's symptoms followed a whip lash injury sustained in a car accident, and in the other, symptoms were associated with the sudden appearance of a frost bite type infection during a period of extreme emotional stress: ".. *maybe triggered by an emotional thing because it was almost exactly to the day that I split up with my boyfriend after two years. I was in a very very bad emotional condition. Extremely distressed, and that is when it happened. Almost exactly.*" (JM). Thus, both physical damage to the nervous system and emotional trauma might be implicated in the onset of the condition. As to the precipitators of individual Raynaud's attacks, where details were given, a range of triggers were mentioned, including cold and temperature change, drafts, aerobic exercise, pressure on the affected area, and 'stress'. Interestingly, a common source of 'stress' appears to be that of contact with the medical profession: "*Any sort of emotion, it will go..um, whenever I go and see the specialist, and I am het up...*" (JM);

"Its the same if I have to go for a medical examination. As soon as I get home, the stress of it all brings on an attack." (HC). Moreover, a common theme throughout the interviews involved doctors' failure to both understand the degree of discomfort suffered, and to provide answers: [the doctors] *"can do nothing. I just have to cope with it best I can."* (HC).

3. Effects of symptoms

Questions addressing the effects of symptoms uncovered three main areas - namely practical, social and psychological effects. On the practical side, symptoms intermittently interfered with participants' ability to use their hands so that hobbies (eg. knitting, dress making and baking) and full time employment were difficult to maintain. Moreover, indirect practical effects of Raynaud's symptoms include the unpleasantness and inconvenience of regular hospital tests[12].

Socially, effects may be directly associated with symptoms, or more indirectly with the psychological effects of those symptoms. In the former case, the need to be continually warm may control the life of the sufferer:*"I am frightened to go anywhere in winter....If I go to a relation's or friend's for a meal, I make discreet enquiries of how warm the house is before I dare to go there."* (AP). Further, it may create problems in the work environment: *"I work in an extremely cold environment, and its extremely noticeable then. ..... Really, I should change jobs."* (JM). The need to be continually warm becomes both a physical and psychological need: *"My main aim in life is to find some way of keeping my feet warm. I can then attempt to do other things."* (AP). Further, in severe cases, where symptoms make a huge impact on the sufferers' day to day existence, loss of confidence may occur such that social interactions may be avoided: *"I feel I've spoilt so many evenings, I don't go out much now."* (HC).

4. Treatments (home and prescribed)

The medical treatments offered to the Raynaud's sufferers ranged from advice to keep warm (in all cases), to drug treatments such as nifedipine (see chapter 4), and to surgical intervention. Moreover,

[12] Although adequate treatment may not be forthcoming, Raynaud's patients are monitored regularly to map the progression of their illness.

in one case, a stress management approach had been suggested (though not taken up). Generally, medical treatments were claimed to produce only short term benefits and/or intolerable side effects.

Methods of dealing with individual "attacks" mainly involved warming the affected area. The use of heaters, gloves, hot water, and sitting on the hands were mentioned (though the inconvenience of the battery pack for heated gloves was such as to prevent their use); however, it was noted that in some instances, no method of warming would curtail attacks. Relaxation techniques such as relaxation tapes and involvement with a self-help chronic pain group were claimed to improve the condition in terms of ability to cope; however, acupuncture and the use of a TENS[13] machine appeared to worsen symptoms.

Concluding comments

It should be noted at this point, that the interview location appeared to have no noticeable effect on the nature of the data obtained; indeed, all interviewees described symptom and onset details, effects of their symptoms, and personal techniques of dealing with those symptoms whether in a novel or familiar environment. (See Appendix 1.2).

In summary, therefore, the interview data reveals that the symptoms, and the effects of those symptoms, are wider in range than a text book description might suggest. In severe cases, Raynaud's is debilitating, and may prevent the sufferers' involvement in previously valued activities; indeed, if allowed, it may control the life of the sufferer. However, one must note that the interview sample was small, and moreover, that the richest data was obtained from the sufferers of Raynaud's Phenomenon. A larger scale postal questionnaire was therefore utilised to obtain a more representative picture of the symptoms of Raynaud's. This dealt only with Raynaud's symptoms, the effects of symptoms, and the onset of both symptoms and the condition as a whole as medical and behavioural treatments for the condition will be discussed in later chapters.

[13] TENS machine: Transcutaneous Nerve Stimulation (see chapter 6).

3.3 THE RAYNAUD'S QUESTIONNAIRE

Method

Participants

One hundred and eleven Raynaud's patients who had been referred to Southampton General Hospital since 1985 were approached by letter with the opportunity of taking part in a study of the effects of behavioural treatments for Raynaud's symptoms. They were informed that the study would involve the completion of a questionnaire, and an invitation to participate in a subsequent treatment programme[14]. 85 people expressed an interest, and of these, 61 completed and returned the questionnaire. Approximately a year later, 18 further questionnaires were completed by participants replying to a local newspaper advertisement recruiting for the main treatment study. Overall, 79 people completed the questionnaire: 28% were diagnosed as Raynaud's Disease patients; 23% as Raynaud's Phenomenon; 17% were unsure of their diagnosis; and 32% were undiagnosed. Table 3.2 details the gender, marital status, occupation and approximate ages of the respondents.

Diagnosis	**Raynaud's Disease**	**Raynaud's Phenomenon**	**Unsure**	**Undiagnosed**
Median Age	50 years +	50 years +	50 years +	31-50 years
Gender	19 F; 3 M	16 F; 2 M	10 F; 3 M	20 F; 5 M
Marital status	17 partner; 5 single	14 partner; 4 single	8 partner; 5 single	18 partner; 7 single
Occupation	14 working; 2 student; 4 housewife; 3 retired	7 working; 1 student; 5 housewife; 3 retired; 2 disabled	4 working; 2 housewife; 6 retired; 1 unemployed	13 working; 4 student; 1 housewife; 6 retired; 1 unemployed

Table 3.2: Demographic details of the questionnaire respondents

[14] See chapter 8.

Design

An exploratory postal questionnaire designed to address a) reports of Raynaud's symptoms, b) subjective trigger(s) of individual "attacks" and precipitators of the condition as a whole, and c) the effects of symptoms. The questionnaire may be found in Appendix 1.3.

Procedure

85 questionnaires were posted in February 1992 to those who had expressed an interest in the behavioural study. Of these, 61 were returned and coded in line with the coding frame set out in Appendix 1.4. A further 18 questionnaires were coded in November 1993.

Results

Data was cross-tabulated to allow Chi-square comparison initially across the four diagnostic groups [sic], and secondly, across the 40 participants (50.1%) known to be diagnosed as sufferers of either Raynaud's Disease or Phenomenon. Participants were free to not respond to questions if they so wished. Consequently, the number of respondents to each question varies. Please refer to Statistical Appendix 1.

a) Symptoms

Three categories were used in the analysis of areas of symptom involvement in the body: i) FHF: Fingers, hands and feet; ii) FHFE: fingers, hands, feet and extremity involvement (eg. nose, ears, tongue, lips); and iii) FHFEIO: the added involvement of internal organs. In line with expectation, analysis revealed significant differences in site of symptom manifestation across the four groups: Chi-square (df 6) = 13.37 $p < 0.05$ (Statistical appendix 1, table 1.a, page S1). As is evident from table 3.3, the symptoms of the Primary and undiagnosed participants were confined to the extremities (hands, feet, nose, ears...) whereas those in the Secondary and Unsure categories mentioned involvement of the internal organs such as the oesophagus, and lungs. However, there were no statistically significant differences between the Raynaud's Disease and Phenomenon participants.

Areas of symptom involvement	FHF	FHFE	FHFIO
Disease	18	4	0
Phenomenon	9	7	2
Unsure	12	0	1
Undiagnosed	21	4	0

Table 3.3: The areas of symptom manifestation across the 4 "diagnostic" groups (n =78)

For 80.5% of respondents, the site of symptom manifestation had not changed since the onset of the condition; however, the symptoms of 16.9% had spread to surrounding areas; whereas, in 2.6% of respondents, symptoms had diminished. The role of medication in the latter case was not assessed.

The reported skin colour changes did not differ significantly across the four groups. However, patterns did occur in the data. Most of the undiagnosed respondents (56.5%) described a single colour change - predominantly associated with blanching of the skin. The Phenomenon respondents tended to report the characteristic vasospastic attack (44.4%), rather than two colours (26.8%), or a single colour change (28.8%). The sufferers of Raynaud's Disease were inclined to report two colour changes in the skin during an attack (47.6%) with only 28.6% and 23.8% respectively describing the characteristic vasospastic attack or a single colour change.

In agreement with the earlier interview data, 20.5% of respondents felt the term "attacks" inappropriate as their symptoms were continuous. Moreover, 37.2% were unable to attach a time scale to their attacks as symptom duration is determined by the time taken to warm the affected area. The remaining participants reported symptom duration ranging from 10 minutes to 2 hours; however, given the 'until warm' response above, one wonders to what extent the latter periods were arbitrarily chosen to allow appropriate completion of the questionnaire.

As to reported symptom frequency, no statistically significant differences were uncovered across the four groups, nor between the two Raynaud's groups. However, as one might expect, overall symptom frequency was greater in winter than in summer: 26% do not experience symptoms in the summer,

64% report 1-4 attacks per day, and only 10% experience 5 or more attacks daily; in contrast, 3.8% do not experience attacks in the winter[15], 48.1% experience 1-4 daily attacks, and 48.1% report five or more attacks per day during the winter.

An area not touched upon in the analysis of the interview data, though mentioned in the previous chapter, is that of the occurrence of Raynaud's symptoms in the immediate relatives of respondents. Overall, 13.7% reported relatives medically diagnosed as suffering from Raynaud's; however, this percentage increases to 46.6% when the qualifier of 'medically diagnosed' is lifted. As the reader may recall, there is no conclusive evidence of Raynaud's as an inherited condition; and indeed, the latter, rather high incidence of undiagnosed familial Raynaud's reported here may merely be indicative of cold sensitivity. In contrast, 13.7% claim to be related to Raynaud's sufferers. This must support the need for further epidemiological research into Raynaud's as an inherited condition.

b) The onset of the condition and of individual "attacks"

Onset age	Childhood	17-40 years	40 years +
Raynaud's Disease	10	10	2
Ray' Phenomenon	1	11	6
Unsure	6	4	2
Undiagnosed	15	9	1

Table 3.4: The age of symptom onset across the diagnostic groups (n = 77)

As suggested by table 3.4, the age of Raynaud's symptom onset differed significantly across the diagnostic groups: Chi-square (df 6) = 16.71 $p < 0.02$ (Statistical Appendix 1, table1.b, page S1): symptom onset was, on the whole, later for the Raynaud's Phenomenon participants than the other groups. This finding sits well with the textbook description of Raynaud's in that 44.2% of the respondents' initial symptoms occurred in adulthood, prior to the fortieth birthday; however, the reported onset age differs from expectation in that 41.6% claim to have experienced their first symptoms in childhood. Perhaps, childhood onset is not as rare as first imagined; alternatively, the

[15] owing to a combination of medication, change in life style and mild symptoms

high incidence of childhood onset might be a function of an inability to recall the precise moment of symptom onset.

One might expect medical advice to have been sought most rapidly in those with the most severe symptoms, and perhaps where symptoms initially occurred later in life. Indeed, the Raynaud's Phenomenon participants tended to seek medical advice within the first year of symptom onset; whereas this was not the case for the other participants: Chi-square (df 2) = 11.68 $p < 0.01$ (Statistical Appendix 1, table 1.c, page S1).

Information regarding the events preceding the initial Raynaud's symptoms was available for all 79 respondents; however, 63 could recall no particular event that "sparked off" their symptoms (onset occurred during childhood for 32 of these respondents). As one might expect, therefore, given the low number of remaining responses, chi-square analysis revealed no group differences in conditions associated with initial symptoms. 16 respondents noted that their symptoms coincided with particular events in their life. Pregnancy was mentioned by 6, puberty by 1, injury or illness by 6, and 'stress' by three participants:"*..it was at this time that Raynaud's first showed and was diagnosed. This was a period of great stress and anxiety*" (JM). Consequently, there may be grounds to assume some action of hormonal factors in the onset of the condition.

As to the precipitators of individual attacks, analysis revealed no statistically significant differences across the diagnostic groups. All of the respondents mentioned cold temperatures; however, 50% mentioned other precipitators in addition to the effects of cold. These included pressure on the fingers or affected area, dampness, and 'stress'. Situations classed as stressful by the participants included social interaction, relationship problems, lack of control of a situation, and interestingly, worry that the ambient conditions may be such as to elicit an attack of Raynaud's. Indeed, overall, 33.3% (26 of the 78 respondents who expressed an opinion) claimed that stressful situations often triggered their symptoms: "*I believe very strongly that stress can sometimes affect the variation in strength of Raynaud's and Scleroderma. ..... I have suffered two very stressful periods and both times have had a physical reaction.*" (GM); "*My Raynaud's is much more obvious at times of stress - my*

fingers blacken dramatically when I am anxious or upset." (JM); "*Many stressful situations have sparked off my Raynaud's. ...as soon as I start to worry about things, I tend to lose my fingers. My top lip is another giveaway - that goes blue when I am stressed.* " (SW).

However, cold was seen as the most common precipitator by 87% of the respondents; 7.8% gave equal weighting to cold in conjunction with other precipitators such as dampness, pressure and stress; and 5.2% (a Raynaud's Phenomenon respondent) felt that stress was the most common trigger of symptoms. Generally, factors involved in the elicitation of attacks had not changed since the onset of the condition (82.4% ie. 61 of the 74 who responded to the question). Furthermore, 13.5% (10) were unsure about any such change. Indeed, only 4.1% (3) of the sample felt that other factors (physical pressure and emotional stress) had taken on a precipitating role as their condition had progressed.

A related point is that of participant awareness of the onset of an attack. 88.6% were aware that an attack had occurred only on experience of the characteristic symptoms. Similarly, 4.9% could predict the occurrence of attacks only from prevailing cold ambient conditions. This suggests that expectation might play a role in the onset of symptoms ie. anticipation of a combination of cold and the related attack is likely to increase physical tension in the sufferer, and thus heighten the sympathetic nervous response to the cold. The remaining 6.5%, in contrast, felt able to predict an attack through the occurrence of a particular undescribable bodily "*sensation*".

c) The subjective effects of Raynaud's symptoms

Two questions addressed the subjective effects of symptoms: initially the broad "What are the effects of Raynaud's in your life ?"; and secondly the more focused query as to the effects of Raynaud's on occupation. 32 participants (40.5%) did not respond to the initial question, and 41 (52%) repeated their earlier symptom description. One is therefore lead to believe that failure to respond is a function of the ambiguous nature of the question: the participants assumed that a further description of the symptoms was required. Of the remaining 6 participants, 2 mentioned social effects such as confinement to the home during particularly cold patches, 3 described the practical effects such as inability to use the hands during vasospasm "*In cold weather, after cycling home, it takes ages to*

unlock the front door.." (GB), and one noted that owing to her condition, she was unable to continue in her previous line of work. Interestingly, the question as to the effects of symptoms on occupation revealed that only 8.5% (6 of the 71 respondents) had been forced to change their occupation because of their Raynaud's symptoms (the majority being sufferers of Raynaud's Phenomenon). This suggests that in general, the physical characteristics of a vasospastic attack do not interfere with the working life of the sufferer.

Conclusions

From the initial interview data, four areas of interest emerged: symptom details; precipitators of both the condition and of individual attacks; the role of symptoms in the life of the sufferer; and the home and medical treatments employed. The former categories were addressed in the questionnaire study; the latter category will be discussed in the subsequent chapters.

As to the interview and questionnaire data, symptom descriptions generally suggested that the sample were characteristic of Raynaud's patients as a whole (area of symptom manifestation according to diagnostic group; attack frequency according to ambient conditions etc.). However, both interview and questionnaire data underline the heterogenous nature of Raynaud's ie. the report of symptoms being confined to one side of the body; the apparent lack of discrete Raynaud's attacks in some sufferers; and the suggestion that childhood onset is less rare than textbook descriptions might imply.

However, there are a number of flaws with both the design and analysis of the data. Firstly, the small size of the data set in conjunction with a relatively large number of categories on some questions, is consistent with type II errors. This means that statistically significant results may be implied where differences are no more than trends. However, as only three statistically significant differences are presented, and the differences, in the main, sit well with the published literature, the flaw, though worth considering, may be overlooked. That said, the study fails to provide more than limited anecdotal and retrospective evidence of a link between particular events (injury, hormonal changes,

trauma) and the onset of symptoms owing, perhaps in part, to the ambiguous nature of specific questions. It may be suggested that the design of the questionnaire both constrained responses, and to a degree, misled the respondents. However, the questionnaire did reveal a number of interesting points including the role of anticipation in the onset of individual attacks. Moreover, the results indicate that preventative treatments might be inappropriate as sufferers are only aware of an attack once it has commenced. In contrast, treatments or methods of coping that could be put into action once an attack has been triggered might prove useful. As such, a preventative behavioural approach is likely to be limited; the behavioural management of symptoms, on the other hand, may be a possibility.

The subsequent chapters will address the question of treatment; indeed, in the next chapter, currently available medical treatments will be discussed as a basis to the introduction of the behavioural approach.

Chapter 4

MEDICAL TREATMENTS

4.1 INTRODUCTION

Although there is currently no cure for the symptoms of Raynaud's Disease and Phenomenon, a range of palliative procedures are generally on offer to the sufferer. These include both surgical and medicinal approaches; moreover, particularly where secondary conditions are identified, symptom progression is carefully monitored through regular hospital tests - including tests of internal organ involvement, lung capacity etc. At risk of labouring the point, it should be underlined that, at best, current treatments can only hope to control, as opposed to cure, the symptoms of Raynaud's.

The aims of this chapter are to describe the avenues available to the medical profession for the treatment of Raynaud's. Clearly, the course of action depends to a certain degree on diagnosis ie. where secondary conditions are identified, the associated conditions will be treated in preference to, or in tandem with the primary symptoms. For example, Methotrexate may be prescribed to a patient presenting with Raynaud's Phenomenon associated with Arthritis. Given the range of associated secondary disorders, this chapter will focus predominantly on the management of primary Raynaud's symptoms; however, the chapter would be incomplete without at least a brief overview of some of the treatments specific to Raynaud's Phenomenon.

Within the current medical approach, there are three broad areas for discussion: the general practitioners' initial advice on day to day coping with the condition; surgical intervention; and lastly, pharmacological treatments. These will be addressed in turn below.

4.2 MEDICAL ADVICE

The initial advice that patients receive on diagnosis is to keep warm, and where applicable, to give up smoking. In brief, people are advised to avoid the situations that might trigger symptoms. In line with this, the Raynaud's and Scleroderma Association suggest a range of sensible methods for keeping warm[16], including advice on clothing to maintain the core temperature of the body (several thin layers to trap air and insulate the body), and guidance as to the availability of a range of chemical and electrical hand, foot and body warmers. Moreover, where Raynaud's Phenomenon is diagnosed, patients are advised to take particular care of affected areas. For instance, characteristic dryness and reduced motility of the mouth in Scleroderma may be associated with dental decay, fungal infection, and inflammation of the gums. Indeed, a number of over the counter remedies are available for such afflictions. Patients are further advised to avoid substances known to be associated with cold sensitivity. For example, beta blockers, amphetamines, cocaine, caffeine, diuretics and the combined contraceptive pill; in some cases hormone replacement therapy is not advised. In contrast, vasodilating substances such as alcohol (in moderation) may be suggested.

Alternative "home treatments" include swinging the arms rapidly around in a circular motion; thereby, forcing blood out toward the fingers, the introduction of dietary therapy, and finally, direct hand warming through regular careful immersion in warm water or wax.

Although conclusive evidence of a link between diet and Raynaud's symptoms is limited, dietary therapy aims to reduce symptoms through dietary supplements (eg. fish oil or evening primrose oil) and/or through the elimination of specific associated foods or food groups from the diet - the foods to be removed from the diet being specific to the patient. Dietary supplements have been investigated in the treatment of Raynaud's; indeed, DiGiacomo et al (1989) reported objectively measured positive effects of fish oil supplements in Raynaud's Disease. Moreover, subjective effects of Idoloba (a supplement containing Ginkgo biloba leaf extract) are currently under investigation.

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[16] For example, in *"Raynaud's: a guide for health professionals."* OS Roath (ed): Chapman and Hall, 1989.

As to hand warming, Goodfield and Rowell (1988) report positive effects: 12 Raynaud's Phenomenon patients warmed their hands for 5 minute periods at 4 hourly intervals on alternate weeks over a 6 week period. Subjective effects included a reduction in group mean attack frequency of 18% and improved skin texture. Objective blood flow measures were performed; however, all measurements (pre- and post-treatment) were taken in the laboratory at ambient temperatures of 28 C. Personal experience (see chapter 8) leads me to suggest that such temperatures are not consistent with low blood flow in even Raynaud's patients; indeed, increased sweating occurs at ambient temperatures of 24 C (Guyton, 1976).

Several methods are available for use in the "home management" of Raynaud's symptoms. For many with mild Raynaud's, such self-help methods are all that are required; however, in others, these measures do little to confront the inherent pain and discomfort such that drug or surgical intervention may be required.

4.3 SURGICAL PROCEDURES

A number of surgical procedures are on offer to Raynaud's sufferers. The most common surgical intervention is that of cervical or lumbar sympathectomy; however, the removal of calcium deposits in the fingers is advocated in the management of certain secondary disorders such as CREST syndrome.

Surgical sympathectomy was a popular Raynaud's treatment in the 1950's and 60's. Based on the assumption of a heightened sympathetic nervous reaction to cold in Raynaud's sufferers, sympathetic innervation to the hand (cervical sympathectomy) or foot (lumbar sympathectomy) is interrupted. In more detail, the pre-ganglionic fibres of the terminal sympathetic branches are isolated, the outer layers stripped and the exposed communications between the arteriole and the nerve are divided. As previously mentioned, the peripheral blood vessels in the skin are extremely sensitive to the sympathetic neurotransmitters norepinephrine and epinephrine. Consequently, even if sympathetic innervation to a particular set of vessels is physically removed, norepinephrine and epinephrine released from the adrenal medulla on sympathetic excitation may still elicit Raynaud's attacks. As such, post ganglionic fibres are not removed in order to prevent blood vessels becoming excessively sensitised to these

circulating catecholamines. In spite of this, the effects of cervical sympathectomy (warm hands, improved skin condition) are short lived[17]. Indeed, Baddely (1965) followed up 36 primary Raynaud's patients who had undergone upper-dorsal sympathectomies between 1950 and 1957. Within a period of at least 6 years, 20.3% of patients remained symptom free, 34.4% who had shown an initial improvement in symptoms, relapsed during the first spell of cold following treatment, and the symptoms of the remaining 45.3% actually deteriorated following surgery. Further, Trafford's (1985) subjective questionnaire based evaluation of sympathectomy reports a rather disappointing 81.4% (of 571 respondents) failure rate of cervical sympathectomy. It should come as no surprise to the reader therefore, that cervical sympathectomy is generally no longer used for the treatment of Raynaud's Disease (Roath, 1989). That said, more recent work by Francaviglia et al (1994) appears to support the use of spinal cord stimulation (SCS) as a means to decrease the pain, ulcers and Raynaud's attacks in Raynaud's Phenomenon associated with progressive Systemic Scleroderma: under general anaesthetic, 15 patients were implanted with electrodes (in the cervical epidural space) to stimulate the spinal cord with up to 4v of current on a short cyclical basis. Long term follow-up suggested clinical improvement (in frequency, intensity and duration of attacks) in 93% of the sample.

The disorders of Raynaud's Phenomenon often require surgery. For example, in Carpel Tunnel Syndrome, trapped nerve(s) in the wrist may require surgery for their "release". Further, as mentioned above, where infection occurs in the calcium deposits associated with CREST syndrome, under general anaesthetic, a dental drill is used to remove the calcinotic nodules in the fingers. Where possible, skin is preserved; however, in many cases, skin grafts are required.

Surgical intervention, though useful in some instances of Raynaud's Phenomenon, is generally not employed in the treatment of Raynaud's Disease. However, a range of pharmacological treatments, offering at least some respite from the condition, are available.

[17] In contrast, the prognosis for lumbar sympathectomy in Raynaud's treatment is good.

4.4 PHARMACOLOGICAL TREATMENTS

In the late 1960's and early 70's, given the limited long term benefits of surgical sympathectomy, and the associated risk of permanent neurological deficit, chemical sympathectomies came into use. Reserpine and Guanethidine were used to mirror the effects of sympathectomy through their action as neurotransmitter depleters. However, both have been replaced by alternative treatments (discussed below) owing to a range of side effects and the short term benefits of treatment. For example, intra-arterial reserpine is associated with arterial damage, breast cancer[18] and depression; guanethidine with diarrhoea and impotence.

In later years, a variety of pharmacological remedies including vasodilators, vasoactive agents, prostaglandins, and rheologically active drugs have been made available for the treatment of Raynaud's disorders. As the purpose of this thesis is an investigation of the behavioural management of symptoms, the above treatments will not be reviewed in depth; however, the (supposed) action of the type of pharmacological treatment, examples of the drug or therapy, and the associated side effects will be outlined.

A number of pharmacological agents may be classified as vasodilators. For example, serotonin antagonists (eg. ketanserin), and calcium channel blockers such as Nifedipine. The latter, reduces the frequency and severity of vasospasm (Waller et al, 1986) and decreases vascular resistance in the finger tip (Winston et al, 1983) through its action in the prevention of vessel smooth muscle contraction. It is however, ill-suited in the treatment of all Raynaud's patients as many will experience side effects such as hypotension, swelling of the ankles, dizziness, palpitations, headaches and flushing that out weigh the benefits of treatment. However, side effects may be transient and dosage dependant.

[18] Report from the Boston Collaborative Drug Surveillance program: reserpine and breast cancer. Lancet 2 669-71, 1974.

Other vasoactive agents such as the alpha-adrenergic receptor antagonists prazosin and methyldopa prevent vasoconstriction through their action as false neurotransmitters ie. blocking the action of norepinephrine on the post-synaptic alpha-adrenoceptors. Indeed, Wollersheim et al (1986) revealed a reduction in the frequency and duration, though not severity, of vasospastic attacks in a double-blind, placebo-controlled trial of prazosin. As with vasodilators, side effects, including postural hypotension and dizziness, are common.

Parenteral treatment is used exclusively for the disorders of Raynaud's Phenomenon. For example, infusion of prostaglandins such as Iloprost, thought to inhibit platelet mechanisms and/or endothelial-promoted mechanisms, are used where digital integrity is threatened. Infusion occurs over a three or five day period (requiring a hospital stay) during which vasodilatory side effects such as headache, flushing and nausea tend to be experienced. Here again, however, side effects are dose related and diminish once infusion is complete. The positive effects (including increased blood flow, and decreased severity, frequency and duration of Raynaud's attacks) associated with Iloprost infusion last anything from 6 weeks to 6 months (Dowd et al, 1982). As treatment is repeated at six monthly intervals, Iloprost infusion is expensive. As such, trials are currently being run on the efficacy of Cicaprost, a synthetic prostaglandin that may be administered orally (eg. Lau et al, 1993).

Alternative treatments include the use of drugs that alter the consistency of blood to improve flow. For example, aspirin and Dipyridamole achieve this through the inhibition of platelet aggregation, and the rheologically active drug Pentoxifylline through the increase of red blood cell deformability. Moreover, Moriau et al (1993) describe a series of studies in which Piracetam is shown to be effective in the treatment of Raynaud's through its action of platelet function inhibition and rheological effect of reduction in blood viscosity. Further, the authors suggest that the treatment is not associated with adverse side effects.

Although currently available medical treatments offer a range of benefits to the Raynaud's sufferer, for many sufferers, side effects and the need for lifelong dependence on drugs is not acceptable. There is, therefore a move towards the behavioural approach discussed in chapter 6 and subsequent chapters.

However, before going on to review the published literature describing the behavioural approach to treatment, the reader's understanding would greatly benefit from a description of the common methodological techniques adopted in assessing the effects of such treatments.

Chapter 5

METHODOLOGY

5.1 INTRODUCTION

The main purpose of this chapter is to introduce and briefly describe a range of subjective and objective measures used in the assessment of behavioural and pharmacological treatments of Raynaud's. In particular, the chapter will focus on techniques employed in the main empirical study of this thesis (as described in chapter 8).

Behavioural and medical approaches differ in that greater involvement and commitment to treatment is required of those adopting the behavioural approach. Where traditionally treated patients take drugs as and when prescribed, those involved with behavioural treatments need to invest more time and energy in practising techniques of temperature control, and in attending training sessions. The latter part of this chapter will therefore address the issue of compliance in the behavioural approach to treatment (although the points raised are, of course, equally applicable to the medical approach).

5.2 SUBJECTIVE MEASURES OF THE EFFECTS OF TREATMENT

When the behavioural approach was initially introduced, treatment effects were exclusively assessed through subjective report; indeed, a range of techniques is available to obtain such information from patients. The most common, and perhaps easiest method of obtaining self-report data, is simply to ask participants their views as to the effects of the treatment(s) that they have received. However, such a technique is open to response bias on the part of the participant: behavioural treatments may involve 10 or 12 meetings of up to an hour in duration with a therapist; volunteers may therefore find it difficult, or even inappropriate, to state that their symptoms have not improved given the time invested by both themselves and the clinician. Moreover, during the course of treatment, the clinician and trainee may

build up some relationship akin to friendship which will further undermine the likelihood of a negative description of the effects of treatment.

An alternative approach is to obtain subjective information in questionnaire format, or indirectly through asking participants to keep a diary of their symptoms both before, during and, for a period after, attending a behavioural treatment programme. The latter technique is not however, problem free: though theoretically simple to maintain an accurate diary record of symptoms over a short period of time, the period required in any adequate study may be up to a year in duration; clearly, participants' motivation levels will not be constant throughout the required period. It is further suggested that self-monitoring is reactive ie. the actual act of recording symptoms (without behavioural or medical intervention) is associated with a reduction in subjective symptom levels (Keefe et al, 1980). This phenomenon is perhaps a function of greater awareness of symptoms and how they relate to external stimuli, or possibly due to changes in participants' interpretation of the severity of symptoms required to constitute an attack. However, a series of studies evaluating self-monitoring of habitual activities reported no such reactive effects eg. McNamara, 1972 (nail-biting), Hall, 1972 (overeating), and Berecz, 1972 (smoking). That said, the monitoring of Raynaud's symptoms differs fundamentally from the monitoring of behaviours that are directly under the control of the perpetrators - though difficult, people can stop smoking, overeating or biting their nails. In contrast, the Raynaud's sufferer is going to suffer from vasospastic attacks (medical or behavioural treatments aside) on contact with everyday conditions that precipitate those attacks. Moreover, smoking and nail-biting behaviours are all or nothing: the nail-biter, for example, can not leave a "quick nibble" out of his or her diary on the justification of it not being an example of nail-biting behaviour; an individual's Raynaud's attacks, in contrast, vary in severity along a continuum. Through monitoring symptoms, the sufferer may unconsciously adjust the point along that continuum at which an attack is considered to have occurred. Therefore, through monitoring, the sufferer may genuinely feel that symptoms have improved.

The standard technique of symptom data collection is to regularly obtain completed diaries through the post or at treatment sessions. However, personal experience leads me to suggest that, at least towards the end of the inquiry period, participants may complete, perhaps, a whole month's diary data

retrospectively before posting the information on. Clearly then, the accuracy of diary data should be considered; indeed, it is not unknown for entries to be fabricated. A related point lies with the potential negative effects diary keeping has on participant compliance. This issue will be discussed further in section 5.7; however, briefly, diary keeping increases the participants' treatment programme requirements (in terms of time and energy). Many experimental volunteers, particularly where symptoms are mild, may find that they are too busy to comply with all of the requirements of the study such that they withdraw at an early stage. Indeed, it is not uncommon for participants to fail to attend a training session rather than to attend without a completed diary. Therefore, though useful in theory, one need carefully balance the potential inaccuracies and extra commitment required of symptom diaries with the benefits, in terms of richness of data, that the technique might offer.

The questionnaire approach, in contrast, is less open to such negative effects as, generally, questionnaire information is collected less frequently than diary data (eg. once prior to, and once or twice on completion of a course of treatment). Moreover, participants provide symptom information in a range of formats (including open questions and Likert scales[19]) such that the internal validity of responses may be assessed. Further, as a plus for the researcher, questionnaire data is more readily coded for analysis than is diary information. However, questionnaire data is generally collected retrospectively; consequently, it is also open to response bias and inaccuracies. Nonetheless, questionnaire data is less likely to be influenced by the relationship between clinician and trainee as questionnaires may be completed in private; furthermore, data collection requires little in the way of extra work on the part of the participants, suggesting that the technique would have less of a negative effect on patient compliance.

Therefore, the questionnaire approach is perhaps the most appropriate form of subjective symptom data collection; however, it must be noted that subjective data, is, by its very nature, somewhat unreliable. Therefore, subjective measures of the effects of treatment are best used in conjunction with objective measures such as those described below.

[19] Likert Scale: a 3, 5 or 7 point scale through which effects of treatment may be rated.

5.3 INDIRECT MEASURES OF PERIPHERAL BLOOD FLOW

As Raynaud's is a condition that affects peripheral blood flow, the most obvious means of assessing the effects of treatment is to compare pre- and post-treatment digital flow. Though invasive techniques such as Angiography might be advantageous in terms of providing a direct, and accurate measure of blood flow, non-invasive procedures are utilised as they limit the discomfort experienced, and risk to, the patient.

Non-invasive methods of assessing peripheral blood flow may be classified in terms of those that measure actual blood flow (eg. Plethysmography, Laser Doppler Flowmetry, microscopy, Ultrasound techniques, radioisotope clearance, and gauges of finger systolic pressure), and those that measure digital skin temperature as an index of flow (eg. Thermography, and Thermometry). Plethysmography, Laser Doppler Flowmetry, and Thermometry are most commonly associated with the evaluation of behavioural and medical treatments. Therefore, these will be described in detail; whereas, the chapter will include only a brief mention of the other techniques - though, the interested reader is referred to a simple review by Mani (1989), or by Freedman et al (1982).

Plethysmography is a means to evaluate blood volume in a peripheral limb over a finite period of time. There are two versions of the technique: venous occlusion plethysmography and photoplethysmography. The former involves measuring pressure changes inside an airtight container in which the forearm, or finger (ie. digital plethysmography), is placed. Venous flow is stemmed with a blood pressure cuff inflated to above 40 mm Hg which allows arterial flow into the limb, but prevents venous return. Immediately after occlusion of the veins, ie. until the back pressure of the blood opposes arterial flow, the pressure in the airtight chamber, being equal to the volume of the limb, provides an index of the rate of arterial flow into the limb. The latter, photoplethysmography, as the name suggests, measures blood volume on the basis of the reflection and absorption of light by the digital tissues. Its use is somewhat limited, however, as it is not sensitive enough to differentiate between degrees of peripheral blood flow. Measurements of finger systolic pressure are obtained in a similar technique to that of venous occlusion plethysmography: a cuff is wrapped around the finger, and

inflated to a pressure in excess of arterial systolic pressure. The cuff is then slowly deflated, releasing small spurts of blood identified by slight tapping sounds. The pressure at which the first sound is detected represents systolic pressure. Similarly, Laser Doppler Flowmetry is not unlike photoplethysmography: the technique, which measures blood volume and direction (up to 1 mm below the skin) utilises a low powered helium neon tube which emits red light that is reflected by red blood cells. The reflected red light, being proportional to blood flow, provides an index of peripheral flow. Other techniques used in the detection of blood flow include Doppler ultrasound (in which blood flow away from the point of contact is associated with a slow wave form; flow toward that point, with a faster wave form), and radioisotope clearance techniques (in which the decay of a radioactive isotope eg. ^{133}Xe, injected into the blood stream, is converted to an index of the rate of blood flow). Moreover, microscopy techniques may also be employed, though generally for diagnostic purposes; however, flow velocity may be calculated by filming blood cell flow through the microscope.

Thermography is a technique that relies on the circulatory system's thermoregulatory role. Briefly, the thermograph provides a series of visual temperature maps of the skin by way of some infrared sensing device. Temperatures are differentiated on the map by a range of colours (eg. white indicating warmth; black, cold etc.). The technique is useful as data is not confounded by the need for physical contact between the skin and recording device.

A thorough review of the history and methodological flaws associated with the techniques described above is outside the scope of this thesis. However, the increased accuracy in measuring blood flow that such procedures provide, is generally outweighed by factors such as cost, limited availability, and the fact that, in the main, measurements can only be recorded over a limited period of time (seconds or minutes in some cases). Consequently, a far simpler technique is more commonly used in the assessment of the effects of treatment(s): namely, thermometry ie. measuring skin temperature directly at the surface of the skin. Thermistor (or temperature detecting) probes are attached to the palmar distal phalange(s) with surgical tape. Temperature fluctuations produce changes in the electrical resistance of the thermistor that are measured and stored in some commercially available temperature data logging device. Ideally, when logging temperature data in the laboratory, the ambient temperature

and humidity should be controlled as digital skin temperature is influenced by changes in ambient conditions; moreover, the skin beneath the probe should be cleaned with surgical spirit before positioning the thermistors as although the tape allows sweat to evaporate from the skin, evaporation is not possible from the skin beneath the thermistor probe. Clearly, the surgical tape must be tight enough to hold the thermistor firmly in place, but not so tight so as to disrupt digital blood flow.

Despite the acceptance of digital skin temperature as an index of blood flow, the assumption is somewhat flawed. Skin temperature is influenced by a variety of factors. For example, arterial blood flow may be cooled by venous blood in adjacent vessels; sweating, ambient conditions and tissue hydration may have an effect (ie. heat loss to the atmosphere through evaporation of perspiration); the basal metabolic rate will influence skin temperature as heat is released in the course of every chemical reaction in the body (ie. where there is greater metabolic tissue activity, the surrounding tissues and skin will be warmer); posture is also associated with skin temperature in that skin blood flow is lower when a person is upright than when supine (Zoller et al, 1972); similarly skin temperature is influenced by clothing, the metabolic effects of smoking and exercise, and the natural circadian fluctuations related to routine patterns of activity such as eating, drinking, and sleeping. Therefore, such factors need be accounted for when monitoring finger skin temperature during any treatment study.

Further, in interpreting changes in temperature data, one must also allow for the "thermal lag" between initial circulatory increase or decrease and the recording of that temperature change; moreover, it should be noted that thermistor probes can detect only the temperature of the skin - not the temperature of the underlying tissues as there is no constant temperature gradient between the tissues and the point of recording on the skin. However, if direct temperature detection techniques are to be adopted in a simple study in which pre-treatment blood flow is compared with post-treatment flow, the factors above will influence both sets of measurements and consequently not confound the results.

Therefore, within the confines of an adequately controlled experiment, digital skin temperature provides an acceptable correlate of peripheral blood flow, and as such, an appropriate means of assessing the effects of treatment. However, the demonstration of treatment-related finger temperature elevations in

the laboratory provides only circumstantial evidence of the efficacy of treatment. If the true benefits of any treatment are to be uncovered, such beneficial effects need be identified away from the laboratory setting. To this end, researchers employ ambulatory temperature monitoring as a means to assess the effects of treatment.

5.4 AMBULATORY TEMPERATURE MONITORING

Laboratory based measures of temperature change are useful in the analysis of digital temperature control; however, in terms of the efficacy of behavioural treatments in the control of vasospastic attacks, entirely laboratory based research is somewhat limited as vasospasms tend not to be elicited in the laboratory. Ambulatory temperature monitoring systems, in contrast, enable physiological data to be collected from mobile patients as they carry on with their usual activities. The systems used for ambulatory temperature monitoring are similar to those required for laboratory based monitoring[20] in that thermistors are attached to the palmar distal phalanges of the non-dominant hand with surgical tape, and the wires secured with tape at the wrist to reduce movement artifacts. The wires are hidden below the volunteer's top layer of clothing, and connected to a meter logging device worn belted around the patient's waist. Up to 24 hours of temperature data may be recorded in any one "session", although for practical purposes (most commercially available loggers are heavy and cumbersome) monitoring takes place over a shorter period of time ie. 3-6 hour periods. The practicalities of collecting temperature data from a mobile participant differ from laboratory data collection. In the laboratory, participants are asked to move as little as possible; during ambulatory monitoring, in contrast, movement effects may confound results. Consequently, movement of the thermistors and logger should be reduced through the use of some kind of electrode cement to maintain contact between the skin and the electrode, or, at the very least, the leads and electrodes should be adequately secured to the arm (Turpin, 1985). Moreover, during a period of monitoring, the surgical tape may loosen or get dirty. Soiled surgical tape may have no direct effects on data collection, but may have the effect of increasing task-related tension in some wearers. This in turn, given the recognised link between physical tension

[20] However, less convenient techniques were used in early research eg. Radiotelemetry, in which temperature data is transmitted to a receiver up to 100 metres from the Raynaud's volunteer.

and peripheral blood flow, may confound the temperature data. However, such potential deleterious effects may be controlled for by providing the Raynaud's patient with fresh surgical tape for replacement purposes.

Ambulatory monitoring of finger temperature further differs from laboratory monitoring in that in the laboratory, both the volunteers' activities and the ambient conditions tend to be controlled. In contrast, away from the laboratory, a range of activities and ambient conditions may apply such that it is difficult to correlate temperature fluctuations with particular activities. Therefore, during the monitoring period, to facilitate interpretation of data, participants are generally asked to complete hourly diary reports of their activities and symptoms. It should be noted that the completion of activity and symptom diaries during the course of a monitoring period is not open to the same biases as are symptom diaries: in the former case, diaries need only be maintained over a short period, and inaccuracies are less likely owing to the initial novelty value of monitoring activities within a limited time span.

Through ambulatory temperature monitoring, assuming that vasospasm occurs, and that activity diaries are accurately maintained, information as to the relative ambient and finger temperatures required to provoke vasospasm may be obtained. Statistical comparisons of these temperatures, before and after treatment, might therefore prove a valuable means of treatment appraisal.

A further means of evaluating peripheral blood flow through changes in digital temperature is that of the Cold Challenge test described below.

5.5 THE COLD CHALLENGE TEST

As previously described, peripheral blood flow alters as a function of ambient temperature and humidity. During exposure to cold, peripheral flow will be reduced. In warm ambient conditions, flow will increase. Following exposure to cold temperatures, peripheral flow in non-symptomatic fingers returns to a little above starting temperatures quite rapidly; Raynaud's fingers, in contrast, rewarm at a

slower rate. Further, as depicted in figure 5.1 below, if digital temperature is plotted across the period of cold stress and subsequent digital recovery, a characteristic difference between the non-symptomatic and Raynaud's trace is evident.

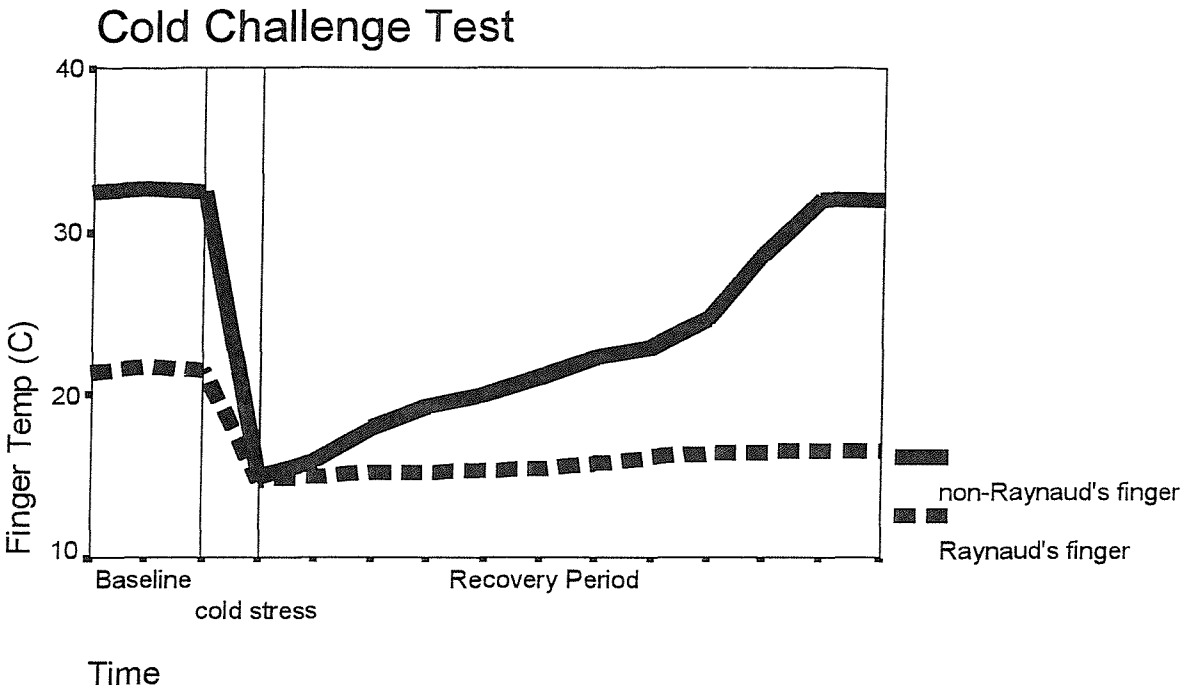

Fig 5.1: The Characteristic rewarming curves of the Raynaud's and non-Raynaud's finger following cold stress.

The cold challenge test involves finger or whole body cooling over a predetermined period and the recording of either the subsequent recovery rate, or the ability to maintain hand temperature under cold stress conditions. A range of practical means of cooling volunteers exist, including a) the use of a "cold chamber" in which the ambient temperature is gradually reduced over time; b) the employment of a whole body water-perfused blanket in which the perfusion of cool water is preceded by perfusion of warm water to reduce sympathetic innervation to the peripheral blood vessels; c) direct finger contact with a stimulus (eg. a metal plate) that gradually cools over time; and d) the immersion of the hand in a "cold water bath". The former, whole body techniques, are useful in that many Raynaud's sufferers report their entire body to be cold during vasospasm of the fingers (personal communication). Consequently, such procedures may be more conducive to the elicitation of vasospastic attacks than

local cold stress techniques. However, owing to factors such as ease of use, the need to limit participant discomfort, and the association between cold objects and vasospasm, local cold stress tests are most commonly employed in the evaluation of the effects of treatment.

The cold stress technique used in the main empirical investigation of behavioural treatments for Raynaud's in this thesis is that of the cold water bath. Full procedural details are described in chapter 8. However, in brief, thermocouples[21] are attached to the palmar distal phalanges with surgical tape, and baseline finger temperature is recorded in some data logging device. A plastic glove is then placed over the hand which is in turn immersed into a cold water bath, maintained at 15C, for 5 minutes. Immediately prior to the start of the recovery period (which continues for a predetermined period, or until digital skin temperature has returned to baseline levels), the hand is withdrawn, and the glove removed. The 15 C water temperature is not an arbitrary choice; indeed, 15 C is the temperature at which maximum vasoconstriction occurs. At lower temperatures, the peripheral vessels actually begin to dilate to prevent cold injury to the exposed extremities (eg. Lewis, 1929 as referenced in Greenfield et al, 1951) - possibly owing to cold induced paralysis of the contractile mechanism of the vessel wall, or blocking of the nerve impulses to the vessels.

The Cold Challenge test has two uses. Firstly, in the assessment of pharmacological, surgical and behavioural treatments for Raynaud's, the test may be used to compare pre-treatment rewarming rates with those following treatment. Clearly, with beneficial treatments, the post-treatment cold challenge test will produce faster recovery rates than the initial test (and graphical presentations more akin to those of non-symptomatic volunteers). Secondly, the method provides a test for use in the diagnosis of Raynaud's. Indeed, Naidu et al (1994) confirmed such a role through the use of an ultrasound device to measure digital artery closure during cold stress; moreover, Neilsen (1978) combined local cold stress (10, 15 or 20 C) and total body cooling (15 C). Raynaud's patients were differentiated from

[21] Thermocouples: temperature detecting probes similar to thermistors.

non-symptomatic controls in that only the former experienced digital artery closure (as calculated from measurements of finger systolic pressure). However, local cooling alone induced digital artery closure in only 60% of the Raynaud's patients. Further, Englehart et al (1986) suggest that total body cooling may provide a means to enable diagnostic differentiation between Raynaud's Disease and Raynaud's Phenomenon. The 1986 study used laser Doppler Flowmetry to measure peripheral flow during total body cooling via a 15 C water perfused blanket. The results suggested that peripheral flow is significantly lower in Raynaud's Phenomenon than Raynaud's Disease.

The validity of such papers is somewhat undermined by published articles that suggest that cold challenge results are not reproducible. For example, Wigley et al (1987) employed cold challenge methodology (local cooling) in a double-blind placebo-controlled evaluation of nifidepine as treatment for Raynaud's. The effects of the drug aside, the results suggest that the participants adapted to cold provocation as lower temperatures (10 or 15 C compared with 20 or 25 C) were required to elicit vasospasm in later cold challenge tests - whether the participants were receiving treatment or not. The ambient conditions of the laboratory did not differ significantly across the three challenge tests of the 10 week period of assessment. However, Thomas (1990) compared rewarming times to cold stress in 8 healthy and 8 Raynaud's Disease volunteers on two separate occasions. Results suggested that the two groups were most readily discriminated in terms of the recovery time to 6 C in digital temperature: the Raynaud's patients rewarming at a significantly slower rate than the non-symptomatic controls, and that follow-up rewarming times to 6 C were not statistically different from the initial times. Moreover, Patel (1991) describes a series of cold challenge studies in which rewarming times to 6 C are replicated over three separate occasions in 8 healthy volunteers, an further, that a reduction in baseline finger temperature at the follow-up cold stress test slows rewarming to 6 C in Raynaud's Disease patients, but not in non-symptomatic controls. In contrast, Wigley et al (1987) made no mention of baseline

temperatures; thus their apparent failure to replicate may be a function of varying pre-challenge digital skin temperatures.

The Cold Challenge stress test is, therefore, a useful tool in the evaluation of treatments for Raynaud's Disease and Phenomenon. However, in addition to evaluative techniques that focus entirely on the detection of peripheral blood flow, complementary procedures may be used to record correlates of physiological arousal in the Raynaud's sufferer.

5.6 THE MEASUREMENT OF PHYSIOLOGICAL AROUSAL

Measures of physiological arousal in the assessment of treatments for Raynaud's are useful for two reasons. Firstly, they provide a guide to general physical effects of any pharmacological or behavioural strategy of symptom control: ie. What is the impact of the technique on the body ?; Through what mechanism(s) might treatment-related symptom alleviation occur ? Secondly, and subsequently, such measures may provide objective evidence that a particular behavioural technique is achieving its aim. With the pharmacological approach, the clinician can be fairly certain that the administration of a drug will elicit particular physiological effects (whether or not the effects are of benefit in the control of symptoms). In the field of behavioural medicine, however, the onus is on the Raynaud's sufferer to regularly engage in a prescribed technique of symptom control. Conscious awareness of the benefits and sensation of the appropriate administration of such techniques may be slow to appear. Participants might, for a time, simply be "going through the motions" ie. not really feeling any of the supposedly associated bodily sensations. Here, physiological recording may be of benefit in that although the trainee may state, and genuinely believe, that the technique is being carried out appropriately, actual objective measures of the assumed physiological correlates of the technique, may imply that it is not.

The main correlates of physiological arousal monitored in the course of evaluative treatment studies include heart rate, respiration rate, and skeletal muscle activity. Skin conductance/resistance (GSR) is also readily accessible to physiological recording, but is rarely used in the assessment of the behavioural approach to treatment.

The nature of neural transmission is electrical; thus, techniques applied to the recording of heart rate, and muscle tension and skin conductance, generally measure electrical current within the organ or area of interest. For example, skin conductance techniques (eg. via the use of a galvanometer - hence the abbreviation GSR or Galvanic Skin Response) involve measuring the changes in electrical potential between two electrodes placed on the skin. The basis for their use in Raynaud's research lies with the historical finding that changes in skin resistance are associated with changes in skin blood flow, and, via the action of sympathetic innervation, with sweat gland activity.

Heart rate is measured via electrocardiography (ECG/EKG) in which the potential of the electrical current traversing the heart muscle and initiating each contraction is recorded on an electrocardiogram ie. a paper record of the electrical event. Increased activity, via an increase in sympathetic nervous activity, is associated with an increase in heart rate; sleep and low levels of activity are associated with a reduction in heart rate - owing to higher levels of parasympathetic nervous activity and subsequent reduction in sympathetic activity. Therefore, the effects of behavioural treatments, such as relaxation, for example, may be recorded as an index of heart rate: a relaxed state is associated with a reduction in heart rate; physical tension with an increase. However, there are a number of problems with the employment of ECG techniques. Firstly, it is a well established fact that heart rate will, at least initially, be influenced by the rather unnerving process of probe attachment and recording. Obviously, this obstacle may be overcome through adequate adaptation periods to enable participants to accustom to the machinery, and the use of complementary measures of physiological arousal such as respiration

rate[22]; however, a sudden noise or disturbing thought can quicken heart rate irrespective of the effects of physical tension or relaxation. Therefore, secondly, a range of physiological states may elicit an increase in heart rate suggesting that although useful in conjunction with other measures of physiological arousal, the employment of ECG measures alone would be ill advised. Finally, an area worthy of note is that of probe attachment. The point of ECG probe contact may be directly above the heart area ie. under the clothes. This may be regarded as intrusive. Alternatively, recording may be via a light emitting probe used in the measurement of finger pulse. Here, accurate recording requires external light to be minimised; therefore, a black velvet bag is placed over the hand, which, as personal experience allows me to unequivocally state, warms the hand, and may have a knock-on effect on the temperature of the contralateral hand.

Skeletal muscle tension is recorded through a process called Electromyography (EMG): action potentials, propagated during muscle fibre contraction, are "picked up" by electrodes, amplified and recorded as an index of muscular activity. For increased accuracy, needle electrodes may be inserted into the muscle; however, within the field of behavioural research, where patient comfort is a priority, probes tend to be stuck to the surface of the skin. Though in the past, probes have been attached with sticking plaster, suction or elastic bands ! (Lippold, 1967), modern day probes tend to consist of a spongy substance perfused with electrode jelly that is attached directly to the skin, and held in place both by the elastic property of the sponge, and by an attached sticky backing. The measurement of EMG requires the attachment of two or three probes to the skin surface: ie. one or two "active" electrodes along the length of the muscle to be monitored; and one "ground" electrode positioned at any inactive point (ie. at a site of limited muscle tissue eg. the elbow) on the body. The ground electrode acts as a reference point from which changes in muscle tension between the "active" electrode(s) may be evaluated; however, the magnitude of recorded action potentials between the electrodes is small.

[22] The measurement of respiration rate, as described in published articles (eg. Freedman et al, 1983c), requires the attachment of nasal thermocouples to the trainee. This sounds exceedingly uncomfortable, and, one assumes, not conducive to positive relaxation effects of treatment.

Consequently, the signal is amplified in the EMG apparatus to within a discernable range to facilitate the differentiation between degrees of muscle tension.

The area of electrode attachment obviously depends on the muscle site of interest. Thus, in tension headache, EMG electrodes may be placed on the Semispinalis capitis and Splenius capitis muscles (ie. those at the back of the neck); where headache tension is associated with jaw pressure, the electrodes would be placed at the site of the masseter muscle. As described in chapter 6, ahead, the aim of relaxation treatments for Raynaud's is to achieve whole body relaxation. Therefore, in evaluative research of the behavioural approach, researchers, ought, ideally record muscular tension of the whole body. Jacobson (1940), as referenced in Lippold (1967), utilised a technique in which electrodes were placed at various points over the body (eg. on the upper and lower arms and legs, the neck and abdomen etc.). The electrodes were connected in two groups to the EMG amplifier system; thereby providing integrated muscle tension of the whole body. Clearly, such a set up is not practicable within the framework of a behavioural investigation of the efficacy of treatment given the number of probes that would have to be simultaneously attached to the participants. Single muscle areas have been considered as an index of whole body tension eg. the frontalis muscle; however, there is no physiological evidence to support the frontalis muscle in such a role. Indeed, the benefits of obtaining a correlate of forehead tension in participants suffering from a disorder of the peripheral limbs, are no greater than those of acquiring a correlate of gluteus maximus muscle tension. That aside, within the behavioural approach to treatment, EMG biofeedback (see chapter 6 for details of biofeedback techniques) has been used extensively as a means to "teach" general relaxation and symptom control - owing, perhaps, to the high reliability of frontalis EMG recording (Goldstein, 1972).

An extensive review of the physiology of muscular activity is beyond the scope of this thesis; however, the interested reader is referred to any physiological text book such as Cacioppo and Tassinary (1990) or Greenfield and Sternback (1972).

The employment of EMG recording is not without its problems. Skin resistance, for example, may exert an influence. Clearly, surface sweat need be removed, and, as most skin resistance occurs in the top, horny layers of the skin, these should be removed via light rubbing with sand paper and vigorous rubbing with electrode jelly. Though only mildly uncomfortable, the process may be rather unpleasant for participants receiving EMG biofeedback on a regular basis; moreover, where repeated application of EMG probes is required, it is important that the site of attachment does not differ across sessions[23]. Lippold (1967) suggests means to ensure exact electrode placement - ranging from dyeing the skin for the duration of the study, the more permanent tattooing of the skin (!), photographing the attached electrodes, and maintaining a permanent record through tracing onto paper the electrode position along with marks on the participant's skin (eg. moles etc). However, despite such safeguards, direct correlates of muscular activity can not be obtained with surface electrodes; indeed, electrical activity that did not originate in the muscle (group) in question may be detected eg. cardiac output, skin resistance changes. Further, electrical activity at the surface of the skin is inversely related to the depth of the muscle (Davis, 1959 as referenced in Goldstein, 1972). Therefore, there may be differences in the measured potentials between two sites on the same limb or area of the body - particularly in participants who are overweight - or between participants with varying muscle mass. Moreover, as reported by Buchthal et al (1954) in Goldstein (1972), there is a notable difference in the duration of action potentials between the old and the young (ie. the duration increases with age), and in warm and cold muscles and ambient temperatures. Clearly, in any investigation of the effects of behavioural treatments in which EMG monitoring is employed, both the temperature of the room, and the temperature of participants' muscles (through an enforced period of inactivity prior to recording) need

[23] Although in larger muscles, the effects of small errors in electrode position will be minimal.

be controlled. Along similar lines, gross movements on the part of the participants should be limited: surface electrodes detect action potentials directly below the point of contact; with gross movements, the skin may move to temporarily cover an adjacent area of muscle; thereby introducing errors into the recorded trace.

5.7 COMPLIANCE

If the techniques described in the sections above are to be put to good use in the evaluation of Raynaud's treatments, it is, therefore, important that the methodological design of any empirical investigation is sound, and, that the prescribed treatment techniques are indulged in as instructed. The former issue, as to appropriate experimental design, will be reviewed in chapter 6; the latter point, regarding techniques of improving patient compliance, will be discussed below. The issue of patient compliance (ie. the lack of compliance to instructions), is not exclusively a problem of the behavioural approach to medicine; indeed, failure to comply to instructions reduces the efficacy of pharmacological as well as behavioural treatments. For example, despite clear instructions to the contrary, many patients fail to complete a course of antibiotics. However, given the greater personal involvement, and the consequent greater need to comply with instructions with behavioural treatments, the issue is particularly germane in the behavioural approach. Nevertheless, as noted by Ley (1988), whatever the treatment approach, compliance will be higher where patients are suffering from a serious condition, where the costs in terms of time and money are low, and where a participant's social network is supportive of the treatment ideology. Therefore, although one might expect higher rates of behavioural treatment compliance in Raynaud's Phenomenon patients than in those suffering from Raynaud's Disease, such an assumption might be offset by the effects of relatives and friends denigrating the behavioural approach to treatment.

In any behavioural treatment study, there are two areas in which patient compliance is pertinent. As noted above, treatment may involve perhaps 10 or 12 meetings with a therapist during the course of training and assessment. That patients attend all arranged meetings, is therefore, required if the efficacy of treatment is to be adequately assessed. Secondly, as in the conventional medical approach, the prescribed treatment must be "taken" regularly between appointments if it is to have the required effect: behavioural techniques must be practised regularly if the accumulative effects are to be established.

An initial step into the area of patient compliance must involve the assessment of current levels of adherence to instructions (- clearly, some people have more "will power" than others), and of attendance at training and evaluation sessions. Clearly, the latter assessment merely requires some register of attendance; the former, however, is less simple. Two means of assessing patient compliance to training instructions are available. Firstly, that of the therapist's subjective judgement. In the same way as a piano teacher might deduce that a pupil has not practised a new piece of music since the last lesson (the pupil is perhaps struggling to remember the notes), the behavioural therapist might use his or her expertise to conclude that a relaxation exercise, for example, has not been appropriately rehearsed. A number of factors might apply here, including a) the observation that the trainee does not remember the practised exercise, or b) the absence of previously observable physical effects associated with a relaxed state (eg. the appearance of having just woken up at the end of the exercise; rapid eye movement during training).

The second means of assessing patient compliance to treatment instructions is simply to ask the participants about their practice behaviour - either directly, or indirectly through patient practice diaries. Here, however, the subjective nature of such information must limit its reliability; indeed, reported practice levels might be inaccurate as a function of lack of understanding on the part of the

patients ie. participants might not have fully comprehended the practice requirements. Moreover, at a more sinister level, participants might state that they have complied with the training instructions quite fully, where in fact they have not. Indeed, as with symptom diaries, where practice diaries are seen as an integral part of the training process, attendance at the next training session might warrant fabrication of diary entries.

The reliability of the assessment of patient compliance will obviously be improved through some overlap between subjective assessment on the part of the participant and the clinician. Assuming therefore, that low compliance rates have been identified, how might adherence to training instructions be improved ?

Given the association between lack of understanding and low patient compliance, an important area to consider is that of patient understanding of both procedural instructions, and what is required of them in involvement with the treatment study. Indeed, compliance will be further improved if the participants understand why they should attend a given number of training and assessment sessions; why it is important to practice taught techniques of temperature control between sessions; and why they have been asked to keep practice diaries etc. Clearly, this requires adequate communication skills on the part of the clinician: information should be provided both orally, and in an clear, simple, written format, pitched at an appropriate level of understanding. Further, written information should be well "sign posted" (ie. it should be stated at the outset what information is included in the hand out and why and how it is presented), and if it is hoped that any hand out will be read once participants have left the laboratory, written information should be both interesting to the reader, and well presented - perhaps with the inclusion of diagrams, and relevant jokes etc.

In line with the above, Ley (1982) suggests that compliance is related to a) the level of a patient's understanding of their role in the treatment process, b) the patient's ability to remember such treatment-related information, and c) the degree of patient satisfaction with both presented information, and the potential personal benefits of treatment. It is therefore advised that prior to handing out written information to be taken away, the clinician should briefly describe the content, and then go through the hand out with the patient. Further, it would be beneficial to encourage participants to ask questions to clarify points that are not completely understood, and perhaps to subtly ask volunteers to describe their understanding of the experimental protocol such that errors might be corrected. As to patient recall of treatment details, Ley (1972b) in Ley (1988) documents a "Primacy effect" in recall of treatment-relevant detail. Therefore, in the preparation of hand outs (and with the spoken word), it is worth considering that recall is better for information that is first heard, so important information, such as the need for practice or completion of treatment, might be stated first. Moreover, as memory for later information is liable to be poor, it may be useful to change the presentation order of treatment information at subsequent training meetings.

The suggestions above are also applicable to the improvement of compliance, in terms of attendance at training and assessment sessions. However, further improvements in attendance might be achieved through making the training and assessment experience pleasant and unthreatening for the participant (allowing, of course, for the unappealing assessment procedures such as cold challenge); thus the clinician might ensure that sessions are run on time, or that (s)he appears sympathetic to the concerns and problems of the participants - be they treatment related or not. Thus, with relaxation treatments, patients might worry that the treatment is not actually reducing symptoms, nor is likely to do so as the sensation of relaxation, and the effects on blood flow, are often slow to emerge. To avoid this leading to patient withdrawal, the clinician might state positively that such insecurities are common in the early stages of training, that the effects of relaxation exercises are cumulative, and that it is worth

persevering with treatment ie. discuss the progress of treatment with the participant. Though, without presenting an unrealistic picture of the benefits of a treatment that may, indeed, prove to be ineffective, it is worth noting that patients are more likely to comply to treatment instructions if they feel that the treatment is likely to be effective for them.

The underlying concept in the sphere of patient compliance is therefore, communication: clear communication of ideas will improve both memory and understanding of patient requirements; adequate communication of the potential effects of treatment will improve patient satisfaction with that treatment. Consequently, it is through patient-clinician communication that patient cooperation may be improved. Moreover, through improved communication, a reduction in deception on the part of participants when reporting treatment practice exercise levels might be achieved. In a non-judgemental manner, the clinician might attempt to empathise with the participants' difficulties with practice by, perhaps, mentioning the apparent "resistance" to practice that most people - the clinician him/herself included - experience at some point during training.

In terms of the behavioural approach to the management of Raynaud's symptoms, adherence to instructions to practice relaxation exercises is generally not high. Barr Taylor et al (1983), for example report that although subjective report indicates a 71% adherence rate, objective measures suggest that only 39% comply with instructions to practice between training sessions. Given the importance of patient compliance to assessment treatment protocols, a number of techniques have been suggested to improve compliance rates. To facilitate practice between training sessions, with standard relaxation techniques, trainees are generally supplied with a tape recording of the exercises. This has been put to good effect in the evaluation of techniques to aid patient compliance. For example, Riley et al (1986) assessed the effects of providing a rationale for treatment. Half of the 52 participants viewed an introductory video in which the rationale behind the technique of Progressive Relaxation (ie. muscles

are tensed, then relaxed, so that the skill of relaxation may be acquired through the participant distinguishing through the two muscular states - see chapter 6 for details). The remaining participants viewed a similar video in which the rationale information had been edited out. Following instruction in the technique, participants were asked to practice the exercise regularly, by listening to tape recorded procedural instructions. The audiotape was sealed in a tape recorder, in which the use of the rewind button was logged as an index of practice rates (ie. in order to listen to the instructions, the tape would have to be rewound to the start). This objective measure of compliance suggested that providing a rationale for treatment does not improve compliance rates. In contrast, as a means to assess patient compliance, Martin et al (1981) suggest providing trainees with separate tapes for each practice session so that they may listen out for, and note down, "cues" (eg. tones, musical notes etc) that may (or may not) be recorded on each tape. Quite how participants are expected to attain complete physical and mental relaxation with one ear out for a cue, is not explained. However, it is suggested that not only would this technique provide an index of compliance through the participants' record of cues, but further, help ensure that the participants actually listen to the tapes as asked. However, the act of listening to a relaxation tape does is necessarily correlated with practice; moreover, in the long term, the implementation of such a technique may lead to patient withdrawal from a training programme.

Within the field of Raynaud's research, the issue of patient compliance is rarely high on the researchers' agenda; indeed, a paper by Jobe et al (1986) reports the employment of a rectal temperature probe in the assessment of the effects of treatment ! Therefore, the steps that I have taken to improve patient compliance in the study described in chapter 8, will be outlined in chapter 7. However, firstly, the body of literature describing the effects of behavioural treatments in the management of Raynaud's symptoms will be described to allow sound methodological practices to be uncovered and methodological flaws discussed.

Chapter 6

BEHAVIOURAL TREATMENTS

6.1 INTRODUCTION

Given the limitations of the medical approach to the treatment of Raynaud's disorders (as outlined in chapter 4) many sufferers are turning toward behavioural alternatives and/or complementary behavioural treatments. As with current pharmacological treatments, it should be noted that the behavioural approach is palliative in nature. However, whereas in the former case, the patient depends on the action of prescribed drug(s) for relief, through the behavioural techniques, the sufferer takes on an active role in the alleviation of symptoms.

The term General Behaviour Modification refers to a range of behavioural techniques including simple life style changes (such as those previously described) and more rigorous techniques that require the acquisition of particular skills eg. relaxation procedures. The purpose of this chapter is to describe such treatments, to consider their action in the control of digital blood flow, and to review the large body of evaluative literature regarding the efficacy of the approach in the management of Raynaud's symptoms.

6.2 GENERAL BEHAVIOUR MODIFICATION

As suggested above, behaviour modification encompasses a range of techniques to reduce the effects of symptoms. Some simple techniques, such as the avoidance of known precipitators, have already been discussed; however, a number of other intimated means of symptom reduction have been empirically tested with regard to Raynaud's. These include Transcutaneous Nerve Stimulation (TENS), conditioning treatments, relaxation, and biofeedback techniques. In the main, attention has focused on the latter treatments. In line with this, therefore, relaxation and biofeedback will be considered

separately in the sections 6.3 to 6.5 below; whereas the lesser known techniques will be discussed in this section.

Transcutaneous Nerve stimulation (TENS), employed primarily as a means to reduce pain, involves the application of 2 Hz of electrical stimulation to disorder-specific areas of the body. A number of TENS investigators report incidental generalised vasodilation and associated finger temperature increases in parallel with the analgesic properties of treatment (eg. Kaada, 1982 and Abram et al, 1980) - the former study involving Raynaud's patients. That said, TENS induces visible muscular contraction at the point of application which may, at least in part, account for reported temperature increases. Consequently, controlled studies are required. In one such study, Mulder et al (1991) applied the TENS cathode to a Raynaud's-appropriate site (the web between ossa MCP I-II of the hand) or a Raynaud's-inappropriate site (the middle of the tractus iliotibialis region of the leg). However, poor subjective effects on symptoms, and only short lived temperature increases, were reported. Moreover, the reader may recall that the interviews presented in chapter 3 suggest that TENS actually worsens symptoms.

The effects of Pavlovian-type conditioning treatments are more encouraging. For example, Hayduk (1980, 1982) successfully trained Raynaud's patients to associate cold ambient conditions with a vasodilatory response, and the ability to vasodilate at both room temperature, and at minus 14 C (!) was claimed to remain at one year follow-up. Further, Jobe et al (1982) produced a vasodilatory response to cold challenge (whole body cooling) in 8 Raynaud's Disease and 7 non-symptomatic participants whose hands were warmed to 43 C through the infusion of warm water at the same time as the body was exposed to cold (0 C). A vasodilatory response to cold was not exhibited by untreated Raynaud's and non-symptomatic controls. Follow-up data suggested that the positive effects of treatment remained some 3 years later. Moreover, Jobe et al (1986) reported classical conditioning effects to be comparable with those of relaxation and biofeedback. However, with this in mind, most empirical research has focused on the relative effects of relaxation and biofeedback in the management of Raynaud's symptoms as the conditioning procedure is somewhat uncomfortable for Raynaud's patients (ie. a series of 10 minute exposures to ambient conditions of 0 C wearing only indoor clothing).

Relaxation in the control of peripheral blood flow will be described and evaluated in sections 6.3 and 6.5; biofeedback in sections 6.4 and 6.5 below.

6.3 RELAXATION TREATMENTS

The employment of relaxation treatments for Raynaud's grew from the association between physical relaxation and sympathetic nervous flow to the peripheral blood vessels as outlined in chapter 1. To recapitulate, physical tension, associated with anticipation of onset or continuation of a Raynaud's attack, will heighten sympathetic nervous activity such that vasospasm may be elicited or maintained. Increased tension may actually prolong a Raynaud's attack through amplifying the effects of a trigger. Consequently, relaxation may help reduce or even avoid the effects of tension; thereby decreasing the duration and intensity of a Raynaud's attack.

Three main relaxation treatments have been utilised in the behavioural management of Raynaud's symptoms: namely Progressive Relaxation, Applied Relaxation and Autogenic Training.

The technique of Progressive Relaxation was developed by Jacobson (1938) because people generally do not know how or when to relax. Jacobson suggested that when an untrained person relaxes, there remains a degree of "residual tension" within the muscles such that although (s)he may consider her/himself relaxed, the trainee may still display signs of physical tension such as irregular respiration or increased pulse-rate. The purpose of Progressive Relaxation, is therefore, to instill passive awareness of the state of the musculature such that a true state of physical relaxation can be achieved. Thus, Progressive Relaxation training requires the flexing of a particular muscle or muscle group to the point at which the trainee "cultivates the muscle-sense". (Jacobson, 1974, p 43) ie. until the muscle (group) is contracted as far as possible and the physical sensation of muscular contraction may be compared with the contrasting sensation of the flaccid muscles in the body. The trainee is then taught to counteract muscle tension through the release of tension to an extreme point of relaxation. By comparing the two extremes of contraction and relaxation, the trainee learns to sense when a muscle is

relaxed. As the "muscle-sense" develops, the need for the initial contraction phase diminishes, and relaxation may occur in isolation.

The term 'progressive' is an appropriate label for the technique for three reasons: firstly, both muscle flexion and relaxation occur progressively in a gentle, steady manner; secondly, when a state of extreme relaxation has been achieved in one muscle or muscle group, another muscle (group) is explored such that the trainee is able to relax progressively more of the body; and thirdly, as the technique is practised, progressively less pronounced levels of muscle tension are identified. As training progresses, the skill of relaxation may be applied in everyday settings. For example, when sitting quietly to read a book, minimal tension is required in task-appropriate areas of the body (eg. the arms to hold the book, the back to maintain posture etc.), but the rest of the body may yield to the sensation of complete physical relaxation.

The principles of Progressive Relaxation have lead to the development of the technique of Applied Relaxation. This requires the acquisition of the skill of Progressive Relaxation, and the introduction of a series of breathing exercises that associate breathing with relaxation. The technique is described in detail in chapter 8 (and in Appendix II).

Autogenic Training (AT), also described in chapter 8, differs from Progressive Relaxation in that the former evokes a state of deep relaxation through cognitive rather than physical means. Indeed, AT is a western form of meditation (or inward focusing of attention). Its origins lie in the "Autohypnosis" work of Oskar Vogt and his student Johannes Schultz (ie. "Autohypnosis" in the integration of mental and physical functions) eg. Schultz (1953) as referenced in Pelletier (1977). Briefly, the technique, which was introduced to the west by Dr Wolfgang Luthe (eg. Luthe, 1964), involves the learning and frequent repetition of a series of mental exercises that bring about deep psychophysiological relaxation, inner peace and calm, and an improved sense of well-being. Three concepts underlie Autogenic Training: firstly, the requirement that the trainee focus the mind inwardly through the repetition of a series of verbal statements that evoke feelings of heaviness and warmth in the body; secondly, passive rather than active concentration is required such that the trainee does not attempt to control the relaxation

process; and finally, specific postures are introduced to reduce distracting sensory input from the body and environment.

Relaxation techniques have been used in the treatment of a range of disorders such as tension headache (Spinhoven et al, 1992), generalised anxiety disorder (Borkovec et al, 1993), genital Herpes (Koehn et al, 1993), and Raynaud's. However, in Raynaud's research, the relaxation techniques tend to be employed in conjunction with, or in comparison with, biofeedback techniques and other treatments; indeed, Surwit et al (1982) favourably investigated the combined effects of Prazosin and Autogenic Training. Literature describing the evaluation of more than one treatment will be discussed in section 6.5; however, the aims of this section are to evaluate research employing relaxation techniques in isolation in the control of digital skin temperature.

An extensive literature search revealed that only one paper directly assesses the effects of relaxation in control of digital skin temperature; moreover, not only is the paper concerned with temperature control just in non-symptomatic volunteers, but, further, it does little to support the case for relaxation as a means to finger temperature control. Boudewyns et al (1976) report a series of experiments in which the subjective arousal effects of an audio relaxation tape were compared with those of an assumed "stressful situation" underpinned by the threat of electric shock. Finger temperature increases from baseline were shown to occur during the relaxation period, and decrease during the period of stress. The reported digital temperature increases were shown to be no greater than those demonstrated by non-treatment control participants. However, as described in the next section, a series of comparative studies of relaxation, biofeedback (and no) treatment, suggest some benefit of relaxation in the management of Raynaud's symptoms.

6.4 BIOFEEDBACK TREATMENTS

Feedback is described as "*a method of controlling the system by reinserting into it the results of its past performance.*" (Wiener, 1948). In terms of the control of peripheral blood flow, biofeedback may, therefore, be used as a means to provide the trainee with information about current finger skin

temperature (in temperature biofeedback) or muscular tension (in Electromyogram [EMG] biofeedback). The trainee can use this information to somehow adjust future muscle tension or digital temperature; thereby indirectly controlling peripheral blood flow.

As with relaxation, biofeedback techniques have been employed in the treatment of a range of disorders such as epilepsy (Finley, 1976), essential hypertension (Patel and North, 1975) and Raynaud's Disease (eg. Freedman et al, 1983c). Indeed, biofeedback became the "wonder drug" of the 1970's given its application to such a range of disorders. However, it is clear that the explanation above does little to explain the supposed mechanism(s) involved in biofeedback mediated control of digital skin temperature. Therefore, before going on to review the biofeedback literature, a more critical appraisal of the enigma of biofeedback is required.

A good starting point in considering the mechanism(s) of biofeedback must be to describe the equipment used in biofeedback treatments. To simplify matters somewhat, it should be noted that although it is alleged that any physiological process under autonomic control might be influenced through the application of biofeedback techniques, the basic biofeedback apparatus is similar for any such process. Probes (eg. thermistors in temperature biofeedback; electrodes in EMG Biofeedback) "pick up" information about the current state of physiological function. This information is processed in the "biofeedback box", and is then fed back to the trainee in some analogous form to enable the process to be voluntarily controlled. In practice, the analogue is generally auditory (ie. a tone that changes in frequency), or visual (ie. a needle that moves in response to the direction of change in peripheral blood flow) in format.

As to the mechanism of biofeedback, Shellenberger and Green (1986) note that in most investigations of the effects of biofeedback, the equipment itself is implicated as the means of physiological change. For example, *" The paradigm that underlies biofeedback research in general is that of operant conditioning .."* (Sappington et al, 1979, p166); *"The findings are consistent with an operant conditioning model of biofeedback learning..."* Barrett et al, 1988 p45), and as quoted by Shellenberger and Green, *"it is the contingency between the target behaviour... and the*

reinforcement... that is responsible for the increase". (Furedy, 1979 p 83); "*the unique contribution of EMG biofeedback had been consistently confounded with both the inclusion of other relaxation methods during training and regular home practice of nonfeedback relaxation*". (Alexander and Smith, 1979, p 124); and *"There appears to be no basis for the claim by many clinicians that awareness of the feedback relevant response is necessary in order to achieve self-control over the response".* (Carlson, 1978, p7). Indeed, biofeedback is viewed either with regard to an operant conditioning model (the apparatus being a *stimulus* to the *response* of temperature change), or in homage to the pharmacological approach to treatment, the biofeedback apparatus is viewed as a *drug,* which if "taken as prescribed" will lead to the temperature change *response*. In effect, the biofeedback apparatus itself is seen as possessing "magical" properties in that merely attaching the equipment to a volunteer, will elicit the required response. Further, it is suggested that conscious involvement in the process of temperature change is unnecessary: *"In summary,* [Biofeedback] *stimuli have innate and acquired properties that enable them to control behaviour".* (Engel, 1979, p171).

However, returning to the biofeedback apparatus, one finds a box that translates physiological signals to some auditory or visual form, a number of electrodes or probes, and some connecting wires. Obviously, without some conscious involvement, the apparatus alone can not influence peripheral blood flow, or any other physiological process. At best, the biofeedback equipment can provide a window through which physiological processes can be seen and therefore regulated. Biofeedback can not be utilised in isolation. Indeed, the biofeedback apparatus operates as a means to evaluate the efficacy of some physical or cognitive strategy of control employed by the trainee. For example, finger blood flow may be influenced by cognitive means such as the use of thermal imagery (ie. imagining oneself in a hot bath, hands near a warm flame etc.), or visualising increased blood flow to the hands. Alternatively, physical strategies that elicit increased muscle tension of the lower arm, general relaxation, or more specific relaxation of the muscles of the lower arm will increase peripheral blood flow. In the former case, temperature elevations may be mediated through the increase in blood volume associated with increased muscular activity; in the latter cases, relaxation will increase peripheral blood flow through an associated decrease in sympathetic nervous activity. The nature of the strategy aside, a degree of trial and error must, at least initially, be involved in the implementation of biofeedback for the control

of physiological processes; indeed, through the "biofeedback window", trainees may witness their errors in the use of a chosen strategy, and therefore, "fine tune" that strategy to enable autonomic control.

Armed with an understanding of the limits of biofeedback training, we are now in a position to evaluate its role in the control of finger skin temperature.

In the early years of clinical use of biofeedback as a stand alone treatment in its own right, a host of published papers addressed the effects of EMG and temperature biofeedback in temperature control for both Raynaud's and non-symptomatic volunteers. Further, the realisation that biofeedback has no mystical properties has been slow to filter through to all research groups. Indeed, papers that place biofeedback on a par with pharmacological or relaxation treatments have been published as recently as 1994, and one must assume that such work is ongoing. For purposes of completeness, however, the work will be reviewed here.

Since the early 1970's, biofeedback has been evaluated in the control of finger temperature in both non-symptomatic and Raynaud's volunteers. Looking firstly at temperature control in non-symptomatic volunteers, Keefe (1975) claimed to demonstrate control in eight volunteers trained to increase or decrease finger skin temperature relative to forehead temperature over 12 successive biofeedback sessions. By the final session, all participants were able to shift their finger skin temperature in the required direction by up to $2^{o}F$. In later work, Keefe and Gardener (1979) went on to assess the comparative effects of less (5) or more (20) biofeedback training sessions. Temperature control improved with each training session in the five, but not the 20 session condition; indeed, in the longer condition, the greatest temperature increases from baseline occurred in the third training session and was not improved upon.

A number of methodological flaws underlie both Keefe papers. Beyond the inappropriate status granted to biofeedback, the Keefe and Gardner paper confounds gender effects with those of treatment: male participants received fewer treatment sessions, female participants more. Further, in neither study was participant body temperature permitted to adapt to the ambient conditions of the laboratory.

Consequently, participant's attempts to increase finger temperature might be countered, or aided, by a natural temperature fluctuations. In line with this, the apparent occurrence of the greater temperature effects in Keefe and Gardner's third training session may be coincidental in that they may be attributable to naturally occurring temperature changes. It should be noted, however, that failure to allow adequate adaptation periods is a common flaw in the experimental evaluation of temperature control techniques. Indeed, such an explanation would be relevant in the finding by Surwit et al (1976) that non-symptomatic volunteers are better able to decrease than increase finger skin temperature using biofeedback techniques.

The effects of "pure" biofeedback in non-symptomatic volunteers have been assessed through a series of papers manipulating different aspects of the biofeedback process. Indeed, Taub and Emurian (1976) controlled the direction of skin temperature changes, Ohno et al (1977) investigated bidirectional control of finger skin temperature in a double blind design[24], and Crockett and Bilsker (1984) compared biofeedback mediated hand and toe temperature control. Some earlier work addressed temperature control in children. Lynch et al (1976) investigated differential control of hand temperature (ie. the ability to increase the temperature of one hand whilst decreasing the other) in 4 children. Further, in an attempt to isolate the mechanism of control, a second experiment evaluated differential control of the index and middle finger temperature of the dominant hand; however, only small magnitude changes were noted - owing perhaps, to the lack of adequate baseline and adaptation periods in the experimental design. Hunter et al (1976) went further through the comparison of biofeedback mediated control in normal children and children with learning disabilities. Moreover, the format of the biofeedback signal was altered to associate temperature control with movement of a toy train around a track, and the effects of both contingent and non-contingent biofeedback were assessed. Here again, however, the failure to allow finger temperature to adapt to ambient conditions, and the employment of inadequate temperature baseline measures (ie. of only 2 minutes duration) undermine the results. This point is

[24] Double-blind study: A comparison of the effects of visual and auditory biofeedback (or other treatments) in which neither the clinician nor trainee is aware of the allocation of mode of biofeedback; and moreover, the trainee is not informed of the connection between the biofeedback analogue and skin temperature.

further underlined by the fact that recorded finger temperature increases associated with treatment were to the order of a rather unimpressive 0.2 C. [A point of interest, though not directly relevant to this thesis, was the finding that voluntary digital temperature control appeared more pronounced in children with learning disabilities than children without.] Along similar lines, Delaney et al (1992) compared standard (audio and visual) and novel biofeedback (a computer screen image that enlarged to its complete form as finger temperature increased), concluding that novel biofeedback is associated with greater temperature control than the standard variety. With "novel biofeedback", one must assume some role of motivational factors. Indeed, in the latter study, the children were provided with a strategy of temperature control in the form of a tennis ball placed in the hand, supposedly, to prevent pressure being applied to the thermistor by the child; however, slight pressure on the ball might elicit short term increases in peripheral blood flow, and therefore, associated temperature elevations. Thus, in the novel biofeedback condition, the children would have been more highly motivated to exert such pressure on the ball - particularly as it had been suggested that they should not press on the ball.

As to temperature control in adults, Surwit (1977), and Kim and Carlson (1991), compared the effects of single and multi-mode biofeedback. In the former case, visual temperature biofeedback was compared with a combination of visual and a complicated binary visual and auditory feedback system; in the latter, the effects of frontalis and multiple site EMG biofeedback as a means of "stress reduction" were compared. Both papers suggest that the type of biofeedback administered is irrelevant in the control of finger temperature. However, such a conclusion might, one assumes, be preempted by the overall failure of participants to demonstrate temperature control. Donald and Hovmand (1981) taught 30 university undergraduates to maintain a constant state of muscle tension in the arm, before introducing them to visual and auditory biofeedback as a means to control finger temperature - under cold (10 C), normal (24 C) or warm (38 C) ambient conditions. Only slight temperature changes were recorded. Stoffer et al (1979) attempted to investigate the true benefits of biofeedback treatment through the introduction of placebo biofeedback in "yoked-sham" form ie. in which temperature control responses are determined by biofeedback signals generated by other participants. Contingent and Yoked-sham biofeedback produced comparable temperature elevations (approximately 0.5 C), which Stoffer et al forward as evidence of the limited use of biofeedback in the control of finger skin

temperature. However, the paper merely underlines the earlier point that biofeedback is not a valid treatment in its own right; indeed, in both the contingent and yoked-sham conditions, participants were unable to assess the efficacy of their chosen strategy of temperature control, owing, in both cases, to insufficient training sessions to permit mastery of the strategies, and in the latter case, to the fact that participants had no structured means of learning the effects of their chosen technique. Lastly, Kluger and Turskey (1982) alternated sessions of temperature biofeedback and no feedback as a means to increase the duration of the temperature elevations achieved through biofeedback training. However, at risk of labouring the point, it must be noted that the results of any empirical design that employs biofeedback as a strategy of control in its own right, are severely undermined by the inappropriate theoretical stance taken: Biofeedback may only be viewed as an aid, rather than a means to, autonomic control.

Later work, in which biofeedback was still seen as a "pure" treatment, was pioneered by an American group lead by Robert Freedman. Not only did the group claim to demonstrate positive effects of biofeedback in the control of finger skin temperature, but also, with the aid of sympathetic nerve blocks, identified the presence of a beta-adrenergic vasodilating mechanism at work during biofeedback mediated temperature control (Freedman et al, 1984a, 1988a). The validity, or otherwise, of such a mechanism aside, the group fail to see that the identified non-neurally mediated mechanism must have been initiated by some underlying technique(s) adopted during the biofeedback training sessions.

Guglielmi and Roberts (1994) and Okouchi (1991) plainly view biofeedback as a means to control finger temperature without conscious awareness on the part of the biofeedback trainee. They equate biofeedback with pharmacological treatments in that those treated need not understand the underlying mechanism of treatment for the effects to be applied to the peripheral vasculature ie. it is clear that the patient does not need to be consciously aware of the specific vasodilatory mechanism of nifedipine (for example) for it to exert its effects. Similarly, it is assumed that conscious awareness is not required in biofeedback induced control of finger skin temperature. In line with this, the researchers above promote double-blind studies of the effects of biofeedback. As noted above, however, biofeedback provides the

trainee with a means to evaluate the efficacy of chosen strategies of temperature control. Consequently, double-blind studies must hinder the acquisition of such control; their use is misplaced in the analysis of the effects of biofeedback treatment of finger skin temperature.

As to the use of "pure" biofeedback in Raynaud's research, early work focused on single case studies. For example, Schwartz (1973) described the use of temperature biofeedback in the alleviation of the cold feet of a male Raynaud's sufferer; however, the benefits of treatment reported were only subjective in nature as the investigation lacked objective measures of temperature change. Similarly, Blanchard and Haynes (1975) describe a single case study of a 28 year old woman, infrequently suffering from the vasospastic attacks (approximately 1 per month), who learned to increase her hand temperature by 3.7 F through 12 feedback sessions; moreover, at regular follow-up assessment sessions, she maintained that her clinical symptoms had virtually disappeared. However, in the latter study, the effects of seasonal temperature changes were not addressed with regard to the management of symptoms.

However, the articles reviewed above manipulated biofeedback as a conditioning treatment. Consequently, other than providing suggested formats of future experimental design, work by Adair and Theobold (1978), Sappington (1977) and Sappington and Fiortio (1985) - who positively review the effects of biofeedback in the treatment of Raynaud's Phenomenon -actually, accomplishes very little. Indeed, if the true effects of biofeedback are to be disclosed, some experimental manipulation of biofeedback in conjunction with behavioural strategies of temperature control are required. Such empirical studies are reviewed below.

6.5 BIOFEEDBACK AND RELAXATION TECHNIQUES IN THE CONTROL OF FINGER SKIN TEMPERATURE: A REVIEW OF THE LITERATURE

As the behavioural approach gained in credibility, larger groups of Raynaud's patients were recruited. Freedman et al (1979, 1981) treated 10 Raynaud's Disease and Phenomenon patients with 12 sessions of "pure" finger temperature biofeedback and reported significant subjective symptom improvement at one year follow-up, and May and Weber (1976) reported symptom improvement and associated control

of finger skin temperature through biofeedback training. Further, research began to address the comparative effects of "pure" biofeedback with those of other techniques, initially with non-symptomatic volunteers, and then with Raynaud's patients.

Keefe (1978) assigned 60 female students to one of six combinations of response specific instructions (ie. "increase your finger temperature"), thermal suggestion (ie. "imagine your hand is getting warmer"), or rest, with or without temperature feedback training. The results indicate that thermal suggestion (with and without biofeedback), and response-specific instructions with temperature biofeedback are conducive to temperature control. That "biofeedback with rest" is not associated with temperature control, bodes well with the current state of thinking that biofeedback is an aid, rather than a means, to autonomic control. Further, the work suggests a potential strategy of control in the guise of thermal imagery or thermal suggestion. In line with this, Grabert et al (1980) demonstrated that visual temperature biofeedback training combined with thermal imagery is associated with greater autonomic temperature control than either imagery or biofeedback alone.

Convinced of the beneficial effects of biofeedback, Freedman et al compared "pure" temperature biofeedback with Autogenic Training, EMG biofeedback and response-specific instructions (1983b, 1985b: follow-up), and "pure" temperature biofeedback with Autogenic Training (1993). In keeping with their beliefs, only temperature biofeedback was associated with finger temperature elevations - a finding which appears to oppose the suggestion that biofeedback is not a valid treatment in its own right. However, a number of points should be considered in the evaluation of these papers. Firstly, as described above, the association between temperature control and response-specific instructions has been dismissed (Keefe, 1978); therefore, temperature deviations would not have been expected within such a treatment condition. Secondly, the connection between EMG biofeedback (ie. muscle tension) and finger temperature is not immediately obvious unless carefully explained. No such explanation was offered in this study; thus participants would have been unable to use the EMG feedback tone as an evaluative mechanism of some chosen strategy of temperature control. Thirdly, the described execution of Autogenic Training suggests inadequate learning of the skill of relaxation. The reader is asked to compare the following description of training, taken from Freedman et al (1983b), with the Autogenic

explanation was offered in this study; thus participants would have been unable to use the EMG feedback tone as an evaluative mechanism of some chosen strategy of temperature control. Thirdly, the described execution of Autogenic Training suggests inadequate learning of the skill of relaxation. The reader is asked to compare the following description of training, taken from Freedman et al (1983b), with the Autogenic Training protocol described in chapter 8: *"Autogenic training subjects listened to the tape recorded instructions of Surwit, Pilon and Fenton (1978) for 3 min after which they repeated the phrase 'my hands are warm and heavy' for 21 min."* (p 684). In contrast, the instructions given to the temperature biofeedback participants were conducive with temperature control: *"The operation of the feedback apparatus was explained and they* [participants] *were told to increase the temperature of their fingers as much as possible using the tone as a guide."* (p684: emphasis added). Participants had been indirectly instructed to use some other strategy of temperature control, and, to assess the efficacy of that treatment through reference to the biofeedback tone. Consequently, though assuming some conditioning effect, Freedman et al have actually provided an example of biofeedback as an aid, rather than means to temperature control.

As to the treatments in relation to Raynaud's, Jacobson et al (1973) investigated the effects of biofeedback and autohypnosis in the management of Raynaud's symptoms: a poor hypnotic subject, who had a three year history of Raynaud's Phenomenon, was trained in the technique of autohypnosis, and as this had limited effects on his finger skin temperature and blood flow, temperature biofeedback was introduced as an operant treatment ie. the participant was instructed to relax and then "*increase his hand temperature by whatever method he desired*" (p740). The authors claim that temperature elevations and colour changes were achieved through the participant's conditioned ability to "*drive the machine*". However, the effects of autohypnosis may have been slow to materialise. Consequently, through the autohypnosis training, the participant may have been equipped with a strategy of temperature control. Along similar lines, therefore, Kelso et al (1985) compared the effects of temperature biofeedback and "modified" autogenic training in two female students, suggesting temperature control benefits of the former treatment, but not the latter. That said, although the order of treatment presentation was different for each participant, equipment failure undermines the validity of the "Biofeedback-Autogenic Training" participant's data. In the

"Autogenic Training-Biofeedback" condition, therefore, the participant is provided with a (poorly acquired) strategy of temperature control, for use with biofeedback, that is clearly associated with minimal digital temperature elevations.

Two research groups, lead by Guglielmi and Freedman respectively, further address the issue of the comparative role of "pure" biofeedback and other behavioural treatments in temperature control of larger groups of Raynaud's sufferers. Guglielmi (1980) compared temperature biofeedback, EMG biofeedback, and no treatment in 36 sufferers of Raynaud's Disease. Subjective diary data revealed a decline in attack frequency across all treatments - though no statistically significant physiological changes were recorded. Guglielmi therefore suggests a placebo explanation of the subjective effects; indeed, as previously stated, symptom report is associated with subjective symptom reduction without behavioural, medical or surgical intervention (Keefe, 1978). Consequently, in a later study, Guglielmi et al (1982) introduced a double blind design (ie. all involved in the data collection (clinicians and participants) were naïe to the nature of the study). The results revealed no significant differences across treatments ie. no objective improvements were measured, and subjective benefits were maintained. Here, however, proponents of biofeedback as an aid, rather than means, to temperature control would have predicted such an outcome in that "pure" temperature and EMG biofeedback may be viewed as "non-treatments" on a par with the no treatment condition.

A number of well designed studies by Freedman and co-workers assessed the relative roles of biofeedback and relaxation in the treatment of Raynaud's (eg. Freedman et al, 1984b, 1991). However, a particularly good example is provided by Freedman et al (1983c): subjective symptom and ambulatory finger temperature data were collected both prior to, during and after the treatment programme; the study protocol included adequate finger temperature stabilisation and baseline periods, and attempted to minimise movement effects; and, moreover, pre- and post-treatment measures of ability to raise finger skin temperature and response to cold were employed. The comparative effects of autogenic training, EMG biofeedback, temperature biofeedback and temperature biofeedback under local cold stress conditions (ie. a stimulus progressively cooled to 20 C) indicate that only the temperature biofeedback and cold stress temperature biofeedback are

associated with statistically significant finger temperature increases (approximately 0.5 degrees C) from baseline during training, and that only temperature biofeedback participants are able to increase digital temperature at will both at post-treatment and 1 year follow-up. Subjective attack frequency was also significantly lowered in the two temperature biofeedback groups, and post-treatment ambulatory temperature recordings indicated that vasospastic attacks were elicited at lower ambient temperatures for the temperature biofeedback treatment groups than the other two groups. Does this paper demonstrate therefore, that biofeedback is a valid, powerful treatment in its own right ? Closer inspection of the procedure adopted suggests not; indeed, the objections voiced earlier in regard to the work of Freedman et al (1983b), also apply here: the connection between EMG biofeedback and finger temperature was not clearly explained; the skill of autogenic training was not adequately taught or explained to the participants (personal experience of autogenic training predicts an amount of scepticism on the part of those initially introduced to the technique); and finally, in both temperature biofeedback conditions, participants were instructed to use biofeedback as a guide to temperature control such that the use of personal strategies of temperature control was sanctioned by the experimenter. Consequently, this paper supports the use of biofeedback procedures as an aid to temperature control. It should be noted that Freedman's early studies differ from his later work only in terms of the technology employed in measuring and assessing the "effects" of treatment. For example, Freedman (1989b) used Venous Occlusion Plethysmography to evaluate changes in blood flow during behavioural training sessions; Freedman et al (1983c), thermometry. In the 1989 paper, slight temperature increases elicited by autogenic training were not associated with increased peripheral blood flow. Freedman, and others of the same camp, would consider this an indication of the redundant nature of Autogenic training in the management of Raynaud's symptoms; however, such a finding might also be indicative of inadequate training in the AT technique, for example. It would be inappropriate to criticise the Freedman group experimental procedure as, generally, it is methodologically sound; however, the true benefits of such well designed methodological studies, are lost through poor evaluation of results - unavoidable owing to inappropriate regard to the role and capabilities of biofeedback.

In summary, therefore, a large body of often well conducted empirical research misses the point of the role of biofeedback in the management of Raynaud's symptoms; thereby leading to the inappropriate interpretation of results. It can not be stated too often that Biofeedback is an aid, not a means, to digital temperature control.

Though the papers above have been dismissed as being of limited use in the evaluation of behavioural means of treatment, the work does provide the researcher with an evaluation of a range of methodological techniques to apply to further research in the area. For example, the need for adequate adaptation periods and baseline measures of temperature. Such methodological implications will be discussed further in the next chapter; however, prior to that, research adopting a "window" or "mirror" model of biofeedback will be reviewed.

Kluger et al (1985) claim to have compared the finger temperature control effects of temperature biofeedback, autogenic training, and finger pulse volume biofeedback in 29 non-symptomatic university undergraduates; thereby assuming some specific treatment effect of biofeedback. Closer inspection of the available procedural information, however, reveals the implicit suggestion of thermal imagery as a technique of temperature control in the two biofeedback conditions: *"They were informed about the exact procedure and asked to relax while concentrating on the task. The use of somatic strategies (ie. tensing muscles) was discouraged, but general cognitive strategies (thinking about warm sensations) were acceptable"* (p164: emphasis added). The results revealed no treatment group differences in measured temperature effects. This might suggest firstly, that autogenic training is a viable strategy of temperature control, and secondly, that although thermal imagery alone is less useful, the role of biofeedback, in "fine tuning" the strategy, greatly improves the effects of thermal imagery. (Although, clearly, without adequate non-treatment control groups, such a suggestion would be difficult to prove). In a similar vein, Ford et al (1983) looked at the effects of relaxation and biofeedback in a range of disorders including Raynaud's, tension headache, migraine and essential hypertension (48 participants suffering from Raynaud's Disease or Phenomenon). Subjective benefit was reported across all conditions; indeed, treatment was associated with a reduction in Raynaud's symptom frequency, severity and duration. However, the treatment effects reported were subjective

in nature, and as previously documented, such reports are notoriously subject to bias. Despite problems with non-compliance, Burke et al (1991) noted modest symptom improvements in two cases of severe rheumatoid arthritis following treatment with relaxation combined with temperature biofeedback, suggesting benefits of such an approach in the management of Raynaud's symptoms.

However, such papers do little to isolate the specific effects of biofeedback above those of acquired strategies of control. One might assume that combined biofeedback and strategies of temperature control (eg. relaxation) would yield larger temperature elevations, and symptom reduction, than the acquired strategies in isolation. Indeed, the comparison of biofeedback and relaxation, with relaxation alone, in 4 lupus patients supports this assumption (Bertino et al, 1991). However, an adequate review is difficult, owing to the limited procedural information presented - though such an obstacle is not present, however, in a limited number of published articles assessing the effects of biofeedback and relaxation in the control of finger skin temperature. For example, Surwit et al (1978) suggest that autogenic training effects are not enhanced by temperature biofeedback as, in their study, subjective clinical improvements in Raynaud's symptoms were reported by all 30 trainees ie. those trained in Autogenic Training, and those trained in Autogenic Training and temperature biofeedback. Moreover, subjective improvements were associated with treatment-related ability to maintain hand temperature under cold stress ("cold chamber"). However, the work is marred by a) lack of adequate baseline measures of skin temperature from which to objectively assess treatment-related changes and an adaptation period limited to 10 minutes duration; by b) failure to teach the skill of autogenic training to the participants: autogenic-type phrases were presented orally such that the participants were not actively involved in the training. Consequently, they were not relating <u>their</u> mind to the state of <u>their</u> body; and indeed, an unmotivated volunteer would have been able to ignore the instructions completely if (s)he so chose; by c) failure on the part of the authors to describe the association between autogenic training and the visual temperature biofeedback signal to the participants; and by d) confounding with instructions to increase digital skin temperature at will using proprioceptive cues of warmth and digital pulsation as indirect biofeedback. The latter clearly prevents evaluation of the exact role biofeedback plays in improving temperature elevation through Autogenic training. That aside, the longer term effects of treatment were evaluated in 19 of the initial

sample (Keefe et al, 1979b). Questionnaire and diary data revealed that patients maintained a lower attack frequency at follow-up, but that cold stress test performance had returned to pre-training levels. Moreover, the results are supported by Surwit et al (1980), and by Keefe et al (1980) who assessed the effects of autogenic training, autogenic training with temperature biofeedback, and progressive relaxation in 21 female participants through patient self-report and cold stress test. Here again, however, the treatments were confounded with indirect biofeedback in the form of instructions to increase hand temperature at will using proprioceptive cues to success; moreover, the effects of biofeedback in conjunction with progressive relaxation were not assessed, and participants received only 3 sessions of behavioural training.

In contrast, Jacobson et al (1979) taught a relaxation technique to 12 participants prior to the introduction, for half of the volunteers, of two bi-weekly 5 minute temperature biofeedback training sessions. Results indicated that the relaxation group was better able to increase digital skin temperature than the biofeedback-assisted relaxation group, and that subjective symptom improvement was not correlated with ability to increase temperature in the laboratory. Here again, however, evaluation of the research is limited by inadequate published details of the training protocol.

Therefore, the trend appears to suggest that biofeedback does not ameliorate the effects of relaxation or other strategies of digital temperature control (and indeed, that biofeedback may actually hinder the treatment effects of relaxation training). That said, this review clearly supports an effect of behavioural treatment in temperature control with both non-symptomatic and Raynaud's volunteers; however, a range of methodological failings (including lack of adequate adaptation and baseline periods, the confounding of factors such as gender in experimental design, insufficient training in behavioural techniques, failure to include non-treatment or other appropriate control groups, and, of course, the assumption that biofeedback is a means of operant conditioning valid as a treatment in its own right) undermine the validity of many of the empirical findings, and prevent analysis of the true contribution of biofeedback in the behavioural approach to the treatment of Raynaud's symptoms.

Why such methodological failings have continued to appear in two decades worth of published material, perhaps lies with the interpretation attached to empirical results by the authors; indeed, on initial inspection, "pure" biofeedback as a treatment alone is apparently successful as a means to manage Raynaud's symptoms - even where behavioural techniques are inadequately taught to the participants. Indeed, "if it ain't broken, why fix it ?" Consequently, the literature survey presented here, implies that an adequate assessment of the relative roles of biofeedback and behavioural temperature control strategies is yet to be published. However, on a more positive note, the literature review above has identified a number of sound methodological practices applicable to further research in the area. The limitations and benefits touched on in this chapter will be expanded upon in the following chapter as an introduction to a large empirical investigation of the behavioural approach.

Chapter 7

METHODOLOGICAL AND DESIGN CONSIDERATIONS

7.1 INTRODUCTION

The current behavioural literature, as reviewed in chapter 6, suggests that there is, as yet, no adequate empirical investigation of the efficacy of the behavioural approach to treatment. Indeed, as previously described, a range of methodological flaws undermine the validity of claims as to the efficacy of the behavioural approach: the main shortcoming being the assumption that biofeedback is a valid treatment in its own right - rather than an aid to symptom control. Indeed, for a number of published articles, this fundamental fault is the only blot in an otherwise commendable experimental design. Therefore, the aim of this chapter is to outline the main empirical failings and strengths of previous research as an introduction to the study described in chapter 8.

7.2 EVALUATION OF BEHAVIOURAL TREATMENTS FOR RAYNAUDS: METHODOLOGICAL CONSIDERATIONS

The methodological flaws of previous Raynaud's behavioural research may be categorised under two headings: firstly, theoretical problems (eg. the biofeedback model adopted, the strategies of temperature control employed and procedural training provided, and the positioning of EMG electrodes as both an objective measure of muscular relaxation and as a feedback site); secondly, empirical flaws (eg. failure to implement adaptation and baseline periods, the confounding of variables, failure to employ comparison control groups, the employment purely of subjective measures of treatment effects, and the issue of compliance). Clearly, there may be some overlap between the two.

As noted above, the fundamental flaw inherent in Raynaud's behavioural research is that of adoption of an inappropriate theoretical model of biofeedback. At risk of labouring the point, biofeedback apparatus merely provides an aid, rather than means to finger temperature (and symptom) control. One

can not simply learn to control finger temperature from the evidence provided by a biofeedback tone unless some strategy of control is implemented. Clearly, this strategy may be self-imposed ie. each participant might, unprompted, chose a technique of their own eg. "thermal/thematically-relevant imagery[25]", or "nothing" (such that temperature is not controlled). However, for experimental validity ie. to control for the possibility of different strategies being used within one treatment group (and to avoid the possibility of participants merely listening to, rather than acting on, a change in biofeedback tone), it is helpful to introduce some technique of temperature control such that the experimenter is aware of the strategy being employed. To reiterate, biofeedback must be clearly introduced as nothing more than an aid to temperature control to be used in conjunction with an appropriate (taught or personal) strategy of control ie. the trainee must be fully aware of the role of biofeedback as an aid to the effects of that strategy. In line with this, the employment of double-blind procedures as an avenue to the "true" effects of biofeedback is clearly inappropriate: if the trainee is not fully aware of the relationship between the biofeedback signal, the taught (or self-appointed) strategy of control, and finger temperature, how could (s)he possibly master voluntary finger temperature control ?

As to choice of strategy, techniques chosen should be comparable in terms of the requirements of acquisition ie. previous research has, for example, often compared Autogenic Training with thermal imagery. Though both are, arguably, applicable to the control of finger skin temperature, the two differ fundamentally on the basis of training requirements to mastery. Indeed, (thermal) imagery is something that we all indulge in - when asked to imagine oneself in a warm bath or lying on a warm beach, we can all immediately conjure up the sensations associated with such an experience. In contrast, the concept of AT is generally new to the trainee; the ideas and procedures often do not sit comfortably with expectations of a relaxation technique. Indeed, both the skill, and to a degree, personal acceptance of the technique, have to be acquired. Therefore, the therapist can not expect similar results when (s)he asks the trainee to use AT to control finger skin temperature as when (s)he suggests thermal imagery; indeed, in the former case, the treatment effects of AT will be confounded with those of learning a new skill.

[25] eg. imagining increased blood flow to the fingers.

The fact that AT has to be learned, has implications for the number of actual training sessions required. It is not enough to gain a brief insight into AT (or any other relaxation treatment) if it is to be useful in the voluntary control of finger skin temperature. In much (though not all) of the reviewed research, perhaps 2 or 3 brief sessions of AT training are provided. Though not equivalent in terms of the degree of skill required, learning to drive may prove a useful analogy. After the second driving lesson, with continual prompting from the instructor, the novice driver may be able to start the car, turn left, and bring the car to a poorly positioned standstill. If the novice was provided with no further instruction (ie. the AT training were to end prematurely), the skill of driving would not be mastered and, quite rightly, the driver would fail his/her driving test. The point being that any acquired technique of temperature control must be adequately taught if its true benefits are to be evaluated. A related area is that of the requirement to practice training exercises between training sessions. Few papers document such a need. However, practice is essential to the acquisition of any new skill - AT and other relaxation procedures being no exception.

A final theoretical problem lies with the choice of EMG electrode positioning. To my knowledge, no empirical investigation of the behavioural effects of Raynaud's has actually measured muscle tension at an area that may have immediate, be they short term, effects on peripheral blood flow. As previously outlined, the frontalis muscle is generally adopted as the site of EMG probe attachment. Though useful as an index of relaxation of the head[26], and as an index of general relaxation in conjunction with other measurement points on the body, frontalis EMG is of no specific benefit in the evaluation of behavioural treatments for Raynaud's (though it may for headache). EMG measurement of some area closer to the site of symptom manifestation eg. the flexor carpi ulnaris or flexi carpi radialis muscles of the forearm, may, in contrast, prove more relevant. (In this empirical investigation, given the importance of minimising the effects of gross movement on recorded muscle tension, the ulnaris muscle was chosen over the radialis muscle as in the latter muscle, small movements of the fingers have a greater impact on muscle tension).

[26] ... if one controls for the effects of eye and facial movements on the trace.

At the more basic level of empirical flaws, a number of (early) studies fail to include adequate adaptation and baseline periods to enable participant finger skin temperature to both adjust to and stabilise to laboratory conditions. It is surely obvious that those hoping to record treatment-related finger temperature increases, or compare pre- and post-treatment baseline hand temperatures, require a relatively constant temperature baseline as a starting point - particularly during or just after the elicitation of vasospastic attacks[27]. Indeed, at the initial stages of an attack, laboratory ambient conditions may warm the whole Raynaud's body, reduce sympathetic innervation to the peripheral blood vessels, and might, therefore, quicken the occurrence of the hyperaemic phase of the attack. In contrast, if a patient enters the laboratory during the hyperaemic phase, the throbbing, warm fingers will gradually lose heat to the relatively cooler environment. Further, the implementation of controlled ABA experimental designs, in which pre-treatment subjective and objective measures of effect are repeated once the treatment programme has been completed, would not be possible without adequate pretreatment baselines of both subjective symptom details and current medical treatments, and of objectively measured control of finger skin temperature.

The confounding of variables, briefly touched on within the realm of theoretical problems, further constitutes a serious empirical flaw. The reader may recall Keefe and Gardner (1979) in which the number of behavioural training sessions and gender were experimentally undifferentiated. Sound methodological experimental design dictates that the researcher need control for non-manipulated factors that might account for experimental findings. Therefore, in assigning participants to treatment groups, it is important to ensure, as far as possible, that individual group membership does not differ significantly on factors such as gender, age, onset and diagnosis of Raynaud's, or procedural instructions given to participants (ie. beyond those required to differentiate between treatment groups). Moreover, it is not enough to show that a particular treatment is associated with some factor of temperature control. One must also demonstrate that where a treatment is associated with the control

[27] Personal experience (chapter 8) enables me to state that attacks are not uncommon on arrival at the laboratory. Behavioural treatment studies tend to occur during the winter months: the sudden change from the warm ambient conditions of a car, to the freezing conditions of the cold morning air, may elicit vasospasm. Where trainees are seen at the time appointed, visual evidence of an attack is available to the clinician.

of autonomic process, similar control is absent in participants not trained in the behavioural technique: the manipulation of non-treatment Control groups is required.

A related point. The methodological design of some previous investigations allow non-specific factors to be indicted in the explanation of results. For example, the employment entirely of subjective assessment procedures invites biased evaluative responses from participants. Factors such as the need to validate time spent by the therapist, or to constructively account for personal time and effort expended in the pursuit of treatment, may encourage a positive description of the effects of that treatment. Given the association with such non-specific effects, one might wonder why subjective measures are employed at all: subjective evaluation of treatment is essential to the evaluative process as a whole ie. it is of little practical use for a treatment to be associated with an increase in digital skin temperature if, subjectively, the treatment offers no symptomatic benefit. Therefore, the researcher would be advised to combine subjective and objective measures of effect. However, even with the employment of objective measures, non-specific factors may still influence the results of a treatment study. For example, the non-specific psychological effects of the therapist's attention, or the therapist's underlying suggestion that the treatment offered will be of benefit, may reduce anxiety about the condition ("someone is actually taking an interest"; "At last, I have a technique to lessen the duration, severity, and frequency of my symptoms"). This, in turn, might have a positive knock-on effect in reducing the role of attack anticipation, and thereby improve symptoms for a limited period of time. Indeed, initially, participants may genuinely believe that their symptoms have improved. However, if high expectations are not met (eg. the treatment offered is not a cure-all), worries that the treatment might be a waste of time, that the benefits are not worth the substantial personal effort required, or even that yet another treatment has failed ("Nothing works. I'm stuck with this affliction for life."), may increase the effects of anticipation at the onset of vasospastic attack, and perhaps return symptoms to pre-treatment levels.

Thus, expectation may produce (or increase) objective symptom improvement - irrespective of the benefits of the actual treatment offered. Other non-specific factors might emerge from the friendship built up between patient and therapist ie. the participants might enjoy and look forward to attending the

training sessions. The pleasure gained from the experience might, in the short term, improve both subjective and objective measures of treatment (feeling comfortable in the treatment situation must aid relaxation, and therefore, potentially, the effects of treatment). Clearly, such non-specific factors are not readily controlled for ie. purposefully not setting up a rapport with participants, for example, though reducing the non-specific effects related to patient-clinician friendship, may have deleterious effects on patient attendance at a series of training and assessment sessions. However, non-specific effects are generally associated with symptom improvement of only limited duration; therefore, in line with the methodological design of a number of later behavioural studies, "non-specific" explanations of empirical results might be identified through the evaluation of longer term effects of treatment eg. at 3 months, 6 months, a year, or more after the training period has ended.

To return to the effects, on patient compliance, of attempting to control for variables that might permit "non-specific" explanations of results: the researcher must strike a balance between the design of a strict, water-tight empirical investigation, and the needs of the clinical group. The measure of treatment efficacy relies quite heavily on patient attendance at meetings, and on the patient taking the treatment seriously (eg. practising treatments as required); therefore, patient comfort must be a priority. Although in the short term, unpleasant, painful or boring procedures during treatment and/or assessment may be tolerated (or a poor rapport between the therapist and patient may be overlooked), in a larger scale study, such negative effects may not be tolerated, and patients may simply withdraw from the study. Therefore, in addition to adopting techniques to improve patient compliance (such as those outlined in chapter 5), some measure of patient enjoyment of both the treatment exercises, and the whole treatment process, along with subjective measures of motivation, may be of use in maintaining patient numbers and in the assessment of treatment effects.

Despite the range of methodological and theoretical flaws inherent in the Raynaud's behavioural research, most empirical investigations include some sound methodological practices. For example, ensuring that skin temperature control is assessed under relatively constant laboratory conditions (such that skin temperature fluctuations during training are attributed to the behavioural trainee rather than to some change in ambient temperature or humidity), and, to improve the reliability of the objective

measures, the introduction of a range of techniques for the assessment of finger skin temperature control.

The behavioural approach described in chapter 8, acknowledges the flaws and sound methodological practices outlined above. For example, the study rests on an ABAA design in which pre-treatment measures are compared with post-treatment measures, and, to allow for the effects of non-specific factors, the post-treatment measures are repeated at follow-up. Moreover, assessment is both subjective and objective in nature, and outcome is compared in both treated and non-treated Raynaud's patients[28].

As to the evaluative procedures adopted, questionnaire and face to face questioning is supplemented by objective tests of voluntary control of finger skin temperature under normal laboratory, cold stress, and ambulatory conditions. Practical considerations prevent the utilisation of physiological measures of heart rate, breathing etc. Indeed, although modern electrocardiographs are small and compact, and probes may be attached directly over the heart area, pulse rate monitors available in the Psychology department are large, noisy, and to the uninformed, rather unnerving. Though, this in itself is not an insurmountable problem (ie. patients will adapt to the noise of the machinery over a period of training, and a clear explanation of the machinery, will help reduce patient worries), the technique of probe attachment does not sit well with an objective measure of finger skin temperature. Indeed, the probe is attached to a volunteers' finger, and detects finger pulse through the refraction of a beam of light shone directly onto the finger. External light needs to be minimised; therefore, the whole hand is enveloped in a black velvet bag throughout the course of measuring - which must influence finger skin temperature of both the measured and contralateral hands. The use of nasal thermocouples is avoided in line with the need to maintain a high level of patient compliance. However, as noted in chapter 5, it is by no means given that the employment of measures of physiological arousal (eg. heart rate and breathing) offer any evaluative benefit with regard to the role of behavioural strategies of Raynaud's symptom

[28] Non-symptomatic patients are not included in the design as the purpose of the investigation is to show that biofeedback will improve on the efficacy of relaxation treatment in the management of Raynaud's symptoms - the ability of non-symptomatic volunteers to control hand temperature is not relevant to this thesis.

management. Therefore, their exclusion does not severely impair the validity of the experimental design.

An area of great concern in the objective analysis of any treatment is that accurately calibrated measurements at the start of a study, remain accurate both during, and at the end of the empirical investigation. Unless accuracy is maintained, the true effects of treatment may not be assessed. Indeed, with temperature logging equipment, for example, although accuracy of the logger equipment may remain intact, sensitivity of the thermistor probes may alter with time (eg. Taub and School, 1978). Therefore both the logger and probes should, ideally, be periodically checked for accuracy. However, within the time scale of the study described in chapter 8, it was not admissible to have the logger equipment checked or recalibrated by the manufacturer during the data collection period. (Indeed, the equipment was used 3-4 times daily over a 7 month and later 2 month period). However, as two separate temperature and EMG loggers were utilised during data collection, the potential effects of logger "wear" on the accuracy of recorded data were cut by half. To similarly reduce the effects of use on probes, fresh probes should preferably be used at every session. With EMG probes, this presented no problem; and indeed, was a necessity given the nature of probe attachment. However, the cost of thermistor probes meant that some test of probe accuracy was required periodically throughout the course of the study. Indeed, prior to, and during the study, calibration of temperature probes was assessed[29] through logging the temperature of boiling water (100 C) and the temperature of ice in water (0 C). This assessment procedure was repeated at monthly intervals, and where the logged temperature differed from the expected temperature, new thermistor probes were used. Clearly, this technique falls somewhat short of 100% accuracy; however, its use does ensure continual monitoring of the state of the thermistor probes and a relatively high turn over of probes.

As noted in chapter 5, ambulatory monitoring of finger temperature differs from laboratory monitoring in that in the laboratory, the researcher may readily interpret temperatures in relation to participant activities. With ambulatory monitoring, in contrast, the participants are asked to provide some written

[29] - following an adequate baseline.

account of their activities ie. complete a diary of activities, of vasospastic attacks and of the use of hand and body warming aids during the monitoring period. Standard monitoring diaries provide a measure of activities and symptoms retrospectively over a period of an hour to enable description of the relationship between measured temperature fluctuations, symptoms and volunteers' activities. To improve the accuracy of monitoring data, the design of diaries requires some time scale of events such that symptoms may be directly related to ambient conditions and activities ie. activities should be recorded in chronological order. Moreover, the clinician requires information such as the participants' location, activities, and use of stimulants known to influence peripheral blood flow eg. tobacco, alcohol, caffeine, during monitoring. Further, an index of volunteers' mood may be useful in the interpretation of data as feelings of frustration or annoyance may have some effect on circulation in general.

The main purpose of ambulatory temperature monitoring is to assess the ambient temperatures and conditions associated with an attack of Raynaud's, and to see whether these change following a course of treatment. Therefore, in the subjective documentation of vasospastic attacks, precise details are required. For example, the benefits of treatment may be overlooked if the severity or duration of an attack, for example, is not assessed; indeed, a treatment may have no effect on the ambient conditions required to precipitate an attack, or on the frequency of attacks, but merely provide the trainee with some coping strategy to limit the duration, or to (unconsciously) reassess the severity of attacks. Therefore, the diary data collected needs to be quite rich in detail. However, the point of ambulatory monitoring is to objectively assess treatment effects away from the laboratory when participants are, as far as possible, carrying out their normal, everyday activities. Consequently, it is important to maintain a balance between richness of data collected, and the degree to which collection of that data interferes with the normal activities of participants. In line with this, the format of the diary should be conducive to rapid completion that requires little in the way of thought on the part of the participants - perhaps with a range of simple, well explained scales or boxes to tick etc.

The main areas of concern in the design of the empirical investigation of this thesis have, therefore, been identified, and will be put to good effect in the next chapter. Although the reader is aware that the purpose of the main investigative study of this thesis is to evaluate the relative efficacy of relaxation,

The main areas of concern in the design of the empirical investigation of this thesis have, therefore, been identified, and will be put to good effect in the next chapter. Although the reader is aware that the purpose of the main investigative study of this thesis is to evaluate the relative efficacy of relaxation, and relaxation supplemented by biofeedback, as an aid to finger temperature control and subsequent symptom improvement, (s)he has little idea of the range of hypotheses to be assessed in the course of the investigation. Therefore, the experimental hypotheses and assumptions are provided in list form - in relative order of importance - in the following section.

7.3 HYPOTHESES

a. Initial baseline ability to control finger skin temperature (under laboratory, cold stress and ambient conditions) will not differ across the experimental and non-treatment control participants.
b. The behavioural treatment participants will report subjective seasonally adjusted symptom improvement at "post-treatment" assessment. The control participants will not.
c. All behavioural treatments will objectively improve ability to control finger skin temperature (under laboratory, cold stress and ambient conditions) at "post-treatment" assessment. Such an improvement will not be shown by the Control participants.
d. The combined effect of biofeedback and relaxation on subjective symptom report will be greater than that of relaxation alone.
e. The combined effect of biofeedback and relaxation on control of finger skin temperature (under laboratory, cold stress and ambient conditions) will be greater than that of relaxation alone.
f. The degree of effect of the behavioural treatments will be a function of patient compliance to practice instructions and/or enjoyment of the treatment exercises.
g. Raynaud's Phenomenon patients - given the association between symptoms and identified physical abnormalities - will obtain less benefit from the behavioural approach when compared with Raynaud's Disease patients.

Chapter 8 A BEHAVIOURAL APPROACH TO THE MANAGEMENT OF RAYNAUD'S SYMPTOMS

8.1 METHOD

Design

The purpose of the investigation was to evaluate the subjective and objective outcome of Behavioural treatments for Raynaud's Disease and Phenomenon. The efficacy of treatment was assessed subjectively by questionnaire, and objectively through tests of voluntary control of finger skin temperature under normal and cold stress conditions, and through periods of finger skin temperature monitoring in everyday situations. 40 Raynaud's sufferers were assessed prior to treatment. 30 of these volunteers went on to receive one of six behavioural treatments; the remaining 10 acting as non-treatment control participants. The treatments under investigation were Applied Relaxation (AR), Applied Relaxation with finger temperature biofeedback (ART), Applied Relaxation with flexor carpi ulnaris muscle EMG biofeedback of the non-dominant arm (ARE), Autogenic Training (AT), Autogenic Training with finger temperature biofeedback (ATT), and Autogenic Training with flexor carpi ulnaris muscle EMG biofeedback of the non-dominant arm (ATE). The assessments were repeated for both experimental and control participants on completion of the treatment programme, and in a follow-up assessment 4-5 months after treatment.

Participants

40 Raynaud's sufferers ranging in age from 12 to 78 years were recruited for the study. 16 (5 male; 11 female) had taken part in the previous questionnaire study (chapter 5), and the remaining 24 (4 male; 20 female) were recruited through an advertisement in a local newspaper. All travelling expenses were reimbursed.

Nine participants had been diagnosed as suffering from Secondary Raynaud's. The underlying condition for four of these participants was Scleroderma; another had Scleroderma and Systemic Lupus Erythematosis (S.L.E.), two were unsure of the condition underlying their symptoms, and the

other two had CREST syndrome (oesophageal hypomotility, sclerodactyly and telangiectasia) and Carpel tunnel syndrome respectively. Two had undergone surgical procedures for Raynaud's (lumbar and/or cervical sympathectomy), and seven were taking prescribed medication for their symptoms (mostly the calcium channel blocker nifedipine). Six of the volunteers were diagnosed as Primary Raynaud's Disease according to the criteria of Allen and Brown (1932). These participants were not taking prescribed medication for their condition, but two had undergone a lumbar or cervical sympathectomy prior to the study. Fifteen were unsure of their diagnosis (due, perhaps in part, to the confusion surrounding Raynaud's terminology within the medical profession). Only three of these volunteers were taking prescribed medication for their condition (nifedipine, adelat, unknown). The remaining ten participants were undiagnosed, and taking no prescribed medication. Inclusion of these participants was based on subjective description of symptoms, a 2 year history of symptoms, and in all cases, visible evidence of attacks during the assessment sessions.

Participants were given the choice of receiving a Behavioural treatment, or of acting as a control for the duration of the study with the option of trying the most beneficial treatment once the study had been completed. Ten participants volunteered to be controls. The remaining thirty were, within the constraints of balancing the groups for gender, randomly assigned to one of six treatment groups: Autogenic Training (AT); Applied Relaxation (AR); Autogenic Training with finger temperature biofeedback or flexor carpi ulnaris muscle EMG biofeedback (ATT/ATE); and Applied Relaxation with finger temperature biofeedback or EMG biofeedback (ART/ARE).

9 volunteers (2 AT, 2 ATT, 1 AR, 1 ART, 1 ARE and 2 Control participants) withdrew from the study before the second assessment session because of ill health or the time commitment involved. Of the nine, three were undiagnosed, three were unsure of their diagnosis, and three were suffering from Secondary Raynaud's. One Secondary Raynaud's sufferer was taking prescribed medication for her condition. Of those who withdrew from the study, two (1 ARE, 1 ATT) did so before the first treatment session, three (2 AT, 1 ART) attended only the first session, two (AR, ATT) attended 3 sessions of treatment, and the Controls attended only the first assessment session. Further, 1 Control and one ATT participant failed to attend the final assessment session.

Demographic, symptom and treatment details are displayed in Tables 8.1 and 8.2 below.

Treatment Group	Gender	Mean Age in Nov 1993 (range)	Diagnosis	Mean duration of symptoms (range)
AT	1 m; 4 f	41.8 (12-67) yrs	1 S (S.L.E and Scleroderma), 2 US, 2 UD	23 (6-35) yrs
ATT	1 m; 3 f	44.8 (25-62) yrs	2 US, 2 UD, 1 S (Scleroderma)	21.8 (2-50) yrs
ATE	1 m; 4 f	38 (29-47) yrs	2 P, 1 US, 2 UD	19.2 (12-29) yrs
AR	1 m; 4 f	48 (35-57) yrs	1 P, 2 US, 1 UD, 1 S (Carpel Tunnel syndrome)	25 (3-57) yrs
ART	1 m; 4 f	54 (46-68) yrs	2 US, 1 UD, 1 S (Scleroderma)	9 (3-19) yrs
ARE	1 m; 3 f	52.3 (31-73) yrs	2 P, 1 UD, 1 S (Scleroderma)	25.8 (11-40) yrs
Control	2 m; 8 f	51.8 (26-78) yrs	1 P, 5 US, 1 UD, 3 S (C.R.E.S.T. syndrome, Scleroderma)	25.4 (2-67) yrs

Table 8.1: Demographic and symptom details of the treatment study participants (P = Primary Raynaud's, S = Secondary Raynaud's, US = those unsure of their diagnosis, UD = undiagnosed)

Treatment Group	Smokers	Prescribed medication	Surgical procedures	Experience of Relaxation techniques
AT	1 S	1 S (Nifedipine)	1 S: Cervical sympathectomy	1 UD: Yoga; 1 UD: breathing (Labour)
ATT	none	1 S (Nifedipine)	none	1 US, 1 UD: Yoga
ATE	1 US	none	none	none
AR	none	1 S (Nifedipine)	1 S: Carpel Tunnel; 1 P: Lumbar sympathectomy	1 US: breathing (Labour); 1 UD: Relaxation tape
ART	none	1 S, 2 US (Nifedipine); 1 S (Illoprost/ Warfarin)	none	2 US: Relaxation tape
ARE	1 UD	1 S (Nifedipine)	none	none
Control	none	1 S, 1 US (no details)	1 S, 1 P: Cervical sympathectomy	none

Table 8.2: Past and present treatments for participants' Raynaud's symptoms (P = Primary Raynaud's, S = Secondary Raynaud's, US = those unsure of their diagnosis, UD = undiagnosed)

Apparatus

THE MEASUREMENT OF DIGITAL SKIN TEMPERATURE

Digital skin temperature was measured in both ambulatory and laboratory situations. In both cases, temperature data were logged and stored at 10 second intervals in a Grant Instruments' Ltd. 1206

series Squirrel meter logger. Two Grant Instruments Ltd. EU-U-V2-2 temperature probes with type U thermistor and lemo plug were attached to the finger pads of the index and middle fingers of the non-dominant hand (unless these fingers were missing or ulcerated). The skin was first cleaned with surgical spirit to increase contact adhesion, and the thermocouples were secured with 2.5 cm micropore surgical tape. A Grant instruments Ltd. CM-U-V2-2 temperature probe (with type U mini thermistor and lemo plug), attached to the meter logger, recorded ambient temperature. The logged data was down loaded from the squirrel logger through a RS-232-C serial interface lead to an IBM RM Nimbus PC loaded with a Grant instruments Ltd. software package Grantware "Squirrelwise" version V.

When monitoring temperature in an ambulatory situation, the squirrel logger was carried in a camera case worn belted around the waist. The thermocouple wires were threaded up the sleeve and down the front of the outer layer of clothing as shown in fig 8.1.

Fig 8.1: The equipment used in the Ambulatory Temperature Monitoring of digital skin temperature

EMG MEASUREMENT

EMG was monitored using a myolog system which consisted of a Grant Instruments Ltd Squirrel meter/logger SQ8-4V-AS with a specification of 0-500mv, and an Aleph One Ltd. Myolink amplifier powered with four 1.2 volts RS seal nickel cadmium rechargeable batteries. [see fig 8.2]. Three electrodes were connected to the squirrel via the myolink. Two were Oxford Instruments silver/silver chloride electrodes, with a 1.5 cm diameter point of contact, attached to the extensor muscle of the non-dominant arm, and the third, a Syncor solid state "Tracet", with a $4cm^2$ area of contact, was attached to the tip of the ulnar at the elbow of the non-dominant arm as a ground electrode. The electrode site was cleaned with surgical spirit and then rubbed dry prior to electrode attachment. In parallel with the measurement of finger skin temperature, a sample of EMG data was averaged at 10 second intervals. The data were downloaded through an RS-232-C serial interface lead to an Acorn/BBC PC with a Grant Instruments Ltd. software package "Squirrel wise", and information was stored on 5.25" 3M floppy diskettes.

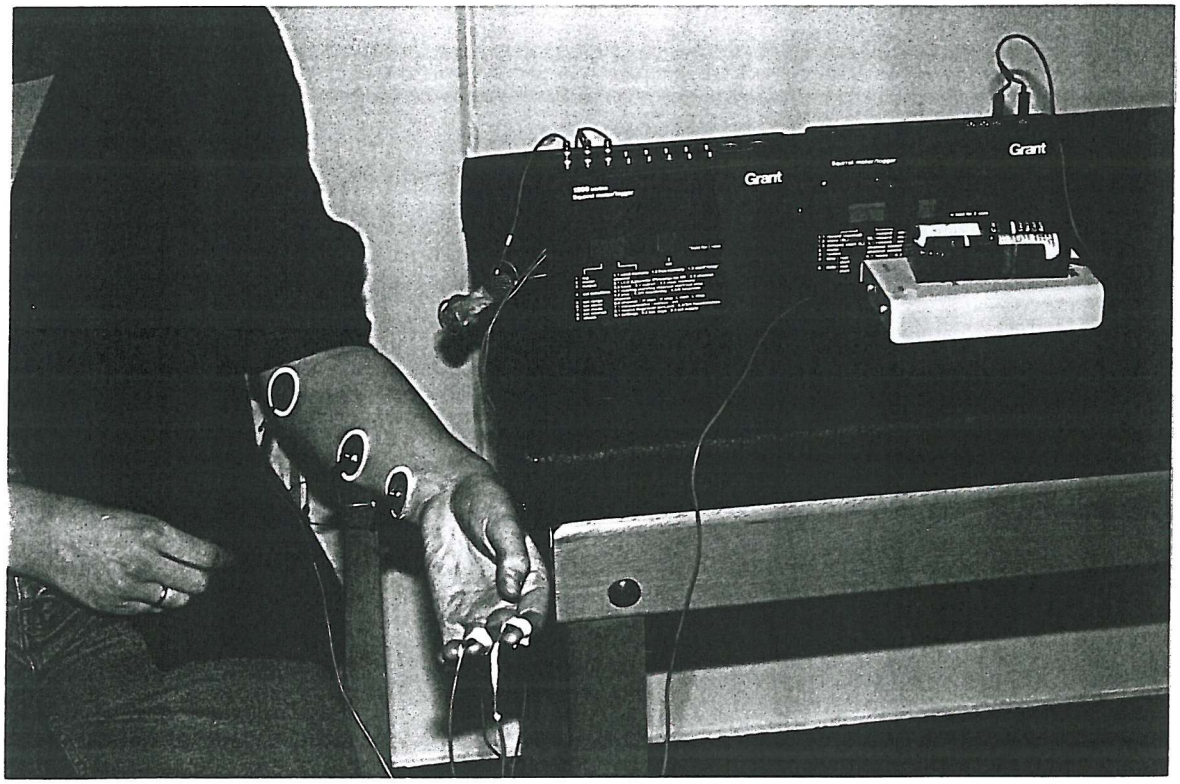

Fig 8.2: Apparatus used in the monitoring of flexor carpi ulnaris muscle EMG

THE COLD CHALLENGE TEST

The equipment used in the "Cold Challenge Test" consisted of a Grant W38 water bath cooled to a temperature of 15 degrees Celsius by a connected Grant CZ1 refridgerated cooler. Five thermistor probes were attached to the fingers and thumb of the non-dominant hand with two pieces of 2.5 cm micropore surgical tape, so that the wires were secured to the second phalange. A further probe, measuring ambient temperature, was attached to the table below the cold challenge equipment. Immediately prior to immersion of the hand in the water bath, the thermistors were covered with a polythene disposable glove which was secured with surgical tape at the wrist. The thermistor wires were connected to an Isolated Thermocouple system (Southampton General Hospital Medical Engineering) which was in turn connected to an Amstrad PC1512 by way of an RS-232-C serial lead. The temperature data was stored on Dysan 100 floppy diskettes. (See fig 8.3).

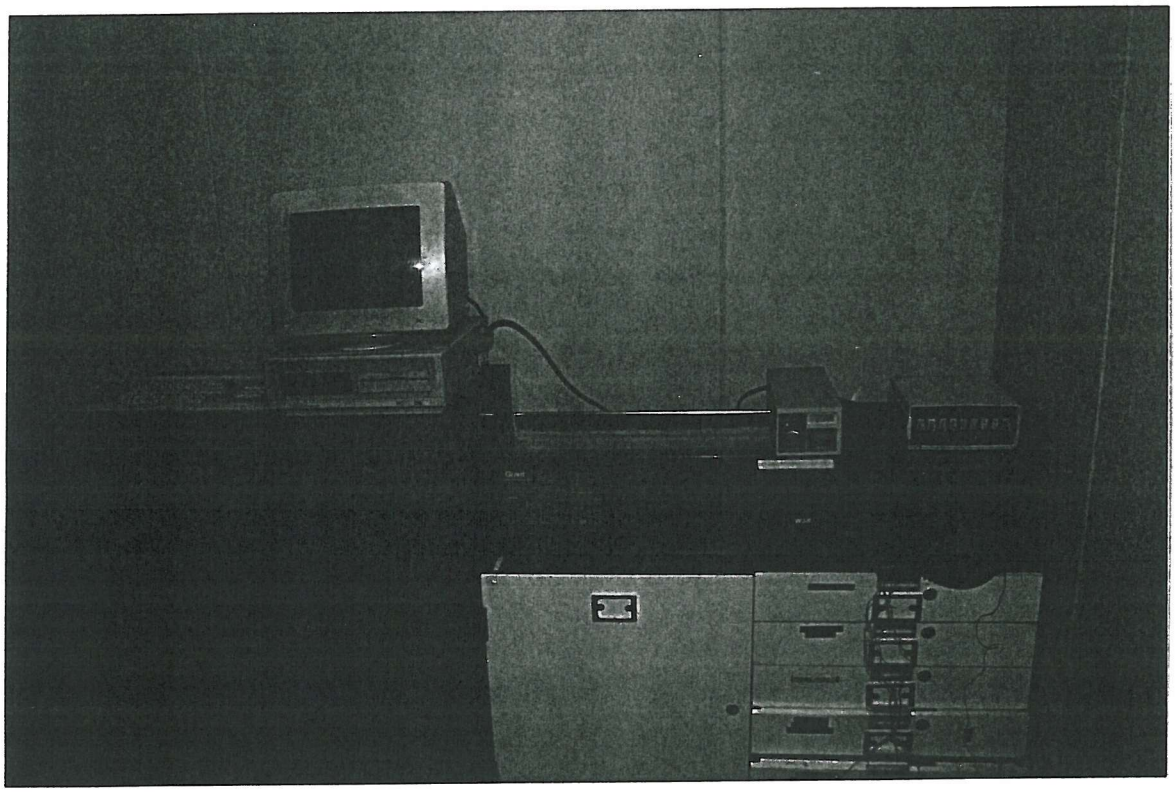

Fig 8.3: The "Cold Challenge Test" equipment

EMG BIOFEEDBACK

Fig 8.4 below displays the arrangement of the equipment used to provide EMG Biofeedback during the training sessions.

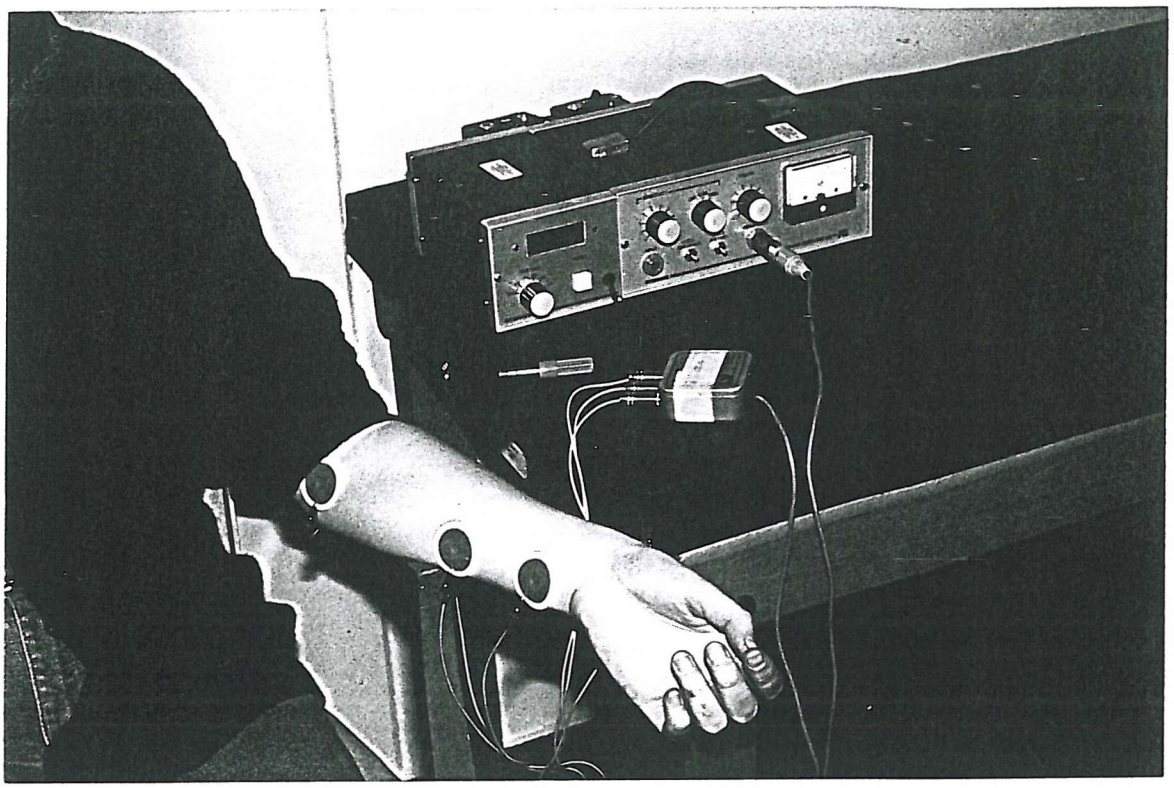

Fig 8.4: The Aleph One Myophone - used to provide auditory feedback of tension in the flexor carpi ulnaris muscle during ATE and ARE Biofeedback training sessions

The Aleph One Ltd Myophone affords both auditory feedback (a tone that increases with muscle tension) and visual feedback (a needle displaced by a change in muscle tone); however, for this study, only the auditory output was used. As with EMG measurement, three electrodes were used. Two Syncor solid state "Tracets" were attached to the extensor muscle of the non-dominant arm, and a third to the elbow of the non-dominant arm as a ground electrode. The electrode site was cleaned with surgical spirit and then rubbed dry prior to electrode attachment. The electrodes were connected to the Myophone by way of an Aleph One Ltd Myophone probe which amplifies the signal at the site of the electrodes to allow it to be "fed back" to participants.

FINGER TEMPERATURE BIOFEEDBACK

Auditory temperature Biofeedback was provided by an Aleph One Ltd Thermophone which emits a tone that decreases with a rise in digital skin temperature. (See fig 8.5). A single thermistor probe is attached to the finger pad of the index finger of the non-dominant hand with surgical tape (unless

prevented by finger loss or infection), and the finger pad is cleaned with surgical spirit and rubbed dry prior to the attachment of the probe.

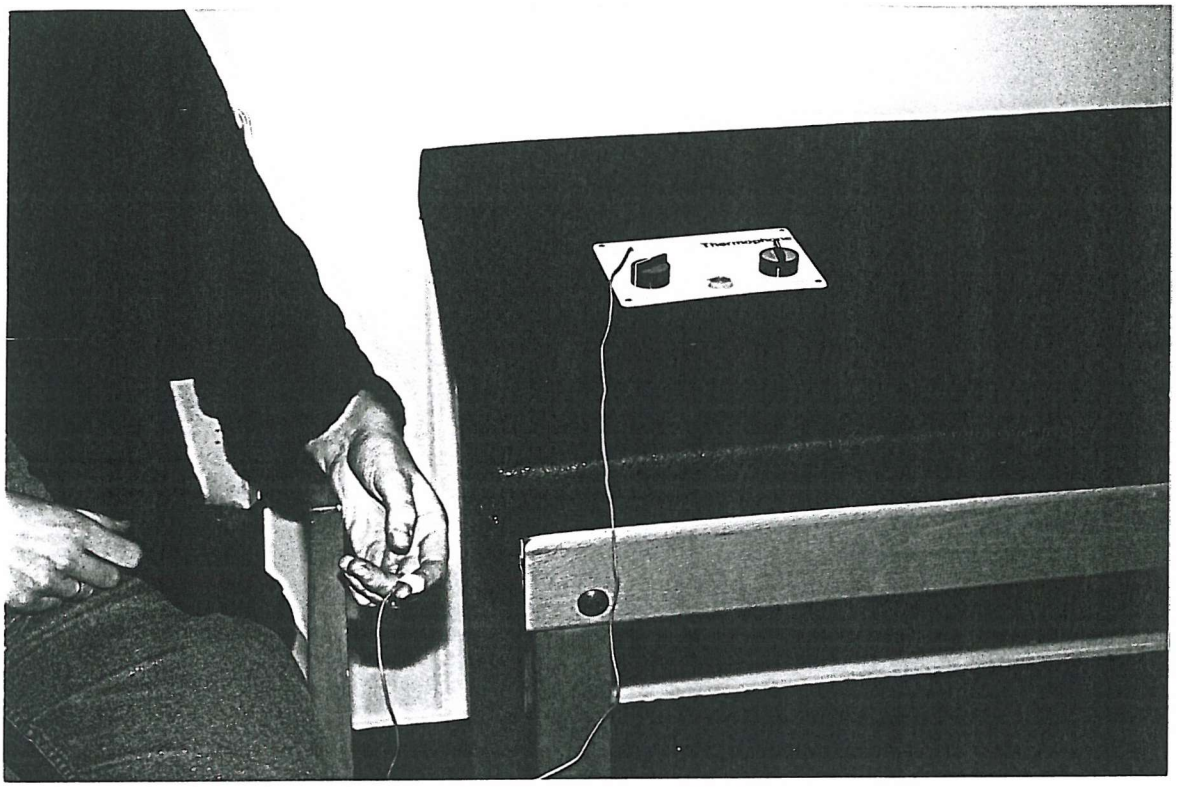

Fig 8.5: The Finger Temperature Biofeedback equipment used during ATT and ART training sessions

THE LABORATORY

All training and assessment sessions (other than Ambulatory temperature monitoring) took part in a soundproof room located in the Psychology Department of Southampton University. Room temperature ranged from 19-25 degrees celsius during the sessions with a mean temperature of 21.5 degrees Celsius. Humidity was constant at 75% RH. Two comfortable chairs were loaned by Southampton General Hospital.

Materials

AMBULATORY TEMPERATURE MONITORING DIARIES

Participants' activities during the monitoring period were recorded at hourly intervals in an Ambulatory Monitoring diary. The diary provides a chronologically accurate description of the

activities, clothing and Raynaud's attacks along with a mood rating sheet. The full diary may be found in Appendix 2.1.

TRAINING SESSION HAND OUTS

At the end of each training session, participants were given a hand out outlining the relaxation exercise(s) to practice before the next training session. The first of these included an overview of general Relaxation treatments and more specific details of either Autogenic Training or Applied Relaxation. Audiotapes of the relaxation exercises were provided where appropriate, and participants were given a note book to list practice sessions ans the subjective effects of the exercises during those sessions. The practice sheets may be found in Appendix 2.2.

Procedure

THE QUESTIONNAIRES (see Appendix 2.3)

Three questionnaires were completed by each participant. The first "pre-treatment" questionnaire concerned the volunteers' Raynaud's symptoms, "home remedies" for their symptoms, their views of "non-conventional" treatments for Raynaud's and other disorders, and past and present treatments for Raynaud's symptoms (ie. surgical, medical and "non-conventional"). The second "post-treatment" and third "follow-up" questionnaires asked about symptom improvement since completion of the first questionnaire ("Do you feel that the treatment you received at the University has had any effect on your Raynaud's symptoms ?"), along with changes in medication, non-conventional treatments, home remedies and lifestyle. There were two versions of both "follow-up" questionnaires - one for the control participants and the other for the experimental participants. The experimental participants' questionnaires differed from those of the controls in that they also assessed motivation and practice of the relaxation exercises along with general, and Raynaud's specific, subjective effects of treatment. In the main, the questionnaires were completed during the assessment sessions, although those recruited through the Raynaud's and Scleroderma Association were sent the first questionnaire with the recruitment letter and details of the study.

VOLUNTARY CONTROL OF DIGITAL SKIN TEMPERATURE (including EMG of the flexor carpi ulnaris muscle), AND THE COLD CHALLENGE TEST

The effects Voluntary control of finger skin temperature on digital temperature (and on muscle tension of the flexor carpi ulnaris muscle of the non-dominant arm), and the response of digital skin temperature to the cold challenge test were evaluated in each of three laboratory assessment sessions: the first before the treatment sessions, the second on completion of the training programme, and the last 4-5 months after the completion of training. Measurements of finger skin temperature and muscle tension were not taken until the participants had adapted for half an hour to the ambient conditions (room temperature and humidity) of the laboratory. Details of the explanation and instructions given to all participants during the initial pre-treatment assessment meeting may be found in Appendix 2.5. However, an outline of the assessment meeting procedure may be found below.

Initially, the theory behind the use of Behavioural treatments for Raynaud's (including the role of relaxation on the activity of the Sympathetic Nervous System, and the role of the SNS on peripheral blood flow) was outlined for the participants (see Appendix 2.5a). The pre-treatment questionnaire (Appendix 2.3a) was then completed, and the tests to be carried out in the session described.

Following the half hour period of adaptation to the ambient conditions (during which the participants were free to ask questions), the tests of voluntary control of finger skin temperature, and the Cold Challenge Test (as described in Appendix 2.5b) were performed.

AMBULATORY TEMPERATURE MONITORING

A second session was arranged for the Ambulatory temperature monitoring. On arrival at the department, the monitoring equipment was attached as explained above. The participants were asked to indulge in their usual daily activities (at least as far as possible - given the unusual monitoring situation) over a three hour period before returning to the department for removal of the equipment. During the monitoring period, activity diaries were completed at hourly intervals, and were checked for clarity on return to the department.

THE TRAINING PROTOCOL

Each experimental participant attended 6 training sessions at between one and four weekly intervals (mean interval 2.7 weeks). Two weekly intervals was the ideal attendance rate, but for many volunteers, factors such as ill health, holiday commitments and work pressure made this impossible. The individual sessions are detailed below, but all meetings began with a 30 minute period to allow adaptation of finger temperature to the temperature of the room, and a ten minute baseline of digital skin temperature.

During the adaptation period, temperature detecting probes (and/or the biofeedback probes and electrodes) were attached, and where appropriate, participants were reminded of the action of the biofeedback equipment. The structure of the rest of the session was also outlined (including a brief description of the particular session's relaxation exercises), and participants questions were answered. In later sessions, the volunteers' subjective interpretation of the effects of practising the previous session's exercises were discussed. As this included levels of practice, means of increasing practice levels were considered where appropriate. Finally, volunteers' travelling expenses were reimbursed, and subsequent training sessions booked.

A. Applied relaxation (with or without biofeedback)

Session 1: Progressive Relaxation

The volunteer sat in a comfortable arm chair, wearing just indoor clothing. During the 30 minute adaptation period, the treatment of Applied Relaxation was explained (Appendix 2.6a), and the structure of the training sessions were outlined. For 10 of the volunteers, Applied relaxation was being offered in conjunction with EMG or Temperature Biofeedback. Here, the appropriate Biofeedback equipment was utilised during the relaxation exercises such that these participants received two forms of Behavioural treatment simultaneously. Additional instructions given to the Biofeedback participants regarding the use of the biofeedback equipment are outlined in Appendix 2.6b. During the baseline period, participants either chatted or read a magazine. Those who chose to read were instructed to keep the non-dominant hand in the "relaxation" position and turn the pages

only with the other hand. Following the baseline, the "Progressive Relaxation" exercise (see Appendix 2.6c) was read aloud to the participant with the proviso that (s)he was free to stop the narration should they encounter anything unclear.

The participants remained in their comfortable relaxation position for the 10 minute post-training baseline period, during which they were questioned about the effects of the relaxation exercise, (and where appropriate, their impression of changes in the biofeedback tone). Moreover, the need for twice daily practice of the exercise was stressed to the volunteer. Tape recordings of the exercise were provided for this purpose, along with a handout of practice details. (Appendix 2.2). In addition, the most ideal times for practice of the exercises were discussed with the individual, and a "relaxation diary" was provided to log practice sessions. It was suggested that the completed diaries should be a true reflection of practice as practice rates would be a factor looked at during the analysis of the results; therefore, any missed practice session should be noted by date and a gap in the diary.

Session 2: Release-only Relaxation

Following the 30 minute adaptation period and 10 minute pre-training baseline of finger skin temperature, the Progressive Relaxation exercise from the previous session was repeated (with Temperature or EMG Biofeedback for the biofeedback participants), and details of the subjective effects of the exercise were noted. The participant remained in their preferred relaxation position for a 10 minute post-training, and a 5 minute pre-training baseline of finger skin temperature. During this, details of the second exercise were explained to the participant (Appendix 2.6d). The Release-only exercise (Appendix 2.6e) was then read aloud to the participants such that they were instructed to relax different areas of the body in sequence without first tensing the muscles in these areas. Following the exercise, participants continued to sit (in the same position) for a further 10 minute baseline measure of finger skin temperature. They were subsequently provided with an audio tape of the exercise to practice twice a day until the next session, and a hand out of practice details.

Session 3: Cue-controlled Relaxation

In the manner of the previous training sessions, following the adaptation and pre-training baseline period, the release-only relaxation exercise was repeated, but rather than suggesting that the volunteer wake up, attention was drawn to their breathing as described in Appendix 2.6f.

For 5 breaths, the volunteer heard the words INHALE and RELAX as they inhaled and exhaled respectively. They were then instructed to concentrate on their breathing. They were told to breathe calmly and regularly, thinking INHALE as they inhaled and RELAX as they exhaled. After about 10 such breaths, the initial breathing instructions were repeated such that in total, there were four blocks of both hearing and thinking INHALE and RELAX. On being told to wake up as in the manner of previous exercises, the participant remained in the same position for a 10 minute post-training baseline of finger skin temperature.

Session 4: Differential Relaxation #1

The session began with the cue-controlled relaxation of session 3. Once the participants had reached a relaxed state through controlled breathing, they were instructed to maintain a state of relaxation in the rest of the body whilst moving the eyes, hands, arms, feet, and legs in sequence initially from a sitting position and subsequently, a standing position. The instructions to participants are detailed in Appendix 2.6g. As in the previous training sessions, during the 10 minute post-training baseline, the participant's thoughts and views on the effects of the exercise were noted.

Session 5: Differential Relaxation #2

The second session of differential relaxation differed from the first in that there were only two blocks of cue-controlled relaxation rather than the four of previous sessions. In addition, on completion of differential relaxation in the sitting and standing positions, participants were asked to maintain physical relaxation as they walked around the room. (The temperature logger and Biofeedback equipment being carried by the experimenter). The instructions given to the participants may be found in Appendix 2.6h.

Session 6: Rapid relaxation

The Rapid Relaxation session began with about ten breathes of cue-controlled relaxation in the sitting position. This was followed by movement of the eyes, hands, arms, feet and legs in the standing position with the eyes open. The volunteer then began walking around the room, maintaining a calm and regular breathing pattern as in the previous session. It was then pointed out to the volunteer that there were a number of green stickers placed strategically around the room (on the door, the wall and the lamp shade). Participants were instructed to stop at each one, breathe in, think RELAX, and breathe out slowly. It was further suggested that they repeat this "Rapid Relaxation" exercise twice more before moving on to the next sticker, and that they should be aware that with each breathe, they are relaxing more and more.

As in all sessions, the exercise ended with a 10 minute post-exercise baseline of finger skin temperature. During this, the participant was provided with a sheet of the exercise for practice, and a number of stickers to be placed around the home/office to cue practice of the rapid relaxation breathing exercise firstly when they were in no hurry to do anything, or go anywhere, and in time, when cold and in a hurry.

B. Autogenic Training (with or without biofeedback)

The Autogenic Training sessions all had the same structure in that they began with a 30 minute period of adaptation of finger skin temperature to the conditions of the room (temperature and humidity), followed by a 10 minute baseline of digital skin temperature. A relaxation exercise was then introduced, followed by a 15 minute pre/post exercise baseline of digital skin temperature, and a second relaxation exercise. The session ended with a 10 minute post-relaxation baseline of finger skin temperature. In session 1, both AT exercises were new. In later sessions, the exercise learned in the previous session was repeated as the first exercise, and a new exercise was introduced later in the session. During the baseline period following the training exercises, the subjective effects of the exercises were discussed.

Session 1: The Short Stitch and Heaviness # 1

During the adaptation period, the volunteer was given a brief history and explanation of Autogenic Training (Appendix 2.7a), details of the positions adopted during repetition of the exercises (Appendix 2.2a), and where appropriate (the ATT and ATE treatment groups), a demonstration of, and instructions for the use of the Biofeedback apparatus (Appendix 2.6b).

The Autogenic Training exercises themselves consist of three repetitions of a series of phrases. The completion of each repetition is marked by cancelling the effects of the exercise. The method of "cancelling out" is described in Appendix 2.2a. The purpose of cancelling out between exercises is to learn the difference between relaxation and physical tension. During the exercises, a deep state of physical relaxation may be reached. By cancelling out, one is rapidly brought out of the relaxed state. Therefore, cancelling out prevents the participants feeling drowsy as is often the case with common relaxation techniques.

All exercises began with a body check for physical tension (the order of which is listed in Appendix 2.2a) in which the tension in named areas of the body was observed, and posture adjustments made to reduce the tension.

The exercises introduced during the first Autogenic Training session were the "Short Stitch" exercise and the "Heaviness #1" exercise. These exercises and the appropriate instructions to participants are detailed in Appendices 2.7b and 2.7c respectively.

Sessions 2-6

The structure of the second and subsequent training sessions were the same. During a 30 minute adaptation period, practice, the subjective effects of the exercises, and Raynaud's symptoms since the last training session were discussed. Then, following a 10 minute baseline of finger skin temperature, the exercise learned in the previous session was repeated. The trainer said the first repetition aloud, and the trainee went on to complete the later repetitions either aloud or to his/herself. Following a 15 minute pre/post exercise baseline of finger skin temperature, during which the effects of the exercise

were discussed, a new exercise was introduced. Here, the first two repetitions were said aloud by the trainer, and the final repetition by the trainee (again either aloud or quietly to his/herself). The session ended with a 10 minute post-exercise baseline of finger temperature. To avoid temperature changes being attributed to placement of the hands, The volunteers remained in the same position throughout the session (except when cancelling out exercises).

Details of the exercises ("Heaviness #2", "Warmth", "Regulation of Heart beat and observation of breathing", "Solar Plexus and forehead", and "Organ specific formulae"), along with the explanations given to the volunteers for each new exercise are detailed in Appendices 2.7d-2.7h.

8.2 RESULTS

As allocation to treatment and control groups was not strictly random, it is necessary to check for pre-treatment group differences that might confound the results. Such differences need be assessed both subjectively through analysis of the pre-treatment questionnaire, and objectively through comparison of the groups' ability to control digital skin temperature in the laboratory, in cold stress conditions, and finally, during ambulatory recording of digital skin temperature.

ANALYSIS OF THE PRE-TREATMENT QUESTIONNAIRE

Data were coded in line with the coding frame outlined in Appendix 2.4a, and compared using Chi-square analysis. In general the groups were alike; indeed, there were no group differences in age, gender, or use of medically prescribed drugs or surgery (past or present) as treatment for Raynaud's and associated symptoms. Furthermore, symptoms and symptom severity did not differ significantly between groups: 21% (8) considered their symptoms mild, 63% (24) moderate, and 13% (5) severe. (Details were lacking for one (3%) Control participant); 29% (11) of the sample described a characteristic vasospastic attack i.e. blanching of the skin (white), cyanosis (blue) and hyperaemia (red); 21% (8) described two of the three associated skin colour changes; and 47% (18) reported just one such colour change. Again, no details were available for one Control participant (3%).

Views offered about non-conventional treatments for Raynaud's did not differ between treatment groups: the views of 39% (15) were positive, 37% (14) neutral, and 24% (9) negative. Finally, a variety of home remedies were described e.g. the avoidance of stressful situations, or keeping warm. However, all reported means of combating symptoms may be categorised in terms of avoidance techniques i.e. avoiding the triggers of attacks - be they the cold, stress or others.

The groups did differ on two factors - namely, the perception of "stress" as a trigger of their attacks, and secondly, in terms of previous experience with Relaxation techniques. Indeed, the Control participants were far less likely to perceive "stress" as a cause of their Raynaud's attacks when compared with both the relaxation groups: Chi-square (df 2) = 11.14 $p < 0.01$; and the biofeedback groups: Chi-square (df 3) = 13.61 $p < 0.01$. (Statistical Appendix 2.1; Tables 1.a.I and 1.a.II; page S2). That participants were given the option of experimental or control status at the recruitment stage might factor in this difference. Perhaps, those who did not connect their symptoms with "stress", opted to be control participants as they were unable to see the potential benefits of relaxation for their symptoms despite a clear explanation that the investigation concerned the role of Relaxation and associated treatments in the reduction of physical tension - not emotional "stress".

As to experience of relaxation techniques, most participants (82%) reported no such experience, however, the distribution of the 18% (7) who did, revealed that 40% (4) of the No biofeedback, and 33% (3) of the Temperature biofeedback groups (as compared with none of the Control or EMG biofeedback participants) had encountered relaxation techniques in the past: Chi-square (df 3) = 9.52 $p < 0.05$. (Statistical Appendix 2.1; Table 1.b.I; page S2). It should be noted that previous relaxation was in the form of Yoga or relaxation classes during pregnancy.

ANALYSIS OF THE PRETREATMENT VOLUNTARY CONTROL OF FINGER SKIN TEMPERATURE

As some participants had experienced relaxation techniques in the past, and relaxation may be associated with increased digital skin temperature, group differences in ability to control finger skin temperature in the laboratory were assessed.

A number of participants had described their middle fingers as the initial and/or main site of symptom manifestation (personal communication); therefore, only the middle finger was chosen for

analysis. A temperature difference, indicative of any change in temperature during voluntary control, was calculated for both the middle finger and room temperature by subtracting the temperature in the last minute of the baseline period from that of the last minute of the voluntary control period for each participant. Using the middle finger "difference" temperature as the dependent variable, an analysis of variance was performed comparing the biofeedback and control groups, and the relaxation and control groups. The room temperature difference, room starting temperature and middle finger starting temperature were co-varied in the analysis.

This analysis revealed significant differences in the relaxation group's ability to control middle finger temperature: $f(2, 34) = 6.078$ $p < 0.01$. (Statistical Appendix 2.1; Table 2.a.I; page S2). Indeed, as highlighted in table 8.3, the finger temperature of the Applied Relaxation group increased during the voluntary control session despite a concurrent room temperature decrease.

Group	AT	AR	No Relax		No Bio	Temp Bio	EMG Bio
Mean finger temperature difference	-0.12 (C)	1.58 (C)	-0.21 (C)		0.90 (C)	0.65 (C)	0.65 (C)
Mean room temperature difference	0.07 (C)	-.01 (C)	0.09 (C)		0.05 (C)	0.06 (C)	-0.01 (C)

Table 8.3: Room and middle finger temperature changes during the voluntary control session.

Given that member(s) of the Applied Relaxation group appear able to increase their finger temperature "at will" prior to treatment, and the Applied Relaxation group were not, in the main, those with previous experience of relaxation techniques, one must ask whether there were any differences in the techniques used to bring about finger skin temperature change.

Methods of temperature control employed were assessed subjectively through participants' descriptions of strategies used, and objectively through changes in the tension of the flexor carpi ulnaris muscle of the dominant arm.

Data on the subjective methods used were available for only 30 (75%) of the participants. Table 8.4 illustrates that two thirds of the participants used "thermal imagery" e.g. imagining oneself in a hot bath, on the beach, or sitting close to a fire, to try to increase finger temperature. The remaining participants claimed to have used the techniques of "willing blood/warmth to the fingers" or "relaxation".

Group	**Thermal Imagery**	**Willing blood/warmth to fingers**	**Relaxation**
AR	6	3	2
AT	10	2	1
No Relax	4	2	0
No Bio	6	2	0
Temp Bio	3	3	1
EMG Bio	7	0	2
Total	20 (66.7%)	7 (23.3%)	3 (10%)

Table 8.4: The reported methods of attempted control of finger skin temperature

Objective evidence of the strategies used may be gained from the changes in tension of the flexor carpi ulnaris muscle of the non-dominant arm. A reduction in muscle tension from baseline would suggest the employment of a relaxation strategy. In contrast, increased muscle tension might suggest an attempt to force blood to the finger tips. Owing to teething problems with the EMG data loggers, EMG data is available only for 16 participants. Chi-square analysis revealed no significant group differences in objective methods used (tension, relaxation or no change in flexi carpi ulnaris muscle). Overall, 37.5% (6) appeared to use a relaxation strategy, 25% (4) tensed the flexor carpi ulnaris muscle during the voluntary control period, and for the remaining 37.5% (6), there was no change in muscle tension from baseline.

ANALYSIS OF THE PRE-TREATMENT COLD CHALLENGE TEST

For the purpose of this study, the cold challenge test was used to measure time taken for finger temperature to rewarm by 6 C following timed immersion and subsequent removal of the hand in a 15 C cold water bath. As the reader may recall, a 6 degree C rise in finger temperature is considered a

potential tool for differentiating Raynaud's and non-Raynaud's sufferers. Therefore, an improvement in the time taken to attain a 6 degree rise might suggest positive effects of treatment.

The time taken to rewarm must depend to a certain extent on finger temperature prior to immersion and room temperature during the cold challenge test; therefore, such factors need be taken into account when statistically comparing cold stress recovery across groups.

The cold challenge test ended once all fingers had returned to starting temperature, or 45 minutes after removal of the hand from the water bath - whichever was the shorter. It should be noted that of the 40 original participants, one Secondary Raynaud's volunteer withdrew part way through the test owing to the unpleasant effects of cold on her ulcerated fingers. The fingers of the remaining 39 participants recovered by 6 degrees C within the rewarming time permitted.

Table 8.5 describes the time taken for middle finger temperature recovery by 6°C from the lowest temperature reached during immersion in the water bath. That the groups do not differ in their ability to rewarm is supported by an analysis of variance of time to rewarm by 6 C covarying room and middle finger pre-immersion temperature: Relaxation groups: $f(2,38) = 0.505$ $p>0.5$; Biofeedback groups: $f(2,34) = 1.318$ $p>0.10$. (Statistical appendix 2.1; tables 3.a.I and 3.a.II, page S3).

	No Bio	Temp Bio	EMG Bio		AT	AR	No Relax
Time (mins)	18.0	22.7	26.7		21.4	23.1	18.9

Table 8.5: The time taken to increase finger temp by 6C following cold challenge

ANALYSIS OF TEMPERATURE CHANGES DURING PRETREATMENT AMBULATORY MONITORING OF FINGER SKIN TEMPERATURE

Ambulatory Temperature Monitoring involves the collection of finger and ambient temperature data concurrently with subjective information (in diary form) concerning participants' mood, symptoms, activities, and views about ambient conditions during the monitoring period. The ambulatory

monitoring diary may be found in Appendix 2.1, and the coding frame used for analysis in Appendix 2.8.

Subjective data were available for only 30 (75%) of the volunteers. Chi-square analysis revealed no group differences in mood, symptoms or activities. Overall, 76.7% (22) did not consider the monitoring period at all stressful/anxiety provoking; and only 23.3% (8) felt that the 3 hour period to be mildly stressful. Furthermore, morale was generally high. 93.1% (27) were not at all depressed during the monitoring period; 84.9% (24) experienced no frustration; and 74.7% (23) found their activities satisfying.

Participants had been asked to carry out their normal everyday activities during the monitoring period. Given the intrusive nature of the monitor and the need to complete the diary at intervals, perhaps "normal everyday activities" were not strictly possible. Indeed, people tended to remain inactive and in the warm: 73.3% (22) did not experience cold ambient conditions during the monitoring period; 65.5% (20) felt no need to wear gloves; and 63.3% (19) were predominantly sedentary during the 3 hour period. The use of stimulants such as caffeine, alcohol and nicotine was confined to 9 (30%) of the participants.

Although most participants remained in the warm as much as possible, 60% (18) actually experienced Raynaud's symptoms (12.3% throughout the monitoring period). However, severity ratings suggest that the symptoms were somewhat less severe than average: 55% reporting very low severity.

Looking further at the subjective data in terms of Raynaud's diagnosis (i.e. Primary Raynaud's, Secondary Raynaud's, those Unsure of their diagnosis and the Undiagnosed), no differences in terms of "mood" across the 4 categories were discovered. Moreover, and perhaps somewhat surprisingly, there were no differences in terms of the occurrence of symptoms, nor the duration or severity of those symptoms. However, Chi-square analysis did reveal that the Primary, Secondary and Unsure participants were more likely to stick solely to warm environments during the monitoring period - the undiagnosed venturing more out into the cold. (Chi-square f (df 6) = 12.63 $p < 0.05$. Please refer to

statistical appendix 2.1; table 4.a.I, page S3) . Further, although not statistically significant, a trend in the data suggests that the Secondary Raynaud's participants were more likely than the other diagnostic (sic) groups to wear gloves during the monitoring period.

As to the analysis of objective data, in line with previous analyses in this section, only middle finger temperature data were compared across groups. Mean objective middle finger and ambient temperatures recorded during the monitoring period are shown in table 8.6.

Temp (C)	No Bio	Temp Bio	EMG Bio		AT	AR	No Relax
Ambient temp	20.8	19.9	20.3		20.9	19.4	21.5
Middle Finger temp	23.8	22.5	22.5		23.0	23.4	23.0

Table 8.6: Ambient and middle finger temperatures (C) during the ambulatory monitoring period.

Analysis of covariance (co-varying ambient temperatures during monitoring) revealed no statistically significant group differences in middle finger temperature during the monitoring period: Relaxation groups ($f(2,32) = 0.912$ $p = 0.412$); Biofeedback groups ($f(2, 32) = 0.267$ $p = 0.768$). Please refer to statistical appendix 2.1; tables 4.b.I and 4.b.II (page S4).

As previously stated, 60% (18) of participants claimed to have experienced symptoms of Raynaud's during the monitoring period. The mean ambient and finger temperatures during these periods may be found in Table 8.7 below. As above, an analysis of covariance uncovered no group differences in middle finger temperature during periods of described Raynaud's symptoms.

Temp (C)	No Bio	Temp Bio	EMG Bio		AT	AR	No Relax
Ambient temp	20.3	20.3	18.8		20.0	19.5	20.1
Middle Finger temp	22.1	20.9	19.3		22.3	20.1	19.4

Table 8.7: Ambient and middle finger monitoring temperatures (C) during periods of described Raynaud's symptoms

A SUMMARY OF THE PRE-TREATMENT ASSESSMENT SESSION

- The No Biofeedback and Temperature Biofeedback groups had had some experience of relaxation techniques prior to the study.
- Members of the AR groups demonstrated a pre-treatment ability to increase finger temperature "at will".
- Subjective reports suggest that most participants used thermal or thematically relevant imagery as a means to increase finger skin temperature, and limited objective data does not dispute this.
- There were no baseline group differences in Cold Challenge recovery times to 6 degrees C.
- During Ambulatory monitoring, participants tended to be fairly inactive and stick to warm environments.
- Raynaud's symptoms, claimed to have been experienced by participants during ambulatory temperature monitoring, were very mild.
- There were no group differences in ambient or finger temperatures during baseline ambulatory monitoring.

THE AUTOGENIC TRAINING AND APPLIED RELAXATION TRAINING SESSIONS

A. AUTOGENIC TRAINING

Session 1: Short Stitch and Heaviness #1

Because of the time commitment involved, one ATT participant withdrew from the study before the first training session.

For display purposes, the final minute of each section of the training session (ie the 3 baseline and 2 exercise periods), was considered representative of the section as a whole as it was assumed that any effects of relaxation might be more noticeable towards the end of an exercise period.

The temperatures during session 1 are displayed in fig 8.6, and the mean room and middle finger temperatures during the 5 sections of the training session are detailed in table 8.8.

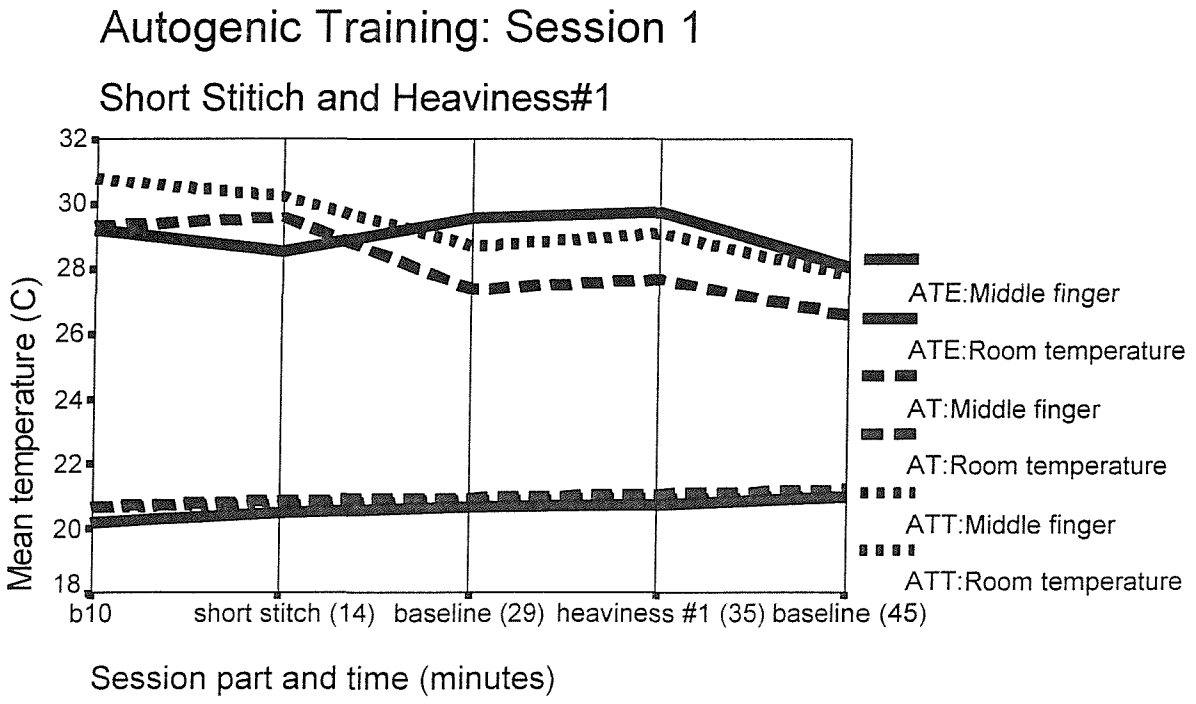

Fig 8.6: The mean middle finger and room temperatures (C) during each of the 5 sections of the first AT training session

Temp variable	Treatment group	Baseline	Short Stitch	Baseline	H#1	Baseline
Middle finger	AT	29.32	29.59	27.37	27.69	26.58
	ATT	30.78	30.25	28.70	29.10	27.81
	ATE	29.19	28.52	29.56	29.75	28.08
Room temp	AT	20.68	20.85	20.94	21.05	21.18
	ATT	20.68	20.74	20.85	20.90	20.98
	ATE	20.17	20.51	20.63	20.72	20.99

Table 8.8: The mean middle finger and room temperatures (C) during the first AT training session

There appears to be very little effect of the relaxation exercises on finger skin temperature; however, subjective reports of the effects of the exercises suggest that the AT group considered themselves to have relaxed during the short stitch exercise; whereas none of the ATT or ATE group members did so. Further, during the Heaviness #1 exercise, 100% (5) of the AT group, 50% (2) of the ATT group, and 60% (3) of the ATE group reported subjective relaxation effects of the exercises.

Session 2: Heaviness #1 and Heaviness #2

Two AT participants withdrew from the study before the second training session because of ill health or their predicted failure to practice the exercises between sessions. Figure 8.7 provides a graphical display of the fluctuations in room and middle finger temperature of the 12 participants who completed this session, and table 8.9, the mean room and middle finger temperatures of the individual Autogenic Training treatment groups during each of the 5 sections of the session.

Temperature variable	Treatment group	Baseline	H#1 exercise	Baseline	H#2 exercise	Baseline
Middle finger	AT	31.94	32.52	31.30	32.07	31.35
	ATT	31.64	31.79	31.75	31.93	30.17
	ATE	29.11	29.33	30.33	30.6	30.32
Room	AT	20.56	20.62	20.80	20.86	21.00
	ATT	20.62	20.71	20.85	20.89	21.02
	ATE	20.21	20.19	20.35	20.47	20.36

Table 8.9: The mean middle finger and room temperatures during the second AT training session

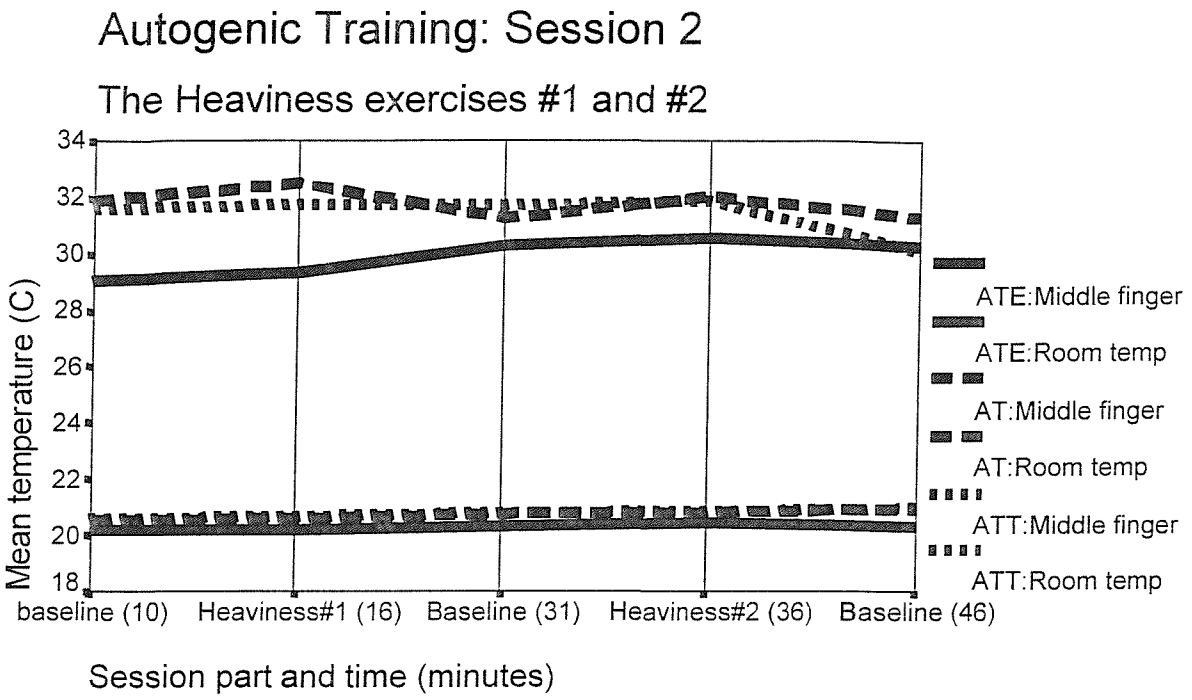

Fig 8.7 Room and middle finger temperature fluctuations during the second Autogenic Training session.

Trends in the data suggest that only the AT finger temperature increased slightly during both Heaviness exercises; however, approximately two thirds of the participants (across all three treatment groups) felt that they had relaxed during the exercises. The failure to find objective evidence of relaxation may be related to lack of practice of the exercises. Indeed, although the participants had been asked to practice the Heaviness #1 exercise twice daily between the two training sessions, recorded practice rates were universally poor across the treatment groups: 60% rarely practised (i.e. less than once per day); 20% practised once per day, and only 20% practised at the required frequency.

Session 3: Heaviness #2 and The warmth exercise

All 12 remaining AT participants attended the third training session. Mean room and finger temperature fluctuations are presented in figure 8.8 and table 8.10.

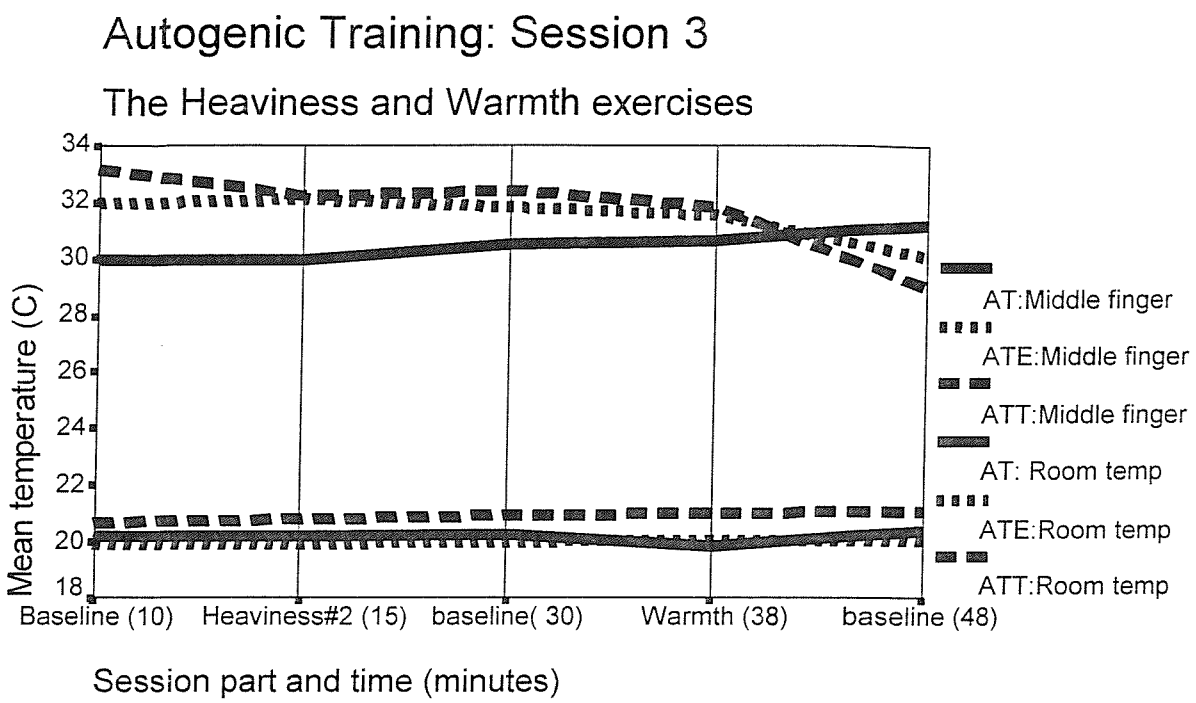

Fig 8.8 Room and middle finger temperature fluctuations during the third Autogenic Training session.

Temperature variable	Treatment group	Baseline	H#2 exercise	Baseline	Warmth exercise	Baseline
Middle finger	**AT**	29.96	30.00	30.56	30.66	31.21
	ATT	33.16	32.23	32.44	31.87	29.02
	ATE	31.96	32.13	31.84	31.56	30.10
Room	**AT**	20.20	20.18	20.29	19.84	20.44
	ATT	20.72	20.82	20.98	21.04	21.13
	ATE	19.95	19.94	20.02	20.09	20.06

Table 8.10: The mean middle finger and room temperatures during the third AT training session

Subjective reports of the session revealed that only half of the participants felt that they had experienced feelings of relaxation during the Heaviness #2 exercise, and 75% during the Warmth exercise. These feelings were not confined to one particular group. The objective data, however, reveal trends toward relaxation during the Heaviness #2 exercise, only for the ATE group, and during the Warmth exercise, only for the AT group.

Data on practice rates were available for only 75% of the participants; however, in line with the objective data, the ATE group were the most consistent and frequent in practising the exercises: all ATE participants for whom data were available claimed to have practised at least once per day.

Session 4: The Warmth and Cardiac/breathing exercises

Although 12 Autogenic Training participants attended the fourth training session, the data for one AT participant were lost; therefore, figure 8.9 and table 8.11 display the temperature fluctuations of 11 participants (2 AT, 4 ATT and 5 ATE).

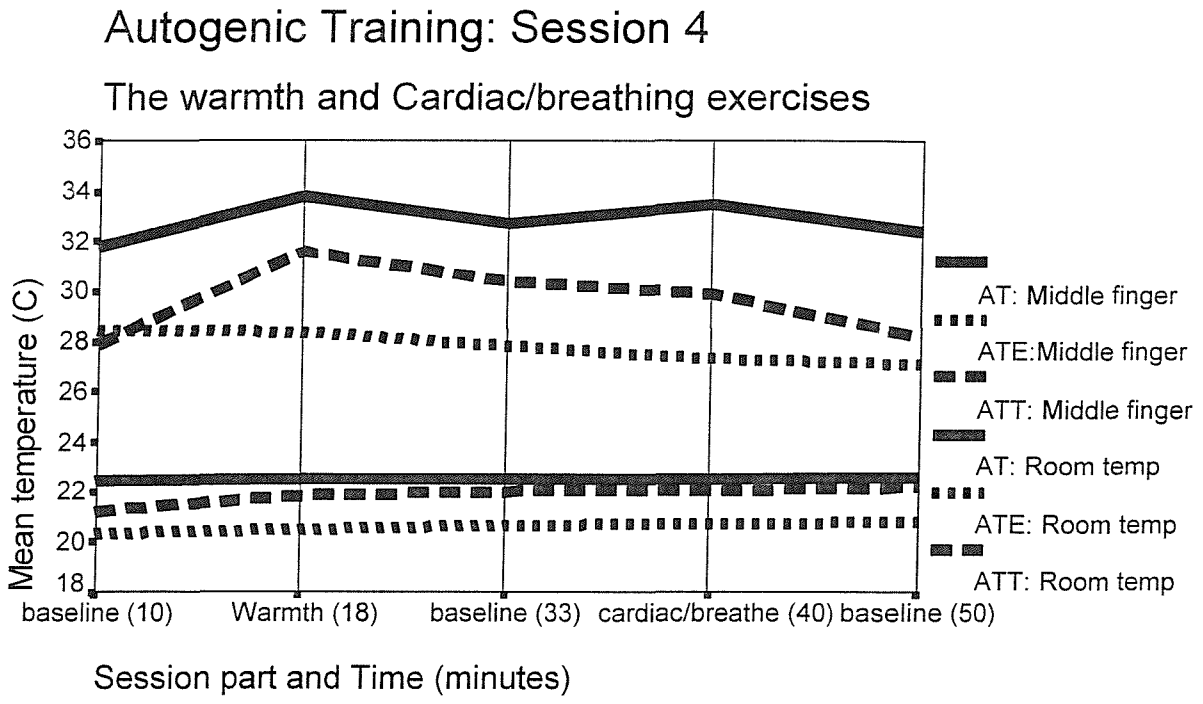

Fig 8.9 Room and middle finger temperature fluctuations during the fourth Autogenic Training session.

Temperature variable	Treatment group	Baseline	Warmth	Baseline	Cardiac/ breathing	Baseline
Middle finger	**AT**	31.82	33.83	32.76	33.50	32.45
	ATT	27.89	31.60	30.43	29.92	28.24
	ATE	28.41	28.40	27.83	27.34	27.11
Room	**AT**	22.42	22.51	22.52	22.52	22.59
	ATT	21.23	21.85	22.01	22.04	22.18
	ATE	20.34	20.49	20.68	20.70	20.81

Table 8.11: The mean middle finger and room temperatures during the fourth AT training session

As is evident from figure 8.9, the AT group show finger temperature increases during both the "Warmth" and "Cardiac/Breathing" exercises, and the ATT group, during the "Cardiac/Breathing" exercises. These data fit well with the subjective reports of the effects of the exercises: all of the AT participants felt that they had relaxed during the "Warmth" exercise, and the ATE group were either unsure of the effects or felt tense during the exercise. However, the results tally less well with practice rates as 80% of the ATE group had practiced at least once per day between training sessions.

Session 5: The Cardiac/breathing and Solar Plexus/forehead exercises

One female ATT participant withdrew from the study before the fifth training session owing to motivational factors. Therefore, the following analysis represents the temperature fluctuations of 11 of the original 15 Autogenic Training participants (3 AT, 3 ATT, 5 ATE). Table 8.12 and figure 8.10 show mean room and finger temperatures during the session.

Temperature variable	Treatment group	Baseline	Cardiac/ breathing	Baseline	Solar Plexus + Forehead	Baseline
Middle finger	AT	30.63	33.01	31.07	32.15	31.04
	ATT	28.01	29.43	28.02	29.01	28.13
	ATE	31.04	33.03	30.00	30.76	29.82
Room	AT	21.98	22.01	22.37	22.84	22.85
	ATT	22.01	22.09	22.44	22.93	23.02
	ATE	20.03	20.05	20.05	20.09	20.12

Table 8.12: The mean middle finger and room temperatures during the fifth AT training session

Clearly, all three treatment groups increased their finger skin temperature during the Cardiac/breathing exercise, and the Solar Plexus/forehead exercise - although during the latter exercise, temperature increases in the AT and ATT groups were probably mediated by an approximate 1 degree C increase in room temperature.

Subjective reports of effects of the exercises suggest that 60% and 40% of the ATE group relaxed during the Cardiac/breathing and Solar Plexus/Forehead exercises respectively, yet all AT participants

felt that they had relaxed during both exercises.

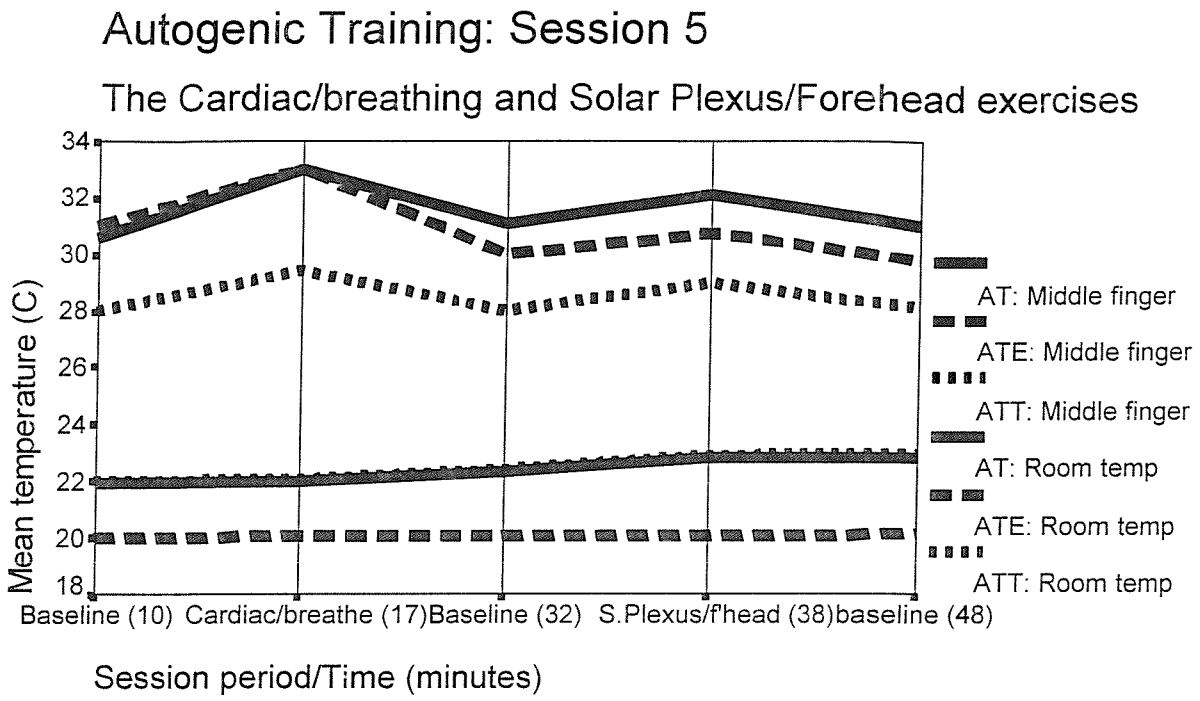

Fig 8.10 Room and middle finger temperature fluctuations during the fifth Autogenic Training session.

In line with the objective data, however, practice rates indicate that most (60%) members of all groups practised the "Cardiac/breathing" exercise at least once per day between training sessions.

Session 6: The Solar Plexus/forehead and the full exercise (Raynaud's specific formulae)

11 participants attended the final Autogenic Training session. As is evident from figure 8.11 and table 8.13, only the AT and ATE groups appear to have increased their finger skin temperature during the two exercises of the session - the AT group increases being more pronounced than those of the ATE group.

As one might have hoped, practice rates for the 11 participants to "stay the course" were consistent: 89% practising at least once/day. However, subjective effects of the exercises appeared not to be related to practice rates, but were reflected in the objective temperature change data: the At group felt, and indeed, did relax during the solar plexus/forehead exercise.

Temperature variable	Treatment group	Baseline	Solar Plexus + Forehead	Baseline	Full exercise	Baseline
Middle finger	AT	26.32	27.20	24.51	25.93	25.92
	ATT	33.03	32.87	31.34	31.97	30.39
	ATE	32.95	33.79	32.53	33.17	30.85
Room	AT	21.09	21.42	21.86	21.89	21.99
	ATT	21.82	21.85	21.97	22.01	22.03
	ATE	20.34	20.47	20.79	20.97	21.33

Table 8.13: The mean middle finger and room temperatures during the sixth AT training session

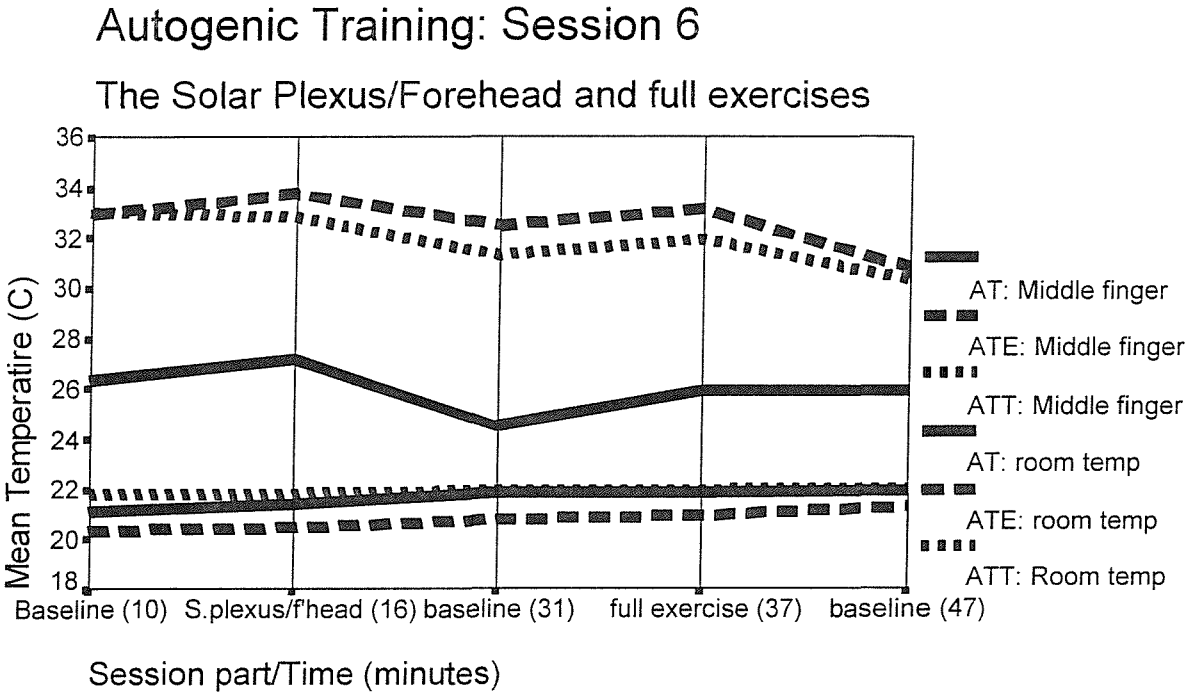

Fig 8.11 Room and middle finger temperature fluctuations during the sixth Autogenic Training session.

Summary of the Autogenic Training sessions

a. **Subjective effects of the exercises during the training sessions**

- The AT and ATT groups generally reported feelings of relaxation during the training exercises.
- The ATE group were generally undecided as to the effects of the relaxation exercises.

b. **Objective effects of the exercises during the training sessions**

- From the second session onwards, the AT group consistently demonstrated trends toward finger temperature increases during the relaxation exercises.
- The ATE group began to demonstrate and maintain finger temperature increases from the third to fifth training sessions; thus suggesting that the skill of relaxation takes longer to acquire through ATE training than through AT training alone. Indeed, one might expect this as the initial stages of Autogenic Training require that distractions - such as biofeedback apparatus (!) - in the training room be kept to a minimum.
- The ATT group demonstrated only occasional temperature increases during the relaxation sessions.

c. **Subjective practice rates of the exercises between the training sessions**

- Practice rates for the ATE group were consistently good.
- Practice rates for the ATT group were consistently poor.
- The AT group claimed to practice the exercises; however, discussion with the AT group members revealed that rather than practising the exercises as taught, they would resort to thinking warm thoughts during the practice periods.

In summary therefore, the AT and ATE groups appeared to gain most from the training sessions in terms of finger temperature elevations.

B. APPLIED RELAXATION

Session 1: Progressive Relaxation

A male ARE participant withdrew from the study before the first training session; therefore, table 8.14 and figure 8.12 display the group mean room and middle finger temperatures of 14 participants during the final minute of each section of the session.

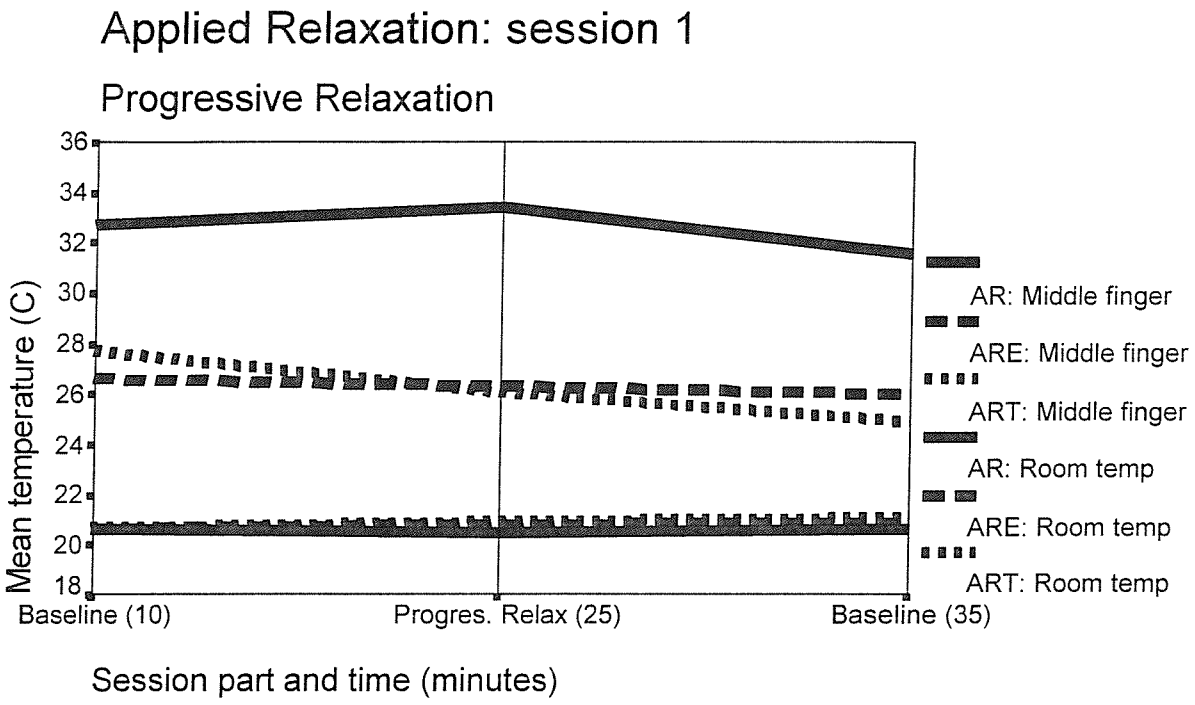

Fig 8.12 Room and middle finger temperature fluctuations during the first Applied Relaxation session.

Temperature variable	Treatment group	Baseline	Progressive Relaxation	Baseline
Middle finger	AR	32.70	33.45	31.58
	ART	27.74	26.09	24.92
	ARE	26.63	26.33	26.01
Room	AR	20.63	20.52	20.66
	ART	20.69	20.94	21.11
	ARE	20.66	20.82	20.89

Table 8.14: The mean middle finger and room temperatures during the first Applied Relaxation training session

Clearly, only the AR group showed any trend toward temperature increase during the period of progressive relaxation. In contrast, 64.3% (9) of the participants (spread equally across the groups) felt that they had relaxed during the exercise.

Session 2: Progressive Relaxation and Release-only relaxation

Only 13 participants attended the second training session because one female ART volunteer withdrew as she had been prescribed a vasoconstricting drug to combat high colour of the face. As evident from figure 8.13 and table 8.15, both the AR and ARE groups increased their middle finger skin temperature during the Progressive relaxation exercise, and showed a trend in the same direction during the Release-only exercise.

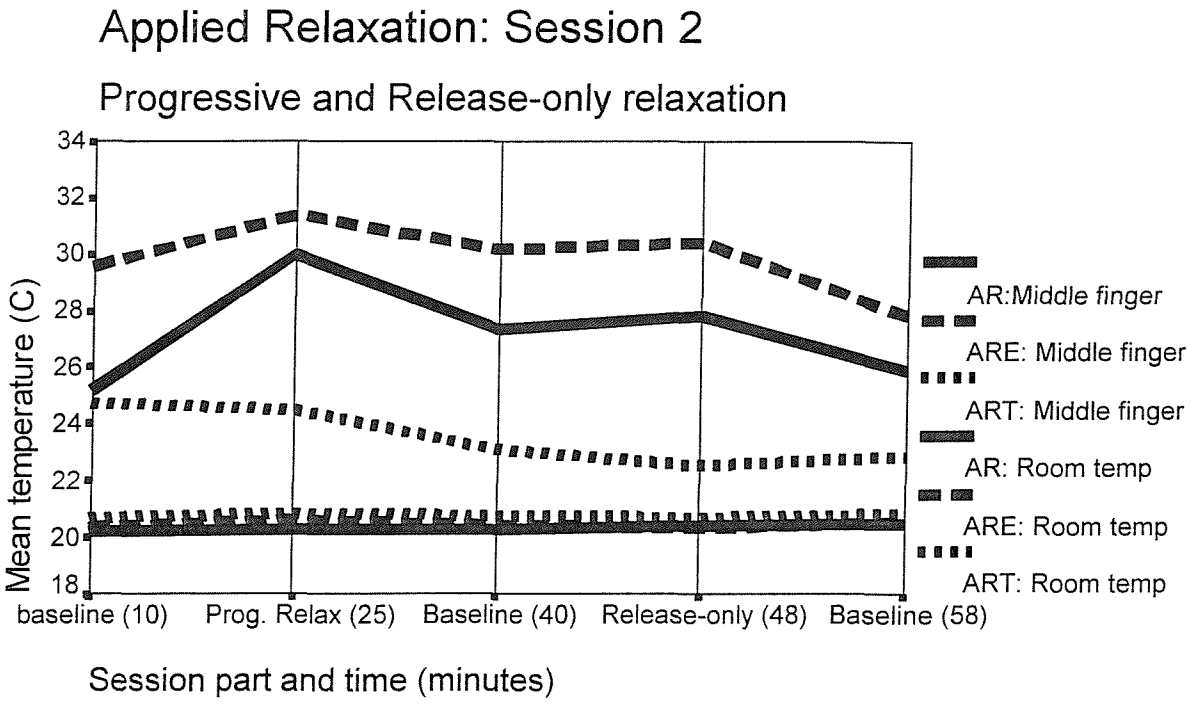

Fig 8.13 Room and middle finger temperature fluctuations during the second Applied Relaxation session.

One might assume that the marked increase in finger temperature during the Progressive relaxation exercise might be related to practice since the first training session. However, there were no

significant group differences in reported practice rates: 82% of the participants (for whom data were available) claimed to have practised at least once per day.

Temperature variable	Treatment group	Baseline	Progressive Relaxation	Baseline	Release only Relax	Baseline
Middle finger	AR	25.20	30.00	27.35	27.85	25.88
	ART	24.75	24.50	23.11	22.55	22.88
	ARE	29.58	31.40	30.17	30.41	27.83
Room	AR	20.20	20.28	20.26	20.43	20.47
	ART	20.71	20.86	20.78	20.72	20.83
	ARE	20.38	20.62	20.44	20.32	20.54

Table 8.15: The mean middle finger and room temperatures during the second Applied Relaxation training session

In contrast, the subjective effects of the exercises tied in well with the observed effects in that all of the AR participants and 50% of the ARE group as compared with only 25% of the ART group considered themselves to have relaxed during the Progressive relaxation exercise. This was not the case during the Release-only exercise in that most (76.9%) of the participants described feelings of relaxation during the exercise.

Session 3: Cue-controlled relaxation

An AR participant withdrew before the third training session as she felt that she couldn't do the treatment justice given the time commitment involved. Consequently, table 8.16 and figure 8.14 display the session temperature fluctuations of 12 participants (4 of each treatment group).

Temperature variable	Treatment group	Baseline	Release only Relaxation	Cue-controlled Relaxation	Baseline
Middle finger	AR	29.16	31.72	30.75	28.53
	ART	21.41	22.76	22.34	22.09
	ARE	26.92	27.80	26.98	26.63
Room	AR	20.08	20.11	20.15	20.31
	ART	20.90	21.06	21.07	21.25
	ARE	20.29	20.35	20.40	20.24

Table 8.16: The mean middle finger and room temperatures during the third Applied Relaxation training session

All groups increased their finger skin temperature during the Release-only exercise, but to differing degrees: the AR group showing the most prominent increase; the ARE group, the least. However, the temperatures of all three groups decreased slightly during the subsequent Cue-controlled section of the exercise.

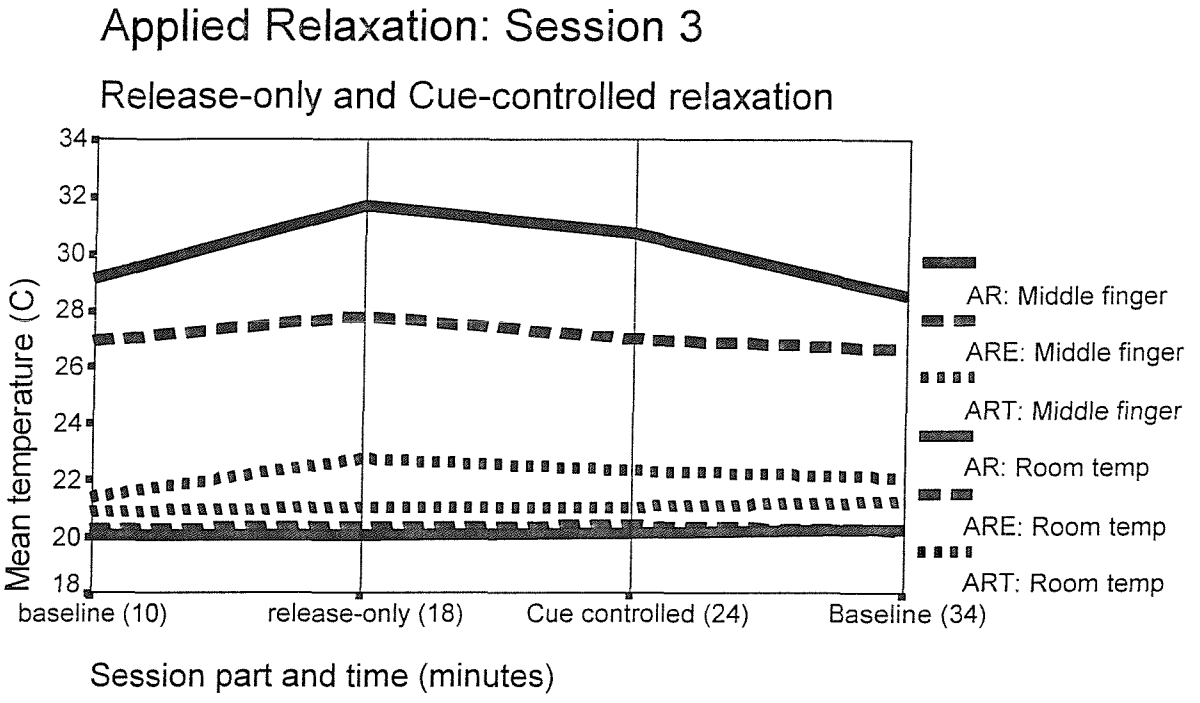

Fig 8.14 Room and middle finger temperature fluctuations during the third Applied Relaxation session.

Subjectively, there were no significant differences in the degree of relaxation across the treatment groups for the exercise as a whole. Similarly, group practice rates did not differ - 75% of all participants claimed to have practised the Release-only exercise twice daily since the previous session.

Session 4: Differential Relaxation

All 12 of the remaining Applied Relaxation volunteers attended their fourth training session. Temperature fluctuations are displayed in figure 8.15 and table 8.17. Reported practice rates did not

differ across the three treatment groups: 92% (11) claimed to have practised at least once/day since the previous training session, and 67% (8), twice per day.

As shown in the figure and table below, the finger temperature of the ARE and ART groups increased during the Release-only/Cue-controlled part of the exercise. The AR group showed a slight increase in finger temperature during this part of the exercise, but the initial warmth of their finger during the baseline period may have prevented further temperature elevations.

Temperature variable	Treatment group	Baseline	Release only/ Cue-controlled	Differential #1	Baseline
Middle finger	AR	32.20	32.72	31.24	30.31
	ART	26.10	27.92	27.45	26.03
	ARE	28.20	30.56	29.71	28.88
Room	AR	20.63	20.54	20.66	20.72
	ART	21.41	21.40	21.46	21.47
	ARE	20.55	20.42	20.46	20.53

Table 8.17: The mean middle finger and room temperatures during the fourth Applied Relaxation training session

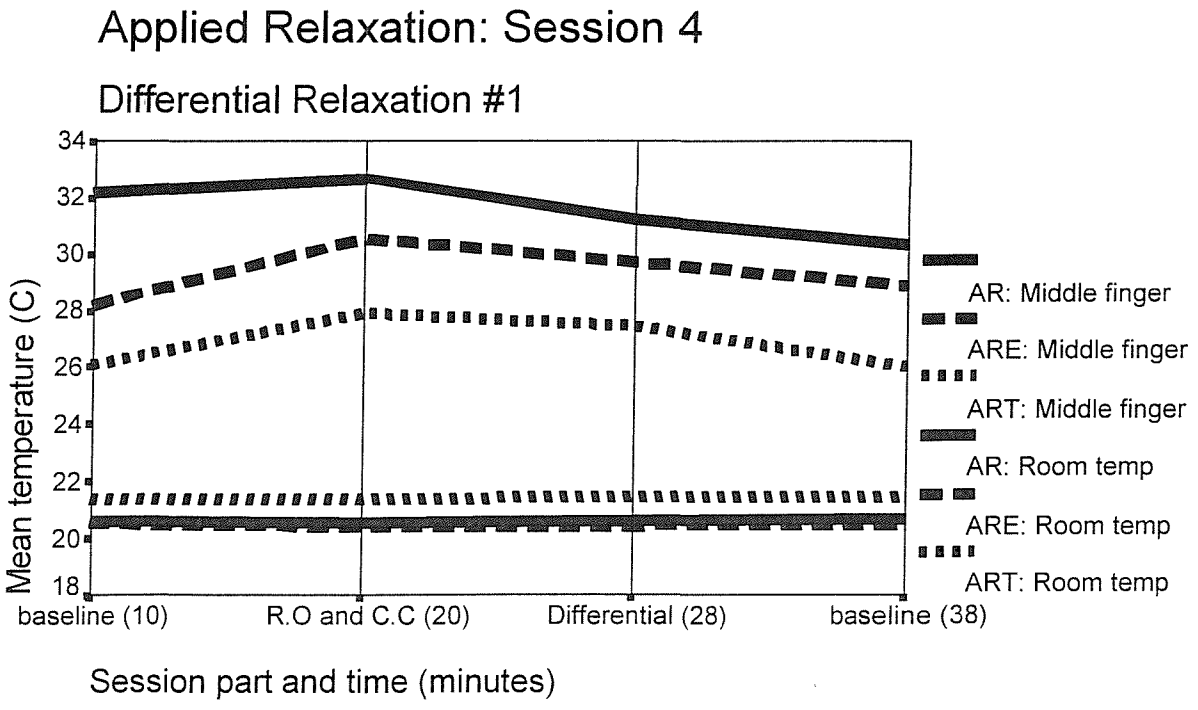

Fig 8.15 Room and middle finger temperature fluctuations during the fourth Applied Relaxation session.

All three groups showed a decrease in finger skin temperature during the Differential Relaxation part of the exercise. Interpreting these decreases is not as straight forward as in the previous sessions given the physical movement required during the Differential relaxation section of the exercise. It is therefore, necessary to consider the possible causes of temperature change during a period of conventional relaxation and when carrying out the necessary movement s of Differential relaxation. (The reader will recall that this exercise required the trainee to maintain a feeling of relaxation whilst moving their eyes, hands, arms, feet and legs in both a sitting and standing position.)

As previously noted, an increase in digital blood flow may result from the decrease in sympathetic nervous activity associated with physical relaxation. Alternatively, peripheral blood flow is likely to increase (for a short period) in line with physical movement following a period of inactivity. Furthermore, in warm ambient conditions (ie at temperatures above skin temperature), fingers will warm as heat is transferred to the skin.[30]

Skin temperature decreases may be similarly explained firstly by greater physical tension in the body increasing sympathetic nervous activity, and thereby reducing blood flow to the peripheral vessels, and secondly through the loss of warmth through evaporation to the surrounding cooler air. Here, the amount of heat lost will be proportional to the amount of blood in the peripheral vessels: the greater the blood flow at the surface, the greater the loss of heat to the cool ambient surroundings. Moreover, this loss of heat will be greater as the surface area of the hand in contact with the cool air increases. Therefore, movement of the hand through the air, as required during the Differential relaxation exercise, must have a cooling effect on digital skin temperature. Given that the ambient temperature is relatively constant and a number of degrees lower than the finger skin temperature, one might expect heat loss to be greater from a warm hand than cool hand simply because there is more blood/heat at the surface of a warm hand to be lost. Indeed, if one were to measure heat loss and the effect on skin temperature during a discrete period, there is little doubt that this would be the case. However, one must recall that blood flow to the fingers is a continuous action such that the

[30] Obviously, the relatively low ambient temperatures of the training session rule out any such explanation in this case.

heat lost through evaporation from a relaxed warm hand will rapidly be replaced by a continual rich supply of blood. In a cooler hand however, although the loss of heat through evaporation will be somewhat lower, this loss will increase the vasoconstrictive effects of cold on the peripheral vessels resulting, in the longer term, in an overall cooling of the hand.

The groups' decrease in finger skin temperature during the Differential relaxation exercise following an increase during the more conventional release-only and cue-controlled parts of the exercise is wholly compatible with the association between relaxation and the control of peripheral temperature. Differential relaxation involves maintaining a state of physical relaxation in areas of the body not involved in the movement of some other part of the body. For example, physical tension in the biceps muscle is not a pre-requisite of the action of moving the eyes. The point being that during the Differential Relaxation phase of the exercise, the quiet, unmoving, relaxed participant is required to move - an action which is bound to interfere with both physical and mental relaxation (and associated finger temperatures) at least until the art of Differential relaxation has been mastered.

The subjective reports of the effects of the exercises suggest that most of the participants (73%) felt relaxed during the training session - presumably during the Release-only and Cue-controlled section of the exercises as it is unlikely that a trainee could fully maintain a feeling of relaxation during their first experience of having to stand and move whilst relaxed.

Session 5: Differential Relaxation part 2 (walking)

All 12 participants attended the fifth training session. Of the 10 participants for whom data were available, all claimed to have practised the previous session's exercise at least once per day, and 80% twice a day.

Table 8.18 and figure 8.16 display the temperature fluctuations of the training session. In line with the subjective reports of the effects of the exercises, the AR group's finger temperature increased during the cue-controlled phase of the exercise, the ART group did so too, but to a lesser degree, and

the ARE group temperature actually decreased during this part of the session. (Subjectively the AR group felt relaxed, the ART group were unsure as to the effects of the exercises, and the ARE group felt tense throughout). As in the previous session, the group finger temperatures decreased during the Differential Relaxation exercise.

Temperature variable	Treatment group	Baseline	Cue-controlled Relaxation	Differential #2 Relaxation	Baseline
Middle finger	AR	30.88	33.08	29.51	28.44
	ART	26.23	27.37	24.58	24.16
	ARE	30.92	30.19	27.56	29.62
Room	AR	20.33	20.27	20.62	20.49
	ART	21.26	21.34	21.61	21.60
	ARE	21.04	21.10	21.52	21.17

Table 8.18: The mean middle finger and room temperatures during the fifth Applied Relaxation training session

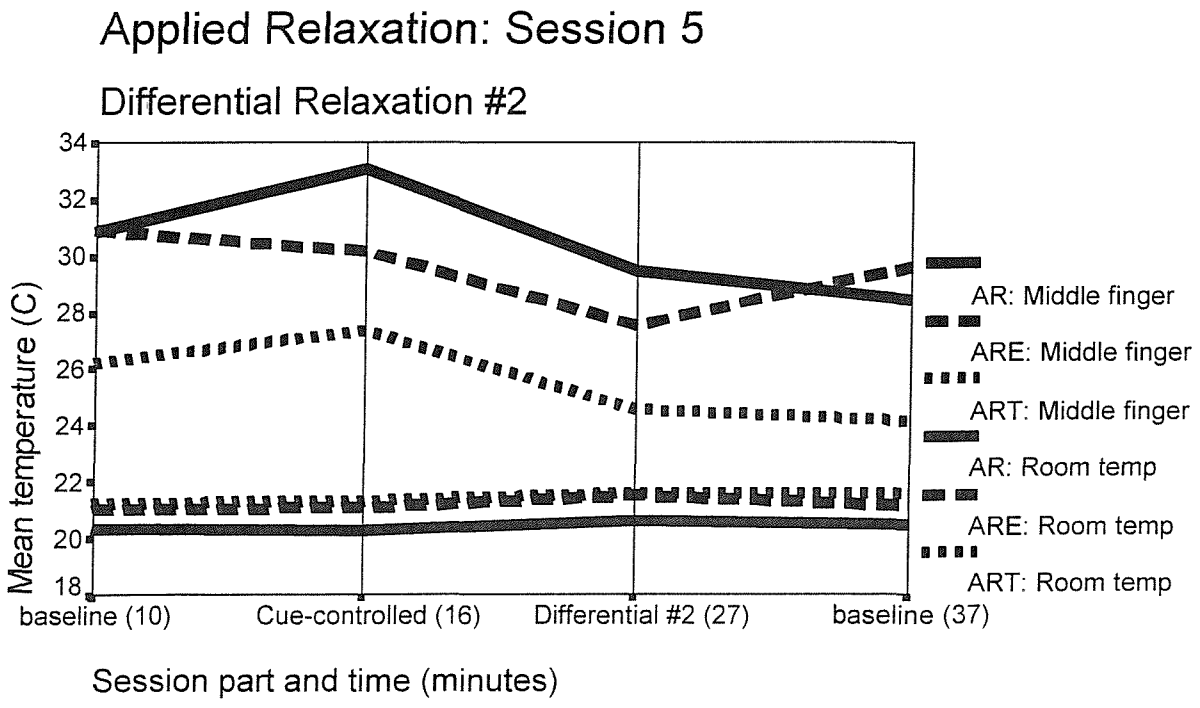

Fig 8.16 Room and middle finger temperature fluctuations during the fifth Applied Relaxation session.

Session 6: The full exercise (Differential Relaxation and controlled breathing)

Of the 12 participants to attend the final training session, practice rates were good: 91% claiming to have practised at least once per day. Subjectively, the AR group considered themselves to have relaxed during the session, the ART group were mixed in their opinion, and the ARE group felt tense throughout the exercise. In contrast, as shown in table 8.19 and figure 8.17, the objective results paint a different picture.

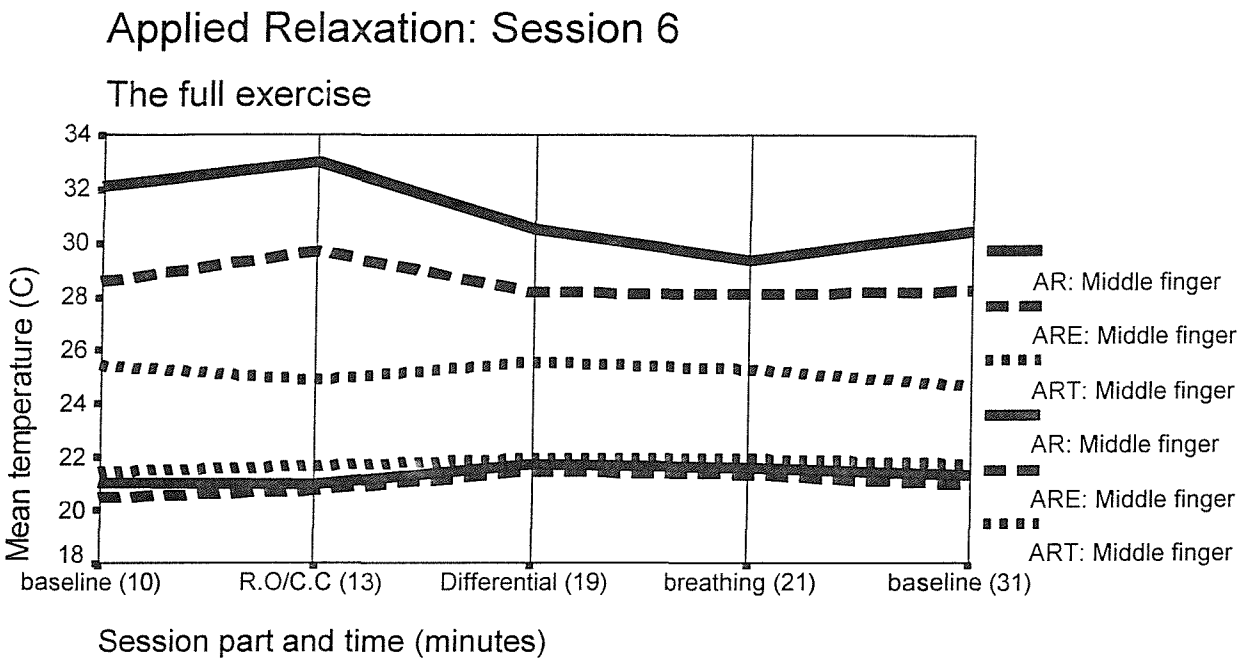

Fig 8.17 Room and middle finger temperature fluctuations during the sixth Applied Relaxation session.

Temperature variable	Treatment group	Baseline	Cue-controlled Relaxation	Differential Relaxation	Breathing	Baseline
Middle finger	AR	32.13	33.04	30.54	29.36	30.45
	ART	25.46	24.90	25.57	25.29	24.68
	ARE	28.57	29.73	28.17	28.09	28.23
Room	AR	21.04	20.98	21.73	21.62	21.35
	ART	21.46	21.68	21.95	21.93	21.75
	ARE	20.47	20.75	21.47	21.35	20.94

Table 8.19: The mean middle finger and room temperatures during the sixth Applied Relaxation training session

The AR and ART groups increased in temperature during the Cue-controlled section of the exercise, and gradually decreased in temperature as the exercise progressed despite a slight increase in room temperature during the exercise. In contrast, the ART group finger skin temperature remained relatively constant (and low) throughout the session.

Summary of the Applied Relaxation sessions

a. **Subjective effects of the exercises during the training sessions**

- The AR groups reported feelings of relaxation throughout the sessions.
- The ARE group felt relaxed only during the first 4 sessions.
- The ART group were generally of mixed opinion in terms of the subjective effects of the exercises.

b. **Objective effects of the exercises during the training sessions**

- The AR group consistently demonstrated trends toward finger temperature increases during the relaxation exercises, and the ART group did so from the third training session.
- The ARE group showed relaxation-related temperature increases during the early stages of training, but failed to do so in the latter two training sessions.
- The ART group only began to demonstrate temperature increases by the third training session.

c. **Subjective practice rates of the exercises between the training sessions**

- Practice rates for all of the three groups were adequate throughout training.

In summary therefore, the AR group demonstrated both objective and subjective temperature increases during the training period, and must therefore have gained the most Raynaud's-related benefit from the treatment. This benefit appears not to be a function of practice between sessions.

THE EFFECTS OF TREATMENT

The post-treatment assessment data were collected over a three month period between March and June 1994. The training period (but not the number of sessions) for several participants was extended due to illness. Therefore, training and assessment sessions were carried out concurrently during the 3 month period. Between recruitment and the post-treatment assessment sessions, 7 Experimental (2 AT, 2 ATT, 1 AR, 1 ART, and 1 ARE) and 2 Control participants withdrew from the study. 2 further experimental participants (ATT and ATE) withdrew before the follow-up assessment session.

As the reader will recall, the effects of treatment were assessed both post treatment and at follow-up i.e. 3-4 months after treatment. In line with the pre-treatment analysis, the effects were assessed both subjectively (via questionnaire) and objectively (via the tests of voluntary control, cold challenge and ambulatory monitoring). Analysis of the data was approached in two ways. Firstly, the subjective questionnaire data were manipulated to provide a subjective description of any effects of treatment both after the training period, and again at follow-up. In contrast, the pre-treatment, post treatment and follow-up data of each of the three objective tests (i.e. voluntary control, cold challenge and ambulatory monitoring) were compared in an incomplete blocks design analysis of variance to assess test performance of the biofeedback and relaxation groups over the three time periods of the study.

THE SUBJECTIVE EFFECTS OF TREATMENT

A. Analysis Of the Post-Treatment And Follow-Up Questionnaires

The post-treatment and follow-up questionnaires provide data on symptoms, on lifestyle changes, and on changes in home, medical and/or surgical treatment(s) - other than those experienced as part of the study. Moreover, in the case of the experimental participants, the frequency at which the relaxation

exercises were practised was also assessed. Data were coded in line with the coding frame outlined in Appendix 2.4a, and compared using Chi-square analysis.

a. The Post-treatment questionnaire

Chi-square analysis revealed, that despite the loss of volunteers since the pre-treatment assessment, the Experimental and Control participants were comparable: there had been no reported change in experience of relaxation techniques (other than those introduced as part of the study), or use of home remedies during the training period; the medication of most (90%) of the participants had not altered (whereas 7% had actually reduced their Raynaud's medication); and only one participant (AR) had undergone surgery for her condition (Carpel Tunnel). Moreover, there were no significant differences between the experimental groups in reported practice rates (83% claiming to practice once or twice a day between training sessions), consistency of practice (70% practising consistently), motivation to practice (52% finding it easy to motivate themselves to practice), or enjoyment of the exercises practised between sessions (57% claiming to enjoy doing the exercises at home).

The main question of interest was that of symptom change since the completion of the pre-treatment questionnaire. Data were coded to two categories: i. Subjective improvement in Raynaud's symptoms; and ii. no improvement in Raynaud's symptoms, but improvement in other areas of life (e.g. Sleeping habits).

More of the experimental participants described symptom improvement than was the case for the Control participants: AR versus AT versus controls (Chi-square (2) = 8.22 $p< 0.05$); No biofeedback versus Temperature biofeedback versus EMG biofeedback versus controls (Chi-square (3) = 15.22 $p<0.05$). Indeed, none of the Control participants felt that their symptoms had improved, as compared with 57% (13) of the Experimental participants; thereby suggesting that the behavioural

approach is, on the whole, better than no behavioural treatment for the subjective relief of Raynaud's symptoms. (Please refer to statistical appendix 2.2; tables 1.a.I and 1.a.II; page S5).

Given the tendency for those who received treatment to describe an improvement in their symptoms, and the lack of such a trend amongst the Control participants, it is worth considering which factors are associated with symptom improvement.

Not surprisingly, symptom improvement is associated with post-treatment views of "alternative treatments". Where symptoms are reported to have improved, views of alternative treatments are positive: Chi-square (2) = 13.33 $p < 0.05$. (Statistical appendix 2.2; table 1.b.I; page S5). Furthermore, there is a strong association between subjective symptom improvement and the perception of "stress" as a trigger of attacks i.e. those who completed treatment were more likely to report positive effects on symptoms if they had perceived stress as a symptom precipitator at the start: Chi-square (1) = 9.12 $p < 0.5$. (Statistical appendix 2.2; table 1.c.I; page S5). This presumably stems from the premise that "relaxation", being the opposite of "stress", will only benefit those whose symptoms are related to stress. Yet, as the reader may recall, during the first treatment session, the role of the relaxation technique in reducing physical tension, rather than psychological "stress" was clearly explained to the participants. Presumably, the point was not presented clearly enough, and should be laboured in future work. Alternatively, the strong association between the perception of stress as precipitator and pre-treatment optimism about treatment outcome suggests that those who expected to gain the most from treatment, did so.

b. The Follow-up questionnaire

2 participants (1 ATT and 1 ATE) chose not to attend the final assessment session. Details of the 29 participants who completed the study are reported in table 8.20.

None of the participants underwent surgery or used other non-conventional treatments between the two post-training assessment sessions. However, 4 (14%) experienced changes in their life(style) that might influence symptoms e.g. the onset of puberty, marital separation and increased exercise. 7% (1 Control; 1 ARE) began a course of new "Raynaud's" medication, and one Control participant reduced his medication.

Treatment Group	Gender	Diagnosis	Age (mean and range)
AT	2 F; 1 M	2 unsure; 1 undiagnosed	mean: 38 range: 12-67
ATT	2 F; 0 M	1 unsure; 1 undiagnosed	mean: 56 range: 49-62
ATE	4 F; 1 M	1 R Disease; 2 unsure; 2 undiagnosed	mean: 38 range: 29-47
AR	3 F; 1 M	1 Disease; 1 R Phenomenon; 1 unsure; 1 undiagnosed	mean: 54 range: 45-57
ART	3 F; 1 M	2 Phenomenon; 2 Unsure	mean: 54 range: 46-67
ARE	2 F; 1 M	1 Disease; 1 Phenomenon; 1 unsure	mean: 59 range: 48-73
Control	5 F; 3 M	2 Phenomenon; 3 unsure; 3 undiagnosed	mean: 53 range: 26-68

Table 8.20: Demographic details of the 29 participants who completed the study

As is clear from the table above, there are age and diagnostic differences between the Relaxation groups: the Applied Relaxation participants are older and include sufferers of Secondary conditions whereas this is generally not the case for the Autogenic Training volunteers. (Please recall that this was not the case at the start of the study). If one assumes that those without an identified physical "cause" for their symptoms are likely to benefit most from behavioural treatment, it might be

surmised that the AT patients will show greatest subjective and objective improvement in their symptoms following treatment.

As in the second assessment, the experimental participants claimed that their symptoms and/or other areas of their life had improved since training had ended. In contrast, the control participants made no such claim: AR versus AT versus Controls (χ^2 (2) = 6.50 $p < 0.05$); No biofeedback versus temperature biofeedback versus EMG biofeedback versus Controls (Chi-square (3) = 10.95 $p < 0.05$). (See statistical appendix 2.2; tables 2.a.I and 2.a.II; page S6). Overall, 52.4% (11) of the experimental participants felt that their Raynaud's symptoms had improved when compared with previous years (e.g. RS: "I feel able to control the body's symptoms", SG: "It has shortened the length of attacks - especially in the finger area"); 38.1% (8) noted treatment-related improvements in other areas of their life e.g. a decrease in general anxiety, an increase in motivation, coping better with day to day activities, and improved sleeping patterns. 9.5% (2) experienced no subjective benefit from treatment.

As to subjective ratings of symptom severity at follow-up, both the Experimental and Control participants reported mild or no Raynaud's symptoms. One might therefore, assume a role of seasonal factors in ratings of symptom severity rather than failure of the treatment. Similarly, that subjective symptom severity did not differ between the Experimental and Control participants, might also be related to a lack of commitment to the practice of the exercises. 95.2% (20) claimed to have practised the exercises since the previous assessment session. (An ATE participant had experienced no subjective benefit at all during the 5 month treatment period, and therefore, felt no compunction to practice further). The level of practice however, was poor: 71.4% (15) rarely practised i.e. perhaps once a week if they remembered; and only 23.8% (5) practised once a day or more. Of the latter group, only 60% (3) were practising at the required rate of at least twice a day. Similarly, practice was inconsistent, with only 14.3% (3) practising both consistently and frequently. Interestingly, most

claimed to have started with good intentions (practising frequently for a short period after the second assessment session), but lost the motivation to practice as the weeks passed. 61.9% (13) claimed to use the relaxation techniques during Raynaud's attacks, and as a consequence of this, 6.7% (2) no longer felt the need to use previously invaluable "home remedies" to relieve their symptoms. However, the use of relaxation techniques during an attack is not subjectively associated with a reduction in the severity of attacks.

What, therefore, led over 50% of the experimental participants to claim symptom improvement following treatment ? Looking at the participants who completed the study, the perception of "stress" as a precipitator of Raynaud's symptoms and subjective improvement in symptoms were strongly linked: Chi-square (2) = 6.14 $p < 0.05$ - statistical appendix 2.2; table 2.b.I.; page S6. Indeed, 63.6% (7) of those who reported symptom improvement saw stress as a precipitator of attacks; of those whose symptoms did not improve, 90.9% (10) felt that stress was not involved in the onset of their symptoms. Similar trends are mirrored by the experimental participants in isolation. Although not statistically significant, 70% (7) who perceived stress as involved in their symptoms, reported symptom improvement, whereas only 10% (1) experienced no subjective benefit from treatment. Further, trends suggest that subjective symptom improvement is proportional to how much the experimenter felt at ease with and liked the participants (as rated on a three point scale: like - neutral - dislike). Of those whose symptoms improved subjectively, 72.7% (8) were "liked"; none were disliked. Moreover, 50% (1) of those who experienced no subjective benefit were actually disliked by the experimenter. However, whether the experimenter's view was conditioned by the participants' view of treatment, or vice versa, is a moot point.

Looking at the diagnosis of the participants who completed training, trends in the data suggest that those who were unsure of their diagnosis, or were undiagnosed, claimed greater benefit from

treatment: 75% (9) of the unsure/undiagnosed participants reporting symptomatic improvement as compared with only 22.2% (2) of the Raynaud's Disease or Phenomenon participants.

In summary, therefore, there were no significant differences in the subjective effects of any particular treatment - nor any such differences in terms of practice rates. However, the experimental participants did claim subjective symptom improvements not experienced by the control participants.

THE OBJECTIVE EFFECTS OF TREATMENT

In order to evaluate the effects of treatment through the tests of voluntary control of finger skin temperature, the cold challenge test, and the period of ambulatory monitoring, an incomplete blocks design split plot analysis of covariance was performed for each of the three sets of data. As clearly displayed in table 8.21, the incomplete cells in the design were those of temperature biofeedback without relaxation, and EMG biofeedback without Relaxation. The reader will recall the position of this thesis in stating that biofeedback is not a valid treatment in its own right.

	Independent Variable: Relaxation Technique			
Independent Variable: Biofeedback Technique		Autogenic Training	Applied Relaxation	No Relaxation
	Temperature Biofeedback	*	*	
	EMG biofeedback	*	*	
	No Biofeedback	*	*	*

Table 8.21: The design of the analyses performed. N.B. there are no data in the shaded cells

As shown in the table above, two between subject independent variables were entered into the analysis: namely, Relaxation technique and Biofeedback technique. Time of testing (i.e. pre-treatment, post-treatment or follow-up) was also entered as the within subjects independent variable.

Given the different tests used to assess treatment effects (voluntary control, cold challenge and ambulatory monitoring), the dependent variables and covariates entered into the analysis differed across the assessment tests. Consequently, specific details of each set of variables will be described below under the appropriate test heading.

To supplement these analyses, *a priori* univariate analyses will be used to compare the results of specific groups on the three objective tests. The a priori comparisons of interest, and the reasons for making the comparisons are listed in table 8.22.

COMPARISONS OF INTEREST	WHY CARRIED OUT
1. No Treatment versus All treatments combined (Controls) v. (AT, ATT, ATE, AR, ART, ARE)	To see whether treatment carries any positive effects over no treatment.
2. Biofeedback versus No Biofeedback (ATT, ATE, ART, ARE) v (AT, AR)	To assess the role of biofeedback over and above that of relaxation.
3. No treatment versus each of the 6 individual treatments (Controls) v. (AT, ATT, ATE, AR, ART, ARE)	To see whether any individual treatment carries any positive effect over that of no treatment.

Table 8.22: *A priori* comparisons used to complement the main analyses within each of the three data sets

The main incomplete blocks design split plot analysis of covariance described above will allow effects of treatment to be evaluated at three different levels: firstly, changes over time (i.e. any differences between participants at the pre-treatment, post-treatment and follow-up assessments); secondly, differences between the Relaxation groups (i.e. Applied Relaxation, Autogenic Training and No Relaxation); and finally, differences between the biofeedback groups (i.e. Temperature biofeedback, EMG biofeedback and No Biofeedback). However, such an analysis will reveal only the presence of differences between groups - not the nature or direction of differences. Thus, should the main analysis uncover statistically significant effects, *Post Hoc* tests will be used to communicate the nature of the differences.

It should be noted at this point that for each of the three objective tests of the effects of treatment, the main analysis described above will be carried out twice. In the first analysis, Biofeedback will be entered into the analysis first so as to describe the overall effects of Relaxation within the model. In the second main analysis, relaxation will be entered into the analysis before biofeedback; thereby, describing the overall effects of Biofeedback. Where the F-ratios for a particular main or interactive effect differ, both F-ratios will be described in the text. For the purpose of standardisation, the F-ratio related to the first analysis (the overall effects of relaxation) will be listed first, and that related to the second analysis (the overall effects of Biofeedback) will follow.

A. The Voluntary Control of Finger Skin Temperature

Given that the three assessment sessions took place at different times of year, one must consider the effects of the outside temperature on clothing worn by the participants - in terms of number and thickness of layers and the nature of the garment. However, all participants dressed appropriately for the time of year, so their different clothing at the three assessment sessions would, to some extent, reduce the effects of differences in the outside temperature. In contrast, room temperature during the sessions would differ across the testing times in line with seasonal use of central heating. Therefore, room temperature during the assessment sessions was taken into account in all analyses performed.

To recapitulate, the test of voluntary control of finger temperature involved a short baseline period, followed by a period during which participants were asked to try to increase their finger temperature. During the session, the temperature of both the middle and index fingers was measured - as was the temperature of the soundproof room. As in the pre-treatment analysis, only the middle finger and room temperature data were included in the analysis. A temperature difference, indicative of any change in temperature during voluntary control, was calculated for both the middle finger and room temperature by subtracting the temperature in the last minute of the baseline period from that of the last minute of the voluntary control period for each participant. A positive result being indicative of

an increase in temperature during the voluntary control period, and a negative result, a decrease. The middle finger temperature difference (at each of the three assessment sessions) was entered into the analysis as the dependent variable, whilst the room temperature difference, and the middle finger and room temperatures at the start of the voluntary control period were included as covariates.

A.I. Voluntary Control of finger skin temperature

98 middle finger temperatures[32], or "difference scores", were entered into each of the two incomplete blocks design split plot analyses of covariance. The outputs of these analyses are shown in Statistical Appendix 2.3 Tables 1.a.I and 1.aII (Page S7 and S8). The text below will interpret the covariate, the main, and the interactive effects of the effects of relaxation and biofeedback.

A.I.a. Covariates: *evaluation of the role of the covaried variables on the middle finger temperature difference during the test of voluntary control*

The main analyses demonstrate a significant relationship between middle finger skin temperature and middle finger starting temperature: $F(1, 33) = 5.15$ $p<0.05$, and between middle finger skin temperature and room temperature throughout the period of voluntary control: $F(1, 33) = 14.30$ $p<0.001$. (Statistical Appendix 2.3 Tables 1.a.I and 1.aII, Page S7 and S8). The analyses do not suggest a relationship between middle finger skin temperature and room starting temperature. The nature of the covariate relationships are outlined in table 8.23, and described more fully in the text below.

Covariate	**Effect of a single unit increase (1°C) in covariate temperature on finger skin temperature**
Room starting temperature	Increase by 0.34°C
Middle finger starting temperature	Decrease by 0. 1°C
Room temperature difference	Decrease by 4°C

Table 8.23: The effect of a 1°C rise in the temperature of each covariate on middle finger skin temperature during voluntary control

[32] i.e. those of 38 participants at the pre-treatment assessment (28E, 10C), 31 at the post-treatment session (23 E; 8 C), and 29 at the follow-up assessment session (21E, 8C).

Looking only at the covariates in table 8.23 that are significantly related to the change in middle finger skin temperature, a higher starting middle finger skin temperature is associated with a lower rise in finger skin temperature during the test of voluntary control: t = -2.03 $p<0.05$ (95% CI for difference: -0.197, -0.002). Thus, owing to a ceiling effect, as middle finger skin starting temperature increases, middle finger temperature during the voluntary control period may decrease, or increase less than would be the case with a lower starting middle finger skin temperature. Secondly, as room temperature rises during the test period, the difference in middle finger skin temperature between the baseline and voluntary control period decreases: t = -3.31 $p<0.05$. (95% CI for difference: -3.96, -0.32). Therefore, depending on the magnitude of finger temperature increase during voluntary control, a rise in room temperature during the session will either reduce the measured increase in finger temperature, or result in a decrease in that finger skin temperature. Please refer to statistical appendix 2.3 (table 1.a.III, page S8).

A.I.b. Time: *comparison of the pre-, post- and follow-up data*

The main analysis reveals no statistically significant effect of time of testing on middle finger skin temperature: $F(2, 41) = 0.14$ $p> 0.05$ (Please refer to statistical appendix 2.3; table 1.a.I, page S7). Indeed, as outlined in table 8.24, the mean middle finger temperature "difference scores" for the groups as a whole are not significantly different at pre-treatment, post-treatment and follow-up.

	Middle finger temperature difference (°C)	**Room temperature difference (°C)**	**Middle finger starting temperature (°C)**	**Room starting temperature (°C)**
Pre-treatment	0.15	0.05	28.21	22.74
Post-treatment	0.56	0.08	28.32	22.12
Follow-up	0.73	0.17	28.46	21.75

Table 8.24: The mean finger and room (starting) temperatures (°C) over the three assessment sessions.

A.I.c. Biofeedback group: *comparison of the 3 biofeedback groups during the assessment sessions.*

The mean room and middle finger starting and difference temperatures for the biofeedback groups during the three assessment sessions as a whole are displayed in table 8.25.

	Middle finger temperature difference (°C)	Room temperature difference (°C)	Middle finger starting temperature (°C)	Room starting temperature (°C)
No Biofeedback	0.41	0.10	28.68	22.22
Temperature Biofeedback	-0.06	0.10	27.41	22.78
EMG Biofeedback	1.12	0.06	28.38	21.92

Table 8.25: The Biofeedback group mean finger and room (starting) temperatures (°C) pooled across the pre-treatment, post-treatment, and follow-up assessment sessions

Although the table above suggests that the EMG biofeedback group achieved the greatest overall finger temperature increases during voluntary control, and that the Temperature biofeedback group decreased slightly in temperature during the voluntary control sessions, the main analysis of covariance uncovered no statistically significant difference in ability to control finger skin temperature between the biofeedback groups: $F(2, 33) = 2.92$ $p>0.05$; $F(2, 33) = 2.77$ $p> 0.05$. (See Statistical appendix 2.3; tables 1.a.I and 1.a.II, pages S7 and S8) .

A.I.d Relaxation group: *comparison of the relaxation groups across the assessment sessions*

Table 8.26 describes the mean middle finger and room starting and difference temperatures for the relaxation groups during the three assessment sessions as a whole.

	Middle finger temperature difference (°C)	Room temperature difference (°C)	Middle finger starting temperature (°C)	Room starting temperature (°C)
Autogenic Training	0.50	0.09	28.85	22.11
Applied Relaxation	0.66	0.08	28.51	22.36
No Relaxation	-0.12	0.11	27.24	22.34

Table 8.26: The Relaxation group mean finger and room (starting) temperatures (°C) pooled across the pre-treatment, post-treatment, and follow-up assessment sessions

The main analysis of covariance revealed no significant effect of Relaxation group: $F(2, 33) = 1.73$ $P > 0.05$; $F(2,33) = 1.89$. (Please refer to statistical appendix 2.3; tables 1.a.I and 1.a.II, pages S7 and S8).

A.I.e.The interaction between time of testing and biofeedback group

The main analyses revealed no significant interaction between time of testing and biofeedback group: $F(4, 41) = 1.69$ $p > 0.05$; $F(4, 41) = 1.43$ $p > 0.05$ (Statistical appendix 2.3; tables 1.a.I and 1.a.II, pages S7 and S8). The biofeedback groups did not, therefore differ in ability to control finger skin temperature over the three assessment periods.

A.I.f.The interaction between time of testing and relaxation group

Similarly, the main analysis in which relaxation was the second variable entered, uncovered no significant interaction between time of testing and Relaxation group: $F(4, 41) = 1.69$ $p > 0.05$ (Statistical appendix 2.3; table 1.a.I, page S7). However, the main analysis describing the effect of biofeedback on middle finger temperature, revealed a significant relationship between relaxation and time: $F(4, 41) = 2.65$ $p < 0.05$. (Statistical appendix 2.3; table 1.a.II, page S8). Therefore, the relationship between time of testing and the relaxation groups' ability to control finger skin temperature across the three time periods remains unclear. Further Post Hoc analyses as described in section A.III (Pages 167-168) will clarify matters.

A.I.g.The interaction between relaxation and biofeedback group

Biofeedback was included in the study design so that it might be ascertained whether the effects of relaxation techniques on control of finger skin temperature are augmented or hindered by biofeedback. The main analysis of covariance revealed no interactive effect of biofeedback and relaxation in the control of finger skin temperature: $F(2, 33) = 1.55$ $p > 0.05$ (Statistical appendix 2.3; table 1.a.I page S7). Thus, one can assume that where biofeedback was used in conjunction with

relaxation techniques during training sessions (though not home practice), it neither increases nor hinders the patient's control of finger skin temperature mediated through relaxation.

A.I.h.The interaction between time of testing and relaxation and biofeedback group

Finally, the main analysis indicates no interaction between biofeedback group, relaxation group and time: F (4, 41) = 0.51 p> 0.05 (Statistical appendix 2.3; table 1.a.I page S7). Clearly, therefore, relaxation training with or without biofeedback does not affect the ability of Raynaud's patients to control finger skin temperature at will.

AII.The a priori comparisons: univariate analyses of covariance

A.II.a No treatment (Controls) versus all treatments combined (AT, ATT, ATE, AR, ART, ARE)

Table 8.27 describes the mean room and middle finger temperatures of the treated and untreated participants during the three assessment sessions as a whole.

	Middle finger temperature difference (°C)	**Room temperature difference (°C)**	**Middle finger starting temperature (°C)**	**Room starting temperature (°C)**
Experimental	0.71	0.08	28.68	22.24
Control	-0.05	0.12	27.24	22.34

Table 8.27: The treated and untreated (experimental and control) mean finger and room (starting) temperatures (°C) pooled across the pre-treatment, post-treatment, and follow-up assessment sessions

A.II.a.i Pre-treatment effects

An a priori analysis of covariance comparing the control of finger skin temperature of the pooled experimental groups and the controls. revealed a significant effect of room starting temperature on control of pre-treatment middle finger skin temperature: F (1, 35) = 4.99 p <0.05. However, no significant difference in the experimental or Control groups voluntary control of finger skin

temperature was uncovered: F (1, 35) = 1.03 p >0.05. (Statistical Appendix 2.3, table 1.b.I.i, Page S9).

A.II.a.ii Post-treatment effects

The a priori analysis of covariance comparing the control of finger skin temperature of the pooled experimental groups and the controls at the post-treatment stage, revealed a significant effect of room temperature on control of post-treatment middle finger skin temperature: F (1, 25) = 28.09 p <0.001. However, no significant difference in the experimental or Control group's voluntary control of finger skin temperature was uncovered at this time: F (1, 25) = 0.174 p >0.05. (Statistical Appendix 2.3, table 1.b.I.ii, Page S9)

A.II.a.iii Follow up effects

The a priori analysis of covariance comparing the control of finger skin temperature of the pooled experimental groups and the controls at follow up, revealed no significant difference in the experimental or Control groups voluntary control of finger skin temperature: F (1, 23) = 0.221 p >0.05. (Statistical Appendix 2.3, table 1.b.I.iii, Page S10).

The a priori tests thus show no significant difference between Control and Treated patient's voluntary control of finger skin temperature.

A.II.b. Biofeedback (ATT, ATE, ART, ARE) versus no biofeedback (AT, AR)

Table 8.28 displays the mean starting temperatures and changes in temperature during the voluntary control assessments for the biofeedback and no biofeedback groups.

	Middle finger temperature difference (°C)	Room temperature difference (°C)	Middle finger starting temperature (°C)	Room starting temperature (°C)
Biofeedback	0.56	0.08	27.95	22.31
No biofeedback	0.77	0.08	30.19	22.09

Table 8.28 The mean finger and room (starting) temperatures (°C) of the Biofeedback and non-biofeedback groups pooled across the pre-treatment, post-treatment, and follow-up voluntary control assessment sessions

A.II.b.i Pre-treatment effects

An a priori analysis of covariance comparing the control of finger skin temperature of the biofeedback (ATT, ATE, ART, ARE) and the no biofeedback (AT, AR) experimental groups revealed a significant effect of pre-treatment room starting temperature on control of finger skin temperature: $F(1, 25) = 0.047$ $P < 0.05$. However, there were no differences in the pre-treatment control of finger skin temperature between the Biofeedback and No Biofeedback experimental groups: $F(1, 25) = 0.339$ $p>0.05$ (Statistical Appendix 2.3 table 1.b.II.i, page S10).

A.II.b.ii Post-treatment effects

The a priori analysis of covariance comparing the control of finger skin temperature of the biofeedback (ATT, ATE, ART, ARE) and the no biofeedback (AT, AR) experimental groups revealed a significant effect of post-treatment room temperature on control of finger skin temperature: $F(1, 17) = 29.73$ $P < 0.001$. However, there were no significant differences in the post-treatment control of finger skin temperature between the Biofeedback and No Biofeedback groups: $F(1, 17) = 0.173$ $p>0.05$. (Statistical Appendix 2.3 table 1.b.II.ii, page S11).

A.II.b.iii Follow up effects

The a priori analysis of covariance comparing the control of finger skin temperature of the biofeedback (ATT, ATE, ART, ARE) and the no biofeedback (AT, AR) experimental groups revealed no significant differences in the control of finger skin temperature between the Biofeedback

and No Biofeedback groups during the follow up assessment session: F (1, 16) = 0.281 p>0.05 (Statistical Appendix 2.3 table 1.b.II.iii, page S11).

The a priori tests thus show no significant difference between Biofeedback and No Biofeedback Treated patient's voluntary control of finger skin temperature.

A.II.c No treatment versus each of the individual treatment groups (AT, ATT, ATE, AR, ART, ARE)

Tables 8.29-8.34 describe the mean room and middle finger starting temperatures and temperature differences of the control and each of the individual treatment groups during the voluntary control assessments of the study. Please note that the reported middle finger "difference" temperatures are adjusted to allow for the effect of room and middle finger starting temperatures.

At each of the three time periods, a set of comparisons was carried out between the Controls and each of the treatment conditions.

A.II.c.i. No treatment versus AT

	Middle finger temperature difference (°C)	**Room temperature difference (°C)**	**Middle finger starting temperature (°C)**	**Room starting temperature (°C)**
No treatment	-0.02	0.12	27.24	22.35
AT	-0.33	0.09	30.65	21.76

Table 8.29: The mean finger and room (starting) temperatures (°C) of the untreated and the AT group across the pre-treatment, post-treatment, and follow-up voluntary control assessment sessions

A.II.c.i.a Pre-treatment effects

There were no significant differences in pre-treatment control of finger skin temperature between the AT and control groups: Difference = -0.074 P>0.05, 95% CI (-1.750, 1.601) (Statistical appendix 2.3; table 1.b.III.i, page S12).

A.II.c.i.b Post-treatment effects

There were no significant differences in post-treatment control of finger skin temperature between the AT and control groups: Difference = -0.463 P>0.05, 95% CI (-2.984, 2.058). (Statistical appendix 2.3; table 1.b.III.ii, page S12).

A.II.c.i.c Follow up effects

There were no significant differences in Follow up control of finger skin temperature between the AT and control groups: Difference = 0.374 P>0.05, 95% CI (-2.761, 3.508). (Statistical appendix 2.3; table 1.b.III.iii, page S12).

A.II.c.ii. No treatment versus ATT

	Middle finger temperature difference (°C)	**Room temperature difference (°C)**	**Middle finger starting temperature (°C)**	**Room starting temperature (°C)**
No treatment	0.06	0.12	27.24	22.35
ATT	-0.32	0.13	30.79	23.08

Table 8.30 The mean finger and room (starting) temperatures (°C) of the untreated and the ATT participants across the pre-treatment, post-treatment and follow-up voluntary control assessment sessions

A.II.c.ii.a Pre-treatment effects

There were no significant differences in pre-treatment control of finger skin temperature between the ATT and control groups: Difference = -0.318 P>0.05, 95% CI (-1.983, 1.347). (Statistical appendix 2.3; table 1.b.III.i, page S12).

A.II.c.ii.b Post-treatment effects

There were no significant differences in post-treatment control of finger skin temperature between the ATT and control groups: Difference = -0.759 P>0.05, 95% CI (-3.060, 1.543). (Statistical appendix 2.3; table 1.b.III.ii, page S12).

A.II.c.ii.c Follow up effects

There were no significant differences in Follow up control of finger skin temperature between the ATT and control groups: Difference = 1.346 P>0.05, 95% CI (-2.376, 5.068). (Statistical appendix 2.3; table 1.b.III.iii, page S12).

A.II.c.iii.No treatment versus ATE

	Middle finger temperature difference (°C)	**Room temperature difference (°C)**	**Middle finger starting temperature (°C)**	**Room starting temperature (°C)**
No treatment	0.12	0.12	27.2	22.35
ATE	1.44	0.06	26.3	21.73

Table 8.31 The mean finger and room (starting) temperatures (°C) of the untreated and the ATE participants across the pre-treatment, post-treatment and follow-up voluntary control assessment sessions

A.II.c.iii.a Pre-treatment effects

There were no significant differences in pre-treatment control of finger skin temperature between the ATE and control groups: Difference = 0.021 P>0.05, 95% CI (-1.595, 1.637). (Statistical appendix 2.3; table 1.b.III.i, page S12).

A.II.c.iii.b Post-treatment effects

There were no significant differences in post-treatment control of finger skin temperature between the ATE and control groups: Difference = 0.967 P>0.05, 95% CI (-0.952, 2.885). (Statistical appendix 2.3; table 1.b.III.ii, page S12).

A.II.c.iii.c Follow up effects

Significant differences in Follow up control of finger skin temperature between the ATE and control groups were revealed: Difference = 2.924 P<0.05, 95% CI (0.340, 5.502). (Statistical appendix 2.3; table 1.b.III.iii, page S12). This suggests that at follow up, the ATE group had more control over

finger skin temperature than did the Control group. Whether this difference constitutes a treatment effect will be investigated further in the Post Hoc analyses described on pages 167-168.

A.II.c.iv. No treatment versus AR

	Middle finger temperature difference (°C)	**Room temperature difference (°C)**	**Middle finger starting temperature (°C)**	**Room starting temperature (°C)**
No treatment	-0.02	0.12	27.24	22.35
AR	1.29	0.08	29.79	22.38

Table 8.32 The mean finger and room (starting) temperatures (°C) of the untreated and the AR participants across the pre-treatment, post-treatment and follow-up voluntary control sessions

A.II.c.iv.a Pre-treatment effects

Significant differences occurred in pre-treatment control of finger skin temperature between the AR and control groups: Difference = 1.933 $P<0.05$, 95% CI (0.169, 3.697). (Statistical appendix 2.3; table 1.b.III.i, page S12). Indeed, as is clearly shown in table 8.32, before the treatment programme began, the AR group were better able to control their finger skin temperature than were the Controls. This difference will be investigated further in the Post Hoc analyses on pages 167-168.

A.II.c.iv.b Post-treatment effects

There were no significant differences in post-treatment control of finger skin temperature between the AR and control groups: Difference = 1.089 $P>0.05$, 95% CI (-0.877, 3.056). (Statistical appendix 2.3; table 1.b.III.ii, page S12).

A.II.c.iv.c Follow up effects

There were no significant differences in Follow up control of finger skin temperature between the AR and control groups: Difference = 0.830 $P>0.05$, 95% CI (-1.906, 3.567). (Statistical appendix 2.3; table 1.b.III.iii, page S12).

A.II.c.v. No treatment versus ART

	Middle finger temperature difference (°C)	Room temperature difference (°C)	Middle finger starting temperature (°C)	Room starting temperature (°C)
No treatment	0.01	0.12	27.24	22.34
ART	0.44	0.09	24.59	22.54

Table 8.33 The mean finger and room (starting) temperatures (°C) of the untreated and the ART participants across the pre-treatment, post-treatment and follow-up voluntary control assessment sessions

A.II.c.v.a Pre-treatment effects

There were no significant differences in pre-treatment control of finger skin temperature between the ART and control groups: Difference = 1.373 P>0.05, 95% CI (-0.339, 3.084). (Statistical appendix 2.3; table 1.b.III.i, page S12).

A.II.c.v.b Post-treatment effects

There were no significant differences in post-treatment control of finger skin temperature between the ART and control groups: Difference = -0.416 P>0.05, 95% CI (-2.738, 1.907). (Statistical appendix 2.3; table 1.b.III.ii, page S12).

A.II.c.v.c Follow up effects

There were no significant differences in Follow up control of finger skin temperature between the ART and control groups: Difference = -0.182 P>0.05, 95% CI (-2.780, 2.417). (Statistical appendix 2.3; table 1.b.III.iii, page S12).

A.II.c.vi. No treatment versus ARE

Table 8.34 describes the temperature changes of the individual ARE and Control group.

	Middle finger temperature difference (°C)	Room temperature difference (°C)	Middle finger starting temperature (°C)	Room starting temperature (°C)
No treatment	-0.08	0.12	27.24	22.35
ARE	0.82	0.05	31.05	22.17

Table 8.34 The mean finger and room (starting) temperatures (°C) of the untreated and the ARE participants across the pre-treatment, post-treatment and follow-up voluntary control assessment sessions

A.II.c.vi.a Pre-treatment effects

There were no significant differences in pre-treatment control of finger skin temperature between the ARE and control groups: Difference = 1.360 P>0.05, 95% CI (-0.449, 3.170). (Statistical appendix 2.3; table 1.b.III.i, page S12).

A.II.c.vi.b Post-treatment effects

There were no significant differences in post-treatment control of finger skin temperature between the ARE and control groups: Difference = 0.510 P>0.05, 95% CI (-1.456, 2.476). (Statistical appendix 2.3; table 1.b.III.ii, page S12).

A.II.c.vi.c Follow up effects

There were no significant differences in Follow up control of finger skin temperature between the ARE and control groups: Difference = 1.032 P>0.05, 95% CI (-2.191, 4.255). (Statistical appendix 2.3; table 1.b.III.iii, page S12).

Thus it appears that whilst the AR treatment group had better control over finger skin temperature at the pre-treatment assessment session, Autogenic Training with EMG Biofeedback had an apparent effect on participants' ability to increase their finger skin temperature at the follow up assessment session test of voluntary control.

A.III.The Post Hoc Scheffé tests

As the reader will recall, the main analysis revealed a significant interaction between relaxation and time. Moreover, the a priori tests revealed that whilst the AR group appeared to have more control over finger skin temperature at the pre-treatment assessment sessions, the ATE group appeared to have more control over finger skin temperature at follow up when compared with the Control group at this time. To investigate further, Post Hoc Scheffé pair wise comparisons of finger temperature control were carried out to evaluate the source of the significance in these interactions.

Table 8.35 describes the mean finger skin temperature differences (after the effects of the covariates have been removed) of the Relaxation groups during the three periods of assessment.

Treatment Group	Pre-treatment	Post-Treatment	Follow up
Autogenic Training	-0.414	-0.073	0.453
Applied Relaxation	0.588	0.164	-0.165
No Relaxation (Controls)	-0.262	-0.126	-0.388

Table 8.35: Mean finger skin temperature differences (after the effects of the covariates have been removed) of the Relaxation groups during the three tests of voluntary control

Scheffe pairwise comparisons initially address the relationship between the means that differ the most from each other in magnitude. Comparisons that differ by the next largest magnitude are then investigated. For the data displayed in table 8.35, three such comparisons are worthy of note.

The first pairwise comparison of means was carried out between pre-treatment Autogenic Training and pre-treatment Applied Relaxation. This yielded a significant difference in ability to control finger skin temperature at the pre-treatment assessment session: Mean Difference = -1.002 $p < 0.05$, 95% CI (-1.806, -0.199). Please refer to Statistical appendix 2.3, Table 1.c.I, page S13. Clearly, the Applied Relaxation group were better able to increase pre-treatment finger skin temperature than were the Autogenic Training group during the pre-treatment test of voluntary control.

The second pairwise comparison of control of finger skin temperature compared the Autogenic Training group's control of finger skin temperature at pre-treatment and at follow up. A within subjects contrast showed that the difference in the means approached significance: $F(1, 9) = 4.79$ $p = 0.056$. Thus, a strong trend suggests that the Autogenic Training groups' finger skin temperature was under more control at follow up than at the pre-treatment assessment session. Please refer to Statistical Appendix 2.3, Table 1.c.II, page S13.

The third pairwise comparison of means was between the Applied Relaxation group at the pre-treatment assessment session, and the Control group at follow up. As both the level of relaxation and the level of time differed for the two means, an independent means t-test was used. This revealed a significant difference: $t(17.5) = 2.84$ $p = 0.011$ (Statistical Appendix 2.3, Table 1.c.III, page S13), thereby suggesting that the Applied Relaxation group had more control over finger skin temperature at pre-treatment than the Control group had at follow up.

In summary therefore, the Applied Relaxation group had good control over finger skin temperature before the treatments were started, and a non-significant trend in the data suggests that the Autogenic Training group's control of finger skin temperature benefited from treatment.

Turning now to consider the techniques implemented in the attempts to increase finger skin temperature during the post-treatment and follow up voluntary control sessions. Did the experimental participants claim to be utilising a relaxation strategy in an attempt to control their finger skin temperature, and if so, were they actually relaxing flexi Carpi Ulnaris muscle during the two post-treatment periods of voluntary control ?

Subjective report reveals that during the post-treatment assessment session, 95.7% (22) of the experimental participants claimed to have been using a relaxation technique to control their finger

temperature; whereas, only 25% (2) of the control participants claimed to do so[33]. Similarly, at follow-up, 100% of the Experimental participants reported using relaxation techniques; whereas, 87.5% (7) of the Controls described some thermal imagery technique.

Objective EMG data were available for 29 participants (21 E, 8 C) at post-treatment, and 22 (14 E, 8 C) at follow-up. As with the finger temperature data of the test of voluntary control, a "difference" score was calculated by subtracting EMG levels in the last minute of the baseline period from that of the last minute of the voluntary control period for each participant. Using this "difference" score as the dependent variable, two analyses of covariance were performed on the post-treatment and the follow-up data. In both cases, the EMG levels and room temperature at the start of the test of voluntary control, and the difference in room temperature between the baseline and voluntary control periods were entered into the analyses as covariates. Comparisons were made across the biofeedback groups (No biofeedback, Temperature biofeedback, EMG biofeedback), and across the relaxation groups (No Relaxation, Autogenic Training, Applied Relaxation).

The mean EMG levels for the biofeedback groups and for the relaxation groups during the post-treatment assessment session are displayed in tables 8.36 and 8.37 respectively. Please recall that a negative difference score is indicative of a reduction in muscle tension.

	EMG difference score (mv)	**Room temperature (°C)**	**Room starting temperature (°C)**	**EMG starting tension (mv)**
No Biofeedback	**7.82**	**0.14**	**21.90**	**45.60**
Temperature biofeedback	**-23.22**	**0.13**	**22.31**	**54.86**
EMG biofeedback	**-9.19**	**0.03**	**21.92**	**38.29**

Table 8.36: The mean difference in EMG levels of the flexi carpi ulnaris muscle tension during the post-treatment test of voluntary control (Biofeedback groups) .

[33] 50% (4) of the Controls and 4.3% (1) of the experimental participants reported using thermal imagery, and 25% (2) of the Controls reported "willing blood to the fingers" in their attempt to control finger skin temperature.

	EMG difference score (mv)	Room temperature (°C)	Room starting temperature (°C)	EMG starting tension (mv)
No Relaxation	**1.48**	**0.16**	**22.10**	**49.50**
Autogenic Training	**-25.70**	**0.07**	**21.92**	**40.67**
Applied Relaxation	**7.95**	**0.11**	**22.02**	**47.83**

Table 8.37: The mean difference in EMG levels of the flexi carpi ulnaris muscle tension during the post-treatment test of voluntary control (Relaxation groups).

Comparison of post-treatment EMG data across both the biofeedback and relaxation groups uncovered no group differences in levels of relaxation during the test of voluntary control: Biofeedback groups ($F(2, 23) = 1.14$ $p>0.05$); Relaxation groups ($F(2, 23) = 1.44$ $p>0.05$). (Please refer to statistical appendix 2.3; tables 1.d.I and 1.d.II, page S14). Thus, although trends in the data suggest that the temperature biofeedback, EMG biofeedback, and Autogenic Training groups reduced the tension of their flexi carpi ulnaris muscles during the post-treatment test of voluntary control, observed levels of relaxation did not differ significantly across the groups.

Tables 8.38 and 8.39 describe the mean EMG levels for the biofeedback and relaxation groups during the follow-up assessment session.

	EMG difference score (mv)	Room temperature (°C)	Room starting temperature (°C)	EMG starting tension (mv)
No Biofeedback	**17.23**	**0.16**	**21.74**	**44.31**
Temperature biofeedback	**-31.46**	**0.25**	**22.73**	**60.50**
EMG biofeedback	**-35.55**	**0.17**	**20.66**	**66.00**

Table 8.38: The mean difference in EMG levels of the flexi carpi ulnaris muscle tension during the follow-up test of voluntary control (Biofeedback groups).

Comparison of follow-up EMG data across both the biofeedback and relaxation groups revealed no group differences in levels of relaxation during the test of voluntary control: Biofeedback groups (F

(2, 16) = 2.00 p>0.05); Relaxation groups (F (2, 16) = 1.83 p>0.05). (Statistical appendix 2.3, tables 1.d.III and 1.d.IV, page S14).

	EMG difference score (mv)	Room temperature (°C)	Room starting temperature (°C)	EMG starting tension (mv)
No Relaxation	**10.01**	**0.17**	**21.65**	**54.00**
Autogenic Training	**-48.91**	**0.25**	**21.72**	**35.00**
Applied Relaxation	**12.39**	**0.17**	**21.69**	**57.60**

Table 8.39: The mean difference in EMG levels of the flexi carpi ulnaris muscle tension during the follow-up test of voluntary control (Relaxation groups).

Thus, although trends in the data suggest that the temperature biofeedback, EMG biofeedback, and Autogenic Training groups reduced the tension of their flexi carpi ulnaris muscles during the follow-up test of voluntary control, observed levels of relaxation were low and did not differ significantly across the groups.

The objective EMG data shows that the trained participants did not reduce muscular tension any more than the untrained participants. However, one can not assume that these results are a reflection of an inadequate treatment per se; it may be the case that the participants were not adequately trained, or, if adequately trained, did not implement the taught relaxation strategies during the test of voluntary control.

B. Analysis of the Cold Challenge data

To recapitulate, the Cold Challenge test required participants to place their hands into a cold water bath (maintained at 15°C) for a period of 5 minutes. The variable of interest was the time taken to recover by 6°C; however, to allow for variation in room temperature and hand temperature prior to immersion, room temperature during the session and starting middle finger temperature were measured and entered into the analyses as covariates.

B.I.The main analysis: The Cold Challenge Test.

Cold Challenge data were missing for 2 participants; therefore 96 sets of data were entered into the main analysis i.e. 39 participants at the pre-treatment assessment (29E, 10C), 29 at the post-treatment session (21E, 8C), and 28 at the follow-up assessment session (21E, 7C). The uninterpreted output of the incomplete blocks design split plot analysis of covariance may be found in Statistical appendix 2.3, tables 2.a.I and 2.a.II, pages S15-S16. The covariate, main and interactive effects are interpreted in the text below.

B.I.a. Covariates: *evaluation of the role of the covariates on recovery time following Cold Challenge*

The main analysis revealed a significant relationship between recovery from Cold Challenge and both room temperature and finger starting temperature: $F(1, 32) = 17.53$ $p<0.001$; $F(1, 32) = 21.31$ $p<0.001$ respectively. Please refer to statistical appendix 2.3; table 2.a.I, page S15).

The nature of the relationships are outlined in table 8.40, and interpreted more fully in the text below.

Covariate	**Effect of a single unit increase (1°C) in covariate temperature on time taken for middle finger temperature to recover by 6°C**
Room temperature	Reduction by 6.04 minutes
Middle finger starting temperature	Reduction by 1.27 minutes

Table 8.40: The effect of a 1°C rise in the temperature of each covariate on middle finger skin temperature recovery time to 6°C

As is evident from table 8.39, a higher room temperature is associated with a reduction in the time required for finger skin temperature to recover by 6°C following cold challenge: $t = -3.6$ $p<0.05$. Similarly, a higher starting middle finger skin temperature is associated with a reduced recovery time of middle finger temperature to 6°C: $t = -2.9$ $p<0.05$. Please refer to statistical appendix 2.3 (table 2.a.III, page S16). In summary, therefore, the warmer the participant's fingers at the start, and the warmer the room temperature during the cold challenge test, the faster the rate of recovery.

B.I.b. Time: *The comparison of pre-treatment, post-treatment and follow-up data*

There was no effect of time of testing (pre-treatment, post-treatment, and follow-up) on the time taken for fingers to recover by 6°C: $F(2, 41) = 2.14$ $p > 0.05$ (Please refer to statistical appendix 2.3; table 2.a.I, page S15). Indeed, as outlined in table 8.41, when the room temperature and middle finger starting temperature are taken into account, the time to recover by 6°C is relatively consistent across the three assessment sessions of the study.

	Time to recover by 6°C (mins)	**Room temperature (°C)**	**Middle finger starting temperature (°C)**
Pre-treatment	28.40	23.91	26.63
Post-treatment	26.71	22.07	26.51
Follow-up	26.84	22.05	27.38

Table 8.41 The time taken to rewarm by 6°C and the room and middle finger starting temperatures (°C) over the three cold challenge test assessment sessions .

B.I.c. Biofeedback group: *Comparison of the 3 biofeedback groups across the assessment sessions.*

The time taken for the members of the three biofeedback groups to rewarm by 6°C during the three assessment sessions as a whole are listed in table 8.42.

	Time to rewarm by 6°C (mins)	**Room temperature (°C)**	**Middle finger starting temperature (°C)**
No Biofeedback	27.89	22.88	27.39
Temperature biofeedback	29.36	22.80	25.82
EMG biofeedback	29.92	22.68	26.60

Table 8.42 Rewarming times (by 6 °C) for the three biofeedback groups across the pre-treatment, post-treatment and follow-up assessment sessions following immersion in the cold water bath.

The three biofeedback groups did not differ significantly in finger skin temperature rewarming ability: $F(2, 32) = 0.21$ $p > 0.05$; $F(2, 32) = 1.11$ $p > 0.05$. (Statistical appendix 2.3, tables 2.a.I and 2.a.II, pages S15 - S16).

B.I.d.Relaxation group: *comparison of the 3 relaxation groups over the 3 assessment sessions*

Rewarming times to 6°C during the three assessment sessions as a whole for the relaxation groups are listed in table 8.43.

	Time to rewarm by 6°C (mins)	Room temperature (°C)	Middle finger starting temperature (°C)
Autogenic Training	30.94	22.64	26.93
Applied Relaxation	25.94	22.79	26.96
No Relaxation	32.02	23.06	26.43

Table 8.43 Rewarming times (by 6 °C) for the three Relaxation groups across the pre-treatment, post-treatment and follow-up assessment sessions following immersion in the cold water bath.

The main analyses revealed no significant effect of relaxation group on cold challenge recovery when the effects of room temperature and middle finger starting temperature were removed: $F(2, 32) = 2.25$ $p > 0.05$ $F(2, 32) = 1.35$ $p>0.05$. (Statistical appendix 2.3, tables 2.a.I and 2.a.II, pages S15 - S16).

B.I.e. The interaction between time of testing and biofeedback group

The main analysis determining the effects of Relaxation, reveals no statistically significant interaction between biofeedback group and time of testing: $F(4, 41) = 0.63$ $p>0.05$. (Statistical appendix 2.3, table 2.a.I, page S15). The main analysis looking at the effects of Biofeedback further supports these findings: $F(4, 41) = 0.80$ $p>0.05$. (Statistical appendix 2.3, table 2.a.II, page S16). In short, the biofeedback groups' rewarming times following cold challenge were not significantly different at each of the three assessment sessions (pre-treatment, post-treatment and follow-up); thereby suggesting no effect of treatment.

B.I.f. The interaction between time of testing and relaxation group

As with the comparison of the biofeedback groups above, the main analyses revealed no statistically significant interaction between relaxation group and time of testing: $F\ (4, 41) = 0.61$ $p>0.05$; $F\ (4, 41) = 0.47$ $p>0.05$. (Statistical appendix 2.3, tables 2.a.I and 2.a.II, pages S15 - S16).

B.I.g. The interaction between relaxation and biofeedback groups

As previously described, biofeedback was included in the study design so that it might be ascertained whether the effects of relaxation techniques on control of finger skin temperature are augmented or hindered by biofeedback. The main analysis of covariance revealed no additional effect of biofeedback with that of relaxation on rewarming times: $F\ (2, 32) = 2.13$ $p> 0.05$ (Statistical appendix 2.3, table 2.a.I, page S15). Thus, one can assume that, at least where biofeedback is used in conjunction with relaxation techniques during training sessions, it neither increases nor hinders the patient's control of finger skin temperature through relaxation. The question as to whether biofeedback might have had a greater effect if participants been given the opportunity to combine the technique with home practice of the relaxation exercises, must remain unanswered.

B.I.h. The interaction of time, relaxation and biofeedback

In line with the results above, the main analysis revealed no statistically significant interaction between time of testing, relaxation or biofeedback group: $F\ (4, 41) = 1.03$ $p>0.05$. (Statistical appendix 2.3, table 2.a.I, page S15). In short, relaxation training (with or without biofeedback) has not been shown to have an effect on cold challenge recovery times in Raynaud's patients.

B.II. The a priori comparisons:

B.II.a. No treatment versus all treatments combined

Table 8.44 describes the middle finger starting temperature, room temperature and the time taken to rewarm by 6°C for the treated and untreated participants across the three assessment sessions as a whole.

	Time to rewarm by 6°C (mins)	**Middle finger starting temperature (°C)**	**Room temperature (°C)**
Treated participants	27.58	26.94	22.72
Control participants	30.88	26.43	23.07

Table 8.44 The mean rewarming times (by 6°C), and the room and middle finger starting temperatures of the treated and untreated (control) participants across the pre-treatment, post-treatment and follow-up cold challenge tests.

B.II.a.i Pre-treatment effects

An a priori analysis of variance comparing cold challenge rewarming of the pooled experimental groups and the controls, revealed a significant effect of room starting temperature and finger skin starting temperature on control of pre-treatment middle finger skin temperature: $F(1, 35) = 5.55$ $p < 0.05$, and $F(1, 35) = 13.74$ $p < 0.05$ respectively. However, no significant difference in the experimental or Control groups rewarming time was uncovered: $F(1, 35) = 1.49$ $p > 0.05$. (Statistical Appendix 2.3, table 2.b.I.i, Page S17).

B.II.a.ii Post-treatment effects

The a priori analysis of variance comparing the cold challenge recovery of the pooled experimental groups and the controls at the post-treatment stage, revealed a significant effect of room starting temperature on rewarming time: $F(1, 25) = 6.04$ $p < 0.005$. However, no significant difference between the Experimental and Control groups' rewarming times was uncovered at this time: $F(1, 25) = 0.300$ $p > 0.05$. (Statistical Appendix 2.3, table 2.b.I.ii, Page S17).

B.II.a.iii Follow up effects

The a priori analysis of variance comparing cold challenge recovery of the pooled experimental groups and the controls at follow up, revealed a significant effect of room starting temperature on cold challenge rewarming times: $F(1, 24) = 5.86$ $p < 0.005$. However, there was no significant difference between the experimental or Control groups' cold challenge rewarming times: $F(1, 24) = 0.320$ $p > 0.05$. (Statistical Appendix 2.3, table 2.b.I.iii, Page S18).

The a priori tests thus show no significant difference between Control and Treated patients rewarming times following the Cold Challenge Test.

B.II.b.Biofeedback (ATT, ATE, ART) versus no biofeedback (AT, AR)

Table 8.45 displays the biofeedback (ATT, ATE, ART, ARE) and no biofeedback (AT, AR) groups' mean recovery times, middle finger starting temperatures and mean room temperatures during the cold challenge sessions.

	Time to rewarm by 6°C (mins)	**Middle finger starting temperature (°C)**	**Room temperature (°C)**
Biofeedback	29.02	26.25	22.74
No biofeedback	23.28	28.49	22.68

Table 8.45 The mean time taken to rewarm by 6°C, and the room and middle finger starting temperatures of the biofeedback and no biofeedback groups across the pr-treatment, post-treatment and follow-up cold challenge tests.

B.II.b.i Pre-treatment effects

An a priori analysis of covariance comparing the cold challenge rewarming times of the No Biofeedback and the Biofeedback experimental groups revealed a significant effect of pre-treatment finger starting temperature: $F(1, 25) = 7.378$ $p < 0.05$, but no effect of treatment group: $F(1, 25) = 2.152$ $P > 0.05$. Please refer to Statistical Appendix 2.3, table 2.b.II.i, Page S18.

B.II.b.ii Post-treatment effects

An a priori analysis of covariance comparing the cold challenge rewarming times of the No Biofeedback and the Biofeedback experimental groups revealed a significant effect of room temperature: $F(1, 17) = 5.625$ $p<0.05$, but no effect of treatment group: $F(1, 17) = 1.855$ $P>0.05$. Please refer to Statistical Appendix 2.3, table 2.b.II.ii, Page S19.

B.II.b.iii Follow up effects

An a priori analysis of covariance comparing the cold challenge rewarming times of the No Biofeedback and the Biofeedback experimental groups revealed a significant effect of pre-treatment finger starting temperature: $F(1, 17) = 7.294$ $p<0.05$, and room temperature: $F(1, 17) = 7.593$ $p<0.05$, but no effect of treatment group: $F(1, 17) = 0.189$ $P>0.05$. Please refer to Statistical Appendix 2.3, table 2.b.II.iii, Page S19.

The a priori tests thus show no significant difference between Biofeedback and No Biofeedback Treated patient's recovery following Cold Challenge.

B.II.c.No treatment versus each of the individual treatment groups (AT, ATT, ATE, AR, ART, ARE)

Tables 8.46-8.51 describe the recovery times, the middle finger starting temperatures and the room temperatures of the control and individual treatment groups during the cold challenge assessment sessions of the study. A set of comparisons between the Control group and each of the individual treatment groups was carried out at each of the three time periods.

B.II.c.i. No treatment versus AT

	Time to rewarm by 6°C (mins)	**Middle finger starting temperature (°C)**	**Room temperature (°C)**
AT	30.72	27.97	22.80
Control	30.29	26.43	23.06

Table 8.46 The room and middle finger starting temperatures, and the time taken to rewarm by 6°C following Cold Challenge of the AT and control participants across the three assessment sessions.

B.II.c.i.a Pre-treatment effects

There were no significant differences in pre-treatment control of finger skin temperature between the AT and control groups: Difference = -252.9 P>0.05, 95% CI (-755.5,249.8) (Statistical appendix 2.3; table 2.b.III.i, page S20).

B.II.c.i.b Post-treatment effects

There were no significant differences in post-treatment control of finger skin temperature between the AT and control groups: Difference = 369.1 P>0.05, 95% CI (-435.2, 1173.4) (Statistical appendix 2.3; table 2.b.III.ii, page S20).

B.II.c.i.c Follow up effects

There were no significant differences in Follow up control of finger skin temperature between the AT and control groups: Difference = -87.3 P>0.05, 95% CI (-849.9, 675.4). (Statistical appendix 2.3; table 2.b.III.iii, page S20).

B.II.c.ii. No treatment versus ATT

	Time to rewarm by 6°C (mins)	**Middle finger starting temperature (°C)**	**Room temperature (°C)**
ATT	28.29	28.38	22.68
Control	29.46	26.43	23.07

Table 8.47 The mean recovery times (by 6°C), and the room and middle finger starting temperatures of the ATT and untreated participants across the pre-treatment, post-treatment and follow-up cold challenge tests.

B.II.c.ii.a Pre-treatment effects

There were no significant differences in pre-treatment control of finger skin temperature between the ATT and control groups: Difference = -86.9 P>0.05, 95% CI (-565.8, 392.1) (Statistical appendix 2.3; table 2.b.III.i, page S20).

B.II.c.ii.b Post-treatment effects

There were no significant differences in post-treatment control of finger skin temperature between the ATT and control groups: Difference = 88.7 P>0.05, 95% CI (-521.4, 698.9) (Statistical appendix 2.3; table 2.b.III.ii, page S20).

B.II.c.ii.c Follow up effects

There were no significant differences in Follow up control of finger skin temperature between the ATT and control groups: Difference = -509.1 P>0.05, 95% CI (-1428.8, 410.6) (Statistical appendix 2.3; table 2.b.III.iii, page S20).

B.II.c.iii. No treatment versus ATE

	Time to rewarm by 6°C (mins)	**Middle finger starting temperature (°C)**	**Room temperature (°C)**
ATE	30.46	25.33	22.51
Control	33.02	26.43	23.07

Table 8.48 The mean time taken to rewarm by 6°C, and the room and middle finger starting temperatures of the ATE and untreated (control) participants across the pr-treatment, post-treatment and follow-up cold challenge tests.

B.II.c.iii.a Pre-treatment effects

There were no significant differences in pre-treatment control of finger skin temperature between the ATE and control groups: Difference = -167.4 P>0.05, 95% CI (-634.4, 299.7) (Statistical appendix 2.3; table 2.b.III.i, page S20).

B.II.c.iii.b Post-treatment effects

There were no significant differences in post-treatment control of finger skin temperature between the ATE and control groups: Difference = -146.9 P>0.05, 95% CI (-669.0, 375.1) (Statistical appendix 2.3; table 2.b.III.ii, page S20).

B.II.c.iii.c Follow up effects

There were no significant differences in Follow up control of finger skin temperature between the ATE and control groups: Difference = -69.8 P>0.05, 95% CI (-759.4, 619.9) (Statistical appendix 2.3; table 2.b.III.iii, page S20).

B.II.c.iv. No treatment versus AR

	Time to rewarm by 6°C (mins)	**Middle finger starting temperature (°C)**	**Room temperature (°C)**
AR	16.04	28.84	22.59
Control	29.96	26.43	23.07

Table 8.49 The mean recovery times, and the room and middle finger starting temperatures of the AR and untreated (control) participants across the pre-treatment, post-treatment and follow-up cold challenge tests.

B.II.c.iv.a Pre-treatment effects

There were no significant differences in pre-treatment control of finger skin temperature between the AR and control groups: Difference = -345.6 P>0.05, 95% CI (-828.5, 137.2) (Statistical appendix 2.3; table 2.b.III.i, page S20).

B.II.c.iv.b Post-treatment effects

A significant difference in post-treatment control of finger skin temperature was uncovered between the AR and the Control group: Difference = -643.57 P<0.05, 95% CI (-1264.8, -22.3) (Statistical appendix 2.3; table 2.b.III.ii, page S20). This suggests that the AR group showed a significantly faster recovery following cold challenge than did the Control group.

B.II.c.iv.c Follow up effects

There were no significant differences in Follow up control of finger skin temperature between the AR and control groups: Difference = -521.5 P>0.05, 95% CI (-1201.3, 158.3) (Statistical appendix 2.3; table 2.b.III.iii, page S20).

B.II.c.v. No treatment versus ART

	Time to rewarm by 6°C (mins)	Middle finger starting temperature (°C)	Room temperature (°C)
ART	32.37	23.69	22.90
Control	32.52	26.43	23.07

Table 8.50 The mean time taken to rewarm by 6°C, and the room and middle finger starting temperatures of the ART and untreated (control) participants across the pr-treatment, post-treatment and follow-up cold challenge tests.

B.II.c.v.a Pre-treatment effects

There were no significant differences in pre-treatment control of finger skin temperature between the ART and control groups: Difference = -167.8 P>0.05, 95% CI (-636.9, 301.3) (Statistical appendix 2.3; table 2.b.III.i, page S20).

B.II.c.v.b Post-treatment effects

There were no significant differences in post-treatment control of finger skin temperature between the ART and control groups: Difference = -10.8 P>0.05, 95% CI (-668.5, 646.9) (Statistical appendix 2.3; table 2.b.III.ii, page S20).

B.II.c.v.c Follow up effects

There were no significant differences in Follow up control of finger skin temperature between the ART and control groups: Difference = 217.4 P>0.05, 95% CI (-452.4, 887.1) (Statistical appendix 2.3; table 2.b.III.iii, page S20).

B.II.c.vi. No treatment versus ARE

Table 8.50 describes the recovery times, and room and finger starting temperatures of the individual ARE and Control group.

	Time to rewarm by 6°C (mins)	Middle finger starting temperature (°C)	Room temperature (°C)
ARE	27.92	28.20	22.90
Control	29.97	26.43	23.07

Table 8.51 The mean time taken to rewarm by 6°C, and the room and middle finger starting temperatures of the ARE and untreated (control) participants across the pr-treatment, post-treatment and follow-up cold challenge tests.

B.II.c.vi.a Pre-treatment effects

There were no significant differences in pre-treatment control of finger skin temperature between the ARE and control groups: Difference = -66.5 P>0.05, 95% CI (-538.6, 405.5) (Statistical appendix 2.3; table 2.b.III.i, page S20).

B.II.c.vi.b Post-treatment effects

There were no significant differences in post-treatment control of finger skin temperature between the ARE and control groups: Difference = -56.05 P>0.05, 95% CI (-634.9, 522.8) (Statistical appendix 2.3; table 2.b.III.ii, page S20).

B.II.c.vi.c Follow up effects

There were no significant differences in Follow up control of finger skin temperature between the ARE and control groups: Difference = -417.5 P>0.05, 95% CI (-1219.2, 384.4) (Statistical appendix 2.3; table 2.b.III.iii, page S20).

Thus, on the whole, the individual treatments had no effect on temperature recovery times following Cold Challenge; however, the AR group were shown to recover from the post-treatment Cold Challenge more rapidly than were the Control group.

B.III. The Post Hoc tests

As no main or interactive effects of treatment were uncovered in the main analyses of the cold challenge data, no Post Hoc tests are required.

In summary, the analyses of the Cold Challenge data showed no significant main or interactive effects of treatment; however, the a priori analyses revealed that the individual AR treatment group recovered significantly more rapidly than the Control group from the pre-treatment Cold Challenge test.

C. **Analysis of the Ambulatory Monitoring data**

As the reader will recall, the Ambulatory Monitoring test involved the measurement of participants' finger skin temperature over a three hour period of pursuing normal daily activities. Mean middle finger skin temperature during the monitoring period was the main variable of interest for analysis purposes; however, ambient temperatures during the monitoring period were covaried in the analyses.

C.I.The main analysis: Ambulatory monitoring.

Data were missing for 9 participants; therefore 89 sets of data were entered into the main analysis i.e. 36 participants at the pre-treatment assessment (27E, 9C), 29 at the post-treatment session (23E; 6C), and 24 at the follow-up assessment session (19E, 5C). Statistical Appendices 2.3, tables 3.a.I and 3.a.II, pages S21-S22 contain the uninterpreted output of the incomplete blocks design split plot analysis of covariance. The covariate, main and interactive effects are discussed in the text below.

C.I.a. Covariates: *evaluation of the role of ambient temperature on mean finger temperature*

The main analysis revealed a significant relationship between ambient temperature and mean middle finger skin temperature during monitoring: $F(1, 30) = 15.95$ $p<0.001$. Please refer to statistical appendix 2.3, table 3.a.I, page S21. The nature of the relationship is outlined in table 8.52, and interpreted in the text below.

Covariate	Effect of a unit increase (1°C) in mean ambient temperature on mean middle finger skin temperature during monitoring
Mean ambient temperature	Increase by 0.54°C

Table 8.52: The effect of a 1°C rise in the ambient temperature on mean middle finger skin temperature during monitoring

Clearly, a higher ambient temperature during the monitoring period is associated with warmer fingers during monitoring: $t = 4.3$ $p<0.05$. Please refer to statistical appendix 2.3, table 3.a.III, page S22.

C.I.b. Time: *Comparison of pre-treatment, post-treatment and follow-up data*

The main analysis uncovered a significant effect of time of testing: $F(2, 37) = 6.65$ $p<0.01$ (Statistical appendix 2.3, table 3.a.I, page S21). Thus, as described in table 8.53, finger temperatures during ambulatory monitoring were warmer during the warmer months of post-treatment and follow up testing.

	Middle finger skin temperature (°C)	Mean Ambient temperature (°C)
Pre-treatment	23.88	20.44
Post-treatment	25.87	21.78
Follow-up	25.93	22.34

Table 8.53 Middle finger and ambient temperatures (°C) across the 3 monitoring periods of the study.

C.I.c. Biofeedback group: *Comparison of the biofeedback groups across the 3 assessment sessions*

The mean middle finger skin and ambient temperatures of the biofeedback groups during the three ambulatory monitoring sessions are listed in table 8.54.

	Middle finger skin temperature (°C)	Mean Ambient temperature (°C)
No Biofeedback	25.58	21.56
Temperature biofeedback	25.30	21.37
EMG Biofeedback	24.62	21.11

Table 8.54: Middle finger skin and ambient ambulatory monitoring temperatures (°C) of the biofeedback groups across the pre-treatment, post-treatment and follow-up assessment sessions

As is clear from the main analyses, the three biofeedback groups do not differ significantly in terms of finger skin temperature during the monitoring periods: F (2, 30) = 0.41 p>0.05; F (2, 30) = 1.84 p>0.05 (Statistical Appendices 2.3, tables 3.a.I and 3.a.II, pages S21-S22).

C.I.d. Relaxation groups: *Comparison of the 3 relaxation groups over the three assessment sessions.*

The mean relaxation group finger skin and ambient temperatures during the three monitoring sessions as a whole, are listed in table 8.55.

	Middle finger skin temperature (°C)	**Mean Ambient temperature (°C)**
Autogenic Training	25.51	21.63
Applied Relaxation	25.39	20.44
No Relaxation	23.89	22.76

Table 8.55 The mean relaxation group middle finger skin and ambient temperatures (°C) during ambulatory monitoring across the three assessment sessions.

Clearly, the three relaxation groups did not differ significantly in middle finger skin temperature during the monitoring periods: F (2, 30) = 2.44 p>0.05; F (2, 30) = 0.99 p>0.05. Please refer to statistical appendices 2.3, tables 3.a.I and 3.a.II, pages S21-S22.

C.I.e. The interaction between time of testing and biofeedback group

The main analyses revealed a significant interaction between time of testing and Biofeedback group: F (4, 37) = 5.07 p < 0.01; F (4, 37) = 5.02 p < 0.01 (Statistical Appendices 2.3, tables 3.a.I and 3.a.II, pages S21-S22). This interaction will be investigated further in the Post Hoc tests described in section C.III.b, pages 196-197.

C.I.f. The interaction between time and relaxation group

The main analysis revealed no statistically significant interaction between relaxation group and time of testing: F (4, 37) = 0.57 p>0.05; F (4, 37) = 0.62 p >0.05. Statistical Appendices 2.3, tables 3.a.I

and 3.a.II, pages S21-S22). Thus, the mean relaxation group middle finger skin temperatures during ambulatory monitoring did not differ across the three periods of assessment.

C.I.g. The interaction between relaxation and biofeedback groups

The main analysis of covariance of the ambulatory monitoring data showed an interactive effect of biofeedback and relaxation group: $F(2, 30) = 4.42$ $p < 0.025$ (Statistical Appendices 2.3, tables 3.a.I and 3.a.II, pages S21-S22). The nature of this interaction will be further investigated in the Post Hoc tests described in section C.III.a, pages 195-196.

C.I.h. The interaction between time of testing, relaxation and biofeedback group

The main analysis revealed no statistically significant interaction between time of testing, relaxation and biofeedback group: $F(4, 37) = 0.73$ $p > 0.05$. (Statistical Appendices 2.3, tables 3.a.I and 3.a.II, pages S21-S22). In short, this result indicates that the combinations of biofeedback and relaxation have no treatment effect on middle finger skin temperature measured during a period of ambulatory monitoring.

C.II.The a priori comparisons:

C.II.a. No treatment versus all treatments combined

Table 8.56 describes the mean ambient and middle finger temperatures of the treated and untreated participants during the three ambulatory monitoring sessions as a whole.

	Mean middle finger temperatures (^{O}C)	**Mean ambient temperatures (^{O}C)**
Treated participants	25.37	20.99
Control participants	23.94	22.76

Table 8.56 The mean ambient and middle finger temperatures of the treated and untreated participants across the pre-treatment, post-treatment and follow-up ambulatory monitoring sessions.

C.II.a.i Pre-treatment effects

An a priori analysis of variance comparing the mean middle finger skin temperature of the pooled experimental versus the control groups during pre-treatment ambulatory monitoring revealed a significant effect of ambient temperature on control of pre-treatment middle finger skin temperature: $F(1, 33) = 10.965$ $p < 0.05$. However, no significant difference between the experimental and Control groups' voluntary control of finger skin temperature was uncovered: $F(1, 33) = 7.88$ $p > 0.05$. Please refer to Statistical Appendix 2.3, table 3.b.I.i, page S23.

C.II.a.ii Post-treatment effects

The a priori analysis of variance comparing the control of finger skin temperature of the pooled experimental groups and the controls at the post-treatment stage, revealed no significant difference in the experimental or Control groups voluntary control of finger skin temperature: $F(1, 26) = 0.152$ $p > 0.05$. (Statistical Appendix 2.3, table 3.b.I.ii, page S23).

C.II.a.iii Follow up effects

The a priori analysis of variance comparing the control of finger skin temperature of the pooled experimental groups and the controls at follow up, revealed no significant difference in the experimental or Control groups voluntary control of finger skin temperature: $F(1, 21) = 0.720$ $p > 0.05$. (Statistical Appendix 2.3, table 3.b.I.iii, page S24).

The a priori tests thus show no significant difference between Control and Treated patients' finger skin temperature during ambulatory monitoring.

C.II.b.Biofeedback versus no biofeedback

Table 8.57 displays the biofeedback (ATT, ATE, ART, ARE) and no biofeedback (AT, AR) groups' mean ambient and middle finger skin temperatures during the ambulatory monitoring sessions.

	Mean middle finger temperatures (^{O}C)	**Mean ambient temperatures (^{O}C)**
Biofeedback	24.13	21.23
No biofeedback	26.14	20.52

Table 8.57 The mean ambient and middle finger temperatures of the biofeedback and no biofeedback participants across the pre-treatment, post-treatment and follow-up ambulatory monitoring sessions.

C.II.b.i Pre-treatment effects

An a priori analysis of covariance comparing the biofeedback and no biofeedback group's middle finger temperature during pre-treatment ambulatory monitoring revealed a significant effect of ambient temperature: $F(1, 24) = 4.547$ $p<0.05$. However, no significant effect of treatment group was uncovered: $F(1, 24) = 26.711$ $p> 0.05$. Please refer to Statistical Appendix 2.3, table 3.b.II.i, page S24.

C.II.b.ii Post-treatment effects

An a priori analysis of covariance comparing the biofeedback and no biofeedback group's middle finger temperature during post-treatment ambulatory monitoring revealed a significant effect of ambient temperature: $F(1, 20) = 6.093$ $p<0.05$, and a significant effect of treatment group on measured ambient finger skin temperature at this time: $F(1, 20) = 8.242$ $p< 0.05$. (Statistical Appendix 2.3, table 3.b.II.ii, page S25.

C.II.b.iii Follow up effects

An a priori analysis of covariance comparing the biofeedback and no biofeedback group's middle finger temperature during follow up ambulatory monitoring revealed no significant effect of treatment group: $F(1, 16) = 13.906$ $p> 0.05$. (Statistical Appendix 2.3, table 3.b.II.iii, page S25).

C.II.c. No treatment versus each of the individual treatment groups (AT, ATT, ATE, AR, ART, ARE)

Tables 8.58-8.63 describe the mean middle finger and ambient temperatures of the control and individual treatment groups during the ambulatory monitoring sessions of the study. At each of the three time periods, a set of pairwise comparisons was carried out to compare the no treatment with each of the individual treatment groups.

C.II.c.i.No treatment versus AT

	Mean middle finger temperatures (^{O}C)	**Mean ambient temperatures (^{O}C)**
AT	26.11	20.93
No treatment	23.56	22.75

Table 8.58 The mean middle finger and ambient temperatures (^{O}C) of the AT and untreated participants across the pre-treatment, post-treatment and follow-up ambulatory monitoring sessions.

C.II.c.i.a Pre-treatment effects

There were no significant differences in pre-treatment control of finger skin temperature between the AT and control groups: Difference = 2.501 P>0.05, 95% CI (-1.463, 6.466) (Statistical appendix 2.3; table 3.b.III.i, page S26).

C.II.c.i.b Post-treatment effects

There were no significant differences in post-treatment control of finger skin temperature between the AT and control groups: Difference = 3.435 P>0.05, 95% CI (-1.335, 8.205) (Statistical appendix 2.3; table 3.b.III.ii, page S26).

C.II.c.i.c Follow up effects

There were no significant differences in Follow up control of finger skin temperature between the AT and control groups: Difference = 0.038 P>0.05, 95% CI (6.202, 6.278) (Statistical appendix 2.3; table 3.b.III.iii, page S26).

C.II.c.ii. No treatment versus ATT

	Mean middle finger temperatures (^{O}C)	Mean ambient temperatures (^{O}C)
ATT	27.86	22.31
No treatment	24.28	22.75

Table 8.59 The mean middle finger and ambient temperatures (^{O}C) of the ATT and untreated participants across the pre-treatment, post-treatment and follow-up ambulatory monitoring sessions.

C.II.c.ii.a Pre-treatment effects

There were no significant differences in pre-treatment control of finger skin temperature between the ATT and control groups: Difference = 2.287 P>0.05, 95% CI (-2.068, 6.642) (Statistical appendix 2.3; table 3.b.III.i, page S26).

C.II.c.ii.b Post-treatment effects

There were no significant differences in post-treatment control of finger skin temperature between the ATT and control groups: Difference = 2.949 P>0.05, 95% CI (-1.79, 7.689). (Statistical appendix 2.3; table 3.b.III.ii, page S26).

C.II.c.ii.c Follow up effects

There were no significant differences in Follow up control of finger skin temperature between the ATT and control groups: Difference = 5.471 P>0.05, 95% CI (-1.381, 12.32). (Statistical appendix 2.3; table 3.b.III.iii, page S26).

C.II.c.iii. No treatment versus ATE

	Mean middle finger temperatures (^{O}C)	Mean ambient temperatures (^{O}C)
ATE	23.53	21.74
No treatment	24.04	22.75

Table 8.60 The mean middle finger and ambient temperatures (^{O}C) of the ATE and untreated participants across the pre-treatment, post-treatment and follow-up ambulatory monitoring sessions.

C.II.c.iii.a Pre-treatment effects

There were no significant differences in pre-treatment control of finger skin temperature between the ATE and control groups: Difference = -2.208 P>0.05, 95% CI (-5.857, 1.441) (Statistical appendix 2.3; table 3.b.III.i, page S26).

C.II.c.iii.b Post-treatment effects

There were no significant differences in post-treatment control of finger skin temperature between the ATE and control groups: Difference = -2.136 P>0.05, 95% CI (-6.26, 1.987) (Statistical appendix 2.3; table 3.b.III.ii, page S26).

C.II.c.iii.c Follow up effects

There were no significant differences in Follow up control of finger skin temperature between the ATE and control groups: Difference = 1.99 P>0.05, 95% CI (-3.516, 7.495) (Statistical appendix 2.3; table 3.b.III.iii, page S26).

C.II.c.iv.No treatment versus AR

	Mean middle finger temperatures (^{O}C)	**Mean ambient temperatures (^{O}C)**
AR	26.77	20.21
No treatment	23.92	22.75

Table 8.61 The mean middle finger and ambient temperatures (^{O}C) of the AR and untreated participants across the pre-treatment, post-treatment and follow-up ambulatory monitoring sessions.

C.II.c.iv.a Pre-treatment effects

There were no significant differences in pre-treatment control of finger skin temperature between the AR and control groups: Difference = 2.777 P>0.05, 95% CI (-0.921, 6.474) (Statistical appendix 2.3; table 3.b.III.i, page S26).

C.II.c.iv.b Post-treatment effects

A significant difference in post-treatment control of finger skin temperature was revealed between the individual AR and the Control group: Difference = 4.802 P<0.05, 95% CI (0.036, 9.567) (Statistical appendix 2.3; table 3.b.III.ii, page S26). Thus, the individual Applied Relaxation group had a higher finger skin temperature during post-treatment ambulatory monitoring than did the Control group.

C.II.c.iv.c Follow up effects

There were no significant differences in Follow up control of finger skin temperature between the AR and control groups: Difference = 1.323 P>0.05, 95% CI (-4.196, 6.843) (Statistical appendix 2.3; table 3.b.III.iii, page S26).

C.II.c.v.No treatment versus ART

	Mean middle finger temperatures (^{O}C)	**Mean ambient temperatures (^{O}C)**
ART	23.42	20.79
No treatment	23.69	22.75

Table 8.62 The mean middle finger and ambient temperatures (^{O}C) of the ART and untreated participants across the pre-treatment, post-treatment and follow-up ambulatory monitoring sessions.

C.II.c.v.a Pre-treatment effects

There were no significant differences in pre-treatment control of finger skin temperature between the ART and control groups: Difference = -0.277 P>0.05, 95% CI (-4.081, 3.527) (Statistical appendix 2.3; table 3.b.III.i, page S26).

C.II.c.v.b Post-treatment effects

There were no significant differences in post-treatment control of finger skin temperature between the ART and control groups: Difference = -2.593 P>0.05, 95% CI (-7.094, 1.909) (Statistical appendix 2.3; table 3.b.III.ii, page S26).

C.II.c.v.c Follow up effects

There were no significant differences in Follow up control of finger skin temperature between the ART and control groups: Difference = 0.318 P>0.05, 95% CI (-5.181, 5.817) (Statistical appendix 2.3; table 3.b.III.iii, page S26).

C.II.c.vi. No treatment versus ARE

Table 8.63 describes the mean middle finger and ambient temperatures of the individual ARE and Control groups during the ambulatory monitoring sessions.

	Mean middle finger temperatures (^{O}C)	**Mean ambient temperatures (^{O}C)**
ARE	26.31	20.31
No treatment	23.50	22.75

Table 8.63 The mean middle finger and ambient temperatures (^{O}C) of the ARE and untreated participants across the pre-treatment, post-treatment and follow-up ambulatory monitoring sessions.

C.II.c.iv.a Pre-treatment effects

There were no significant differences in pre-treatment control of finger skin temperature between the ARE and control groups: Difference = 3.036 P>0.05, 95% CI (-0.688, 6.761) (Statistical appendix 2.3; table 3.b.III.i, page S26).

C.II.c.iv.b Post-treatment effects

There were no significant differences in post-treatment control of finger skin temperature between the ARE and control groups: Difference = 1.277 P>0.05, 95% CI (-3.229, 5.783) (Statistical appendix 2.3; table 3.b.III.ii, page S26).

C.II.c.iv.c Follow up effects

There were no significant differences in Follow up control of finger skin temperature between the ARE and control groups: Difference = 2.101 P>0.05, 95% CI (-4.833, 9.035) (Statistical appendix 2.3; table 3.b.III.iii, page S26).

Thus, on the whole, the individual treatments had no effect on finger temperature during ambulatory monitoring; however, the AR group were shown to display a greater post-treatment finger skin temperature during ambulatory monitoring than were the Control group.

C.III. The Post Hoc tests

The main analysis of the ambulatory monitoring data uncovered two significant effects: a significant interaction between Biofeedback group and relaxation group (as described in section C.I.g, page 187), and an interaction between Biofeedback and time of testing (section C.I.e, page 186). The reader will recall that the main analysis merely indicated the presence of the interactions, and that Post Hoc tests needed to be carried out in order to isolate the nature of the interactions.

C.III.a. An interaction between Biofeedback and Relaxation group

Table 8.64 describes the mean finger skin temperature differences (after the effects of covariates have been removed) of the combinations of biofeedback and relaxation groups pooled over the three periods of assessment.

	No Biofeedback	**Temperature Biofeedback**	**EMG Biofeedback**
Autogenic Training	-0.228	0.154	-0.329
Applied Relaxation	0.092	-0.141	-0.015
No Relaxation	0.094	N/A	N/A

Table 8.64: Mean middle finger skin temperature differences during ambulatory monitoring across the Relaxation and Biofeedback groups

As the reader will recall, pairwise comparisons are carried out sequentially on the means that differ the most from each other in magnitude. Only one such pair of means were worthy of note in the Relaxation/Biofeedback group interactions. Indeed, as is evident from table 8.63, the largest difference between the means occurs between the Autogenic Training with Temperature Biofeedback group and the Autogenic Training with EMG Biofeedback group. The pairwise comparison, however, revealed no significant difference between the effect of Temperature Biofeedback on Autogenic Training and the effect of EMG Biofeedback on Autogenic Training: Mean difference = 0.48, $P > 0.05$, 95% C I (-0.90, 1.87). (Statistical appendix 2.3, table 3.c.I, page S27) This suggests that neither temperature nor EMG biofeedback had an effect on finger skin temperature during ambulatory monitoring beyond that of Autogenic Training.

C.III.b. The interaction between Time and Biofeedback group

Table 8.65 describes the mean finger skin temperature differences (after the effects of covariates have been removed) of the Biofeedback groups during ambulatory monitoring at each of the three periods of testing.

	Pre-treatment	**Post-Treatment**	**Follow up**
No Biofeedback	0.093	0.519	-0.258
Temperature Biofeedback	0.037	-0.271	0.355
EMG Biofeedback	-0.197	-0.568	0.161

Table 8.65: Mean middle finger skin temperature differences during ambulatory monitoring of the Biofeedback groups over time

The first pairwise comparison was carried out between No Biofeedback and EMG Biofeedback during the post-treatment ambulatory monitoring session. A univariate analysis with biofeedback as the independent variable revealed a statistically significant difference between the effects of No Biofeedback and EMG Biofeedback: Mean difference = 1.09, 95% confidence intervals (0.130, 2.04),

$p < 0.05$. Please refer to statistical appendix 2.3, table 3.c.II, page S27. This suggests that the No Biofeedback group had a higher finger skin temperature during the post-treatment ambulatory monitoring than did the EMG biofeedback group.

The second comparison between No Biofeedback and Temperature Biofeedback at the post-treatment assessment session revealed no statistically significant effect of Biofeedback group: Mean difference = 0.79, 95% Confidence Intervals (-0.299, 1.879) $p > 0.05$. Please refer to statistical appendix 2.3, table 3.c.II, page S27. In light of the results in the first pairwise comparison, this result suggests that both methods of biofeedback are ineffective in increasing finger skin temperature during ambulatory monitoring.

The final comparison was carried out between the EMG Biofeedback group at the post-treatment assessment session and Temperature Biofeedback at follow up. As the means were measured at different assessment periods, a two tailed independent t-test was used to compare the groups. A significant effect was found: $t(12.7) = 2.375$ $p < 0.05$. Please refer to statistical appendix 2.3, table 3.c.III, page S28. This suggests that the temperature biofeedback group maintained a greater finger skin temperature during follow up ambulatory monitoring than did the EMG Biofeedback at post-treatment.

In summary therefore, analysis of the objective ambulatory monitoring data showed a significantly higher finger skin temperature during ambulatory monitoring for the AR group than for the Control group, and no significant additional effect of Biofeedback beyond that of Relaxation.

It would be interesting to see therefore, if any subjective differences between groups were reported during monitoring. The ambulatory monitoring diaries were completed by 28 participants at the post-treatment assessment, and 26 participants at follow-up.

During the post-treatment monitoring periods, there were no group differences in ratings of mood; indeed, levels of reported anxiety, depression, frustration and stress were low. 63% pursued predominantly sedentary activities such as reading, watching television and even sunbathing. 89% (25) of the volunteers reported warm ambient conditions throughout the monitoring period, and 86% (24) did not feel the need to wear gloves. In line with this, 68% (19) reported no Raynaud's symptoms, and more than half of those who did (51%), described symptoms for less than 10 minutes of the 3 hour monitoring period.

At follow-up, the diary data revealed that over 80% of the participants reported no feelings of anxiety and depression during monitoring, and 70% (18) reported no Raynaud's symptoms. In line with this, most participants (77%) were predominantly sedentary, and 85% (22) described warm ambient conditions throughout the monitoring period; indeed, gloves were worn by only two Secondary Raynaud's sufferers.

SUMMARY OF THE EFFECTS OF TREATMENT

- The participants who received treatment claimed symptom improvement at post-treatment and follow-up; the untreated control participants did not.
- The Applied Relaxation group showed a significantly greater control over finger skin temperature at pre-treatment than did the Control group.
- Strong trends in the data suggest that the ATE group have better voluntary control of finger skin temperature at follow up when compared with the Control group.
- Participants who received relaxation (and biofeedback) training, reported implementing relaxation strategies during the test of voluntary control; whereas untreated participants did not. In contrast, objective tests revealed no group differences (treated or untreated) in use of relaxation strategies during the test of voluntary control.

- A higher room temperature and finger starting temperature is associated with faster rewarming following Cold Challenge.
- The individual Applied Relaxation group recovered faster from post-treatment Cold Challenge than did the Control group at this time.
- A higher ambient temperture during ambulatory monitoring is associated with warmer fingers; indeed, the participants fingers were warmer during the spring and summer months of the post-treatment and follow up assessments.
- During post-treatment ambulatory monitoring, higher finger temperatures were recorded by the individual Applied Relaxation treatment group than by the Control group.
- Biofeedback does not appear to have an effect on finger skin temperature measured via ambulatory monitoring.

8.3 CONCLUSIONS

To summarise, the analyses reported above revealed both subjective and objective effects of treatment. The following section will therefore relate these findings to the hypotheses outlined in chapter 7.

Firstly, it was hypothesised that there would be no subjective or objective pre-treatment differences between groups. Clearly such differences did occur. Indeed, pre-treatment questionnaire data revealed that only the experimental participants reported an association between "stress" and symptoms, that the control and EMG biofeedback groups claimed no previous experience of taught relaxation techniques, and that the No biofeedback and Temperature biofeedback groups reported some such experience. Moreover, baseline objective tests revealed greater finger temperature control within the Applied Relaxation than the other groups even before training had begun.

To address the subjective differences, although it had been clearly explained to all participants that the purpose of treatment was to reduce physical tension (rather than emotional stress), the assumed relationship between stress and symptoms appears to have had an impact on participants' choice of assignment to control or experimental conditions. Although this might suggest that the experimental participants envisaged greater benefit and personal relevance of the behavioural approach than did those who chose to be controls, this difference need not be a problem in itself: surely, in a real life setting, given the nature of the treatment, the only people who would be attracted to, and continue with the behavioural approach, would be those with some belief in the personal relevance of treatment. Indeed, involvement in treatment would require too much effort for those not convinced of its possible efficacy.

However, that said, a difference between groups in perception of an association between stress and symptoms might have some influence on the subjective outcome of treatment. Relaxation is seen by many as the opposite, or an antidote to stress. Thus, if stress and symptoms are associated in the

mind of the patient, stress reduction (via relaxation) should also reduce symptoms. Therefore, if someone who associates symptoms and stress feels some general benefit from relaxation training, (s)he is more likely to report subjective symptom improvement than someone who feels the same sensations of relaxation, but does not associate Raynaud's symptoms with stress. Clearly, therefore, either actual symptom improvement (owing to the effects of relaxation on sympathetic nervous flow), or perceived "stress reduction" might explain subjective reports of symptom benefit.

It is not without grounds, moreover, that the perceived role of stress in symptom manifestation might also colour objective tests of finger temperature control. Indeed, if participants view stress as having some role in symptom manifestation, in any "stressful" situation (e.g. cold challenge), finger temperature control might be less pronounced than when compared with a non-stressful situation simply because of the effects of unease and physical tension on sympathetic flow.

The second difference to emerge from the pre-treatment questionnaire was that of differential experience of relaxation techniques. If one assumes that the effects of relaxation training are cumulative, pre-treatment differences could affect treatment outcome. However, in this study, cumulative relaxation skills would be very slight as previous experiences of relaxation techniques were brief, were not generally recent, and were reported by less than 20% of the sample.

Similarly, that certain groups exhibited greater control over their finger skin temperature at the start of the study does not reduce the validity of the findings, nor prevent the efficacy of treatments being judged. Indeed, positive objective effects of treatment could still have been isolated through the observation of <u>increased</u> finger temperature control in those who were already shown to possess the skill, or should a ceiling effect be in place, by increase in finger temperature of the other treated groups.

A second set of hypotheses suggested that experimental participants would report greater subjective symptom improvement than the untreated Controls, and that the experimental participants'

performance on the objective tests would be improved following treatment. As the reader will recall, the behavioural treatments had a pronounced positive effect on the subjective view of symptoms. Moreover, both the Cold Challenge and Ambulatory Monitoring tests indicated that the individual Applied Relaxation group had significantly greater control over post-treatment finger temperature than did the Control group. Further, a strong trend in the data suggests that the ATE group had greater voluntary control over finger skin temperature at follow up than did the Controls. Thus, the second set of hypotheses can not be disproved by this investigation.

It was also surmised that the combined effects of biofeedback and relaxation on both subjective symptom report and performance on objective tests, would be greater than that of relaxation alone. At a subjective level, the hypothesis is clearly refuted by the finding that the Temperature and No biofeedback groups described equal symptomatic benefit and that both described greater benefit than the EMG biofeedback group. (Had the hypothesis been correct, both biofeedback groups would have reported greater subjective benefit than the No biofeedback group). Similarly, at the objective level, biofeedback was not shown to increase the effects of relaxation on finger temperature - indeed, the apparent positive effect of Autogenic Training with Biofeedback was shown only to be a trend in the data.

The penultimate hypothesis was that the effects of training would be related to practice of the exercises. Thus, assuming that the effects of relaxation are cumulative and that the exercises have an effect on peripheral circulation, participants who practice regularly should obtain more subjective and objective symptom benefit than those who practice rarely or not at all. At the subjective level, the AR and ATE groups claimed to have practised the most, and should therefore have reported the greatest symptom benefit. However, no such relationship between subjective benefit and practice levels was uncovered. Looking at the objective data, however, the reader will recall a significant post-treatment effect of AR temperature control over that of the Controls, and a trend in the data suggesting a positive effect of ATE on finger temperature at follow up. The AR and ATE groups were those who claimed to have practised the most - particularly during training. Thus one might

assume a relationship between practice levels and objective effects of treatments; indeed, the findings of this study can not refute the suggestion that failure to practice is likely to result in failure to benefit from treatment.

Finally, it was predicted that the Phenomenon participants would benefit less from treatment than the Raynaud's Disease participants - simply because of the physical abnormalities associated with the Secondary condition. However, appraisal of such differences in treatment benefit is somewhat confined by the unknown diagnosis of the "unsure" participants. Indeed, at a subjective level, only the unsure and undiagnosed groups reported greater benefit from treatment; thus, one might assume that the Phenomenon and Disease patients do not differ in terms of effects of treatment. However, as the "unsure" patients were generally not receiving medical treatment for secondary conditions, one might argue that their diagnosis is in fact that of Raynaud's Disease. If so, the results suggest that those with milder symptoms (i.e. the Raynaud's Disease and Undiagnosed participants) did, indeed, enjoy greater subjective treatment benefit than was the case with the Raynaud's Phenomenon patients.

Therefore, in summary, the behavioural approach to the management of Raynaud's symptoms (as outlined above) appears to offer both subjective and objective benefit to patients if practised regularly.

The final chapter will provide a more in depth discussion of some of the more global points raised in this thesis.

Chapter 9

GENERAL DISCUSSION

9.1 INTRODUCTION

The initial aims of this thesis have been met: the reader, equipped with a basic understanding of Raynaud's, its precipitators and treatments, has encountered both subjective descriptions of Raynaud's and its effects, and an investigation of the efficacy of one behavioural approach to treatment. A number of questions and points of interest have emerged during the course of writing. Some have been satisfactorily addressed above; others, merely touched upon, require further discussion in this final chapter. Moreover, a number of points, as yet unmentioned, need attention if this thesis is to be complete: for example, side effects and ethical considerations as to the future use of the behavioural approach, and details of the training of control participants.

Certainly, there is much to discuss. Therefore, this chapter will initially extend upon points already outlined in the thesis, but not, as yet, brought to a satisfactory conclusion. It will then move on to discuss both a range of practical considerations relevant to the design and implementation of interview, questionnaire, and behavioural training studies, and a number of theoretical suggestions, guidelines and recommendations for future work. These broad areas of discussion will provide the groundwork for a discussion of the future role of the behavioural approach in the management of Raynaud's symptoms.

9.2 AN OVERVIEW OF THE THESIS

On reading through the thesis, six broad areas of discussion emerge as incomplete. These include issues related to the diagnostic classification of Raynaud's patients; the role of "stress" in behavioural research; the role of participant expectation in symptom onset, maintenance and treatment; the role of ambient temperatures on peripheral finger skin temperature, the bearing of the main treatment study on the question of cause of Raynaud's attacks; and finally, given the still widely accepted - although

inappropriate - view of biofeedback as a conditioning treatment, a clear discussion of the difference between conditioning and biofeedback-mediated treatments.

The diagnosis of Raynaud's

In both the initial questionnaire and later treatment study, volunteers were classified as either Raynaud's Phenomenon patients, those suffering from Raynaud's Disease, those diagnosed but unsure of their diagnosis, or those undiagnosed but exhibiting the characteristic symptoms of Raynaud's. Inclusion of the latter category was a matter of necessity given limited interest of formally diagnosed volunteers. However, in recruiting undiagnosed participants for the main treatment study, the diagnostic guidelines outlined in chapter 1 were rigidly adhered to. (Systemic disease was assumed absent given the duration of symptoms and the fact that the volunteers had not approached a doctor with regard to any secondary symptoms). However, one must wonder whether the inclusion of the undiagnosed group was actually appropriate; and moreover, given the author's lack of formal medical qualifications, the validity of judgements as the diagnostic status of such participants.

To address these points, consider the following. I do not suffer from Raynaud's, yet have experienced three cold-induced instances of vasospasm in the fingers - characterised by blanching, cyanosis, and hyperaemia - whilst cycling without gloves in the snow. Two such "attacks" left me unable to get warm all day despite turning on the central heating and taking a hot bath. Let's assume that I were now to read an advertisement recruiting volunteers for an investigation of the behavioural approach to the treatment of Raynaud's. Assuming that my knowledge of Raynaud's was confined to magazine articles on the subject, would I try to get involved ? Possibly. Yet, my "symptoms" are merely an example of the normal adaptive response to cold: anyone cycling without gloves in freezing temperatures would experience such "symptoms".

Let's now assume that one of my "attacks" occurred in February 1993, and the latter two in February 1995. I now have a two year history of symptoms. I might mention these "symptoms" to the doctor in passing: "Oh, that sounds like Raynaud's syndrome/Disease/Phenomenon. Just try to keep warm,

but come back if it bothers you." I am now "diagnosed"; though unsure of my diagnosis. Thus, both before and after "diagnosis", I might legitimately have become involved in a treatment study - despite not being a Raynaud's sufferer. Following a particularly cold winter and one further "attack" (perhaps, after coming in from the cold and spending 10 minutes searching in the freezer), I decide to go back to the doctor, but am offered nothing more than the advice to keep warm. I read another article about Raynaud's, and discover that a number of potential secondary conditions - some life threatening - are associated with my "symptoms". To allay my fears, I am referred to a consultant for tests. These reveal no underlying systemic disease; thus, my symptoms must be indicative of Primary Raynaud's Disease. Clearly, this line of thinking could not be extended to the diagnosis of Raynaud's Phenomenon as if secondary conditions are present, the diagnosis must be Raynaud's Phenomenon. However, poetic licence aside, as the only clear cut diagnosis is that of Raynaud's Phenomenon, the example above could occur.

The paragraphs above aim not to suggest that the problems of Raynaud's sufferers are in any way fabricated; neither is the professional integrity of the general practitioner brought into question. The role of the scenes above is to underline the inherent difficulties of diagnosing Raynaud's, and to illustrate that any objections to the inclusion of Undiagnosed participants in the behavioural study might equally be applied to the inclusion of those unsure of their diagnosis, or those diagnosed as suffering from Raynaud's Disease.

At present, the most credible technique of diagnosis appears to be that of individual opinion based on standard description of the disorder (see chapter 1), which begs the question: To whom should any such opinion belong ? Perhaps only those formally trained in medicine ? But what of those who have spent some 18 months learning about Raynaud's and its treatment, who have met, interviewed and talked with sufferers about their symptoms (yet obtained no formal medical qualification) ? Given the difficulties associated with diagnosis, the diagnosis of Raynaud's Phenomenon aside, it is clear that the latter would be acceptable - particularly as volunteers' symptom history was obtained, that in most cases, vasospastic attacks were witnessed, and that not all of those interested in the study

were included as a number did not appear to sufferer from Raynaud's. (Here, rapid recovery following the first cold challenge test - i.e. within 5 minutes -played a part in diagnosis. Thus, in so far as it is possible to tell, those who took part in the treatment study (though not necessarily all who completed the initial postal questionnaire[34]) were Raynaud's sufferers. Consequently, a lack of formal diagnosis was not considered an obstacle to participant inclusion.

The role of "stress" in Raynaud's research

The experimenter's view aside, a trainee might assume an association between stress and symptoms, and by default therefore, between stress and behavioural treatment. Despite clear guidelines to the contrary, behavioural training volunteers are likely to stamp treatments with "stress reduction" properties. Indeed, treatment study volunteers described at length the "positive stress-reduction" properties of their treatment e.g. how their ability to cope with everyday events had improved. These effects were heartfelt, and should not be dismissed; however, many of the trainees appeared to have failed to grasp (or ignored) the described arousal reduction properties of treatment simply because they were unable to dissociate the ideas of stress and relaxation. Indeed, a number of participants had definite preset ideas as to the stress-reduction effects of treatment which were maintained throughout the treatment programme; thus, treatment was not seen as having any direct physical effect. Indeed, the only effect perceived by the participants in question was that of stress reduction (personal observation).

It is difficult to undermine such pre-set ideas - particularly when one considers the almost universal acceptance of the concept of psychological stress. Consequently, stress must have a role in the behavioural approach to treatment where participants believe that it does. Though, perhaps impossible to remove participants' belief in the concept of stress, methods of reducing its impact might be of use. For instance, the researcher might note that it is not enough to labour the point as to the intended role and mechanism of treatment, but that a clear (and regular) statement is required that the purpose of treatment is not to reduce stress, but to reduce physical tension.

[34]The author had no access to patient medical records, and without meeting patients, had to rely on second hand diagnostic information included in completed questionnaires.

Even with the introduction of such techniques, other participant effects may still have a role. Indeed, although the mechanism of relaxation is assumed to be that of a reduction in physiological arousal, there are sound objections to the complete acceptance of such a model. If the arousal model were entirely correct, it would follow that all relaxation techniques exert the same effects (i.e. a reduction in physiological arousal), and should thus, be equally as effective for any given individual. Physiological arousal is indeed reduced with relaxation; however, the arousal reduction model is incomplete as some cognitive processing on the part of the trainee must occur. Smith (1988) outlines a cognitive-behavioural model to account for both the arousal and cognitive aspects of relaxation which assumes the involvement of cognitive processes in the acquisition of the skill of relaxation. These include focusing of attention (i.e. concentrating on the exercises), passivity (ie. letting the relaxation happen), and receptivity (i.e. acceptance of the ideas and requirements of the technique). The degree of arousal reduction depends on the individual, and on the relaxation technique employed. Thus, at the individual level, the initial degree of physical tension will influence the amount of arousal reduction required, and the initial receptivity of the individual to the concept of relaxation (for example) will influence the cognitive demands of treatment. Moreover, different relaxation techniques will be associated with varied cognitive demands: somatic techniques such as Progressive Relaxation or yoga are relatively undemanding (the trainer's voice directs the trainees attention, and the exercises make use of everyday movements such as tensing the fists); Autogenic Training, in contrast, given that the onus is more on the trainee to carry out the exercises, might require greater cognitive involvement. Thus, in someone physically tense and sceptical at the start of treatment, successful acquisition will depend on a substantial reduction in physical arousal, and a greater shift in attitude toward acceptance of treatment. Smith (1988) suggests that as training progresses, cognitive processing is refined such that the demands of learning to relax are reduced. For example, trainees get more used to focusing their attention as the phrases of the exercises become more memorable; as the pleasant effects of relaxation become more pronounced, participant resistance to practice will be reduced; in noting the failure in forcing relaxation, trainees will become more comfortable with the idea of passively allowing relaxation to happen; and as the benefits of treatment begin to emerge,

acceptance and receptivity toward the taught relaxation technique will increase. In brief, through training, the attitude of the trainee toward relaxation will change.

Clearly, beyond attaining the ability to reduce physiological arousal during training, relaxation trainees undergo attitude changes that must be considered within behavioural research. Thus, a lack of improvement on a post-treatment assessment test might be a function of an attitude-related lack of skill acquisition rather than evidence that treatment is ineffective. In the main study described above, participant attitude was assessed via pre- and post-treatment questionnaire. However, changes in attitude were difficult to assess partly because of problems with the wording of the relevant question (see section 9.3 for a discussion), but mostly because participants tended not to respond to the question. Consequently, despite sound initial planning of the experimental protocol, the effects of attitude could not be adequately taken into account.

The role of participant expectation on the results

To continue with the question of participant attitude, questionnaire data (chapter 3) suggested that, at least for a small number of participants, the onset of an attack might be accurately predicted from prevailing ambient conditions i.e. where symptoms are expected to occur, they do[35]. As expectation appears to have a role in the onset of Raynaud's attacks, it must follow that expectation might also be implicated in any positive effects of treatment i.e. where improvements are expected, they will occur. Perhaps, to fulfil expectations, participants' judgement as to what constitutes an attack (unconsciously) changes, or given the considerable effort expended by participants, the need to obtain "pay back" of some description if continuation with treatment is to remain a viable option, might colour participants' evaluation of the efficacy of treatment. Thus, the physiological effects aside, treatment is likely to be viewed as having some positive effects such as improving the quality of participant's life. Indeed, (non-specific) factors beyond those traditionally associated with behavioural treatments are implicated. This discussion must, therefore, move on to consider how such factors might be accounted for, or avoided, in future research. Although a range of such factors

[35] The role of anticipation in the onset of an attack having been described previously.

might correlate with treatment outcome, not all possibilities can be addressed here; thus, discussion will focus on participant attitude to treatment, and on experimenter effects.

Looking initially at participant attitude, whether positive or negative, (a change in) participant attitude will influence treatment outcome. For example, a positive shift in attitude (eg. toward increased receptivity and passivity) will promote the acquisition of the relaxation technique, and therefore, any positive treatment effects. A negative attitude, in contrast, will be associated with difficulties in maintaining passivity, receptivity, and focusing of attention, which may reduce patient compliance or precipitate withdrawal from the study. Thus, participants' (changing) views of the taught techniques, and of the acquisition of such techniques, must be sought, and any practice-related difficulties discussed. The client and therapist, as described in the main treatment study above, should consider means to reduce distractions when practising exercises at home[36]; the trainer should remain positive about treatment to help reduce participant scepticism; and perhaps, an illustration of how the trainer overcame his/her own obstacles to acceptance and enjoyment of the technique, might be employed. Moreover, within a clinical setting, where attitudes toward the taught techniques do not appear to be shifting in a positive direction, alternative behavioural treatments might be introduced.

A second source of non-specific effects is that of experimenter bias. For example, participant subjective reports as to the efficacy of treatment might be tainted by a need to please the experimenter through a positive attitude to treatment and its effects. Further, the presence of the experimenter at testing might negatively influence participant ability to control finger temperature under assessment conditions (Bregman and McAllister, 1983)[37]. To reduce such effects, Guglielmi et al (1994), for example, propose the implementation of double blind methodology. (To recapitulate, with the double blind approach, neither the experimenter nor the trainee is aware of the treatment received). However, if treatment involves training and practice in a relaxation (or equivalent)

[36] Practising at home was compared negatively with that in a sound proof laboratory

[37] In the study described in chapter 8, the researcher was present at all sessions; thus any such negative effects would presumably influence all participants, and not confound results.

technique, the trainee must be aware of the treatment received; moreover, the very act of training also requires such awareness on the part of the trainer. Thus the double blind approach is clearly inappropriate. However, a single blind approach in which the researcher carrying out the assessment tests is blind to the treatments received[38], and a second researcher teaches the appropriate behavioural technique(s), might be admissible. Advantages would include the reduction in participants' need to please the experimenter, and thus, "pressure to perform" in the assessment sessions - particularly if it were clearly stated that the trainer would have no access to participants' questionnaire or objective test results. Furthermore, withdrawal owing to client-therapist personality clashes would be addressed through the introduction of a second researcher as the contact hours with a "disliked" researcher would be reduced.

Such changes to the research protocol can, at best, reduce the effects of non-specific factors. Nevertheless, that such factors remain, need not be a problem if the effects apply uniformly across all participants, and if such effects are monitored. For example, in the study described above, the follow-up assessment session was included primarily as a means to identify such placebo effects; indeed, it is well documented (eg. Marcer, 1986, p83) that non-specific effects are of limited duration. However, where non-specific factors influence certain participants' results more than those of others, the validity of empirical results might be drastically reduced. Thus, if a study were designed to compare the effects of Autogenic Training with, perhaps, Progressive Relaxation, and the trainer, the number of training sessions, and the practice requirements were constant across groups, any unaccounted for effects, such as "pressure to perform", would, one assumes, influence the results of both sets of participants. However, if a control group were introduced (perhaps, receiving no treatment, but taking part in identical assessment sessions as the experimental participants), clear differences between the control and experimental groups would emerge: the number of sessions attended and associated difficulties in parking/waiting at bus stops would differ; consequently, the amount of attention received from the therapist would not be equal across groups.

[38] When relaxing or using some imagery technique, participants tend to close their eyes and remain relatively still; thus, it would be unlikely that the assessment researcher could accurately gauge the nature of the taught technique.

The example above clearly brings into question the validity of the control group employed in the main treatment study: what role did the described non-specific factors take in the control and experimental groups' post-treatment results; were differences truly a result of the presence or absence of behavioural training ? The only means to reach a conclusive decision would have been to have included an additional control group ie. one consisting of Raynaud's participants who, whilst not receiving training in relaxation, attended an equal number of ineffectual, placebo "treatment" sessions in addition to the assessment sessions. Such a control groups' treatment would have to be known (by the experimenter) not to have any effect on peripheral skin temperature, but appear credible in the eyes of the participants. Both practical and ethical difficulties would, of course, prevent the inclusion of such a control group. Indeed, from a practical stand point, as outlined in chapter 6, there are no adequate placebo versions of biofeedback or relaxation. Further, even if there were, the lack of any improvement in symptoms through such "treatments" might lead to an increase in patient non-compliance and withdrawal; thereby, reducing, rather than improving, the validity of results. Ethically, training in a procedure known to be ineffective would waste the participants' time and energy[39]. If one were to recruit from a non-symptomatic student population, such a protocol might gain ethical approval; however, with such a clinical population, approval might prove somewhat harder to obtain. Thus, within the confines of academic research, a state of stalemate appears to prevail: the occurrence of non-specific factors must be noted and discussed, but it must also be accepted that, often, little can be done to adequately control for such factors.

The role of ambient temperatures on peripheral finger skin temperature

The reader will have noted the significant role of high ambient and finger skin starting temperatures on finger skin temperature control and recovery throughout the objective tests used in this study. Without wishing to detract from the effects of the behavioural treatments, it should be noted that the strong effects of these covariates add additional support to the medical advice given to patients when initially diagnosed i.e. to keep hands and other extremities warm in an attempt to avoid and/or better cope with symptoms should they occur.

[39] Many Raynaud's volunteers being quite ill with associated secondary conditions.

The bearing of the results on the question of cause of Raynaud's attacks

Within the limits outlined above, it might prove appropriate to assume the validity of the results of the main treatment study. From this stand point what bearing might the results have on the question of the cause of Raynaud's attacks ? (The reader is reminded of Raynaud's causal theory of hypersensitivity of the sympathetic nervous system (HSNS), and Lewis' "local fault" theory).

To briefly recapitulate, results suggested that whilst subjective improvement occurred in all treated groups, only short term objective improvements in control of finger skin temperature were shown by the Applied Relaxation group. If attacks could be shown to be controlled by relaxation, the role of sympathetic innervation in the onset of attacks might be supported. If not, a "local fault" explanation of vasospasm might prevail.

Thus, what effect did relaxation have on vasospastic attacks ? Throughout the study, no attacks were recorded during laboratory monitoring, so the effects of laboratory-based relaxation on attacks (and thus, on sympathetic flow at the time of vasospasm) could not be ascertained. However, the Cold Challenge test was designed to provide a simulation of an attack. As the AR group were significantly faster at rewarming following post-treatment Cold Challenge than the control group, it could be suggested that the AR group were better able to cope with attacks than were the Control group. However, during the cold challenge assessment sessions, it was not ascertained whether the relaxation techniques taught were actually implemented by the participants. Thus, though tempting to suggest that the results of this study lead to the conclusion that hypersensitivity of sympathetic innervation might provide the cause of Raynaud's attacks, we do not know for certain that the AR group used Applied relaxation to warm their fingers following Cold Challenge. Consequently, the question as to cause of attacks (local fault or hypersensitivity of the sympathetic nervous system) can not be answered on the basis of the results of laboratory monitoring.

However, vasospastic attacks did occur during ambulatory monitoring (and, of course, periodically throughout the duration of the training programme). The ambulatory monitoring diaries recorded

instances of vasospastic attack, which were, in the main, rated as mild by the participants. However, although information as to participant activities was noted, once again, at no point were instances of the application of relaxation techniques documented during attacks. Thus, the effect of the relaxation strategies on Raynaud's attacks during ambulatory monitoring can not be established, and as with the laboratory monitored data, the ambulatory monitoring data can not provide insight into the cause of Raynaud's attacks.

There are two possible explanations as to why the utilisation of relaxation strategies during attacks were not documented. Firstly, experimental participants may not have actually applied relaxation techniques at the time of vasospasm which would suggest that the taught techniques are not practical as treatment for Raynaud's. Secondly, relaxation techniques might have been applied, but not actually noted in the participant's diaries. (That diary entries were not checked for descriptions of the application of relaxation techniques during data collection, was clearly an oversight on the part of the researcher). Indeed, although participants had been asked to note down their activities, relaxation may not have been considered an activity as such: with both Applied Relaxation and Autogenic Training, relaxation might have been seen more as a passive breathing monitoring exercise rather than a conventional "activity".

General subjective accounts[40] might prove a more useful means to assess the role of relaxation on attacks. Indeed, one female undiagnosed ATT participant described how she successfully used AT to shorten the duration of vasospasm whilst waiting in a cold hall for a yoga class to begin. In contrast, a number of Raynaud's Phenomenon participants noted how, although pleasant to practice, the pain and sensations of cold during attacks were such as to prevent relaxation actually being put into practice during vasospasm. Thus, from the main treatment study, supportive evidence of the role of relaxation in the amelioration of symptoms (as opposed to merely increasing general blood flow) is somewhat limited.

[40] As simply asking whether the taught strategies had any effect on Raynaud's attacks permits bias, the accounts reported here are those that emerged spontaneously during training and assessment sessions.

Conditioning versus Biofeedback as treatment technique

A question that might yield a more positive outcome, is that of the difference between biofeedback and conditioning treatments. As previously stated, biofeedback is seen as an aid, rather than means to temperature control; conditioning treatments might provide a means to such control. The term "conditioning" refers to the modification of behaviour through the setting up of an association between two processes or events. There are two forms of conditioning: Classical, in which a stimulus is paired with a response e.g. a dog might be conditioned to salivate in response to the sound of a bell (Pavlov, 1927); and Operant, in which trainees learn that a particular response will be followed by a particular consequence or action eg. a caged rat might be operantly conditioned to press a lever to obtain food (Skinner, 1938).

In much of the literature, biofeedback is considered a form of operant conditioning and, as such, a stand alone treatment in its own right. Classical conditioning is not considered an appropriate model of biofeedback because a stimulus (e.g. a specific biofeedback tone) must be directly associated with a particular response (i.e. a particular finger skin temperature); thus, a series of tones could only be associated with a series of temperatures, and no means of altering the pitch of the biofeedback tones would be available to the trainee. However, classical conditioning may prove an appropriate means to "learn" to associate cold ambient temperatures (i.e. those associated with the elicitation of an attack) with warm finger skin temperatures. Indeed, through sounding a buzzer at the time of hand immersion in cold water, Menzies (1937) classically conditioned non-symptomatic volunteers to vasoconstrict in response to the sound of the buzzer. If vasoconstriction might can be classically conditioned, why not vasodilation ?

To return to operant conditioning and biofeedback, Simkins (1982), noted that "*many of the biofeedback applications have not yet demonstrated their hoped for potential*" (p4). From the standpoint of this thesis, one might suggest that the apparent failure of biofeedback is a function of the adoption of an inappropriate operant conditioning model of biofeedback. To substantiate this opinion, both operant conditioning and biofeedback will be inspected more closely.

Operant conditioning is traditionally the technique used in shaping the behaviour of animals (and, in line with Behaviourist thinking, equally applicable to learning in humans). Indeed, Thorndyke (1898) described the "law of effect" to explain how animal (and by default, human) behaviour comes to be operantly conditioned. A cat in a cage, for example, might, through the course of a range of cat behaviours, inadvertently catch the latch of the cage with its paw, thereby opening the cage and freeing itself. The cat does not learn from this experience per se, but the action of catching the latch is reinforced by the positive reward of a period of freedom. Other elicited behaviours may not receive such positive reward or reinforcement; thus over time, a set of rewarded behaviours will become more pronounced; others will be eliminated as a consequence of the lack of associated reinforcement. Therefore, through operant conditioning rather than insightful cognitive-mediated learning, the cat comes to "know" how to open the cage and gain its freedom.

The role of operant conditioning in biofeedback-mediated temperature control would presumably lie with the assumption that once a particular behaviour has been performed (e.g. a strategy of hand warming), the likelihood of that behaviour being repeated (or maintained), is dependent on the consequences of that behaviour i.e. whether or not it is rewarded through a reduction in biofeedback tone. Clearly, therefore, some strategy of control must occur in operant biofeedback conditioning. The operant model allows for this in that a range of non-physical[41] behaviours will be unconsciously elicited by the trainee, who, might inadvertently stumble upon a behaviour that is associated with a reduction in the pitch of the biofeedback tone. Over a series of trials, this successful behaviour would be positively reinforced by its repeated association with a reduction in biofeedback tone.

To a point, this appears a credible account of the role of biofeedback in temperature or muscle tension control; however, at the same time, it underlines the point made in earlier chapters that there is no such thing as "pure" biofeedback. Indeed, some strategy of control is clearly employed - be awareness of it conscious, or unconscious. The operant conditioning model would predict unconscious "trial and error" as the means of reaching a successful strategy; the cognitive model

[41] The trainee having been instructed not to use physical strategies.

supported by this thesis, would predict conscious "trial and error" (or in the study described in chapter 8, the conscious use of a taught relaxation strategy) as the means of reaching a successful strategy. It is difficult to predict which theory might be appropriate - particularly as both view biofeedback as providing "knowledge of results" of an unconsciously or consciously chosen strategy. Thus, the role of conscious awareness must differentiate between the two models: if conscious awareness at some early point of training is not required, the operant conditioning model might be accepted; if such awareness is required, the cognitive model must come into play.

Consider again the cat "learning" to escape from its cage. If the cat's paw catches the latch, the cage door will open. Whether it opens a little or completely, the reinforcement effect is still the same i.e. freedom. In contrast, in the biofeedback example, whether the trainee is hot, cold, alive or dead, given the nature of the biofeedback apparatus, a tone (reinforcement) will be emitted when the participant is attached. Thus, any strategy of control will only alter the pitch of the reinforcer. Therefore, reinforcement must be in terms of degree - there being no equivalent "door open", "door closed" with biofeedback. If the biofeedback trainee is to come to control that tone (i.e. control his/her reward), clearly, some cognitive appraisal mechanism must be implemented.

Given this need for cognitive appraisal of the biofeedback tone, the operant conditioning model of biofeedback can not apply. The role of biofeedback must, therefore, be to provide a window to, or knowledge of results of the relative success of a particular strategy of temperature control. Perhaps conscious awareness of the strategy might no longer be required once the skill of temperature control has been attained, but, at the stage of acquisition, it most certainly is.

Thus, the five points outlined at the start of this thesis overview have now been addressed. Consequently, the discussion will move on to practical difficulties to emerge during the course of the described empirical investigations. However, before thus continuing, it should be noted that to meet ethical obligations, a number of control participants were trained in the behavioural approach following completion of data collection. Of the 9 control participants who completed all three

assessment sessions, only 3[42] actually opted for training. Owing to time limitations, training was forced to begin before analysis of the main empirical investigation had been completed. Thus, as no outstanding treatment had emerged from the post-treatment analysis, participants were trained in the technique which, retrospectively, was considered the most pleasant to teach (Applied Relaxation). Given the small number of participants, no data were collected; however, subjective report did suggest that the effects of treatment were viewed in a positive light.

9.3 PRACTICAL CONSIDERATIONS

The points of practical value to emerge through the completion of this thesis are of three types. Firstly, those of relevance to the planning of empirical investigations (including interview, questionnaire, and treatment evaluation); secondly, those related to the analysis of such investigations; and lastly, the question of the acceptability of techniques utilised within this behavioural approach to treatment.

As to the first point outlined above, the areas of participant recruitment and compliance apply equally to the planning of interview, questionnaire and treatment studies, and will thus, be addressed first.

There are three possible routes to the recruitment of Raynaud's volunteers: namely, by referral through hospital consultants, referral through general practitioners, and finally, through advertising. Across individuals, Raynaud's severity differs a great deal: some patients experience secondary symptoms; others, simply painful vasospastic attacks; still others, mild, occasional discomfort. Consequently, the need to seek medical attention for symptoms will differ in a similar manner: questionnaire data suggests that Raynaud's Phenomenon patients seek medical attention at least within the first year of symptom onset; idiopathic symptoms might not warrant referral to a consultant haematologist; mild Raynaud's symptoms may not even reach the attention of the family doctor. Therefore, obtaining a patient sample from only a consultants' referral list for any treatment

[42] 2 Raynaud's Phenomenon participants (1 male, 1 female) and 1 undiagnosed male.

study would prevent the generalisability of the results of that test to the Raynaud's population in general. Perhaps this would not prove a matter of concern with the evaluation of pharmacological interventions as drugs tend to be targeted at severe symptoms. However, with the behavioural approach, those with milder, primary symptoms might benefit most, and thus, through testing with a biased sample (such as those from a consultants referral list), a potentially beneficial treatment might be dismissed. Participant recruitment through general practitioners might provide a preferred alternative given the diagnostic validity associated with recruiting from such a source, and the wider range of Raynaud's patients obtained. However, owing to logistical difficulties such as the greater work load required in recruiting in a number of health centres, coupled with the need to obtain a patient sample within a relatively short period of time, the general practitioner route was not considered a viable option. Moreover, owing to a cross discipline grant application (psychology and haematology), a list of 111 potential volunteers was already available at the start of the recruitment process. Therefore, to increase the validity of the patient sample, recruitment from an alternative source (an advertisement in a local newspaper) was required to redress the balance. Disadvantages, as noted previously, included the problem of attracting people who were not actually sufferers of Raynaud's. However, through combining two such sources of volunteers, it was hoped that a more generalisable evaluation of the effects of treatment could be obtained.

It should be noted that although the problem of generalisability of the patient sample was clearly a negative factor in the initial pilot interviews described in chapter 3 (predominantly Raynaud's Phenomenon patients), even with a range of participant sources, only those with severe symptoms might consider their condition absorbing enough to warrant the interest of a researcher ie. those with only mild symptoms would be less inclined to get involved in an interview study. With a treatment programme, in contrast, sufferers might gain personally from involvement; thus the effects of personal consideration of interest value, might be less pronounced.

Questionnaire difficulties lay mainly with the issues of question ambiguity and participant freedom not to respond to questions. Examples, drawn from both the Raynaud's and treatment study

questionnaires include "What are the effects of Raynaud's in your life ?", and "Please write as little or as much as you like about your views on non-conventional treatments..". The initial question was intended to gauge how being a Raynaud's sufferer might interfere with everyday activities; the latter, a means to assess (changes in) participant opinion toward treatment. In both cases, data were limited by participant misunderstanding of the information required and/or failure to respond. Thus, in the former case, participants tended simply to repeat symptom details; in the latter, participants tended not to respond at all - possibly, one assumes, owing to the need to spend time both considering and wording answers. Thus, although the questionnaires were piloted, it is clear that studies might benefit from closer attention at the questionnaire design stage: unambiguous questions, covering the key points of interest whilst requiring simple yes or no answers, might improve completion rate through reducing "work load" requirements of participants; however, to avoid effects of forced choice questioning, some provision for longer, more spontaneous answers should also be employed.

As to the question of compliance, further consideration, beyond that discussed and implemented in previous chapters, is required in light of the experience gained in carrying out a large scale treatment study. Clearly, both patient practice and session attendance levels require serious attention if the true benefits of treatment are to be discovered. In the described treatment study, a series of steps were implemented in an attempt to improve attendance levels. Initially, following failure to attend, volunteers were contacted to arrange a further appointment. Generally, this appointment was kept; however, often the volunteers failed to attend a second time. Here, participants were contacted, and the potential personal benefits of treatment and/or the needs of the researcher to maintain attendance levels for the purpose of obtaining a Ph.D., were outlined. Generally, this was sufficient to promote attendance (if not compliance to practice). However, where this measure failed, no further action was taken - participants were thanked for their help and interest, and allowed to withdraw from the project - as it was considered that benefits of relaxation would not be exhibited in those who felt obliged to attend sessions against their will. In line with this, it might be noted that with any treatment requiring a great deal of commitment on the part of the patient, only those with sufficient self-will and social support (and, one assumes, severe symptoms) might actually continue with and gain from treatment;

thus, no benefits might be associated with "forcing" participant attendance. However, as a means to increase both self-will and social support, the behavioural treatment might be offered to both the patient and close (non-)Raynaud's relatives or friends (their own health problems considered). Indeed, training two or three people at once would not significantly increase the demands on the trainer or his/her resources.

To turn now to the main treatment study, in an adequately designed investigation, all factors likely to influence body temperature (e.g. food intake, activity levels, and pre-assessment session levels of caffeine and nicotine) ought be taken into account. In practice, however, logistical difficulties prevent such exacting control. For example, ideally, participants should be randomly assigned to session times (to allow for differences in temperature effects related to meal times) and continue to attend at those times throughout the treatment and assessment period. But, given the number of participants and sessions required, the assignment of participants to time slots was far from random and not consistent through the study. Moreover, on consideration, instructions not to drink coffee or smoke cigarettes prior to the sessions were abandoned as piloting experience with student volunteers suggested that people tend either forget such instructions or ignore them[43]. However, most participants were non-smokers, and allowing for travelling time, waiting and adaptation periods, it is unlikely that coffee would have been taken within an hour or so of training or assessment.

A further difficulty with the design of the treatment study was that owing to resource limitations, the biofeedback groups could receive feedback only during the training sessions. Ideally, biofeedback apparatus would have been provided to enable practice of relaxation techniques with appropriate feedback of results at home, such that direct comparison of the effects of biofeedback and non-biofeedback mediated treatment might be obtained. Thus, though clearly not a design fault, as a result, the biofeedback groups differed only marginally from the non-biofeedback groups.

[43] Such instructions might also have interfered with patient compliance.

The second area of practical discussion is that of statistical analysis of results. As the reader may recall, it was noted that owing to the small data set, Chi-square analysis was dogged with problems associated with Type I errors. In essence, given the small data set, the tests performed on questionnaire data were less stringent than might otherwise have been the case. Generally, this fundamental difficulty must bring the validity of the results into question; however, in the questionnaires described in this thesis, significant results were rare, and where they did occur, were generally in the direction predicted in the literature. Thus, although the problem should be noted and considered in any future work, its effects do not severely undermine the results presented here.

The World Health Organisation (WHO), as referenced by Cottreaux (1993), recently published a list of guidelines as to the acceptability of treatments for psychiatric disorders which are equally relevant to the evaluation of treatments for other disorders. Seven main points are outlined including the effectiveness, safety, and side effects of treatments, ethical acceptability and applicability of treatment in a range of settings, the relative costs of treatment in relation to alternatives, and lastly, the possibility of treatment misuse. With these as a framework, the discussion might now turn to the question of general acceptability of the behavioural approach to treatment.

In relation to the efficacy of treatment, three questions might be considered: firstly, the role of treatment in subjective and objective symptom reduction; secondly, the effect of treatment on quality of life; and thirdly, a related point, the retention of any subjective or objective positive effects of treatment. As to the first two points, non-specific factors aside, treatment appears to be associated with subjective, and short term objective symptom improvement, and with subjective improvement in participant quality of life. As to the retention of effects, the reader will recall that the subjective improvement in symptoms reported by the experimental participants at the post-treatment assessment remained at follow-up despite near universal failure to regularly practice the relaxation exercises between the two assessment periods. One might therefore, assume that subjective treatment benefits were retained beyond the cessation of treatment; and consequently, at least at a subjective level, the behavioural approach outlined above complies with the WHO guideline for retention of treatment

effects. (Of course, the apparent retention of subjective efficacy needs to be considered in light of bias inherent in subjective report. One could consider the role of participant need for "pay back" for their and/or the therapist's efforts). Further, the objective effects of treatment were only significant at the post-treatment assessment session. This might suggest that the effects were not retained in the longer term and as such, would not comply with the WHO treatment guidelines. However, the reader will recall that the lack of significant treatment effects at follow up might be associated with lack of practice of the techniques i.e. the equivalent of stopping treatment altogether. As such, it can not be stated definitely that the treatment does not comply with the WHO retention of treatment guidelines.

The issue of safety, side effects, and the possibility of treatment misuse will be addressed together as potential treatment dangers lie with the side effects of treatment. Positive side effects of relaxation training include bodily warmth, peace, calm, sensations of floating, the loss of physical boundaries, and occasionally subjective improvement in other medical conditions. However, such effects might have a negative outcome. Indeed, during the main treatment study, an AT participant, feeling better generally through AT, chose to stop taking prescribed medication for a heart condition[44]. Thus, contrary to the general assumption that even if of no symptomatic benefit, relaxation techniques will not harm, worrying side effects might occur. Lazarus et al (1990) provide an extensive list of documented negative effects - including floating, rapid heart rate, fear of losing control, headache, nausea, intrusive images and thoughts, irritability, guilt, panic, perspiration, numbness, increased tension, feelings of vulnerability, jerks and spasms. Some of these might be attributable to non-specific factors such as a poor client-therapist relationship, or the ambient conditions of the room, others, such as feelings of nausea, might be associated with more physical effects such as "parasympathetic rebound" ie. a sudden shift in autonomic activity (e.g. Gellhorn et al (1963) as cited in Lazarus et al, 1990). For example, intrusive thoughts and general irritability might emerge during training simply because the trainee feels obliged to attend unenjoyable training sessions, or participants may be unable to relax because the room is too light or too dark for their own particular

[44] He was persuaded to continue with the medication.

tastes. It is further suggested that unwanted side effects might simply be uncovered, rather than caused by relaxation training (eg. Jacobsen et al, 1982).

The question of cause aside, negative effects do occur. Indeed, during training, one female AT participant described a fear of the floating sensation associated with relaxation (having experienced negative "floating" sensations as a child), and felt unable to relax completely for fear of losing control. A second AT female experienced panic following shortage of breath during the AT breathing exercise. (She was advised either to avoid the breathing exercise in future, or, to stop immediately she experienced such effects). Lazarus et al (1990) suggest that individuals might respond adversely to certain types of relaxation training. Indeed, AT manuals categorically state that asthmatics should learn AT only under strict medical supervision[45], and Schultz and Luthe (1969) do not advise AT for people with gastrointestinal problems. Therefore, although avoidance of relaxation procedures is not suggested, the need to carefully monitor their use is advised.

To redress the balance a little, it should be noted that a male AR trainee jerked violently during an the early training session, though on questioning, was unaware of his movements, but did describe his experience of a series of pleasant, vibrant colours. Clearly, the subjective nature of judgement must be considered in the evaluation of side effects.

As to the ethical acceptability and cost effectiveness of treatments, commencement of training required University ethical approval. Clearly, therefore, within academic research, the techniques utilised are ethically viable. Further, the only relevant costs within a research setting are those of participant time and energy. In this case, such costs should not prove an obstacle to treatment evaluation as whether the treatment was considered successful or not, most participants reported enjoying the experience, and many offered to take part in any further behavioural investigations.

[45] In asthmatics with small airway obstruction, the airways tend to dilate in response to increased sympathetic innervation; therefore, relaxation, by reducing sympathetic impulses, will actually worsen symptoms (Lazarus et al, 1990)

The final point refers to the applicability of treatments in a range of settings: i.e. can the techniques be taught successfully to anyone ? If, as outlined above, individuals might be more attuned to certain behavioural treatments, personality might prejudice the success of treatment. Thus, Lazarus (1989) proposes a technique termed "*tracking*" in which a client's sequence of emotional responses to stimuli might predict the type of relaxation therapy to implement. For example, those whose immediate emotional response to stimuli is cognitive (rather than image based or sensory in nature), might respond poorly to a somatic relaxation technique such as progressive relaxation. In contrast, where the initial response is sensory, Progressive Relaxation might prove a more suitable treatment. Given participants' acceptance of the stress reduction role of relaxation, anxiety levels are also suggested as predictive of the efficacy of treatment (e.g. Qualls and Sheehan, 1981). Moreover, an equally plausible suggestion is that of locus of control (i.e. whether a person considers her/himself in control of events, or that events are externally controlled by others or the environment). Indeed, Ollendick and Murphy (1977) claim that those with an external locus of control might respond well to Progressive Relaxation as instructions are externally generated (i.e. via cassette tape or the trainer's voice), and, in contrast, that cognitive forms of relaxation might prove more useful in those with an internal locus of control. Though plausible and attractive in theory, there is, as yet, no sound empirical supporting evidence of a role of personality factors. Yet, on a more applied level, the implementation of techniques might improve compliance to treatment simply because perseverance is more likely with a treatment that suits the individual. Clearly, further research is required in this area.

Therefore, the treatments evaluated in this investigation comply with the WHO guidelines - at least within the realm of academic research. However, as the efficacy of the approach in the management of Raynaud's has still not been proven categorically, the following section will outline a range of retrospective thoughts and suggested improvements that might be taken on board in future empirical work in the area.

9.4 GUIDELINES AND RECOMMENDATIONS FOR FUTURE WORK

How might the empirical work of the thesis have been improved ? A number of recommendations have already been made in the course of earlier discussion. These points will be re-stated within a comprehensive list of improvements at the end of the section. However, a further point, worthy of discussion, lies with the need for larger scale studies if the true benefits of non-conventional treatments are to be discovered.

As noted by Simkins (1982) "*Data are not available on a large scale basis to indicate clearly what proportion of the clinical population with a given problem is responsive to this kind of treatment*" (p13). Indeed, in the study described, following patient withdrawal, there were barely enough participants to enable valid statistical analysis. However, even with a statistically viable sample, where small differences in effects of treatment appear (as might occur with the behavioural approach), these might not be picked up unless a very large patient sample is recruited: to detect very small differences, large patient samples are required. Clearly, given the lack of empirical support as to the efficacy of the behavioural approach, grants are unlikely to be available to finance such large investigative studies. However, an alternative means to this end might be to perform a meta-analysis of the relevant behavioural research to date. That sample size and procedures differ across studies, might be overcome through clever use of statistical packages; however, a serious difficulty with the implementation of meta-analysis techniques lies with the disagreement over the appropriate model of biofeedback. For example, a large scale analysis of studies using biofeedback as a treatment in its own right, could provide details only of the large scale effects of a range of non-specific, non-standardised strategies of temperature control i.e. effective strategies could not be divorced from unproductive means of temperature control as strategies of control would be hidden under the guise of biofeedback. Therefore, it must be suggested that if the current approach to treatment is going to continue to be investigated, some standardisation of procedure and theory need be set out and referred to in the design of future investigative studies. Consequently, the true efficacy of the

approach might be outlined through the meta-analysis of a set of methodologically sound investigations.

What might be required in such a sound methodological investigation ? On the basis of this discussion and previous chapters, a by no means exhaustive list of methodological and theoretical improvements to study design is outlined below.

- Adequate adaptation and baseline periods.
- A sufficient sample size to allow for patient diversity, predicted patient withdrawal, and appropriate analysis of results.
- Adherence to predetermined criteria as to the required diagnostic status of participants.
- Single blind studies with long term follow-up to help reduce experimenter effects.
- A move away from the evaluation of effects of treatment in laboratory tests of temperature control, to more "in situ" tests of treatment efficacy in symptom control - requiring refinement of ambulatory monitoring methodology such that implementation of learned treatment skills during attack might be evaluated.
- Adequate controls for seasonal fluctuations during a long treatment study.
- Further research into the possible role of personality factors in treatment outcome - perhaps with a view to a predictive model of personality and treatment efficacy.
- The need to adequately evaluate changes in participant attitude toward treatment, and, where necessary, the introduction of techniques to improve such attitudes such that the skill of relaxation might be appropriately acquired.
- Whilst noting the possibility of a perceived role of stress in symptom outcome, clear statements to the effect that relaxation is a means to a reduction in physiological arousal - not stress reduction.
- The provision of biofeedback equipment for home practice.
- Adequate teaching of the behavioural techniques employed (with tests to that effect).

- Comparative investigations of the efficacy of Classical conditioning and the approach investigated here[45].

9.5 THE ROLE OF THE DESCRIBED BEHAVIOURAL APPROACH IN THE MANAGEMENT OF RAYNAUD'S SYMPTOMS

In light of the discussion and empirical evidence presented thus far, what role might the described behavioural approach take in the future management of Raynaud's symptoms ?

One would assume that relaxation techniques might be of most use in the treatment of mild Raynaud's symptoms, and that the underlying physical abnormalities of Raynaud's Phenomenon conditions will not be improved through such treatment. Therefore, before going on to discuss the future role of biofeedback mediated relaxation for Raynaud's Disease, the reader is reminded that conditions associated with Raynaud's Phenomenon are serious, debilitating and, in some cases, life threatening. The empirical work documented here involved sufferers living and dying with the serious effects of their condition. Consequently, the role of the Raynaud's and Scleroderma Association in providing practical and emotional support for sufferers, and the need for further medical research into treatments for secondary conditions, must be underlined. The behavioural approach may offer emotional and practical support to such sufferers, but only medical intervention might hope to find a cure.

How applicable might the behavioural approach outlined here be within a real world setting ? Clearly, the approach is unlikely to provide a cure for the condition; indeed, at best it might provide the sufferer with a means of coping with symptoms. The approach is likely to give the most benefit to those with mild Raynaud's symptoms - in which case, the mainly negative results for those more

[45] Classical conditioning experiments, such as those outlined by Hayduk and by Jobe in chapter 6, might prove an alternative avenue of investigation. Owing, perhaps, to a large amount of inappropriately supportive research of the efficacy of biofeedback, conditioning research is sorely lacking. Potential questions might include at what stage of Raynaud's Disease might the conditioning approach fail to exert an effect ?

seriously afflicted patients are understandable. But, even within this group, there is positive evidence which could be interpreted to suggest that with regular, sustained practice, Primary Raynaud's patients may benefit from Applied Relaxation in the management of their symptoms.

THE APPENDICES

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APPENDIX 1

Chapter 3: Subjective accounts of Raynaud's (Interview and Questionnaire)

APPENDIX 1.1: The semi-structured interview (guiding questions)

1. Personal details: age, occupation, marital status, children etc.

2. Smoking habits (including smoking habits of family)

3. Duration of condition ?

4. Description of initial Raynaud's attack
   - What were you doing at the time ?

5. Have your symptoms changed ?
   - describe a typical current attack of Raynaud's
   - What does it feel like ?
   - frequency/duration of attacks
   - Do your symptoms prevent you doing anything ?

6. What, if anything, aggravates the condition ?

7. How do you try to prevent an attack ?

8. What do you do to try to stop an attack once it has started ? /Treatments

APPENDIX 1.2: The interview transcripts

1. AP: Tue 22/9/92 (male; 43 years old)

JW: How long have you suffered from Raynaud's ?

AP: Um, Its hard to tell. I think as a child I had the problems with the ears and extremities and so on, but it affects me more in adult life with my feet. I have a complication. When my feet get cold: they tend to sweat, which is unusual, I appreciate. Then of course when they sweat, they get damp. When they get damp they get cold. Its an ongoing thing. A particular problem that I have that's interesting is that I can't wash or have a shower in winter, in the morning because if I lose my body heat, If I stand at a bus stop for 20 minutes on a cold morning, and the concrete is next to my feet, I just get totally cold. If I have a shower it seems to affect me and a bath affects me as well. I just can't regain that body heat. It is sucked out of my feet in particular. So, it takes me about four or five hours to feel warm again..... Unusual, but that is how it is. Another problem that I have is that I am frightened to go anywhere in winter. I will not go anywhere. If I go to a relation's or friend's for a meal, I make discreet enquiries of how warm the house is before I dare to go there. I take with me a bag full of underwear and clothes to wear in case it is cold there.

JW: So how do you cope in everyday situations ?

AP: [*Shows a heater and foot warmer by the desk*] I need personally to be about 70 degrees to be comfortable. Below that I tend to lose body heat and feel very uncomfortable. When people feel my feet they can't feel that they a re particularly cold, but I can so that's how it effects me.

JW: Can you describe exactly how it feels ?

AP: The opposite of a warm burning sensation. In fact it feels very much like a burning sensation. It starts from the toes and goes up my legs progressively. The doctors told me to insulate what she calls the pipe work: wear long johns or underwear or thick socks to try to minimise it. The problem I have is that the

more I insulate, the more I entrap this perspiration, but after a while it makes it a negative thing. So when you are told to keep your feet warm, it does help me, until that point they become saturated, and then it is just a negative thing. If I wear a very thin sock, it can be better than a very thick one in the longer term. OK, so I tend to carry with me at all times umpteen pairs of socks and constantly change them throughout the day. You see I think that in my last employment I had a lino covered concrete floor so I suffered badly. Here, I've got a carpet of sorts, and I'm allowed a large double fan heater, so I tend to regulate my office temperature. I am now going to move to an open plan office....[*phone call interrupts*]

JW: How long does an attack last ?

AP: Until I get warm.

JW: About how long might that be ?

AP: I mean, if I get badly cold, I really do suffer all day, until I get warm somewhere. Um.. yes, that is really the answer. Its temperature for me generally speaking. 70 degrees is my break even point.

JW: Do you still get symptoms in a hotter climate ?

AP: I have not had that. They tell me that on a beach when the wind changes you can turn blue. Drafts do affect me. I mean I can't stand my friends having windows open. That just irritates me. We have to compromise that somehow. If my shoulders are cold that doesn't matter. Its my feet that affect me particularly. I feel happy if I can control the environment, or adjust it - by having prior knowledge about how cold it will be....so I can wear the appropriate clothes. I have to rush off to the toilet and change several times a day. Seriously it can be embarrassing. People get used to it after a while.

JW: Is there anything that Raynaud's stops you doing ?

AP: I think it controls my whole life really. My first aim in life is to be warm more than anything else, rather than to be healthy or rich or.... First thing in the morning I must find a way of getting warm. I'm ok lying down. It seems to be when I am standing up and my feet are on the ground that I have a problem.

JW: Does it help to have a hot bath before you go to bed at night ?

AP: Um.. I'm trying to think. I don't find that useful. I don't suffer indoors because I have the temperature in the house on quite warm. The wife and kids don't mind that so much. If I go elsewhere I find it difficult, and I have tended to spend a lot of time in the bedroom that I can keep warmer than the rest of the house. My main aim in life is to find some way of keeping my feet warm. I can then attempt to do other things.

JW: Do your wife or children have similar symptoms ?

AP: One of my daughters does feel the cold in her feet. But we haven't really... um, I suppose not as seriously as me. They do feel the cold, one of them does.

JW: Have you ever smoked, or have you ever lived with anyone who smoked ?

AP: I have smoked. Yes, I'm ugh.., I smoked in my twenties I guess really. Two or three years I guess really.

JW: Did it seem to be worse then ?

AP: I can't remember. I haven't smoked since I was 23, so that's going back 20 years. I'm Mormon, I don't smoke or drink, so that... I can't remember back that far really.

JW: Does anything else aggravate your condition apart from the cold ?

AP: Well, we have mentioned the drafts so um... People say that stress affects them, but I don't generally find that I'm aware of that. Just drafts, temperature change, and the cold.

JW: What sorts of treatments have you tried ?

AP: Everything that is legal I suppose !! I have tried some pills from the doctor. My doctor is quite good about it. I have tried some pills, but had a very minor effect. I mean, I think the pills made it about a quarter less worse, but we felt that it wasn't worth continuing permanently for that small benefit really.

JW: Did you get side effects ?

AP: Um... I used to get burning ears. Because its a small part of the body, lots of blood was going through faster, and you got a tingle in your ears. I did try to take half the dose. They say that it would have gone eventually, but it wasn't worth the effort.

JW: Is there anything else that you'd like to add ?

AP: I think that the thing I thought about the last 3 weeks was the fact that I really am terrified to go any where. I take with me... If I go in the car, I'll take jumpers and coats and socks particularly, and I'm very unhappy about going anywhere that I can't control or have items with me that I can add to or take off to cope with the situation. I have tried electric gloves and things. Someone lent me some stuff, but I found that the weight of the batteries was a problem really. I think I'd rather rush off to the toilet every hour and appear to be constipated, and change the socks than to carry the big batteries around and look an imbecile. Its a shame really. Also, you use the word "attacks", but I don't really have attacks. I get a constant attack from September to May I suppose really. The problem I have is not winter. The problem is Autumn and Spring. In winter I can normally wear clothes or control the environment because people have heating on. Whereas in the late Autumn or early Spring like now, when the heating is not on, that I suffer most really. I want an air conditioned world. More global warming please.

2. JM: Wed 23/9/92 (female; 23 years old)

JW: Do you or have you ever smoked ?

JM: For about a week once. That was years ago though. Only when extremely drunk do I have a cigarette I think.

JW: Do you live with anyone who does smoke or has smoked ?

JM: My Dad smokes, but it isn't very obvious in the house. He smokes roll ups, so its not as bad as normal cigarettes. My mother doesn't. She used to, but that must be 10- 15 years ago, so... I work with smokers. Its not on the shop floor, its not in the staff room. I will pass through it, but not for any length of time.

JW: Do you find that the condition gets worse in a smoky environment ?

JM: Well, no, the amount of time I'm exposed to it, you wouldn't be able to put it down to that. Its more the fact that I work in an extremely cold environment, and its extremely noticeable then.

JW: For how long have you suffered from Raynaud's ?

JM: Um, almost exactly a year. But I don't know whether I've always had it. Its all a bit strange. It came about obviously very suddenly. I have always had quite bad sort of hand colour. But it started off with er... I had like what I thought was an ingrowing finger nail. Its only in one finger you see. It got very inflamed, and then it moved around to the top of that finger [*points to middle finger of right hand*]. It started going a bit yellow looking, so I only mentioned it off hand to the doctor, and he sort of took a look at it, and said pack your bag, you are going to hospital. OOOh, my God !!! Um, er, you can probably still

see it. Its just in the end. It actually developed like a complete hi****, its extremely painful, and ever since then, it has been cold all the time - just that finger. It warms up very occasionally. The blackness is in that one, that one and my thumb. [*points to middle and index finger of right hand*] But they do go the most bizarre colours. My Mum has what we call bad circulation. They do go very black. Its quite frightening I have to say.

JW: So is it only that hand that's affected ?

JM; Yeah, I have got it on this hand too. Its sort of down there as well [*points to index finger of left hand*], and you can probably see that there is a bit of a dodgy nail. But there just come with cold hands. I've just always had them.

JW: What sorts of thing do you do to try to stop attacks happening ?

JM: Oh, there is nothing that I can do that I know of to try to stop it. Um, I mean sort of wiggling my hands about won't do any good. I mean I have been to see Doctor X. at the hospital and he said its not primary Raynaud's, its secondary (whatever that means !). He seemed to suggest that there is really nothing he can do about it. I have asked him "should I do exercises or whatever ?", and he said " not really because its not really a circulation problem that I have got." He can't explain it. Its there, and I have got to learn to live with it. I mean I can't wear gloves all the time at work. I can't put my hands under my arm pits or anything. When you are working in a shop, you just can't do it. I just live with it really.

JW: Are you able to take a heater in, or anything ?

JM: No, that is a bone of contention at the moment. I mean we haven't got adequate heating in the shop. Really I should change jobs. That's the bottom line. I don't see why I should let it affect my life that much. Um, we work at the counter in the shop. It is right by the door, and I can't sort of huddle round the heater. I just can't do it. I can't wear gloves. There's no facility for doing that so its just a case of living with it.

JW: Does it actually interfere with your job at all ?

JM: Um, sometimes I find that that thumb or both thumbs they will go. They just literally haven't got any blood in them. They are white and I keep going like that (*rubbing them with index finger*), and if I am writing out a credit card or doing any writing, it just feels very odd. But mostly its just incredibly unsightly - people look at my hands and go ugghh ! You know, they go so black. Its more embarrassing rather than anything. When that finger was inflamed (*right middle*), if I touched the end, it was just like you know, I was going up the wall. I mean if you touch the very end it is a bit sore now. It's just a bit funny looking. That's all. That really is the problem.

JW: Does that worry you ?

JM: Well, no, I mean not especially. It does when they go completely black. Its more worrying because I think well what is that doing to me ? I don't really mind what people think: if I have black hands I have black hands. People just think they are dirty or something, but I mean to me it is worrying for them to go that black - they do go extremely purple colour. And when I take a shower or something they'll go red or purple, and I have multicoloured hands - orange as well. Um, you know that can't be good for you. No, its not what they look like really.

JW: Does it actually stop you doing anything ?

JM: No, not at all.

JW: Do you remember what you were doing the first time it happened ?

JM: It would have happened in October, so it was getting towards being cold. No, I mean it just all stemmed from this which I thought was just an injury. I just thought I'd hit my finger and it just started going sore. I wasn't aware of the coldness then. Um, you see I didn't think anything of it. You don't when

you get an infection in the side of the fingernail, you think well you don't go to the doctor about it - you just leave it. When it worked around to the top, and if I touched it or bashed it at all, it was really, really painful. As I said, I just mentioned it in passing. And he sort of said have your hands always been like this ? This black ? So I put them in hot water, and they were going all sorts of different colours. He said he thought I had frost bite. Then I was taken off to the Royal South Hants, and they said well we'll give you a few blood tests here and there. But I mean it was a bit strange actually. I have a feeling or thought that it may be something to do with..., maybe triggered by an emotional thing because it was almost exactly to the day that I'd split up with my boyfriend after two years. I was in a very, very bad emotional condition. Extremely distressed, and that is when it happened. Almost exactly. That's how I know how long ago it was. So, I thought there was a connection to that.

JW: Do you find that you are stressed when it happens now ?

JM: Oh, yes. Its definitely linked. It definitely is a stress. Any sort of emotion, it will go..um, whenever I go and see the specialist, and I'm het up, I'm like [*stressed gesture*] and they go black in front of him, and he's going wow, and looking at them. Its definitely, apart from being related to cold, it is linked to stress, I think, to emotion. They go all sorts of colours. I mean they are going all funny now. Its literally in my hand, I can feel all the blood running out of it and it go all like freezing cold. I get the shakes as well but, so its definitely linked to that, I think. That's why its strange that it should happen at exactly that point you see. ..... Whenever I go and see doctor X, he's getting results of blood tests, and you feel like cos they push you from pillar to post, and I had to have ... what are those things called ?... Barium meals, and they were trying all sorts to find out what it was. And Doppler scans as well, and you don't know what is going on, and even now he can't tell me what it is. Anything that is an unknown quantity, you are just worried to death about. And because he is like, I mean I find him a very difficult man to get on with, so he stresses me out even more, and I find he has no answers. He has a tendency not to expect his patients to ask - to sort of go with the flow so to speak, and I find that I want to know. If anything, you want to know, what is the worst case, and how will I know if it is getting worse. He said well, you'll know. I can see the point of view: he doesn't want to put the thoughts into my mind, so that I'm looking for such and such. But when he came back and said its not primary, its secondary, I said what does that mean, he said well it could be covering something else such as cancer, and you think "Oooh, Oh my God, I have got cancer and I'm going to die". He said he'd retest my blood every three months or whatever, and tell you that you've got slightly elevated levels of such and such, or slightly decreased levels. Of course, this is all Greek to me. You know, I don't know what any of this means. And so they were literally changing colour in front of him. But it is a stress thing definitely.

JW: Has the condition worsened in the last year ?

JM: No, I wouldn't have said so. Apart from...this finger is cold all the time [*right middle*] - it very rarely warms up. It's now in the thumb of the right hand and two fingers of the right hand that go the most predominately black. But, no, it hasn't spread. I mean, I suffer from cold feet - a lot of women do, but its obviously much easier to keep your feet warm, so... you don't really notice, and I don't get cold anywhere else really. I find that my body temperature swings a lot. Once I have got cold, I mean that is it. Nothing will make me warm, or next thing I know, I'm really really hot. It's a bit strange that it will swing about like that. But I wouldn't say that it has got noticeably worse. Its not really in this hand [*left*]. I mean, I get discolouration down there that you'll see sometimes, but no, I wouldn't say that it has got any worse. Its about the same. The doctors did the original blood test, and then did a blood test after that which was the last one I had and the levels which were a bit off, had come down. I assume that I have got better. But I don't know. The doctors don't see it in action. They only take my word for it. And I don't know what their testing for.

JW: Can you just describe exactly what Raynaud's feels like ?

JM: It doesn't really hurt. In fact, the initial injury, which was what Doctor X. said, a lot of people suffer from it, but don't ever go to the doctor about it, just live with it. This initial injury was what showed what was beneath it. That was excruciatingly painful, but now it doesn't hurt. Its just that its cold, and I get a bit of a buzzing sensation. I don't feel anything really. I haven't lost the feeling in that finger, but it is a bit ... I have got like a kinda like scab thing on the top of it. My nails grow. My nails haven't stopped growing.

I have seen sort of...working in a book shop, I have seen lots of medical dictionaries full of horrible finger nails and things of Raynaud's sufferers, and say "Oh dear, I'm well off with that". It doesn't hurt at all. They just go absolutely icy cold, and people will shake my hand and go "UGh ! She's dead". Dead hands, but they don't hurt.

JW: How often do they go "funny", and how long will it last ?

JM: During the summer, I suppose that apart from the fact that the middle finger is cold all the time, and I have just come back from being in Gran Canaria for two weeks, and before I went, he said that there is really nothing you can do, but he had a patient who went to live abroad to help the condition, and he said that being in a hot climate doesn't help it, but I found that it did. It was a bit.... Going swimming would lower the temperature of my hands and they would go bright red, but actually being in that environment, it does actually warm up. It just doesn't stay like it very long. It'll be like it for about half an hour or an hour, and then it will go back to being cold again. But at work at the moment, during the summer, it really is only linked to stress. Say I'm getting hassle at work, or moments of emotion, and it'll be almost instantaneous, it'll go from being warm to being cold just like that. And then it'll last I suppose if I just left them for as long as that feeling lasts. I sit on my hands a lot. I always carry a pair of gloves just incase I need them. And that will bring them round quite quickly. In the winter, it is quite difficult to remember. But, obviously last winter, I was still suffering from this on the end of my finger, but they were cold all the time. And putting them in hot water or something - not the best thing to do, but they will change colour, and they will warm up. So that's a way to get them to warm up. I tend to find that I do spend a lot of time sitting on my hands. I get them to come round. I put them over heaters and things during the winter, but its... sometimes, I just can not get them warm. They will just stay like it all day. And that middle one is just cold all the time. Its a shame really... I can't sort of give time scales.

JW: Is it worse at any particular time of the day ?

JM: Um, not really, I mean at night, you tend to be indoors or in quite a warm environment. But during the day, I am at work, and work equals cold basically. So it is worse in the day. I don't wake up with cold hands. The middle finger may be cold, but as a rule, they are quite toasty feeling. When they warm up, you really feel the warmth. Its not like someone would say I have normally warm hands. You are actually aware of it. Its like when they are cold. I like move them around, and you can feel this cold air moving around with them. But no. When I had the initial sore, I felt towards the end of the day, it was just throbbing - so painful. I was getting like pain shooting up my hands, but that was only when I had that. I don't get that now. One thing that I have noticed that I have told doctor X, and he is going to do an experiment on it (!!), is that if I have been out for the night, I don't drink heavily because I tend to drive, but when I do, if I go out for a major drinking session, next morning, it is so extremely painful. They are like.... they are swollen up - if I try to bend them, you feel like you can barely do it you know. So, you'll have to get me drunk to see !!! Also, I have just started doing... I'm not a particularly active sort of person, I don't consider myself a particularly healthy person, but I have started doing aerobics, and I find that when I'm doing that it is really really like ice, it just goes incredibly cold. And they will go quite a bizarre colour then. But only once again in the one finger. Only in extremes of cold, or I'm extremely het up that the whole hand will go. When I do lots of exercise. When we go out to a dance or something, doing like a lot of dancing or something, then it will go quite quickly very, very cold. But its not an emotional thing at that point. Its to do with exercise. The blood is pumping. Its not like I get stressed when going to aerobics with other people to jump up and down for an hour. Its to do with the way the blood is going.

JW: Does anything else aggravate the condition apart from exercise, emotional situations and cold ?

JM: No, not really.

JW: What treatments have you tried ?

JM: I have discussed this biofeedback thing with Doctor X. But nothing yet has come of that, so I don't know if I could cope with that anyway. I mean, we have thought about trying to do some meditation or something like that just to get the stress under control, to actually try to alleviate that side of it. Its actually

a bit strange, but in 1990, I wasn't diagnosed as having it, but I suffered from an extreme attack of stress, or what they said was stress, and which a homeopath actually diagnosed as M.E. and was very ill for a year. I feel like I'm getting back on form now. I dropped out of University cos I was over stressed, and I couldn't ... my mind went almost completely. I guess it was borderline to a nervous breakdown. So, I don't cope well with stress anyway. I have a very low tolerance to it, and my emotions will swing a lot anyway. So, we thought maybe if I tried something like Yoga, or sort of meditation, or alternative health things, that might help it, but I haven't really got round to it, I have to say. It doesn't really appeal -Yoga, and I tried meditation whilst I was at University, but my mind wandered off, and that was the end of that really.

[*Pause*]

I'm a bit concerned. Has the blood stopped running to the top of my finger, or why hasn't it dropped off or withered or something ? The nail hasn't stopped growing, although they do tend to break off a lot. My nails, I think, are weaker since I have had this, and cos I used to have quite long finger nails, and they are always splitting at the side, or whatever. There is nothing more I can say really. The only time I can guarantee that they are going to go is in an emotional condition. I mean, now like, parts of me a really hot, and parts of me are really cold, which is really odd. But I will just have to learn to live with it. At work sometimes, I get...., in a stressful situation I get hot and bothered, yet my hands go absolutely like ice, and I'll be like holding a pen. I have found if I am writing for any length of time, and I tend to grip the pen quite hard anyway, then I get sort of huge dents. I go like that [rubbing with index finger] and it won't reform, and the blood won't come back into my fingers. So that's another thing. If I am using my hands for anything, then they will go then. It doesn't really bother me. Its just unsightly. It's embarrassing. People shy away because I have these cold clammy hands. It doesn't hurt at all.

JW: What sort of things do you do when you are stressed ? How do you cope ?

JM: It doesn't really trigger anything. I don't sort of reach for a cigarette or alcohol or food. A lot of the stress I suffer at work, you really can't do anything because obviously you are serving the general public, and there is a lot on. When work is mounting up, I tend to relieve it by shouting at people, snapping and being really obnoxious. I don't really get very stressed at home. I tend to find that ... that now I mean, I don't know if there is a link with this, I don't know how many people recognise M.E as a complaint or syndrome or whatever, but when I had that really bad, I couldn't cope with it, and I was actually seeing a psychiatrist for a year, but that was only ten minutes a week. It wasn't like mainstream discussion with him for an hour and a half or something. And I have found that it was a very mental thing. I couldn't cope with it. It was very difficult to cope then. Now, having been through that for a year - six months of which I couldn't work for, I can get on top of my emotions and stress. I sit on my own and like chill out. It sounds like a very contrived thing to do, but I can do, I can get on top of it. I can think myself out of it. That's if I get into an emotional state at home - my parents rowing or something. Then I find that I can actually go away, play some loud music or something, and deal with it and get beyond it. And so yes, stress in the home environment, I can get around it simply by taking a lot of deep breaths and thinking my way around it. At work, it will come as stressful... It will build up, but you will have to work through it because you can't go away. You can't sob in the kitchen. You have to deal with it there and then on the shop floor. You can't just go and shy away from it. Now, its just confronting the stress and dealing with it in that respect. I have been setting myself little challenges to see if I can deal with more stressful things. When a whole part of your life has been affected by what they term stress, you tend to think well, I don't want to do anything if it is going to get me back there again. But, now I set myself little targets of going into sort of what I term dodgy situations. I think I'm more on a level now than I have been for quite some time. And now I go and get this don't I ? I can't believe it !

3. <u>HC: Fri 25/9/92 (female: 41 years)</u>

JW: Do you recall your initial Raynaud's attack ?

HC: I was involved in a car accident on 1/10/87... seemed to be alright at the time, next day had a stiff neck, couldn't move my head or right arm. They said I had a whip lash injury. By December 1987, I began to notice colour changes in my face and hand - going from white, blue to red, and was getting severe pain and funny sensations in the right side of my head, face, tongue, nose, right ear, eye, arm hand and fingers. The pain in my head was so severe, I thought something was going to burst. Also, I couldn't bear the right side of my head and right arm to be touched or knocked.

JW: Have you been diagnosed as suffering from Raynaud's ?

HC: I saw a Rheumatologist in February 1988, who diagnosed Raynaud's Phenomenon. He said it wasn't caused by Rheumatism or Arthritis. He said it was vital to keep warm. He never explained to me what the symptoms of Raynaud's are, or gave me any information about Raynaud's.

JW: Can you describe a typical Raynaud's attack ?

HC: I feel nauseous and tired and have to get my head down. I feel awful. Cold affects me. Using my right arm... any pressure on my right arm sets off some kind of reaction causing terrible pain and discolouration. I don't feel that the doctors take my Raynaud's seriously enough because it just affects my right side. They think it is unusual. The number of times I've been told that Raynaud's either affects the hands or the feet, and not the right side like me. I have no problems on the left side. Sorry, anyway, I also find the constant pain makes noises sound out of proportion, and make me act like a raving lunatic - even the silliest things make me shout at my family. I really feel as if I'm going mad. I find it hard to cope with meeting people..... I feel I've spoilt so many evenings, I don't go out much now.... The doctor doesn't how bad the pain is. I always end up feeling desperate after seeing him.

JW: What medical treatments have you received ?

HC: I was referred to a specialist in 1989 who arranged further investigations. I had an MRI scan done in London, the main nerves tested in Newcastle (which showed nothing), and I had an angiogram done which showed the subclavian artery was being compressed by my right cervical rib. I had the right cervical rib removed in 1989, after which I felt more like my old self. However, a month later, the pain started again. I began to feel tired, and I had to give up my job due to tiredness, and not being able to cope. I was having trouble getting change out of the till with my right hand: my fingers went numb and I dropped things. I lost my confidence.... *[pause]* I'm afraid I don't know how much of my pain is caused by the Raynaud's or by the whip lash injury, or by the stress of the pain, the scar tissue, muscle spasms or depression - I suppose that all of those things complicate things.

JW: What effects do your symptoms have on your lifestyle ?

HC: I have found it hard to cope with the frustration of not being able to do things I used to do like knitting, dress making, decorating, painting, gardening and baking - they all cause too much pain in my neck and shoulder. Even walking can make my arm feel like its being pulled out of the socket. It feels like a ton weight and my hand goes blue. Um.. the attacks are worse in winter, or if I have to go for a medical examination, as soon as I get home, the stress of it all brings on an attack. I feel sick, dizzy, and my nose and hand and ear will go blue and my face very pale. this feeling can last for hours, days or weeks. When the pain is really bad, I get a very bad flaking scalp and sometimes shed skin all over my body. The doctor said it is a nervous reaction. Also, if I jerk my neck, that causes the muscles in my neck and shoulders to go into spasm, and that can bring on an attack. Sometimes, I can do more than others. Sometimes, I am more sensitive than others. I lose the use of my arms and hand, can't hold anything. My hand goes into spasm at times, which is very painful. Sometimes, it goes bluish and curls up and feels numb, and I can't straighten my fingers. Sometimes, even when the colour is normal, I can't stretch or straighten my hand. I worry that I'll lose the use of it permanently.

JW: What methods have you tried to control your symptoms ?

HC: I have tried acupuncture and hydrotherapy, but they brought on Raynaud's attacks. I tried a TENS machine, but within a few seconds, it started to stimulate the nerves. The pain was horrendous, indescribable. I have tried two types of drugs to help Raynaud's, but they opened the blood vessels too much, and I couldn't cope with the side effects. I feel better now thanks to relaxation classes... All the doctors I've seen can do nothing. I just have to cope with it best that I can. The only help I've had is from a chronic pain group. I have found it a great help to meet others in the same situation. Also, I've noticed that when I'm on holiday with nothing to do, I'm a little better.

4. RP: Mon 28/9/92 (male: 66 years old)

JW: For how long have you suffered from Raynaud's ?

RP: I was diagnosed in 1981 as having Raynaud's Disease, but with the benefit of hindsight, I realise that my symptoms began to appear in about 1978. I was put on Adelat soon after diagnosis. Late 1982, I was made redundant after 42 years. This was a very stressful period, and made my symptoms worse. During 1983, my Raynaud's symptoms were worse than they have ever been before or since. Despite wearing good gloves, my fingers were often extremely painful, and I frequently had spasms in my fingers and toes which left the affected nails very sensitive and painful.

JW: How do you try to control your symptoms ?

RP: My symptoms have been much less serious in the last few years. I guess its a combination of reduced stress (I was divorced in 1987 !) and the medical treatment I receive. I cannot remember the last time I had a spasm in my fingers, and its many months since I had one in my toes. I think that the stress of my redundancy and the subsequent domestic stress aggravated the basic symptoms of my Raynaud's Disease.

APPENDIX 1.3: The Raynaud's Questionnaire

This questionnaire has been constructed to help us gain a better insight into the life of a Raynaud's sufferer.

We hope you will be willing to complete all sections: Your answers will be completely confidential. However, if you would prefer not to respond to a particular question or section, for whatever reason, please do not feel pressured into doing so.

Thank you for taking the time to fill out this questionnaire.

CONSENT FORM

I, _____, understand that my responses are completely confidential, and that if I do take part, I am free to withdraw at any future time. I would/would not* be interested in taking part in a Raynaud's treatment programme, although before making up my mind, I would like to receive more details about what this would involve.

* Please delete as applicable.

Signature _____ Date _____

Section 1: About you and your Raynaud's

1. Name: _____

2. Date of birth: _____ /_____ /_____

3. Sex: M/F [Please circle]

4. Which of the following best describes your marital status ? [Please circle]

a) Single b) Married c) Co-habiting d) Separated e) Divorced f) Widowed g) Other

5. Which of the following best describes your occupation ? [Please circle]

a) In full or part time employment b) Working from home c) Housewife(husband)/Mum d) Retired e) Unemployed f) Student g) Other

6. Has your occupation changed since your initial Raynaud's attack ? [Please circle]
No Yes

If yes, is this a consequence of your Raynaud's symptoms ? [Please circle]
Yes No

7. What type of Raynaud's do you suffer from ? [Please circle the appropriate letter]

a) My Raynaud's is medically diagnosed as Raynaud's Disease[1]
b) My Raynaud's is medically diagnosed as Raynaud's Phenomenon[2]
c) My Raynaud's is medically diagnosed, but I am not sure as which type.
d) I am undiagnosed

8. When (if applicable) were you first diagnosed by a doctor as such ? _____

9. Which areas of your body are affected by Raynaud's ? [Please circle all that apply]

a) Fingers b) Hands c) Feet d) Internal Organs e) Tongue f) Ears g) Nose
g) Other: Please state _____

10. Have your Raynaud's symptoms always affected the same areas of your body ?
Yes No [Please circle]

If no, how have your symptoms changed ? _____
_____
_____

11. Please describe a typical Raynaud's "attack", including any skin colour changes that you might experience. _____
_____
_____
_____
_____

[1] Raynaud's Disease, which is also known as Primary Raynaud's, is not associated with any identified underlying disease or disorder.
[2] Raynaud's Phenomenon, which is also known as Secondary Raynaud's, is associated with an identified underlying disease such as Scleroderma.

12. How long does a typical Raynaud's attack last ? _____

13. Please estimate the number of Raynaud's "attacks" that you suffer per day:

a) in the winter _____ b) in the summer _____

14. Which, if any, of your close relatives suffer from Raynaud's Disease or Phenomenon ?

_____

_____

_____

Have they been medically diagnosed as such ? [Please circle]
Yes No

Section 2: Triggers of your symptoms

15. How old were you when you experienced your first Raynaud's attack ? _____

16. Can you recall your first Raynaud's attack ?
Yes No [Please circle]

If you can recall your first Raynaud's attack, did any particularly significant event occur in the months preceeding that attack ? If yes, please give details below:

_____

_____

_____

_____

_____

_____

_____

17. Which, if any, of the following <u>can</u> trigger your Raynaud's symptoms ? [Please circle]

a) Cold weather b) A sudden drop in temperature c) A sudden <u>unexpected</u> drop in temperature
d) "Stressful" situations e) A combination of cold and "stress"
f) Others: Please state _____

If you feel that "stress" is involved in your attacks, please give example(s) of stressful situations that might trigger your attacks.

_____

_____

_____

_____

_____

18. If you have circled more than one trigger in question 17 above, which is the most common cause of <u>your</u> Raynaud's attacks ?

_____

_____

_____

_____

19. Have the circumstances that trigger your Raynaud's symptoms altered in any way since you initially became a Raynaud's sufferer ? [Please circle]
a) I do not remember b) No c) Yes

If they have changed, please describe how they have altered.

20. Can you ever tell that a Raynaud's attack is going to occur before you experience the physical symptoms ?
Yes No [Please circle]

If Yes, how can you tell that an attack is about to start ? What are the signs or feelings associated with the onset of an attack ?

If No, when are you first aware that an attack has started ?

Section 3: What are the effects of Raynaud's in your life ?

Please write as little or as much as you like about the effects of your Raynaud's.

Continue overleaf if necessary.....

APPENDIX 1.4: The Raynaud's Questionnaire (Coding frame)

Section 1: About you and your Raynaud's

1. Name:

2. Sex:
1 = male
2 = female

3. Marital status
1 = Single
2 = Married
3 = Co-habiting
4 = Separated
5 = Divorced
6 = Widowed
7 = Other

4. Occupation
1 = In full or part time employment
2 = Working from home
3 = Housewife(husband)/Mum
4 = Retired
5 = Unemployed
6 = Student
7 = Other

5. Change in occupation since initial Raynaud's attack ?
1 = No
2 = Yes (not because of Raynaud's symptoms)
3 = Yes (because of Raynaud's symptoms)

6. Diagnosis
1 = Raynaud's Disease
2 = Raynaud's Phenomenon
3 = Unsure
4 = Undiagnosed

7. When first medically diagnosed ?
0 = Never
1 = Within 1 year of symptom onset
2 = A year or more after symptom onset

8. Areas affected by Raynaud's
1 = Any or all of fingers, hands, feet (FHF)
2 = Any or all of fingers, hands, feet and other extremities (FHFE)
3 = Any or all of fingers, hands, feet, other extremities and involvement of internal organs (FHFEIO)
4 = Other

9. Have Raynaud's symptoms always affected the same areas of the body ?
1 = Yes
2 = No (symptoms have spread)
3 = No (symptoms have diminished)

10. Symptoms
0 = No details
1 = Vasospastic attack (white -> Blue -> Red)
2 = 2 colour changes in the skin (WB, WR, BR)
3 = 1 colour change (W, B, R)

11. Attack Duration
0 = No "attacks" as such
1 = Until warm
2 = up to 10 minutes
3 = up to half an hour
4 = up to an hour
5 = up to 2 hours
6 = 2 or more hours

14. Daily attack frequency in Winter/Summer
0 = 0 attacks
1 = 1-4 attacks
2 = 5+ attacks
3 = No attacks per se

15. Raynaud's in relatives
0 = No response
1 = No relatives with Raynaud's/Unsure
2 = Yes (not medically diagnosed)
3 = Yes (medically diagnosed)

Section 2: Triggers of your symptoms

16. Onset Age
0 = Can't recall
1 = Childhood
2 = up to 40th year
3 = 40 years +

17. Events preceeding initial attack
0 = No response/events noted
1 = In childhood/can't recall
2 = Hormonal factors 9eg. puberty, pregnancy)
3 = Injury/illness
4 = Trauma/stress
5 = Vibration
6 = Chemical factors

18. Triggers of Raynaud's symptoms
1 = Cold weather
2 = A sudden drop in temperature
3 = A sudden unexpected drop in temperature
4 = "Stressful" situations
5 = A combination of cold and "stress"
6 = Others

19. Most common trigger
1 = Cold weather
2 = A sudden drop in temperature
3 = A sudden unexpected drop in temperature
4 = "Stressful" situations
5 = A combination of cold and "stress"
6 = Others

20. Change in triggers
1 = No/do not recall
2 = Yes (now include factors other than cold)
3 = Yes (fewer precipitators)

21. Prediction of attacks
1 = Can't predict
2 = Yes can predict

Section 3: What are the effects of Raynaud's in your life ?

1 = No response
2 = Description of Raynaud's symptoms
3 = Social effects
4 = Practical effects
5 = Psychological effects
6 = Combination of above

APPENDIX 2

Chapter 8:
A behavioural approach to the management of Raynaud's symptoms

APPENDIX 2.1: The Ambulatory Temperature Monitoring Diary

A. What is the time ? _____

B. Where have you been during the last hour ? Please tick any of the following that describe your location during the last hour, and please state when exactly you were in that location.

An example. Perhaps you were in a warm building (your office) from 5-5.30pm, walked home in mild weather between 5.30 and 5.45pm, spending the time between 5.45pm and 6.00pm inside a warm building (your home) ?

LOCATION	TIME PERIOD(S)
☐ a. Inside: warm building	5.00-5.30; 5.45-6.00
☐ b. Inside: cold building	
☐ c. Outside: wintry weather	
☐ d. Outside: mild/warm weather	5.30-5.45
☐ e. In a cold motor vehicle	
☐ f. In a warm motor vehicle	

C. During the last hour, what were you wearing ? Please tick, and as before, indicate when you were wearing what.

LOCATION	TIME PERIOD(S)
☐ a. Just indoor clothing	
☐ b. Outdoor coat	
☐ c. Warm gloves	
☐ d. Chemical/electrical thermal aids	

D. Did you experience any "Raynaud's symptoms" during the last hour ? Yes/No

WHAT SYMPTOMS ? **TIME OF SYMPTOMS ?** **DURATION OF SYMPTOMS ?**	**HOW SEVERE ?** 1 = The least severe attack that you have ever experienced 7 = The most severe attack that you have ever experienced
	1-------2-------3-------4-------5-------6--------7
	1-------2-------3-------4-------5-------6--------7

E. <u>What were you doing during the last hour ?</u> Please write down <u>all</u> of your activities of the last hour (e.g. eating, writing, gardening, going to the toilet...), and the approximate times that you were engaged in them.

ACTIVITY	**TIME PERIOD(S)**

F. How important to you were the activities of the previous hour ?
[please circle the appropriate number]

1----------2----------3----------4----------5----------6----------7
(not at all) (very)

G. How important to other(s) were your activities of the previous hour ?
[please circle the appropriate number]

1----------2----------3----------4----------5----------6----------7
(not at all) (very)

H. How satisfying were the previous hours activities for you ?
[please circle the appropriate number]

1----------2----------3----------4----------5----------6----------7
(not at all) (very)

I. How well did you cope with your activities during the last hour ?
[please circle the appropriate number]

1----------2----------3----------4----------5----------6----------7
(not at all) (very well)

J. Were you frustrated during the last hour ?
[please circle the appropriate number]

1----------2----------3----------4----------5----------6----------7
(not at all) (very)

K. Please rate your level of depression during the last hour.
[please circle the appropriate number]

1----------2----------3----------4----------5----------6----------7
(nil) (very high)

L. Please rate your level of anxiety during the last hour.
[please circle the appropriate number]

1----------2----------3----------4----------5----------6----------7
(nil) (very high)

APPENDIX 2.2: The Training Session Hand outs

A. AUTOGENIC TRAINING:

The primary symptoms of Raynaud's Disease are three colour changes in the skin and associated pain and numbness. Firstly, the peripheral blood vessels close, preventing blood flow to the affected area. This causes the initial **white**ning of the skin. If blood flow does not return rapidly, the trapped blood releases all of its oxygen to the surrounding tissues, and consequently, it and the surrounding tissues turn **blue**. Once the blood flow returns, the area **red**dens as large quantities of blood (carrying with it a vital oxygen supply) flood back into the affected area.

The opening and closing of the peripheral blood vessels is controlled in the main by the **Sympathetic Nervous System**. When there is an increase in sympathetic nervous activity, the blood vessels close. In contrast, when this activity decreases, the peripheral blood vessels open up, thereby increasing blood flow.

1. Why Relaxation treatments ?

Through relaxation treatments, one is able to exert some control over the sympathetic nervous system i.e. the very system that it is believed to bring about the primary symptoms of Raynaud's. When we are physically tense, the activity of the sympathetic nervous system increases; thereby reducing peripheral blood flow. In contrast, when we are deeply relaxed, sympathetic nervous activity decreases, which increases blood flow to the hands, feet, nose and other peripheral areas of the body.

2. What is Autogenic Training ?

Autogenic Training (AT) was developed by Dr Johannes Schultz in the 1920's as a form of self-hypnosis to relive tension, fatigue and psychosomatic disorders (i.e. disorders for which there is no identified cause). Nowadays, it is used by people form all walks of life to achieve a state of deep relaxation. It involves the frequent repetition of a series of mental exercises which focus conscious attention inwards, thereby relating the mind to the body. The sensations experienced include feelings of warmth and heaviness in the limbs, calmness of the heart, abdominal warmth, regularity of breathing, coolness of the forehead, and warmth at the sites of Raynaud's symptoms. The sensation of heaviness experienced is associated with deep muscular relaxation, and that of warmth with the opening up of the blood vessels of the extremities.

When learning the skill of Autogenic Training, it is important to reduce external distractions. Thus, the initial training and practice should take place in a quiet room. Moreover, specific postures are employed which reduce sensory input from the environment and from the body [see below]. Once the skill of AT has been acquired, external distractions are not such a problem, and as such, rigorous attention to AT positions are no longer a priority.

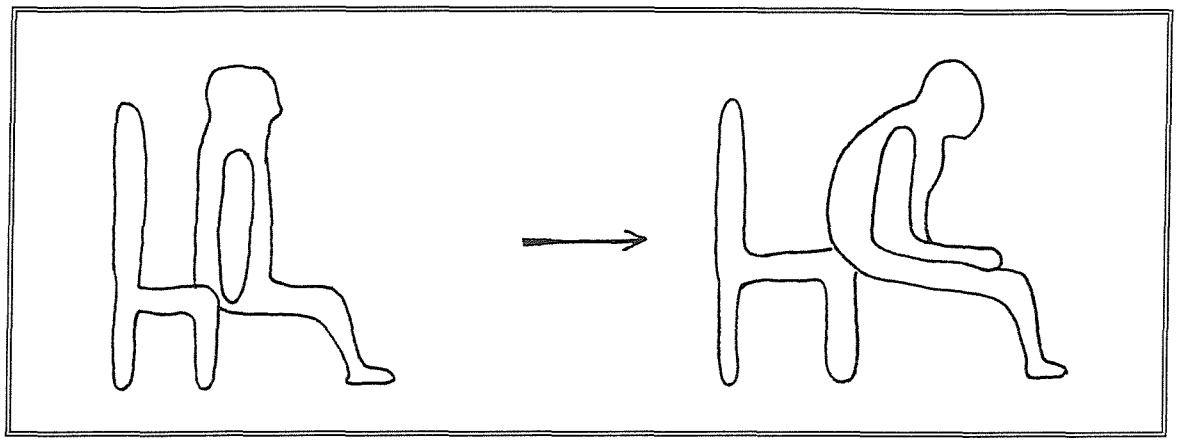
The positive effects of Autogenic Training are often slow to emerge; therefore, the trainee should not be discouraged by nay lack of instant results. Through continued regular practice, the benefits of AT will appear.

Session 1: Short Stitch and Heaviness # 1

1. The basic training positions

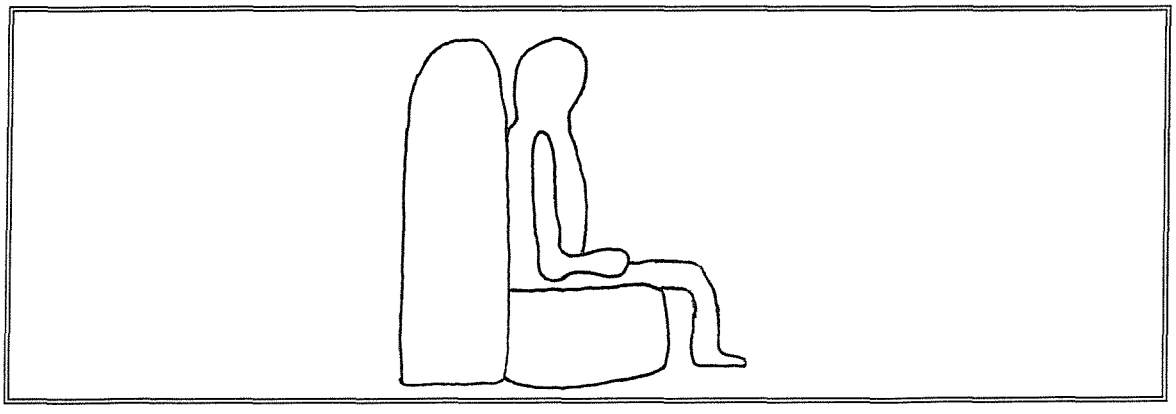
There are four standard positions for AT, andy of which may be used when practising the exercises.

a) **The simple sitting posture**



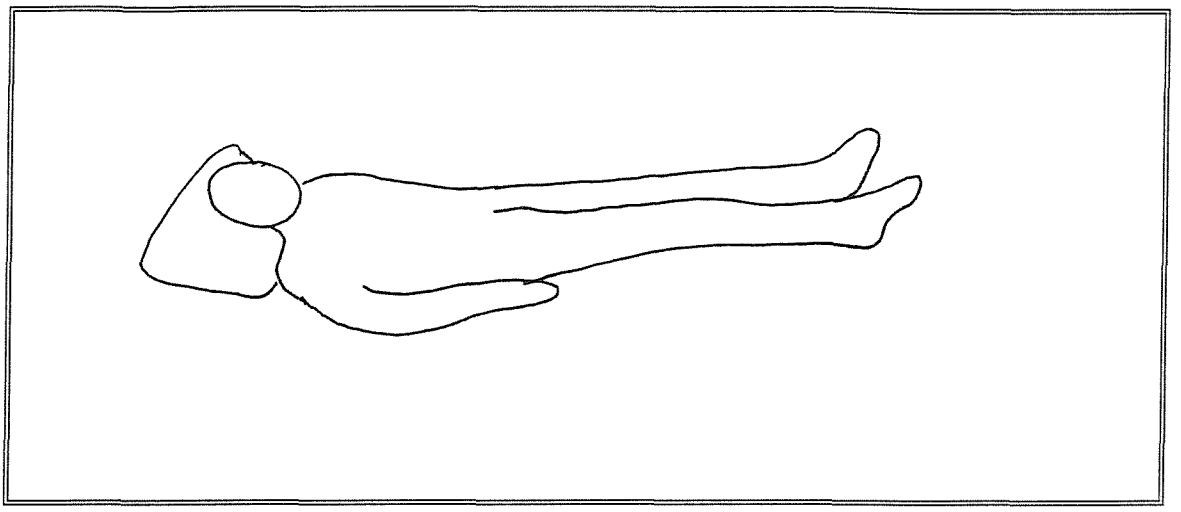
i. Sit right at the front of a hard backed chair, legs slightly apart, the knees at slightly more than a right angle, and the feet flat on the floor. With the arms straight down the sides of the body, stretch the spine and head upwards as though being pulled up by a string.
ii. Imagine that the string has been cut so that the body flops forward in a rolling motion so that the back and the neck are relaxed, but the shoulders and arms don't fall forwards.
iii. Place the hands comfortably on the legs, and mentally check the body for tension (adjust the position if necessary).

b) **The armchair position**



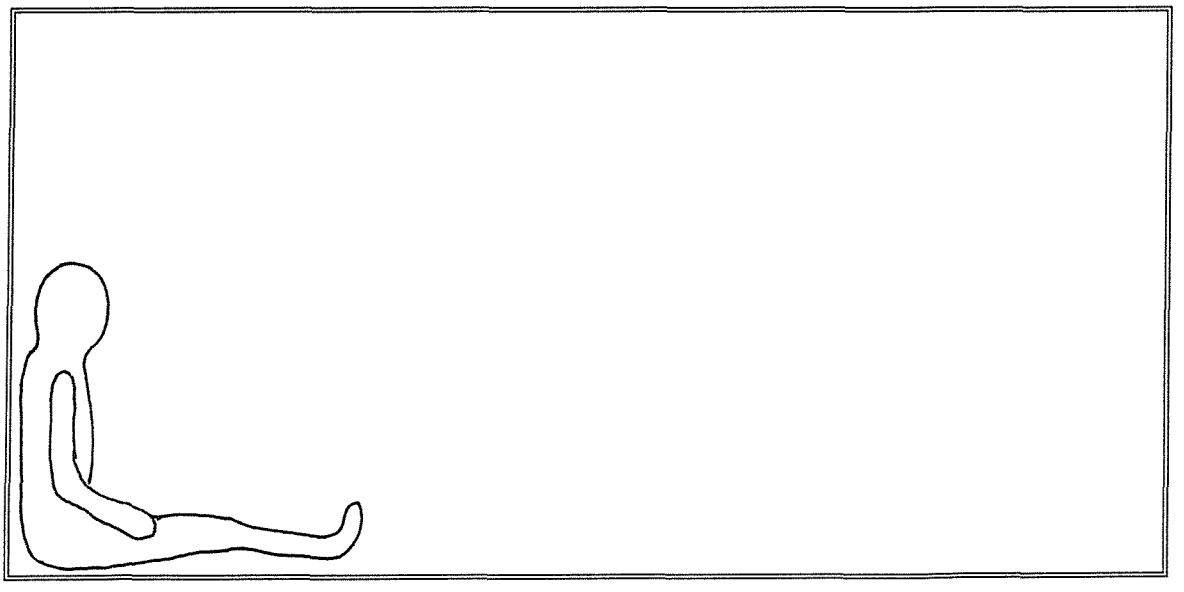
i. Sit back in a comfortable chair, resting the head against the back of the chair. Place the hands comfortably on the legs, and mentally check the body for tension (adjust the position if necessary). If there is no back to rest the head against, keep the head in an upright "neutral" position.

c) **The horizontal position**



i. Lie flat on the floor or on a bed with the head supported by a pillow or cushion. (The head should be straight, not turning to one side). A cushion may also be placed under the knees. Legs should be slightly apart with the feet pointing outwards slightly.
ii. The arms should rest by the side of the body, palms downwards. Above all, the position should be comfortable. (It may be better to so this on top of the bed rather than in it as the weight of the bed clothes may be disturbing).

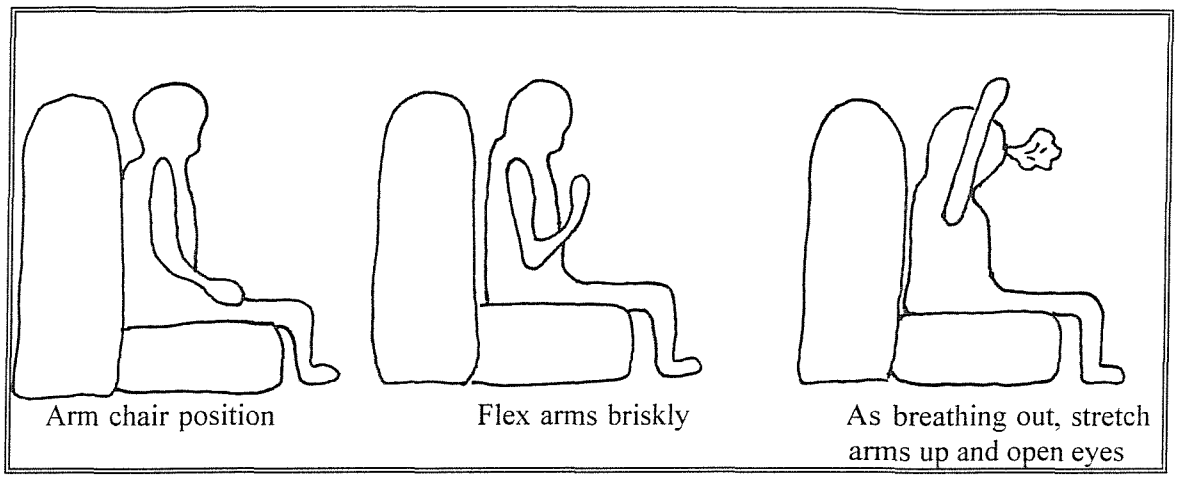
d) **The Continental position**



i. Sit on the floor with your legs straight out in front of you, and your back up against a wall.
ii. Place the hands comfortably on the legs, and mentally check the body for tension (adjust the position if necessary).

2. Termination of the exercises (or how to **CANCEL OUT**).

Cancel out at the end of each exercise, if concentration wanders, or if you experience any visual imagery during an AT exercise.



3. The body check (for tension):
Feet, ankles, lower legs, upper legs, buttocks, lower back, upper back, stomach, chest, shoulders, upper arms, lower arms, hands, back of the neck, back of the head, top of the head, forehead, eyes, jaw, tongue.

4. The "Short Stitch" exercise

Use the short stitch if concentration wanders during a longer exercise i.e. stop the longer exercise and revert to the short stitch. The exercise may also be used as a "booster" throughout the day.

My right arm is heavy
My right arm is heavy
Cancel
Repeat 3-4 times

5. The "Heaviness #1" exercise (arms and legs)

My right arm is heavy (x3)
My left arm is heavy (x3)
Both arms are heavy (x3)
My right leg is heavy (x3)
My left leg is heavy (x3)
Both legs are heavy (x3)
My arms and legs are heavy (x3)
Cancel out
Repeat whole exercise x2

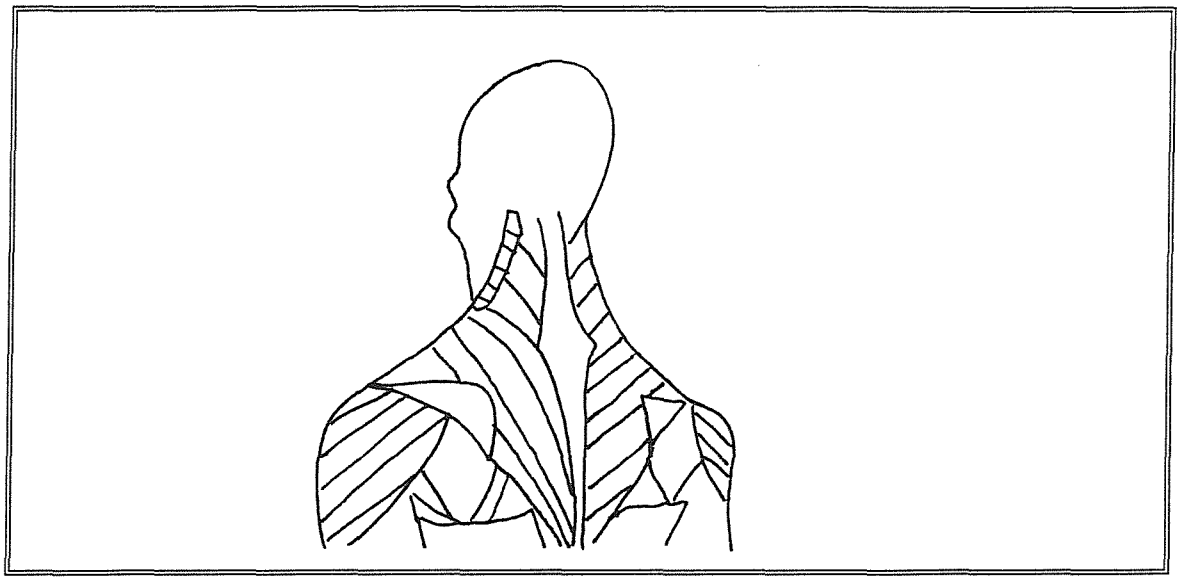
6. Practice - The heaviness exercise twice a day (including the body check), and complete the relaxation diary.

Session 2: The heaviness exercise - part 2

1. The body check and heaviness # 2 (neck and shoulders are heavy)

My right arm is heavy
My arms and legs are heavy
My arms and legs are heavy
My arms and legs are heavy
My neck and shoulders are heavy
My neck and shoulders are heavy
My neck and shoulders are heavy
I am at peace
I am at peace
I am a peace
CANCEL OUT Repeat x2

2. The **Booster** neck and shoulders are heavy



The "neck and shoulders are heavy" part of the exercise may be used as a "booster" at any point during the day (as with the short stitch). As a "booster", it is carried out with the eyes open, and without stopping what you are doing (i.e. without adopting an AT position. So, whenever you think of it, silently repeat "my neck and shoulders are heavy" in bursts of 5, 10, 20 and 25 repetitions (100 repetitions per day). Use the stickers provided as a reminder.

3. Practice

a. Repeat the heaviness exercise twice a day. If there are any difficulties, revert to the short stitch or booster exercises.
b. Use the stickers as a reminder to do the "shoulders and neck are heavy" booster wxercise at intervals throughout the day.
c. Complete the relaxation diary.

Session 3: The Warmth exercise

1. The body check and the warmth exercise

My right arm is heavy
My arms and legs are heavy (x3)
My right arm is warm (x3)
My left arm is warm (x3)
Both my arms are warm (x3)
My right leg is warm (x3)
My left leg is warm (x3)
Both my legs are warm (x3)
My arms and legs are warm (x3)
My neck and shoulders are heavy (x3)
I am at peace (x3)
Cancel out
Repeat whole exercise x2

Please note that later exercises will be shorter than this.

2. Practice

a. Practice the warmth exercise in place of the heaviness #2 exercise in your usual routine.
b. Continue to do the partial exercise (my neck and shoulders are heavy) up to 100 times per day.
c. Complete the relaxation diary.

Session 4: Cardiac regulation and observation of breathing

1. The body check and the heart exercise

My right arm is heavy
My arms and legs are heavy and warm (x6 or 8)
My heart beat is calm and regular (x3)
It breathes me (x3) Just be aware of your breathing. Do not try to control it.
My neck and shoulders are heavy (x3)
I am at peace (x3)
CANCEL OUT

Repeat the exercise twice more.

2. Alternative short stitch exercise

i. With the eyes closed, and maintaining a state of passive concentration (i.e. observation rather than straining to concentrate) repeat three or four times.

My arms and legs are warm and heavy
My arms and legs are warm and heavy
CANCEL OUT

3. Practice

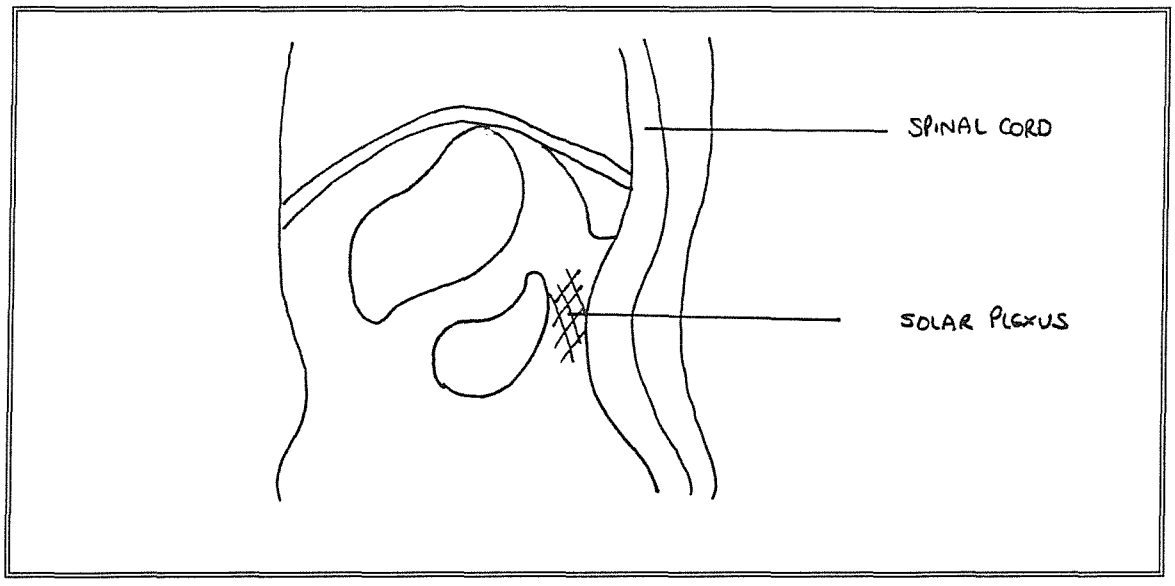
a. Twice per day.
b. Try to use the alternative short stitch and booster exercises throughout the day.
c. Complete the relaxation diary.

Session 5: Abdominal warmth/cool forehead

1. The body check and the abdomen/forehead exercise

My right arm is heavy
My arms and legs are heavy and warm (x6 or 8)
My heart beat is calm and regular (x3)
It breathes me (x3)
My solar plexus is warm (x3) - note a feeling of warmth in the middle of the body
My forehead is cool (x3)
My neck and shoulders are heavy (x3)
I am at peace (x3)
CANCEL OUT

Repeat the exercise twice more.



2. Practice

a. Practice the full exercise twice a day.
b. Practice the booster and short stitch exercises regularly
c. Continue to complete the relaxation diary.

Session 6: Organ specific formulae

1. Use formulae that are specific to your Raynaud's symptoms e.g. My hands (or fingers, feet nose ..) are warm and comfortable.

2. The body check and organ specific exercise

My right arm is heavy
My arms and legs are heavy and warm (x6 or 8)
My heart beat is calm and regular (x3)
It breathes me (x3)
My solar plexus is warm (x3)
My forehead is cool (x3)
My X is warm and comfortable (x3) - where X = site of Raynaud's symptoms
My neck and shoulders are heavy (x3)
I am at peace (x3)
CANCEL OUT

Repeat the exercise twice more.

3. The hold exercise - to be used as an alternative to the short stitch exercise

My right arm is heavy
My arms and legs are heavy (x6)
My neck and shoulders are heavy (x3)
I am at peace (x3)

4. Practice

Continue to practice the long exercise twice daily, and use the alternative exercises at times of poor concentration and when you remember during the day.

Try to keep the exercises as part of your daily routine in the future.

B. APPLIED RELAXATION:

The primary symptoms of Raynaud's Disease are three colour changes in the skin and associated pain and numbness. Firstly, the peripheral blood vessels close, preventing blood flow to the affected area. This causes the initial **white**ning of the skin. If blood flow does not return rapidly, the trapped blood releases all of its oxygen to the surrounding tissues, and consequently, it and the surrounding tissues turn **blue**. Once the blood flow returns, the area **red**dens as large quantities of blood (carrying with it a vital oxygen supply) flood back into the affected area.

The opening and closing of the peripheral blood vessels is controlled in the main by the **Sympathetic Nervous System**. When there is an increase in sympathetic nervous activity, the blood vessels close. In contrast, when this activity decreases, the peripheral blood vessels open up, thereby increasing blood flow.

1. Why Relaxation treatments ?

Through relaxation treatments, one is able to exert some control over the sympathetic nervous system i.e. the very system that it is believed to bring about the primary symptoms of Raynaud's. When we are physically tense, the activity of the sympathetic nervous system increases; thereby reducing peripheral blood flow. In contrast, when we are deeply relaxed, sympathetic nervous activity decreases, which increases blood flow to the hands, feet, nose and other peripheral areas of the body.

2. Why Applied Relaxation ?

A deep state of physical relaxation can be achieved through a variety of relaxation techniques. However, research has shown that the effects of relaxation are not readily transferable to Raynaud's specific situations simply because the effects are far from instant. In theory, you may be willing to spend 10-20 minutes practising a relaxation exercise during a Raynaud's attack, but in practice, few people are able to persevere with the exercise during a painful attack of Raynaud's.

Applied Relaxation, in contrast, allows the Raynaud's sufferer to achieve a deep state of physical relaxation much more rapidly, and can be used in situation that one would not readily associate with a relaxed atmosphere (e.g. when walking or driving in cold, wet, windy weather).

3. What is Applied Relaxation ?

The purpose of Applied Relaxation is twofold. Firstly, to learn how to recognise physical tension in the body, and secondly, how to alleviate it. Applied Relaxation makes this possible by allowing you to discover and contrast the sensations of physical tension and relaxation.

There are 5 stages in the acquisition of the skill of Applied Relaxation. The first stage is quite lengthy, in that once mastered, relaxation takes about 20 minutes to achieve. But with each new stage, the time needed to relax decreases such that by the end of the 5th session, relaxation can be achieved rapidly and applied in situations associated with Raynaud's symptoms.

Session 1: Part 1 - Progressive Relaxation

The first stage of Applied Relaxation is a shortened version of Progressive Relaxation based on the work of Jacobson (1983). It involves tensing and relaxing certain muscle groups in sequence, so that you can learn the difference between the sensation of tension and relaxation in all muscle groups.

Positions

Lie down on a bed or the floor, or sit in a comfortable arm chair.

Listen to and follow the instructions on the tape.

Practice

It is important to practice the exercise twice a day at times convenient to you. Find a quiet room where you won't be disturbed for 20 minutes and listen to the tape.

Please fill in the relaxation diary after each exercise, and bring the tape and relaxation diary with you to the next meeting.

Date	Time	Exercise	What happened ?
		Progressive	*e.g. felt tension at the back of the neck*

If you fail to do an exercise, please fill in the date, leaving other sections blank.

Session 2: Release-only relaxation

The second stage in Applied Relaxation is Release-only relaxation. This involves relaxing the muscles without first tensing them. Having said that, if you have any problems relaxing certain muscle(s), you may briefly tense them first.

Listen to and follow the instructions on the tape (approximately 10 minutes). You will learn to relax the body from head to toe:

Forehead, eyebrows, eyelids, jaw, tongue, throat, lips, face, neck, shoulders, arms, hands, fingertips (breathe with the stomach all of the time), stomach, waist, back, the lower part of your body, buttocks, thighs, knees, calves, feet, tips of toes. Feel yourself relax more and more with each breath).

Practice

Complete the 10 minute exercise twice a day, filling in the relaxation diary as before.

Session 3: Cue-controlled relaxation

In the third stage of Applied Relaxation, you learn to associate a state of relaxation with your breathing pattern. You need first to reach a relaxed state using the release-only technique, and then concentrate on your breathing.

i. Briefly use release-only relaxation to achieve an initial state of relaxation (preferably without the tape).
ii. Once you have achieved a deep state of relaxation, concentrate on your breathing.

As you inhale, think INHALE
As you exhale, think RELAX

iii. Repeat the breathing exercise approximately 30 times (i.e for about 5 minutes).
iv. Stop, and repeat the exercise.
v. Practice twice a day (or more if you like), completing the diary as before.

Session 4: Differential Relaxation #1

You have now mastered the basics of relaxation, and now need to apply the techniques to everyday situations; you need to master differential relaxation.

a. In a sitting position
i. Scan the body for tension. Release any tension with the release-only technique.
ii. Do approximately 10 breaths of cue-controlled relaxation (Exhale = Relax).
iii. Keeping the rest of the body relaxed (scan for tension throughout), move your eyes to look around the room. If you feel any tension in the surrounding muscles, relax them.
iv. Repeat with hands, arms, feet, and legs in turn.

b. Standing up
i. Keep the body as relaxed as possible, move to stand near a wall, but do not lean against it.
ii. Scan the body for tension. Release any tension with the release-only technique.
iii. Do approximately 10 breaths of cue-controlled relaxation (Exhale = Relax).
iv. Keeping the rest of the body relaxed (scan for tension throughout), move your eyes to look around the room. If you feel any tension in the surrounding muscles, relax them.
v. Repeat with hands, arms, feet, and legs in turn.

Practice - twice a day. Keep diary as before.

Session 5: Differential Relaxation #2

Keep the muscle group(s) that are not part of a given activity relaxed e.g. don't tense the biceps muscle for movements of the wrist.

a. In a sitting position
i. Scan the body for tension. Release any tension with the release-only technique.
ii. Do approximately 10 breaths of cue-controlled relaxation (Exhale = Relax).
iii. Keeping the rest of the body relaxed (scan for tension throughout), move your eyes to look around the room. If you feel any tension in the surrounding muscles, relax them.
iv. Repeat with hands, arms, feet, and legs in turn.

b. Standing up
i. Keep the body as relaxed as possible, move to stand near a wall, but do not lean against it.
ii. Scan the body for tension. Release any tension with the release-only technique.
iii. Do approximately 10 breaths of cue-controlled relaxation (Exhale = Relax).
iv. Keeping the rest of the body relaxed (scan for tension throughout), move your eyes to look around the room. If you feel any tension in the surrounding muscles, relax them.
v. Repeat with hands, arms, feet, and legs in turn.

c. Walking around
i. Now continue to scan for tension and release any inappropriate tension as you slowly walk around the room.

Practice - twice a day. Keep diary as before.

Session 6: Rapid Relaxation

You can now relax one part of your body whilst using another. If this ability is to be any use in the treatment of Raynaud's symptoms, it must be achieved rapidly. So you need to learn to relax within about 30 seconds.

Place stickers around home/work environment, and use them as a reminder to carry out a quick 30 second relaxation exercise:

Breathe in.
Say RELAX
Slowly breathe out

Repeat 3 times (always scan and release tension from the body).

Practice - At first practice only when you have plenty of time; after a week or so, start to use the technique in cold weather, if rushed **and during Raynaud's attacks**

Remember: improvement takes practice.

APPENDIX 2.3: The Treatment Study Questionnaire

a. THE PRE-TREATMENT QUESTIONNAIRE

Part A: **Questions about your symptoms**

1. Name:

2. Sex M/F (please delete as applicable)

3. What type of Raynaud's do you suffer from ? (please circle)
a. Primary Raynaud's (also known as Raynaud's Disease)
b. Secondary Raynaud's (also known as Raynaud's Phenomenon)
c. Unsure
d. Undiagnosed

4. For how long have you suffered from Raynaud's ?

5. When did you firs tsee a doctor about your Raynaud's symptoms ?

6. What are your symptoms?

Please describe your symptoms

_____
_____
_____
_____
_____
_____
_____
_____
_____

7. How severe are your symptoms **today** ?

Please rate your symptoms on the 7 point scale below in which
1 = I have no symptoms today
7 = My symptoms today are/have been the most severe that I have ever experienced

1 ----------2----------3----------4----------5----------6----------7

8. How severe are your symptoms **generally** ?

Please rate your symptoms on the 7 point scale below in which
1 = I generally have no symptoms
7 = My symptoms are generally exceptionally severe

1 ----------2----------3----------4----------5----------6----------7

Part B: **Questions about the medical treatments that you have tried for Raynaud's in the past**

9. Have you undergone any surgical procedures - such as nerve blocks - as treatment for your Raynaud's ? Yes/No (Please delete as applicable)

If yes, please list them in the table below:-

Surgical Procedure	Date	Please list any side effects of surgery	Please list any benefits of surgery

10. Are there any **medically** prescribed treatments (i.e. medication) that you have tried in the past, **but no longer take** ? Yes/No (please delete as applicable)

If yes, it would be helpful if you would tell us the **name(s) of the medicines, when** you **started taking them,** and for **how long you took them.** Please tell us about your medicines in the table below.

Name of medicine	When did you first take this medicine ?	For how long did you take it ?

... Continue on the other side of this sheet if necessary.

11. Did you experience side effects with **any** of the medicines **you** mentioned in question 10, above ? Yes/No (please delete as applicable

If yes, please use the table below to state what the side effects were:

Medicines	**Side effect(s)**

12. Did your Raynaud's symptoms improve whilst you were taking any of the medication that you listed in question 10 ? Yes/No (please delete as applicable

If yes, please use the table below to list the medicine(s) that helped, and the beneficial effect that they had on your symptoms:

Medicines	**How did your symptoms improve ?**

Part C: **Questions about the treatments and medicines that you are currently using.**

13. Are you currently waiting for any surgical treatment for your Raynaud's ?
Yes/No (please delete as applicable)

14. Are you currently taking prescribed medicines for your Raynaud's ?
Yes/No (please delete as applicable)

If yes, please write the name(s) of the medicines (and when you started to take them) in the table below.

Medicine	**For how long have you been taking this medicine ?**

... continue overleaf if necessary.

15. Have your symptoms improved as a result of taking these medicines ? Do you experience side effects with the(se) medicine(s) ? Yes/No (please delete as applicable)

Medicine	**Improvements in symptoms**	**Side effects**

... continue overleaf if necessary.

Part D: **Questions about your views and experience of non-conventional treatments**

16. Have you ever tried non-conventional treatments for Raynaud's or for any other medical condition ? (Non-conventional treatments are those that do not involve the use of prescribed medicines or surgery e.g. hypnosis, meditation, relaxation...)

Yes/No (please delete as applicable)

If yes, please list the treatment(s) that you have tried in the table below:

Treatment	**Was this treatment for Raynaud's ?**

17. Did you experience any benefits or side effects with these non-conventional treatments ?
Yes/No (please delete as applicable)

If yes, please list them below:

Treatment	**Benefits of treatment ?**	**Side effects ?**

18. What are your views about alternative treatments ?

Please write as little or as much as you like about your views on non-conventional treatments in the space below. For example:
Do you think that they would work for you ?
What would be your expectations of the outcome of treatment ?
What do you understand by the term "non-conventional" treatments ?
Would you be apprehensive about using such treatments ?

... continue overleaf if necessary.

19. Do you have any "home remedies" for your Raynaud's symptoms ? What sorts of things do you do to cope with your Raynaud's attacks once they have started ? how do you warm your hands and body generally ?

Please write as little or as much as you like about your methods of "treating" your Raynaud's symptoms at home:

... continue overleaf if necessary.

20. What triggers your Raynaud's symptoms ?

... continue overleaf if necessary.

b. THE POST-TREATMENT QUESTIONNAIRE

I. The experimental participant's version

Part A: **Questions about your treatment**

1. Name:

2. How severe are your symptoms **today** ?

Please rate your symptoms on the 7 point scale below in which
1 = I have no symptoms today
7 = My symptoms today are/have been the most severe that I have ever experienced

1 ----------2----------3----------4----------5----------6----------7

3. How severe have your symptoms been on an average day **since you started your treatment** at the university ?

Please rate your symptoms on the 7 point scale below in which
1 = I have no symptoms
7 = My symptoms have been exceptionally severe

1 ----------2----------3----------4----------5----------6----------7

4. Have you undergone any surgical procedures - such as nerve blocks - as treatment for your Raynaud's ? Yes/No (Please delete as applicable)

If yes, what was the surgical procedure ?

_____

_____

5. Have there been any changes to your medication **since** you started this treatment ?
Yes/No (please delete as applicable)

If yes, please tell us which medicine(s) you are no longer taking, and any medicines that you have started to take since the start of your treatment at the university:

Medicine(s) that you are no longer taking	**New medication**

6. Did you practice the exercises in between training sessions ?
Yes/No (please delete as applicable)

7. Did you find it hard to motivate yourself to practice between sessions ?
Yes/No (please delete as applicable)

8. How often did you practice the exercises ? Being as truthful as possible, please tick the phrase below which best describes your general level of practice of the exercises.

- ☐ I never practised the exercises
- ☐ I rarely practised the exercise (i.e. less than once per week)
- ☐ I practised the exercises more than once a week, but less than once per day
- ☐ I practised the exercises once per day
- ☐ I practised the exercises twice per day
- ☐ I practised the exercised more than twice per day

9. Please tick **any** of the phrases below that are true of you and your level of practice of the exercises:

- ☐ My level of practice was consistent throughout the treatment period
- ☐ My level of practice was inconsistent through the treatment period
- ☐ I practised more in the first weeks of treatment than I did towards the end of treatment
- ☐ I practised more at the end of the treatment period than I did in the first few weeks of treatment
- ☐ I practised the exercises more in the days before (or just after) a training meeting than I did for the rest of the treatment period
- ☐ I never practised the exercises during an attack of Raynaud's
- ☐ I practised the exercises whenever I had an attack of Raynaud's
- ☐ I enjoyed practising the exercises
- ☐ I did not enjoy practising the exercises

10. Do you feel that the treatment you received at the University had any effect on your Raynaud's symptoms ?
Yes/No (please delete as applicable)

11. Do you feel that the treatment you received at the university worsened your Raynaud's symptoms at all ?
Yes/No (please delete as applicable)

If yes, in what way did your symptoms worsen ?

12. Do you feel that the treatment you received at the university improved your Raynaud's symptoms at all ? Yes/No (please delete as applicable)

If yes, in what way did your symptoms improve ?

13. Do you feel that the treatment you received at the university had any effect on any other areas of your life? Yes/No (please delete as applicable)

If yes, in what way did the treatment effect your life ?

14. Did you make any changes to your lifestyle (other than those required by the treatment) during the course of treatment ? e.g. move to a warmer office, go abroad
Yes/No (please delete as applicable)

If yes, what changes did you make ?

15. Did you try any other "non-conventional" treatments for Raynaud's (or for any other conditions) during the period of treatment at the university ?
Yes/No (please delete as applicable)

If yes, what were they, and what effects did they have ?

16. What are your views about alternative treatments ?

Please write as little or as much as you like about your views on non-conventional treatments in the space below. For example:
Do you think that they would work for you ?
What would be your expectations of the outcome of treatment ?
What do you understand by the term "non-conventional" treatments ?
Would you be apprehensive about using such treatments ?

... continue overleaf if necessary.

17. Do you have any "home remedies" for your Raynaud's symptoms ? What sorts of things do you do to cope with your Raynaud's attacks once they have started ? how do you warm your hands and body generally ?

Please write as little or as much as you like about your methods of "treating" your Raynaud's symptoms at home:

Thank you for completing this questionnaire.

b. THE POST-TREATMENT QUESTIONNAIRE

II. The control participant's version

1. Name:

2. How severe are your symptoms **today** ?
Please rate your symptoms on the 7 point scale below in which
1 = I have no symptoms today
7 = My symptoms today are/have been the most severe that I have ever experienced

1 ----------2----------3----------4----------5----------6----------7

3. How severe have your symptoms been on an average day **since you completed questionnaire 1** ?
Please rate your symptoms on the 7 point scale below in which
1 = I have no symptoms
7 = My symptoms have been exceptionally severe

1 ----------2----------3----------4----------5----------6----------7

4. Have you undergone any surgical procedures - such as nerve blocks - as treatment for your Raynaud's ? Yes/No (Please delete as applicable)

If yes, what was the surgical procedure ?

_____

_____

5. Have there been any changes to your medication **since** you completed questionnaire 1 ?
Yes/No (please delete as applicable)

If yes, please tell us which medicine(s) you are no longer taking, and any medicines that you have started to take since you completed questionnaire 1:

Medicine(s) that you are no longer taking	**New medication**

6. Do you feel that your symptoms have improved since you completed questionnaire 1 ?
Yes/No (please delete as applicable)

If yes, in what way did your symptoms improve ?

_____

_____

_____

_____

_____

7. Have there been any changes in any other areas of your life since you completed the first questionnaire ? Yes/No (please delete as applicable)

If yes, in what way has your life/life style changed since you completed questionnaire 1 ?

8. Have you tried any "non-conventional" treatments for Raynaud's (or for any other conditions) since you completed questionnaire 1 ?
Yes/No (please delete as applicable)

If yes, what were they, and what effects did they have ?

9. What are your current views about alternative treatments ?

Please write as little or as much as you like about your views on non-conventional treatments in the space below.

... continue overleaf if necessary.

10. Do you have any "home remedies" for your Raynaud's symptoms ? What sorts of things do you do to cope with your Raynaud's attacks once they have started ? how do you warm your hands and body generally ?

Please write as little or as much as you like about your methods of "treating" your Raynaud's symptoms at home:

Thank you for completing this questionnaire.

c. THE FOLLOW-UP QUESTIONNAIRE

I. The experimental participant's version

Part A: **Questions about your treatment**

1. Name:

2. How severe are your sym:ptoms **today** ?

Please rate your symptoms on the 7 point scale below in which
1 = I have no symptoms today
7 = My symptoms today are/have been the most severe that I have ever experienced

1 ----------2----------3----------4----------5----------6----------7

3. How severe have your symptoms been on an average day **since your treatment at the university ended** ?

Please rate your symptoms on the 7 point scale below in which
1 = I have no symptoms
7 = My symptoms have been exceptionally severe

1 ----------2----------3----------4----------5----------6----------7

4. Have you undergone any surgical procedures - such as nerve blocks - as treatment for your Raynaud's ? Yes/No (Please delete as applicable)

If yes, what was the surgical procedure ?

_____

_____

5. Have there been any changes to your medication **since** the end of your treatment at the university ?
Yes/No (please delete as applicable)

If yes, please tell us which medicine(s) you are no longer taking, and any medicines that you have started to take since the end of your treatment at the university:

Medicine(s) that you are no longer taking	**New medication**

6. Did you practice the exercises since the training sessions came to an end ?
Yes/No (please delete as applicable)

7. Did you find it hard to motivate yourself to practice ?
Yes/No (please delete as applicable)

8. How often did you practice the exercises since the end of the training sessions at the university ? Being as truthful as possible, please tick the phrase below which best describes your general level of practice of the exercises.

- ☐ I never practised the exercises
- ☐ I rarely practised the exercise (i.e. less than once per week)
- ☐ I practised the exercises more than once a week, but less than once per day
- ☐ I practised the exercises once per day
- ☐ I practised the exercises twice per day
- ☐ I practised the exercised more than twice per day

9. Please tick **any** of the phrases below that are true of you and your level of practice of the exercises since treatment ended:

- ☐ My level of practice was consistent throughout the treatment period
- ☐ My level of practice was inconsistent through the treatment period
- ☐ I practised more in the first weeks of treatment than I did towards the end of treatment
- ☐ I practised more at the end of the treatment period than I did in the first few weeks of treatment
- ☐ I practised the exercises more in the days before (or just after) a training meeting than I did for the rest of the treatment period
- ☐ I never practised the exercises during an attack of Raynaud's
- ☐ I practised the exercises whenever I had an attack of Raynaud's
- ☐ I enjoyed practising the exercises
- ☐ I did not enjoy practising the exercises

10. Do you feel that the treatment you received at the University had any effect on your Raynaud's symptoms in the months since the training sessions ?
Yes/No (please delete as applicable)

11. Do you feel that the treatment you received at the university worsened your Raynaud's symptoms at all ?
Yes/No (please delete as applicable)

If yes, in what way did your symptoms worsen ?

12. Do you feel that the treatment you received at the university improved your Raynaud's symptoms at all ? Yes/No (please delete as applicable)

If yes, in what way did your symptoms improve ?

13. Do you feel that the treatment you received at the university had any effect on any other areas of your life? Yes/No (please delete as applicable)

If yes, in what way did the treatment effect your life ?

14. Did you make any changes to your lifestyle (other than those required by the treatment) since the treatment came to an end ? e.g. move to a warmer office, go abroad
Yes/No (please delete as applicable)

If yes, what changes did you make ?

15. Did you try any other "non-conventional" treatments for Raynaud's (or for any other conditions) since training at the university came to an end ?
Yes/No (please delete as applicable)

If yes, what were they, and what effects did they have ?

16. What are your views about alternative treatments ?

Please write as little or as much as you like about your views on non-conventional treatments in the space below. For example:
Do you think that they would work for you ?
What would be your expectations of the outcome of treatment ?
What do you understand by the term "non-conventional" treatments ?
Would you be apprehensive about using such treatments ?

... continue overleaf if necessary.

17. Do you have any "home remedies" for your Raynaud's symptoms ? What sorts of things do you do to cope with your Raynaud's attacks once they have started ? how do you warm your hands and body generally ?

Please write as little or as much as you like about your methods of "treating" your Raynaud's symptoms at home:

Thank you for completing this questionnaire.

c. THE FOLLOW-UP QUESTIONNAIRE

II. The control participant's version

1. Name:

2. How severe are your symptoms **today** ?
Please rate your symptoms on the 7 point scale below in which
1 = I have no symptoms today
7 = My symptoms today are/have been the most severe that I have ever experienced

1 ----------2----------3----------4----------5----------6----------7

3. How severe have your symptoms been on an average day **since you completed questionnaire 2** ?
Please rate your symptoms on the 7 point scale below in which
1 = I have no symptoms
7 = My symptoms have been exceptionally severe

1 ----------2----------3----------4----------5----------6----------7

4. Have you undergone any surgical procedures - such as nerve blocks - as treatment for your Raynaud's since you completed questionnaire 2 ? Yes/No (Please delete as applicable)

If yes, what was the surgical procedure ?

_____

_____

5. Have there been any changes to your medication **since** you completed questionnaire 2 ?
Yes/No (please delete as applicable)

If yes, please tell us which medicine(s) you are no longer taking, and any medicines that you have started to take since you completed questionnaire 2:

Medicine(s) that you are no longer taking	**New medication**

6. Do you feel that your symptoms have improved since you completed questionnaire 2 ?
Yes/No (please delete as applicable)

If yes, in what way did your symptoms improve ?

_____

_____

_____

_____

_____

7. Have there been any changes in any other areas of your life since you completed the second questionnaire ? Yes/No (please delete as applicable)

If yes, in what way has your life/life style changed since you completed questionnaire 2 ?

8. Have you tried any "non-conventional" treatments for Raynaud's (or for any other conditions) since you completed questionnaire 2 ?
Yes/No (please delete as applicable)

If yes, what were they, and what effects did they have ?

9. What are your current views about alternative treatments ?

Please write as little or as much as you like about your views on non-conventional treatments in the space below.

... continue overleaf if necessary.

10. Do you have any "home remedies" for your Raynaud's symptoms ? What sorts of things do you do to cope with your Raynaud's attacks once they have started ? how do you warm your hands and body generally ?

Please write as little or as much as you like about your methods of "treating" your Raynaud's symptoms at home:

Thank you for completing this questionnaire.

APPENDIX 2.4: Coding frames for the Treatment study Questionnaires

a. THE PRE-TREATMENT QUESTIONNAIRE

Part A: **Questions about your symptoms**

1. Name

2. Sex
   1 = Male;
   2 = Female

3. Raynaud's diagnosis
   1 = Primary;
   2 = Secondary;
   3 = Unsure of diagnosis;
   4 = undiagnosed

4. For how long have you suffered from Raynaud's ?
   1 = Since childhood;
   2 = As an adult up to 40 years of age;
   3 = unsure of diagnosis

5. When did you first see a doctor about your Raynaud's symptoms ?
   1 = Up to a year following onset
   2 = Up to 5 years following onset
   3 = Over 5 years after symptom onset
   4 = Never

6. What are your symptoms ?
   0 = No details
   1 = Vasospastic attack (WBR)
   2 = 2 colour changes in skin (WB, WR, BR)
   3 = 1 colour change (W, B, R)

7. How severe are your symptoms today ?
8. How severe are your symptoms generally ?
   7 point scale:
   0 = No response
   1 = No symptoms
   2, 3 = Mild
   4,5 = Moderate
   6,7 = Severe

Part B: **Previous surgical and medical treatments**

9. Previous surgical procedures for Raynaud's
   0 = No response
   1 = None
   2 = Yes e.g. Sympathectomy, Carpel Tunnel

10. Previous Prescribed medication for Raynaud's symptoms
    1 = None
    2 = Vasodilatory action
    3 = Calcium Channel Blockers e.g. Nifedipine
    4 = Medication for underlying conditions
    5 = No details

    and :
    1 = No
    2 = Yes

11. Side effects of previously prescribed medication
    0 = no medication used
    1 = Side effects experienced
    2 = No side effects

12. Improvements in symptoms when taking previously described medication ?
    0 = No previous medication used
    1 = Side effects e.g. Headaches, Nausea
    2 = No side effects
    3 = Do not know

Part C: **Current symptoms (surgical/medical)**

13. Are you waiting for surgical treatment for your Raynaud's symptoms ?
    1 = No
    2 = Yes e.g. Sympathectomy, Carpel Tunnel

14. Are you currently taking prescribed medication for your symptoms ?
    0 = No response
    1 = No
    2 = Yes

15a. Have your symptoms improved as a result of taking these medicines ?
    0 = Not taking prescribed medication
    1 = Yes
    2 = No
    3 = Don't know

15b. Do you experience side effects with these medicines ?
    0 = Not taking prescribed medication
    1 = Yes
    2 = No
    3 = Don't know

Part D: **Views/experience of "non-conventional" treatments**

16. Have you ever tried non-conventional treatments ?
    1 = none
    2 = Relaxation
    3 = Other e.g. Aromatherapy, Herbalism, acupunctur

17a. Did your symptoms improve with these non-conventional medicines ?
0 = Not experienced non-conventional medicines
1 = Yes
2 = No
3 = Don't know

17b. Did your experience side effects with these non-conventional medicines ?
0 = Not experienced non-conventional medicines
1 = Yes
2 = No
3 = Don't know

18. Your views about alternative treatments ?
0 = No views expressed
1 = Optimism (positive)
2 = Willing to try (neutral)
3 = Scepticism (negative)

19. "Home remedies"
0 = None
1 = Avoidance of triggers e.g. keeping warm, stress reduction
2 = other e.g. Physical exercise

20. Subjective Trigger of attacks
1 = cold
2 = "Stress"
3 = Cold and "stress"
4 = Other
5 = No details

b. THE POST-TREATMENT AND FOLLOW-UP QUESTIONNAIRES

1. How severe are your symptoms today?
2. How severe are your symptoms generally ?
   7 point scale:
   1 = no symptoms
   2,3 = mild symptoms
   4,5 = moderate symptoms
   6,7 = severe symptoms

3. Surgical procedures since last questionnaire
   1 = None
   2 = Yes e.g. sympathectomy

4. Changes to medication since last questionnaire
   1 = No change
   2 = reduced/stopped
   3 = changed/new medication for Raynaud's

5. Practice exercises between sessions ?
   0 = Control participant
   1 = Yes
   2 = No

6. Motivation to practice
   0 = Control participant
   1 = Always motivation problems
   2 = Some motivation problems
   3 = No motivation problems

7. Practice frequency
   0 = Control participant
   1 = Never
   2 = rarely (less than once/day, but at least once since last training session)
   3 = 1/day
   4 = As asked (twice/day)

8. Consistency of practice
   0 = Control participant
   1 = Rare/Never practised
   2 = Inconsistent practice
   3 = Consistent practice

9. Enjoyment of practice
   0 = Control participant
   1 = Enjoyed practice
   2 = Sometimes enjoyed practice
   3 = Did not enjoy practising

10. Have your symptoms altered since the last questionnaire
    1 = Symptoms improved
    2 = improvement in other areas of life
    3 = No effect

11. Use of other non-conventional treatments since last questionnaire
    1 = None
    2 = Relaxation treatments
    3 = Other e.g. Aromatherapy, Herbalism, Acupuncture

12. Views of non-Conventional treatments
    1 = Optimism/positive
    2 = open minded/neutral
    3 = sceptical/unimpressed/negative

13. Lifestyle changes
    1 = None
    2 = positive changes e.g. Central heating installed
    3 = Negative changes e.g. Death of companion, divorce, separation

14. Changes to Home remedies
    1 = No change
    2 = reduced/stopped home remedies
    3 = changed/introduced new home remedies

15. "Likeability" of participant
    1 = Likeable
    2 = Neutral (including Controls)
    3 = Disliked

16. Participant enjoyment of training sessions
    0 = Control participant
    1 = Enjoyed session
    2 = Neutral (including Controls)
    3 = Did not enjoy the sessions

APPENDIX 2.5: Instructions to participants during the Pre-treatment assessment session

a. THE THEORY BEHIND THE USE OF BEHAVIOURAL TREATMENTS FOR RAYNAUD'S SYMPTOMS

"When a Raynaud's attack occurs, the blood vessels of the extremities, such as the fingers and toes, clamp shut, or go into spasm, preventing blood flow to the affected areas. Blood flow to the extremities is under the control of part of the nervous system known as the Sympathetic Nervous System. A high level of sympathetic nervous activity causes the blood vessels to clamp shut. This is a normal response that allows blood to be diverted away from the extremities toward the core of the body at times of need. In contrast, a low level of sympathetic nervous activity has the opposite effect ie. that of opening up the peripheral blood vessels. Therefore, if we could find a way of decreasing sympathetic nervous activity, we may be able to reduce, if not prevent, the blood vessel spasms characteristic of Raynaud's attacks. Unlike the central nervous system, the sympathetic nervous system is not under our voluntary control. We can not directly open up the blood vessels as we might directly open our eyes. But, there is a way in which we can indirectly exert control over the activity of the sympathetic nervous system, and that is through relaxation. When we are physically tense, the activity of the sympathetic nervous system increases. On the other hand, when we are relaxed, sympathetic nervous activity decreases resulting in greater blood flow to the extremities. So it follows that through relaxation, we may be able to exert some control over Raynaud's symptoms."

b. THE DESCRIPTION OF THE OBJECTIVE TESTS OF MUSCLE TENSION AND BLOOD FLOW GIVEN TO ALL PARTICIPANTS DURING THE "PRE-TREATMENT" ASSESSMENT SESSION

"Relaxation treatments for Raynaud's have been used in the past with quite encouraging results. Having said that, there is little objective evidence that the treatments are effective. So, we are going to use objective tests for comparison before and after treatment. The first test assesses control of finger temperature, or more specifically, your ability to increase finger skin temperature. Temperature detecting probes will be attached to the index and middle fingers of your (non-dominant) hand, and three electrodes to your forearm. The temperature detecting probes pick up the temperature of your fingers and store this information in the attached logger. The electrodes will provide a similar recording of the tension and movement of the muscles of your lower arm.

Both loggers will be switched on for 24 minutes. The first 12 minutes will provide a baseline of your finger temperature and muscle tension at rest. Then for the next 6 minutes, you will be asked to try to increase your finger temperature using any non-physical means. So, for example, do not sit on your hands. It is important that you keep your hands in same position throughout the 24 minutes. But having said that, please do not remain in an uncomfortable position. Use any technique that you feel may help you warm your hands. The final 6 minutes will be a further baseline measure to see whether temperature changes persist once you have stopped trying to warm your hands.

The second test, known as a cold challenge test, measures the response of your fingers to immersion in cool water. Temperature detecting probes will pick up the temperature of your fingers and display the information as a graph on the computer screen. After 5 minutes, I'll put a plastic glove on your hand and ask you to stand up and immerse your hand in the water bath for 5 minutes. The water is at 15 degrees Celsius, so although cool, it is not unbearable. If you wish to remove your hand before the five minutes is over, please do so. After 5 minutes, the glove will be removed, and we will wait

for the temperature of your fingers to return to the baseline level. Your hand should be in the same position during the warming phase as it was in the baseline period. Again, please try to limit movement of your hand and arm. It may take quite a long time for your fingers to rewarm. The computer will be turned off when all fingers have rewarmed, or after 45 minutes - whichever is the shortest."

APPENDIX 2.6: Training instructions given to the Applied Relaxation participants

a. AN OUTLINE OF APPLIED RELAXATION AS A TREATMENT FOR RAYNAUD'S SYMPTOMS

"The method of relaxation that I am going to teach you during this and the next five meetings is called Applied Relaxation. In the initial sessions, you will learn the basic techniques of relaxation - the first of these being Progressive Relaxation. In subsequent meetings, you will learn to reach a relaxed state more rapidly. One of the problems with many forms of relaxation is that they enable you to reach a deep state of relaxation only when lying down in a quiet room. With Applied Relaxation, as the name suggests, you will be able to apply the skill of relaxation to everyday situations."

b. ADDITIONAL INSTRUCTIONS GIVEN TO THE ART AND ARE (BIOFEEDBACK) PARTICIPANTS

I. Temperature biofeedback:

"In addition to the relaxation training, at each session, you will receive a supplementary treatment known as finger skin temperature biofeedback. Temperature biofeedback is a means of making you aware of small changes in the temperature of your fingers achieved through the relaxation technique that you have been taught. You can use this information to learn to control your own finger temperature. The temperature detecting probe of the feedback apparatus is attached to the index finger of your [dominant] hand with surgical tape. When switched on, the apparatus emits a tone which is analogous to the temperature of your finger. As you warm your fingers with AR, the tone will begin to get quieter and may disappear altogether; if your fingers get cooler, the tone will get progressively more high pitched. To demonstrate, listen to the tone with the probe attached to your finger. Now move your finger up to your neck where it is warmer, and listen to the tone quieten. So, a decreasing tone means that the relaxation technique is warming your fingers; a high pitched tone means that you are not relaxing (or relaxing less) and your fingers are cooling down. Use the tone as a guide to the effects of the relaxation technique on the temperature of your fingers."

II. EMG Biofeedback:

EMG biofeedback is a means of making you aware of small changes in your muscular activity. These three electrodes will be attached to your elbow and to the extensor muscle of your [non-dominant] arm - like so. When switched on, the apparatus emits a tone which is analogous to the muscular activity of your arm. As you use the relaxation technique to decrease muscle tension in your arm, the tone will get quieter and may disappear altogether; if your arm becomes tense, the tone will get louder. To demonstrate, make a fist and listen to the tone. Now relax you arm and hand and listen as the tone quietens. To repeat, a quietening tone means that the muscle in your arm is getting progressively less tense ie. the relaxation technique is working; a tone that is getting louder means that your arm is less getting progressively less relaxed. Use the tone as a guide to the effects of the relaxation technique on the muscle tension of your arm."

c. THE PROGRESSIVE RELAXATION EXERCISE

"Sit in a comfortable position with your arms at your sides and your legs uncrossed in front of you. Please try to stay in that position for the rest of the session, although do move if you feel uncomfortable. The exercise will last for about 20 minutes, and will involve tensing and then relaxing muscles around the body. Through this exercise, you will come to learn the difference between relaxed and tensed muscles, so that you will know from the physical sensations experienced, the state of tension in your muscles. After the exercise, there will be a further 10 minute baseline of finger temperature to see if any temperature changes persist after the relaxation exercise.

Close your eyes and listen to my voice as I talk you through the exercise. If you are unable to hear, or do not understand something, please don't be afraid to interrupt me.

Lie down or sit as comfortably as possible. Begin by making a fist with your left hand. Make a tight fist and notice the tension in your hand and arm..... And relax. Let the fingers in your left hand be loose and notice the difference. Once more, make a really tight fist with your left hand. Feel the tension in your hand and forearm..... And relax. Straighten your fingers and notice the difference when you let your hand relax. Now repeat the same thing with your right hand. Make a tight fist, but let the rest of your body stay relaxed. Feel the tension in your hand and arm... And relax. Notice the comfortable contrast again. Once more, tense your right hand tightly. Analyse how it feels. Now do the opposite to tensing: relax and notice the difference. Continue to relax that way for a while.

Now make a tight fist with both hands. Your hands tense, your forearms tense. Think how it feels... And relax. Let your fingers be loose, and feel the relaxation. Observe the difference between tensing a muscle and relaxing it. Continue to relax your hands and forearms more and more.

Now bend your elbows so you can feel the tension in your biceps without tensing your hands. Notice the tension in the upper arm..... And relax. Let your arm relax, and feel the difference again. Now stretch your arms straight out so that you can feel the tension in the muscles at the back of your upper arms - the triceps. Feel the tension and hold it. Hold it....And relax. Bring your arms back to a comfortable position and let the relaxation spread to each part of your hands and arms. Concentrate on the relaxation. Feel the relaxing of your arms without any tension. Notice the feelings of heaviness that comes with relaxation. Experience your arms feeling comfortable and heavy. Heaviness that comes with relaxation. Relaxed and heavy. Relaxed and heavy.

Now let us turn to the face. Wrinkle your forehead by raising your eyebrows. Wrinkle tightly. And hold it as tight as you can... And relax. Now pull your eyebrows together and frown. Notice the tension. And hold it as tightly as you can. And relax, and smooth out your forehead again. Squeeze your eyes tightly. Feel the tension. Let it build and build. And relax, without opening your eyes. Keep your eyes lightly and comfortably closed. And notice the relaxation of the forehead and eyes. Now tense your jaws by clenching your teeth together. Feel the tension everywhere in your jaws. Feel it building, building. Tense... and relax. Part your lips slightly. Now press your tongue hard against the roof of your mouth. Feel the tension in your tongue. Press hard, hard and relax by letting your tongue go back. Now pucker your lips, forming your mouth into an "O". Push tight - as tight as you can and relax. Notice the difference between tension and relaxation. Feel the entire face relaxing. The forehead, eyebrows, jaws, lips and tongue. Let the relaxation become deeper. And notice the feeling of heaviness that comes with relaxation.

Let us now move to the throat, neck and back. Press your head back against the surface as hard as possible, and feel your neck tensing, tensing harder and harder. Roll your head to the right and feel how the tension changes. Then roll it to the left. Once again feel the tension move, and bring your head back to the original position, and bend forwards. Press your chin against your chest as hard as you can. Tight, as tight as you can... and relax. Put your head back into a comfortable position. Let your throat and neck relax. Let the relaxation in your throat and neck become deeper and deeper.

Now pull your shoulders up toward your ears without tensing your arms. Feel the tension, and hold it there. Hold it, and relax. Now pull up your shoulders again without tensing your arms, and move your shoulders around in a circle. Your shoulders should move upwards, forwards, and backwards as much as possible. Feel the tension in your shoulders and upper part of your back. Nice and tense. Let your shoulders go back again...And relax. Notice that contrast, with your shoulders relaxed. The difference between tension and relaxation. Now arch your back, and feel the tension along the spine. Feel it building and building, and let go to a comfortable position. Relax the small of your back once again. Once more arch your back, and try to feel where you have tension in your back. Arch it tight. As tight as you can. Feel the tension building. Hold it. And relax again. Rest comfortably, and let the relaxation spread through your entire back. Feel the heaviness in each muscle as it relaxes. Relaxed and heavy.

Let us now move to the lower part of the body. Tense your buttocks and thighs, pressing your heels against the floor as hard as possible. Hold it. Press hard... and relax. Notice the difference. Once more tense your thighs and buttocks. Keep tensing. Keep tensing, and relax. Notice the contrast. This time stretch your feet and toes away from your face so that the calf muscles are tensed. Think of the tensing. Feel it. And relax the feet and calves. Now bend your feet up towards your face, so you feel the tension in your chin. Feel it building. Feel it getting tight. And relax. Continue to relax for a while. Let the relaxation spread throughout the entire lower part of your body. Notice how heavy your legs become when they are relaxed. Relaxed and heavy. Experience a feeling of warmth spreading through your body. Heavy and warm. Heavy and warm.

Now to the stomach and chest. Begin by observing how you breathe. Are you mostly breathing with your chest or stomach ? Try to alternate between chest breathing and stomach breathing. Observe which is the most relaxing.... The most comfortable. Push out the stomach muscles so that the stomach wall becomes hard. But continue to breathe. And now you must breathe with your chest. Notice the tension and relaxation. Once more, push the stomach out. Make the muscles hard, but continue to breathe. Keep up the tension, but think about it. Think where it is. Think how it feels. Then relax. Notice the comfortable feeling that comes with the relaxation of the stomach muscles. Now pull in your stomach, and feel the tension, but continue to breathe. Relax, and at once change to stomach breathing. Feel the relaxation through your entire chest and stomach. Now pull in your stomach again, and feel the tension. Feel it build as you continue to breathe. Push out the stomach again. As before, keep the tension. And continue to breathe, and this time, pull the stomach in, and again feel it tighten. Feel the tension build. And now relax completely. Let all tension disappear. Breathe with your stomach, and feel how the relaxation spreads through your whole stomach and chest. Breathe slowly. Use your stomach, and then your chest. Notice the difference between breathing in and breathing out. Notice that the feeling of relaxation increases as you breathe out. Now breathe in and fill your whole lungs. Take a deep breathe, and hold your breathe. Notice the tension in your chest and stomach build. Notice it building. Building. And breathe out. Let your chest sink down, and automatically push out the air. Feel the relaxation follow the breathing out. Continue to relax, and breathe calmly and evenly with your stomach. Let the relaxation become deeper with each breathing out. Do not make an effort to relax. Let the relaxation come by itself. Each time you practice this program, you'll experience an increased effect. You'll feel that the relaxation becomes deeper. Comes more quickly, each time you practice in this way. As you learn the difference between tensed and relaxed muscles in this way, you learn the meaning of total relaxation. Continue to relax on your own for a while. Notice the feeling of heaviness and warmth. And notice the feeling of calmness that comes with the relaxation. How the relaxation brings an experience of calmness and certainty. How you can sink into a comfortable state. A deep state of tranquillity and restfulness and relaxation. Experience this as you continue to relax for a while on your own.

Then you can listen to me once again. You can experience this as a comfortable way to relax. It will afterwards make you feel rested with new strength. You will also be able to keep this feeling of calmness and certainty.

Now you can wake up by taking a few deep breaths. Before you open your eyes, you can wake up your body by moving your feet, by bending and stretching your arms, and by moving your head a little. When

you feel that you body is awake, take another deep breath. Open your eyes and feel completely awake and good in every way....

d. AN EXPLANATION OF THE RELEASE-ONLY RELAXATION EXERCISE

"The second stage of Applied relaxation is Release-only relaxation. As the name suggests, this involves relaxing without first tensing particular muscles or muscle groups. Through practice of the last (Progressive Relaxation) exercise, you learned the difference between relaxed and tensed muscles. As such, you now know how it feels when your muscles are relaxed. During this exercise, I will talk you through the exercise, naming the different areas of the body to be relaxed. If you find it difficult to relax certain muscles, it may help to briefly tense any particularly stubborn muscle areas before relaxing them, so that you are reminded of the difference between relaxed and tense muscles. Having said that, the purpose of this exercise is to relax without tension, so only tense the muscles that you are unable to relax directly.

e. THE RELEASE-ONLY RELAXATION EXERCISE

Breathe with calm, regular breaths...
Feel how you relax more and more with every breath...
Just let go...
Relax your forehead.... eyebrows.... eyelids.... jaws.... tongue and throat....lips.... your entire face...
Relax your neck... shoulders... arms... hands... and all the way to out to your fingertips.
Breath calmly and regularly with your stomach all the time.
Let the relaxation spread to your stomach... waist...and back.
Relax the lower part of your body... your buttocks... thighs... knees...calves... feet.... and all the way down to the tips of your toes.
Breath calmly and regularly, and feel how you relax more and more with each breath.
Take a deep breath, and hold your breath for a couple of seconds.
Let the air out slowly... slowly.
Notice how you relax more and more.

When you feel ready, wake up your body by taking a few deep breathes, and by moving your feet, arms and feet. When you feel that your body is awake, open your eyes and feel wide awake and good in every way."

f. THE BREATHING INSTRUCTIONS FOR THE CUE-CONTROLLED RELAXATION EXERCISE

"I am going to watch you for a few moments as you breathe. As you breathe in, I am going to say INHALE; as you breathe out, I am going to say RELAX. Do not try to fit your breathing in with my saying inhale and relax. I shall try to fit the words in naturally around your breathing. Breathe calmly and regularly, but all the time, be aware of your breathing."

g. INSTRUCTIONS TO PARTICIPANTS DURING THE DIFFERENTIAL RELAXATION #1 EXERCISE

"Remain in your relaxed position. Continue to breathe calmly and regularly. Now, keeping your eyes closed, I would like you to move your eyes as though you were looking around the room. The only tension that you should feel is that in the muscles around your eyes. When you move your eyes, you do not need to use any other muscles of the body. Check the rest of the body for muscle tension, and release any tension that you find. Return your eyes to their relaxed position, and continue to breathe calmly and

evenly. Now, in the same way, gently move your hands. When moving your hands, you need only experience tension in your arms as the arm muscles alone are responsible for movement of the hands. As you continue to breathe calmly and evenly, check the rest of your body for muscle tension. If you detect any tension in say the neck, shoulders or legs, release it. You only need to experience tension in the arms when moving the hands. Now return the hands to their relaxed position, and whilst breathing calmly and regularly, lift up your arms, and move them gently around. All the time scan the body for tension, and release any unnecessary tension.

Now, return the arms to their relaxed position, and breathe calmly and regularly for a few moments. Inhale; relax. Keep checking your body for tension as you begin to move your feet. Only the leg muscles are involved in the movement of the feet, so release any tension that you might find in the neck, shoulders or arms. Now, return the feet to their relaxed position, and gently move the legs. As before check that you are only experiencing tension in the muscles that are involved in the movement of the legs. All the time, continue to breathe calmly and evenly. Now, return the legs to their relaxed position. Scan the body for tension once more, and release any that you might find.

"Now, open your eyes, stand up and move to the wall. Do not lean against the wall, but be near enough to it should you feel the need to lean against it. Continue to breathe calmly and evenly. Scan your body for tension, and release any that you do not need. You will need some muscle tension in your legs as they are holding you up, but your neck, shoulders and arms may be relaxed. Now, as you continue to scan your body for tension, slowly move your eyes to look around the room. Note the tension in the muscles around your eyes. Release any tension that you experience in areas other than your eyes or legs. Return your eyes to their relaxed position, and continue to breathe calmly and evenly. As before, move your hands without tensing any other areas of your body; and now your arms - all the time scanning the body for tension and releasing any unnecessary muscle tension. Return the arms to their relaxed position, and once more, concentrate on your breathing: Inhale; Relax.

Now, one at a time, slowly move your feet and legs by lifting your heel from the ground in a walking motion. All the time, check the body for tension, and breathe calmly and regularly. Next increase the motion by lifting each foot from the floor and placing it a step in front. Then return the foot to its starting position.

Now, return to your seat. Scan the body for tension, and release any that you might find. When you feel that you are completely relaxed, take a deep breathe, open your eyes and feel completely refreshed."

h. ADDITIONAL INSTRUCTIONS TO PARTICIPANTS DURING THE DIFFERENTIAL RELAXATION #2 EXERCISE

"Now, one at a time, slowly move your feet and legs by lifting your heel from the ground in a walking motion. All the time, check the body for tension, and breathe calmly and regularly. Next, increase the motion by lifting each foot from the floor and placing it a step in front. Then return the foot to its starting position. When you feel that you can do this without unnecessary tension in your body, walk slowly around the room. As you do so, pay particular attention to breathing. Keep it calm and regular as you scan your body for tension..... Now return to your seat, and check that you are completely relaxed."

APPENDIX 2.7: Training instructions given to the "Autogenic Training" participants

a. A BRIEF HISTORY AND EXPLANATION OF AUTOGENIC TRAINING

"Autogenic training is a means of achieving a state of deep relaxation. It is a western form of meditation (or inward focusing of attention), but unlike other types of meditation such as Zen, it has no philosophical or religious overtones. It was brought to the west by Dr Wolfgang Luthe in the 1930's, but its origins lie in the work of Oscar Vogt at the turn of the century. Vogt observed that individuals could learn to put themselves in and out of an hypnotic state, and as a result, experienced relief from tension, fatigue and psychosomatic disorders. On the basis of this, a student of Vogt, Dr Johannes Schultz, developed Autogenic Training in the 1920's.

Briefly, Autogenic Training involves the learning and frequent repetition of a series of mental exercises that bring about deep psychophysiological relaxation, inner peace and calm, and an improved sense of well-being. The training is based on three concepts. Firstly that the individual focuses the mind inwardly. This is achieved through mental repetition of a series of verbal statements which encourage an "awareness" of particular parts of the body in sequence. This awareness is not in the form of an image of a given part of the body, but more of a feeling or sensation. The statements foster feelings of heaviness and warmth in limbs, calmness and regularity of heart beat, soothing abdominal warmth, self-regulation of breathing and coolness of the forehead. The warmth and heaviness exercises in particular are of interest in the treatment of Raynaud's Disease and Phenomenon as the sensation of heaviness is associated with deep muscular relaxation, and that of warmth with vasodilation of the peripheral arteries. The second recommendation is that a state of passive concentration is maintained during training. Passive concentration means concentrating on letting the relaxation happen rather than trying to control it; indeed trying too hard with autogenic training can only reduce one's chance of success. Lastly, specific postures are used which reduce distracting sensory input from the body and environment. This reduction in external distractions is particularly important during the initial learning stages, although with practice, the exercises can be done anywhere irrespective of environmental input.

The exercises consist of a series of phrases that relate mind to body. The phrases will be repeated in blocks of three with a termination between each repetition and at the end of the exercise. The phrases mention feelings of heaviness, warmth and calmness in particular areas of the body. It is important when doing the exercises to mentally associate the phrase with the body part mentioned. Having said that, do not try to visualise the area, rather, just be aware of it. Do not force the relaxation, but allow yourself to experience the sensations evoked by the phrases.

All exercises begin with a body check for tension. When a body part is named, check to see if there is any noticeable tension in it. If there is, adjust your position to reduce the tension.

The positive effects of Autogenic Training are often slow to surface; indeed, they may not appear at all during the initial training programme. As such, you need to be indifferent to your progress, and maintain a relaxed, detached attitude toward the training as a whole. The effects of training will emerge at their own pace. Having said that, during training you may notice 'Autogenic Discharges'. These are simply signs of the body "de-stressing" itself. For example, you may experience twitching of arms and legs; increased peristaltic movement; dizziness and visual/auditory effects. In some cases, hidden anxiety and tension may surface in the form of headaches, left chest pain, deep anger, or frustration."

b. THE "SHORT STITCH" EXERCISE WITH INSTRUCTIONS TO PARTICIPANTS

"The first "Short Stitch" exercise has two functions. Firstly, it will give you an idea of what to expect from Autogenic Training, and secondly, it is an exercise that you can use in place of later exercises at times of poor concentration. It concentrates on feelings of heaviness in the arm with the phrase 'My right arm is heavy'. We concentrate on the arms to start with because we need to apply the techniques of relaxation to areas that we use a lot first. In doing so, you will learn to associate the phrase 'My right arm is heavy' with a feeling of complete relaxation, so that in time, the phrase on its own will trigger relaxation."

The short stitch exercise - 3 repetitions

My Right arm is heavy x2
CANCEL

c. THE "HEAVINESS #1" EXERCISE WITH INSTRUCTIONS TO PARTICIPANTS

"The Heaviness #1 exercise is the first proper exercise, and the exercise that I will ask you to practice twice a day until our next meeting. The phrases should evoke feelings of heaviness in the limbs. As I have already mentioned, try not to visualise the limbs named, rather just be aware of them and the feelings of heaviness in them."

Heaviness #1 exercise - 3 repetitions
My right arm is heavy x1
My left arm is heavy x3
Both arms are heavy x3
My right leg is heavy x3
My left leg is heavy x3
Both legs are heavy x3
My arms and my legs are heavy x3
CANCEL

d. THE "HEAVINESS #2" EXERCISE WITH INSTRUCTIONS TO PARTICIPANTS

"It is possible to be mentally relaxed, and to feel physically relaxed, yet still have a large amount of tension in the neck and shoulders. As such, the next exercise introduces a phrase that helps reduce such tension, namely 'my neck and shoulders are heavy'. As you say the phrase, you may be aware of heaviness in your shoulders, and as the tension is released, you may feel your shoulders drop. In addition, at this point, we will introduce a phrase that describes the mood of a relaxed person: the phrase "I am at peace" sums up the sensations experienced when AT is working."

Heaviness #2 - 3 repetitions
My right arm is heavy
My arms and legs are heavy x3
My neck and shoulders are heavy x3
I am at peace x3
CANCEL

e. THE "WARMTH" EXERCISE WITH INSTRUCTIONS TO PARTICIPANTS

"As I mentioned before the training began, AT involves phrases that evoke feelings of heaviness and warmth. In this third exercise, the warmth phrases are introduced. The exercise is easy to remember because it combines the last two exercises, substituting "warmth" for "heaviness". One possible draw back of this is that the resultant exercise is quite long - taking up to 10 minutes to complete. Some people find this a bonus, but for those who do not, all later exercises will be shorter."

The warmth exercise - 3 repetitions
My right arm is heavy
My arms and legs are heavy x3
My right arm is warm x3
My left arm is warm x3
Both my arms are warm x3
My right leg is warm x3
My left leg is warm x3
Both my legs are warm x3
My arms and legs are warm x3
My neck and shoulders are heavy x3
I am at peace x3
CANCEL

f. THE "CARDIAC REGULATION AND BREATHING" EXERCISE WITH INSTRUCTIONS TO PARTICIPANTS

"By now, you may find that you are experiencing feelings of warmth and heaviness in each limb as you repeat the phrase that relates to it. The next exercise will enable you to experience these sensations in all four limbs at the same time through the repetition of a single phrase. In addition, we will bring in phrases to regulate heart beat, and to make you aware of your breathing. The phrase associated with heart regulation is "My heart beat is calm and regular" as a calm and regular heart beat is a sign of complete relaxation. The phrase for breathing is somewhat different as the object of Autogenic Training is not to control breathing. Consequently, a phrase such as "My breathing is calm and regular" would be inappropriate. Breathing in requires no effort. Once you have exhaled, air will naturally fill the lungs up again because the pressure in the lungs will be less than that of the environment. Awareness of breathing is important in Autogenic Training. We do not want to use a phrase that forces you to breathe in a particular way. Therefore, the phrase used is "It breathes me". This may sound a little strange at first, but it embodies the idea that you are not controlling your breathing. Each breath is happening by itself. I should make clear at this point that if you have ever had any heart problems or breathing difficulties, you should be careful when using these phrases. If you feel any discomfort, cancel out the exercise immediately and do not use the heart/breathing phrases in further practice of the exercises. This applies even if you have had no heart or breathing problems in the past. Sometimes, the breathing or heart beat becomes erratic on first experience of the phrases. It does not happen to everyone. You may experience no effects at all. If you do, stop the exercise immediately. The purpose of Autogenic training is not to make you feel uncomfortable."

The breathing and heart exercise - 3 repetitions
My right arm is heavy
My arms and legs are heavy and warm x6
My heart beat is calm and regular x3
It breathes me x3
My neck and shoulders are heavy x3
I am at peace x3
CANCEL

g. THE "SOLAR PLEXUS AND FOREHEAD" EXERCISE WITH INSTRUCTIONS TO PARTICIPANTS

"In this exercise, two further phrases are introduced. The first brings about a feeling of warmth in the abdomen; the second, coolness of the forehead. The abdominal warmth is conjured up through the phrase 'My solar plexus is warm'. The solar plexus is the nerve centre of the sympathetic nervous system, and is situated in the centre of the body between the spine and the stomach. As it is difficult to pin point its exact location, it may help to concentrate on either the stomach or the middle back when repeating the phrases. If you have a history of stomach ulcers, it would be wise to concentrate on the back rather than the stomach. You may then experience the relaxed feeling of sitting with your back against a warm fire. The second phrase is 'My forehead is cool'. With this, you should experience the feeling that you have when someone has blown on your forehead. Blow on your own forehead to refresh your memory. It may seem strange to suddenly jump from phrases that elicit feelings of warmth to those that evoke sensations of coolness. But, if you imagine yourself in a warm bath, you would feel uncomfortable if your head was too hot. It is nice to have a cool, clear head when the rest of your body is feeling warm and comfortable."

The forehead and solar plexus exercise - 3 repetitions
My right arm is heavy
My arms and legs are heavy and warm x3
My heart beat is calm and regular x3
It breathes me x3
My Solar Plexus is warm x3
My forehead is cool x3
My neck and shoulders are heavy x3
I am at peace x3
CANCEL

h. THE "ORGAN SPECIFIC FORMULAE" EXERCISE WITH INSTRUCTIONS TO PARTICIPANTS

"In this last session, we will introduce phrases to bring about warmth in the areas of your body that are affected by Raynaud's. So, for example if you only get Raynaud's symptoms in your fingers, the phrase that we will add is 'My fingers are warm and comfortable'. Of course, if several areas of the body are affected, there is the danger that the finished exercise will be too long. As such, the number of repetitions of each phrase (other than the Raynaud's specific phrases) is reduced to one and a short gap between each phrase is introduced. Having said that, the Raynaud's specific phrases will each be repeated three times. To further shorten the exercise, we can at this stage miss out phrases that you feel have no beneficial effects. So, for example, I am not comfortable with the breathing exercise, so I no longer practice it. Other people find the breathing exercise the most pleasant and beneficial phrase and therefore prefer to increase the repetitions of it from three to six. We need to work out a formula that suits you."

The organ specific formulae exercise - 3 repetitions
My right arm is heavy
My arms and legs are heavy and warm
My heart beat is calm and regular
It breathes me
My Solar Plexus is warm
My forehead is cool
ORGAN SPECIFIC FORMULAE X3
My neck and shoulders are heavy
I am at peace
CANCEL

APPENDIX 2.8: The Ambulatory Monitoring Diary coding frame

A. Subjective ambient temperature
1 = Predominantly in warm conditions (over 50 minutes of the hour)
eg. Inside warm building, outside in mild/warm weather, in a warm motor vehicle
2 = Predominantly in cold conditions (over 50 minutes of the hour)
eg. Inside cold building, outside in wintry weather, in a cold motor vehicle
3 = A mixture of the above (less than 50 minutes of the hour in either warm or cold conditions)

B.I Use of gloves/heating aids
1 = No gloves/heating aids during the hour
2 = gloves/heating aids for up to 20 minutes of the hour
2 = gloves/heating aids for up to 40 minutes of the hour
2 = gloves/heating aids for the full hour
B.II Time at which started wearing gloves/heating aids

C.I Raynaud's symptoms experienced during the last hour ?
1 = No
2 = Yes
C.II Time at which symptoms started in the last hour

D. Duration of Raynaud's symptoms of last hour
1 = No symptoms experienced during the last hour
2 = Symptoms lasted up to 10 minutes
3 = Symptoms lasted up to 20 minutes
4 = Symptoms lasted up to 30 minutes
5 = Symptoms lasted upto 40 minutes
6 = Symptoms lasted up to 50 minutes
7 = Symptoms lasted the full hour

E. Symptom severity (during the last hour)
0 = No symptoms experienced during the last hour
1 - 7 = severity rating (1 = least severe "attack" ever; 7 = Most severe attack ever experienced)

F. Nature of activity during the last hour.
1 = Predominantly sedentary (over 50 mins/hour) eg. reading, watching television, driving
2 = Predominantly active (over 50 mins/hour) eg. walking, shopping, cycling
3 = Both active and sedentary (less than 50 minutes active or sedentary activity)

G.I Use of stimulants (eg. Caffein, alcohol, nicotine) during the last hour
1 = No stimulants used during the last hour
2 = Stimulant use for up to 10 minutes
3 = Stimulant use for upto 20 minutes
4 = Stimulant use for up to 30 minutes
5 = Stimulant use for upto 40 minutes
6 = Stimulant use for up to 50 minutes
7 = Stimulant use for the full hour
G.II Time at which stimulant use started in last hour

H-O. Mood ratings on 7 point scale
0 = No response
1 - 7 = severity rating (where 1 = low; 7 = high)

THE STATISTICAL APPENDICES

INDEX: THE STATISTICAL APPENDICES

Statistical Appendix 1: The Raynaud's Questionnaire

1. *The Raynaud's Questionnaire*

a. The site of Raynaud's symptom manifestation: Chi-square analysis (see page 36)

Chi-Square	Value	DF	Significance
Pearson	13.37061	6	0.03751
Minimum expected Frequency - 0.500 Cells with Expected Frequency < 5 - 8 of 12 (66.7%)			

Table 1.a: Comparison of the site of symptom manifestation (FHF/FHFE/FHFEIO) across the diagnostic groups (Primary Raynaud's, Secondary Raynaud's, Unsure, Undiagnosed)

b. The age of symptom onset: Chi-square analysis (see page 38)

Chi-Square	Value	DF	Significance
Pearson	16.70747	6	0.01042
Minimum expected Frequency - 1.714 Cells with Expected Frequency < 5 - 5 of 12 (41.7%)			

Table 1.b: Comparison of the age of symptom onset (Childhood/<40 years/40+ years) across the diagnostic groups (Primary Raynaud's, Secondary Raynaud's, Unsure, Undiagnosed)

c. The duration of the condition prior to diagnosis: Chi-square analysis (see page 39)

Chi-Square	Value	DF	Significance
Pearson	11.68012	2	0.00291
Minimum expected Frequency - 3.367 Cells with Expected Frequency < 5 - 1 of 6 (16.7%)			

Table 1.c: Comparison of the duration of the condition prior to formal diagnosis (<1 year/>1 year) across the known diagnostic groups (Primary Raynaud's, Secondary Raynaud's)

Statistical Appendix 2.1: Pre-treatment analysis

1. *The Pre-treatment Questionnaire*

a. The subjective role of "stress" as a cause of symptoms: Chi-square analysis (see page 119)

Chi-Square	Value	DF	Significance
Pearson	11.14286	2	0.00381
Minimum expected Frequency - 3.333 Cells with Expected Frequency < 5 - 3 of 6 (50%)			

Table 1.a.I: Comparison of the role of "stress" as a subjective precipitator of Raynaud's symptoms (Yes or NO) between the Relaxation and Control groups

Chi-Square	Value	DF	Significance
Pearson	13.61250	3	0.00348
Minimum expected Frequency - 2.667 Cells with Expected Frequency < 5 - 4 of 8 (50%)			

Table 1.a.II: Comparison of the role of "stress" as a subjective precipitator of Raynaud's symptoms (Yes or NO) between the Biofeedback and Control groups

b. Previous experience of Relaxation Techniques: Chi-square analysis (page 119)

Chi-Square	Value	DF	Significance
Pearson	9.52381	3	0.02308
Minimum expected Frequency - 1.575 Cells with Expected Frequency < 5 - 4 of 8 (50%)			

Table 1.b.I: Comparison of previous experience of Relaxation techniques (Yes or NO) between the Biofeedback and Control groups

2. *Pre-treatment Voluntary Control of finger skin temperature*

a. Pre-treatment control of finger skin temperature: Analysis of covariance (page 119)

Source of Variation	Sum of Squares	DF	Mean Square	F	Sig of F
Covariates:	11.920	3	3.973	2.185	0.108
Room starting temp	10.648	1	10.648	5.855	0.021
Finger starting temp	0.142	1	0.142	0.078	0.781
Room temp difference	0.596	1	0.596	0.328	0.571
Main Effects:	22.105	2	11.053	6.078	0.006
Relaxation Groups	22.105	2	11.053	6.078	0.006
Explained	40.121	5	8.024	4.412	0.003
Residual	61.833	34	1.819		
Total	101.953	39	2.614		

Table 2.a.I: Comparison of pre-treatment control of finger skin temperature across the **Relaxation groups** (Autogenic Training, Applied Relaxation, No Relaxation)

3. *The pre-treatment Cold Challenge Test*

a. Recovery time to 6°C: Analysis of Co-variance (see page 122)

Source of Variation	Sum of Squares	DF	Mean Square	F	Sig of F
Covariates:	381882.208	2	19.941.104	1.350	0.273
Room starting temp	10625.764	1	10625.764	0.075	0.786
Finger starting temp	367231.865	1	387231.865	2.597	0.116
Main Effects:	142819.111	2	71409.555	0.505	0.608
Relaxation Groups	142819.111	2	71409.555	0.505	0.608
Explained	479199.754	4	119799.939	0.847	0.505
Residual	4807192.533	34	141388.016		
Total	5286392.308	38	139115.587		

Table 3.a.I: Comparison of pre-treatment recovery to 6°C across the **Relaxation groups** (Autogenic Training, Applied Relaxation, No Relaxation)

Source of Variation	Sum of Squares	DF	Mean Square	F	Sig of F
Covariates:	246346.525	2	123173.262	0.915	0.410
Room starting temp	6005.923	1	6005.923	0.045	0.834
Finger starting temp	237999.071	1	237999.071	1.768	0.193
Main Effects:	371889.505	2	185944.752	1.381	0.265
Biofeedback Groups	371889.505	2	185944.752	1.381	0.265
Explained	7.08270.148	4	177067.537	1.315	0.284
Residual	4578122.160	34	134650.652		
Total	5286392.308	38	139115.587		

Table 3.a.II: Comparison of pre-treatment recovery to 6°C across the **Biofeedback groups** (No biofeedback, Temperature biofeedback, EMG biofeedback)

4. *Ambulatory Temperature Monitoring*

a. Preferred environment during monitoring: Chi-square analysis (page 123)

Chi-Square	Value	DF	Significance
Pearson	12.63690	6	0.04918
Minimum expected Frequency - 0.100 Cells with Expected Frequency < 5 - 10 of 12 (83.3%)			

Table 4.a.I: Comparison of reported warmth of ambient conditions during monitoring (warm or cold) across the Diagnostic Groups (Primary Raynaud's, Secondary Raynaud's, Unsure, Undiagnosed)

b. **Finger skin temperature during monitoring**: analysis of Co-variance (page 124)

Source of Variation	Sum of Squares	DF	Mean Square	F	Sig of F
Covariates:	142.081	1	142.081	11.941	0.002
Ambient Temperature	142.081	1	142.081	11.941	0.002
Main Effects:	21.714	2	10.857	0.912	0.412
Relaxation Groups	21.714	2	10.857	0.912	0.412
Explained	143.310	3	47.770	4.015	0.016
Residual	380.755	32	11.899		
Total	524.065	35	14.899		

Table 4.b.I: Comparison of pre-treatment middle finger skin temperature during monitoring across the **Relaxation groups** (Autogenic Training, Applied Relaxation, No Relaxation)

Source of Variation	Sum of Squares	DF	Mean Square	F	Sig of F
Covariates:	113.932	1	113.932	9.210	0.005
Ambient Temperature	113.932	1	113.932	9.210	0.005
Main Effects:	6.595	2	3.297	0.267	0.786
Biofeedback Groups	6.595	2	3.297	0.267	0.786
Explained	128.191	3	42.730	3.454	0.028
Residual	395.875	32	12.371		
Total	524.065	35	14.973		

Table 4.b.II: Comparison of pre-treatment middle finger skin temperature during monitoring across the **Biofeedback groups** (No biofeedback, Temperature biofeedback, EMG biofeedback)

Statistical Appendix 2.2: Post-treatment and follow-up Questionnaires

1. *The Post-treatment Questionnaire*

a. Subjective improvement in Raynaud's symptoms: Chi-square analysis (page 146)

Chi-Square	Value	DF	Significance
Pearson	8.22572	2	0.01636
Minimum expected Frequency - 3.355 Cells with Expected Frequency < 5 - 3 of 6 (50%)			

Table 1.a.I: Comparison of reported symptomatic improvement (improved; not improved) between the Relaxation and Control groups

Chi-Square	Value	DF	Significance
Pearson	15.22453	3	0.0163
Minimum expected Frequency -2.935 Cells with Expected Frequency < 5 - 7 of 8 (87.5%)			

Table 1.a.II: Comparison of reported symptomatic improvement (improved; not improved) between the Biofeedback and Control groups

b. Subjective views of alternative treatments : Chi-square analysis (page 147)

Chi-Square	Value	DF	Significance
Pearson	13.33333	2	0.00127
Minimum expected Frequency - 0.400 Cells with Expected Frequency < 5 - 3 of 6 (50%)			

Table 1.b.I: Comparison of views of alternative treatments (positive or negative) between those reporting or not reporting symptom improvement

c. Reported symptom improvement and subjective role of "stress" on symptoms: Chi-square analysis (page148)

Chi-Square	Value	DF	Significance
Pearson	9.12294	2	0.00252
Minimum expected Frequency - 5.107 Cells with Expected Frequency < 5 - 0 of 4 (0%)			

Table 2.c.I: Comparison of views of "stress" as a precipitator of Raynaud's symptoms (Yes or No) and reported symptom status (improved or not improved).

2. *The Follow-up Questionnaire*

a. Subjective improvement in Raynaud's symptoms: Chi-square analysis (page 149)

Chi-Square	Value	DF	Significance
Pearson	6.50384	2	0.03870
Minimum expected Frequency - 2.750 Cells with Expected Frequency < 5 - 4 of 6 (66.7%)			

Table 2.a.I: Comparison of reported symptomatic improvement (improved: not improved) between the Relaxation and Control groups

Chi-Square	Value	DF	Significance
Pearson	10.95544	3	0.01197
Minimum expected Frequency - 2.357 Cells with Expected Frequency < 5 - 8 of 8 (100%)			

Table 2.a.II: Comparison of rates of reported symptomatic improvement (improved: not improved) between the Biofeedback and Control groups

b. Reported symptom improvement and subjective role of "stress" on symptoms: Chi-square analysis (page 150)

Chi-Square	Value	DF	Significance
Pearson	6.14668	2	0.0467
Minimum expected Frequency - 2.593 Cells with Expected Frequency <5 - 4 of 6 (66.7%)			

Table 2.b.I: Comparison of views of "stress" as a precipitator of Raynaud's symptoms (Yes or No) and reported symptom status (improved or not improved).

Statistical Appendix 2.3: The comparison of pre-treatment, post-treatment and follow-up data

1. ***The Voluntary Control of finger skin temperature*** (pages154-171)

a. **Main Analysis**: incomplete blocks design split plot analysis of co-variance with two empty cells

Source of Variation		DF	Seq SS	MS	F-ratio	P-value
Covariates						
Room Starting Temp.		1	5.6938	5.6938	1.8626	NS
Middle finger starting temp.		1	15.7280	15.7280	5.1451	<0.05
Room Temperature		1	43.4721	43.721	14.302	<0.001
Main Effects						
Biofeedback		2	17.8807	8.940	2.924	NS
Relaxation		2	10.5840	5.292	1.7312	NS
Time (pre/post/followup)		2	0.7138	0.3569	0.1449	NS
Interaction Effects						
Biofeedback by Relaxation		2	11.0954	5.5477	1.5544	NS
Biofeedback by Time		4	16.6786	4.1696	1.6928	NS
Relaxation by Time		4	23.5777	5.8940	2.3930	NS
Biofeedback by Relaxation by Time		4	5.0030	1.2507	0.5077	NS
Error Terms						
Between Subjects error term		33	100.8787	3.0569		
Within Subjects error term		41	100.9833	2.4630		
Total		97	352.2891			

Table 1.a.I: The effect of **Relaxation** on **voluntary control** of middle finger temperature across **Time of Testing** (pre-treatment, post-treatment, follow-up sessions), **Relaxation group** (Autogenic Training, applied Relaxation, No Relaxation), and **Biofeedback group** (No Biofeedback, Temperature biofeedback, EMG biofeedback) covarying room and finger starting temperatures and room temperature difference Page 154-158.

Source of Variation		DF	Seq SS	MS	F-ratio	P-value
Covariates						
Room Starting Temp.		1	5.6938	5.6938	1.8626	NS
Middle finger starting temp.		1	15.7280	15.7280	5.1451	<0.05
Room Temperature		1	43.4721	43.721	14.302	<0.001
Main Effects						
Relaxation		2	11.5567	5.7780	1.8900	NS
Biofeedback		2	16.9080	8.4540	2.7655	NS
Time (pre/post/followup)		2	0.7138	0.3569	0.1449	NS
Interaction Effects						
Relaxation by Biofeedback		2	11.0954	5.5477	1.5544	NS
Relaxation by Time		4	26.1306	6.5326	2.6522	<0.05
Biofeedback by Time		4	14.1256	3.5314	1.4337	NS
Relaxation by Biofeedback by Time		4	5.0030	1.2507	0.5077	NS
Error Terms						
Between Subjects error term		33	100.8787	3.0569		
Within Subjects error term		41	100.9833	2.4630		
Total		97	352.2891			

Table 1.a.II: The effect of **Biofeedback** on **voluntary control** of middle finger temperature across **Time of Testing** (pre-treatment, post-treatment, follow-up sessions), **Relaxation group** (Autogenic Training, applied Relaxation, No Relaxation), and **Biofeedback group** (No Biofeedback, Temperature biofeedback, EMG biofeedback) covarying room and finger starting temperatures and room temperature difference Pages 154-158.

Covariate	B	Beta	S.E	t-value	sig of t	95% CI for Diff
Room starting temperature	0.34841	0.1226	0.158	2.209	0.030	0.034, 0.663
Middle finger starting temperature	-0.0995	-0.2250	0.049	-2.034	0.046	-0.197, -0.002
Room temperature difference	-3.9687	-0.0326	1.197	-3.314	0.001	-6.355, -1.583

Table 1.a.III: Regression analysis for the within and residual error term: The role of the covariates (Room and middle finger starting temperatures and room temperature difference) on the middle finger skin temperature difference during the test of voluntary control Page 155.

b. **The *A priori* Analyses**: Univariate comparisons (pages 158-166)

1.b.I. The Controls versus the Pooled Experimental Groups

Source	Sum of Squares	df	Mean Square	F-ratio	P value
Corrected Model	20.403	4	5.101	2.189	0.090
Intercept	8.833	1	8.833	3.791	0.060
Covariates:					
Room Temperature	3.721	1	3.721	1.597	0.215
Finger Starting Temp	0.030	1	0.030	0.013	0.910
Room Starting Temp	11.622	1	11.622	4.988	0.032
Main Effects Experimental Groups Vs Controls	2.387	1	2.387	1.025	0.318
Error	81.551	35	2.330		
Total	111.884	40			
Corrected Total	101.953	39			

Table 1.b.I.i: An analysis of covariance comparing the **pooled experimental groups** and **the Controls** in control of **pre-treatment** finger skin temperature P158

Source	Sum of Squares	df	Mean Square	F-ratio	P value
Corrected Model	83.840	4	20.960	9.168	0.000
Intercept	0.169	1	0.169	0.074	0.788
Covariates:					
Room Temperature	64.217	1	64.217	28.090	0.000
Finger Starting Temp	1.825	1	1.825	0.798	0.380
Room Starting Temp	0.349	1	0.349	0.153	0.699
Main Effects Experimental Groups Vs Controls	0.397	1	0.397	0.174	0.680
Error	57.154	25	2.286		
Total	155.919	30			
Corrected Total	140.994	29			

Table 1.b.I.ii: An analysis of covariance comparing the **pooled experimental groups** and **the Controls** in control of **post-treatment finger skin temperature** P159

Source	Sum of Squares	df	Mean Square	F-ratio	P value
Corrected Model	15.480	4	3.870	0.971	0.442
Intercept	0.229	1	0.229	0.057	0.813
Covariates:					
Room Temperature	0.434	1	0.434	0.109	0.744
Finger Starting Temp	11.172	1	11.172	2.804	0.108
Room Starting Temp	0.506	1	0.506	0.127	0.725
Main Effects Experimental Groups Vs Controls	6.302	1	6.302	1.582	0.221
Error	91.643	23	3.984		
Total	109.895	28			
Corrected Total	107.123	27			

Table 1.b.I.iii: An analysis of covariance comparing the **pooled experimental groups** and **the Controls** in control of **follow up finger skin temperature** Page 159

1.b.II. The Biofeedback (ATT, ATE, ART, ARE) versus the No Biofeedback (AT, AR) experimental groups

Source	Type III Sum of Squares	DF	Mean Square	F - ratio	P-value
Corrected Model	15.854	4	3.963	1.294	0.299
Intercept	8.680	1	8.680	2.835	0.105
Covariates					
Room starting temperature	13.320	1	13.320	4.351	0.047
Room Temperature	5.947	1	5.947	1.943	0.176
Middle finger starting temperature	0.651	1	0.651	0.213	0.649
Main Effects Biofeedback versus no Biofeedback	1.038	1	1.038	0.339	0.566
Error	76.543	25	3.062		
Total	108.501	30			
Corrected Total	92.397	29			

Table 1.b.II.i: An analysis of covariance comparing the Biofeedback (ATT, ATE, ART, ARE) and the No Biofeedback (AT, AR) experimental groups in the control of finger skin temperature during the pre-treatment assessment session. Page 160

Source	Type III Sum of Squares	DF	Mean Square	F - ratio	P-value
Corrected Model	87.354	4	21.838	9.803	0.000
Intercept	0.361	1	0.361	0.162	0.692
Covariates					
Room starting temperature	0.854	1	0.854	0.384	0.544
Room Temperature	66.234	1	66.234	29.731	0.000
Middle finger starting temperature	6.769	1	6.769	3.039	0.099
Main Effects					
Biofeedback versus no Biofeedback	0.385	1	0.385	0.173	0.683
Error	37.871	17	2.228		
Total	140.968	22			
Corrected Total	125.225	21			

Table 1.b.II.ii: An analysis of covariance comparing the Biofeedback (ATT, ATE, ART, ARE) and the No Biofeedback (AT, AR) experimental groups in the control of finger skin temperature during the post-treatment assessment session. Page 160

Source	Type III Sum of Squares	DF	Mean Square	F - ratio	P-value
Corrected Model	14.785	4	3.696	0.680	0.616
Intercept	0.686	1	0.686	0.126	0.727
Covariates					
Room starting temperature	0.274	1	0.274	0.050	0.825
Room Temperature	0.162	1	0.162	0.030	0.865
Middle finger starting temperature	7.998	1	7.998	1.471	0.243
Main Effects					
Biofeedback versus no Biofeedback	1.526	1	1.526	0.281	0.604
Error	87.008	16	5.438		
Total	107.014	21			
Corrected Total	101.793	20			

Table 1.b.II.iii: An analysis of covariance comparing the Biofeedback (ATT, ATE, ART, ARE) and the No Biofeedback (AT, AR) experimental groups in the control of finger skin temperature during the follow-up assessment session. Page 161

1.b.III. The Controls versus the Individual Experimental Groups

	AT v. Controls	ATT v. Controls	ATE v. Controls	AR v. Controls	ART v. Controls	ARE v. Controls
Contrast Estimate	-0.0747	-0.318	0.0210	1.933	1.373	1.360
Hypothesised value	0	0	0	0	0	0
Difference (Estimate - Hypothesised)	-0.0747	-0.318	0.0210	1.933	1.373	1.360
Std. Error	0.820	0.815	0.791	0.864	0.838	0.886
P-value	0.928	0.699	0.979	0.033	0.112	0.135
95% CI	-1.750, 1.601	-1.983, 1.347	-1.595, 1.637	0.169, 3.697	-0.339, 3.084	-0.449, 3.170

Table 1.b.III.i: Comparisons of the individual treatment groups with the Control group in control over finger skin temperature at the pre-treatment assessment session. Pages161-166

	AT v. Controls	ATT v. Controls	ATE v. Controls	AR v. Controls	ART v. Controls	ARE v. Controls
Contrast Estimate	-0.463	-0.759	0.967	1.089	-0.416	0.510
Hypothesised value	0	0	0	0	0	0
Difference (Estimate - Hypothesised)	-0.463	-0.759	0.967	1.089	-0.416	0.510
Std. Error	1.209	1.103	0.920	0.943	1.113	0.942
P-value	0.706	0.500	0.306	0.262	0.713	0.594
95% CI	-2.984, 2.058	-3.060, 1.543	-0.952, 2.885	-0.877, 3.056	-2.738, 1.907	-1.456, 2.476

Table 1.b.III.ii: Comparisons of the individual treatment groups with the Control group in control over finger skin temperature at the post-treatment assessment session. Pages161-166

	AT v. Controls	ATT v. Controls	ATE v. Controls	AR v. Controls	ART v. Controls	ARE v. Controls
Contrast Estimate	0.374	1.346	2.924	0.830	-0.182	1.032
Hypothesised value	0	0	0	0	0	0
Difference (Estimate - Hypothesised)	0.374	1.346	2.924	0.830	-0.182	1.032
Std. Error	1.492	1.772	1.227	1.303	1.237	1.534
P-value	0.805	0.457	0.028	0.532	0.885	0.510
95% CI	-2.761,3.508	-2.376,5.068	0.340,5.502	-1.906,3.567	-2.780,2.417	-2.191,4.255

Table 1.b.III.iii: Comparisons of the individual treatment groups with the Control group in control over finger skin temperature at the follow-up assessment session. Pages161-166

c. The Post Hoc Analyses: Scheffé multiple range tests (page 167-168)

1.c.I: Comparison 1

Relaxation Group combinations	Mean Difference	Std. Error	P-value	95% Confidence Interval	
				Lower Bound	Upper Bound
AT x AR	-1.0021388	0.3151142	0.011	-1.8057633	-0.1985143
AT x No Relaxation	-0.1521709	0.3523084	0.911	-1.0506504	0.7463086
AR X No Relaxation	0.8499679	0.3523084	0.067	-0.0485	1.7484474

Table1.c.I: A univariate analysis comparison of the Relaxation group's mean residual scores for difference in finger skin temperature during the test of voluntary control during the pre-treatment assessment session Page 167

1.c.II: Comparison 2

Source	Time	Type III Sum of Squares	df	Mean Square	F-ratio	P-Value
Time	Pre-treatment vs. Follow up	8.058	1	8.058	4.799	0.056
	Post-treatment vs. Follow up	2.688	1	2.688	1.140	0.313
Error (Time)	Pre-treatment vs. Follow up	15.111	9	1.679		
	Post-treatment vs. Follow up	21.209	9	2.357		

Table 1.c.II: A within subjects comparison of the AT group mean residual scores for difference in finger skin temperature during the test of voluntary control over the three periods of assessement. Page 168

1.c.III: Comparison 3

Pre-treatment AR vs. Follow up Control	t-test for Equality of Means			
	t	df	P-Value (2-tailed)	Mean Difference
Equal variances assumed	2.022	20	0.057	0.9765
Equal variances not assumed	2.844	17.475	0.011	0.9765

Table 1.c.III: An independent samples t-test comparing the pre-treatment AR group residual finger skin temperature difference scores with the Control group follow up scores Page 168

d. Post-treatment and Follow-up Flexi Carpi Ulnaris EMG measurements during voluntary control : Analyses of covariance (pages 170-171)

Source of Variation	Sum of Squares	DF	Mean Square	F	Sig of F
Within and Residual	48172.53	23	2094.46		
Regression	3180.69	3	1060.23	0.51	0.682
Biofeedback group	4796.10	2	2398.05	1.14	0.336
(Model)	8702.23	5	1740.45	0.83	0.541
(Total)	56874.76	28	2031.24		
R- squared = 0.153; Adjusted R-squared = 0.000					

Table 1.d.I: Comparison of **post-treatment EMG** levels during voluntary control across the **Biofeedback groups** (No Biofeedback, Temp Biofeedback, EMG biofeedback) covarying starting EMG levels, starting room temperature and room temperature Page 170

Source of Variation	Sum of Squares	DF	Mean Square	F	Sig of F
Within and Residual	47072.85	23	2046.65		
Regression	5865.04	3	1955.01	0.96	0.430
Biofeedback group	5895.78	2	2947.89	1.44	0.257
(Model)	9801.91	5	1960.38	0.96	0.464
(Total)	56874.76	28	2031.24		
R- squared = 0.172; Adjusted R-squared = 0.000					

Table 1.d.II: Comparison of **post-treatment EMG** levels during voluntary control across the **Relaxation groups** (Autogenic Training, Applied Relaxation, No relaxation) covarying starting EMG levels, starting room temperature and room temperature Page 170

Source of Variation	Sum of Squares	DF	Mean Square	F	Sig of F
Within and Residual	43585.16	16	2724.07		
Regression	8541.12	3	2847.04	1.05	0.400
Biofeedback group	10910.26	2	5455.13	2.00	0.167
(Model)	28892.84	5	5778.57	2.12	0.116
(Total)	72478.00	21	3451.33		
R- squared = 0.399; Adjusted R-squared = 0.211					

Table 1.d.III: Comparison of **Follow-up EMG** levels during voluntary control across the **Biofeedback groups** (No Biofeedback, Temp Biofeedback, EMG biofeedback) covarying starting EMG levels, starting room temperature and room temperature Page 171

Source of Variation	Sum of Squares	DF	Mean Square	F	Sig of F
Within and Residual	44332.36	16	2770.77		
Regression	23294.64	3	7764.88	2.80	0.073
Biofeedback group	10163.05	2	5081.52	1.83	0.192
(Model)	28145.64	5	5629.13	2.03	0.129
(Total)	72478.00	21	3451.33		
R- squared = 0.388; Adjusted R-squared = 0.197					

Table 1.d.IV: Comparison of **Follow-up EMG** levels during voluntary control across the **Relaxation groups** (Autogenic Training, Applied Relaxation, No relaxation) covarying starting EMG levels, starting room temperature and room temperature Page 171

2. ***The Cold Challenge Test*** (pages 172-183)

a. **Main Analysis**: incomplete blocks design split plot analysis of co-variance with two empty cells

Source of Variation		DF	Seq SS	MS	F-ratio	P-value
Covariates						
Room Temperature.		1	3220175	3220175	17.526	<0.001
Middle finger starting temp.		1	3914884	3914884	21.307	<0.001
Main Effects						
Biofeedback		2	78326	39163	0.213	NS
Relaxation		2	828042	414021	2.253	NS
Time (pre/post/followup)		2	856941	428471	2.144	NS
Interaction Effects						
Biofeedback by Relaxation		2	782868	391434	2.1305	NS
Biofeedback by Time		4	504532	126133	0.631	NS
Relaxation by Time		4	486581	121645	0.6088	NS
Biofeedback by Relaxation by Time		4	825293	206323	1.0326	NS
Error Terms						
Between Subjects error term		32	5879532	183735		
Within Subjects error term		41	8192175	199809		
Total		95	25569349			

Table 2.a.I: The effect of **Relaxation** on **Recovery by 6°C** of middle finger skin temperature across **Time of Testing** (pre-treatment, post-treatment, follow-up sessions), **Relaxation group** (Autogenic Training, applied Relaxation, No Relaxation), and **Biofeedback group** (No Biofeedback, Temperature biofeedback, EMG biofeedback) covarying room and finger starting temperatures Page 172-175

Source of Variation		DF	Seq SS	MS	F-ratio	P-value
Covariates						
Room Temperature.		1	3220175	3220175	17.526	<0.001
Middle finger starting temp.		1	3914884	3914884	21.307	<0.001
Main Effects						
Relaxation		2	496892	248446	1.352	NS
Biofeedback		2	409476	204738	1.114	NS
Time (pre/post/followup)		2	856941	428471	2.144	NS
Interaction Effects						
Relaxation by Biofeedback		2	782868	391434	2.1305	NS
Relaxation by Time		4	350971	87743	0.4775	NS
Biofeedback by Time		4	640141	160035	0.8009	NS
Biofeedback by Relaxation by Time		4	825293	206323	1.0326	NS
Error Terms						
Between Subjects error term		32	5879532	183735		
Within Subjects error term		41	8192175	199809		
Total		95	25569349			

Table 2.a.II: The effect of **Biofeedback** on **Recovery by 6°C** of middle finger skin temperature across **Time of Testing** (pre-treatment, post-treatment, follow-up sessions), **Relaxation group** (Autogenic Training, applied Relaxation, No Relaxation), and **Biofeedback group** (No Biofeedback, Temperature biofeedback, EMG biofeedback) covarying room and finger starting temperatures Page 172-175

Covariate	B	Beta	S.E	t-value	sig of t	95% CI for Diff
Middle finger starting temperature	-38.173	-0.3033	12.888	-2.962	0.004	-63.860, -12.487
Room temperature difference	-181.43	-0.4830	49.454	-3.669	0.000	-279.99, -82.874

Table 2.a.III: Regression analysis for the within and residual error term: The role of the covariates (middle finger starting temperatures and room temperature difference) on the middle finger skin temperature difference during the cold challenge test Page 172.

b. **The *A priori* Analyses**: Scheffé pair wise comparisons (pages 176-183)

2.b.i: The Controls vs the Pooled Experimental Groups

Source	Sum of Squares	df	Mean Square	F-ratio	P value
Corrected Model	3297597.2	3	1099199.1	7.123	0.001
Intercept	2467742.5	1	2467742.5	15.992	0.000
Covariates:					
Room Temperature	856122.84	1	856122.84	5.548	0.024
Finger Starting Temp	2120566.5	1	2120566.5	13.742	0.001
Main Effects					
Experimental Groups Vs Controls	229358.66	1	229358.66	1.486	0.231
Error	5400879.8	35	154310.85		
Total	33395175.0	39			
Corrected Total	8698476.9	38			

Table 2.b.I.i: An analysis of covariance comparing the pooled experimental groups and the Controls in pre-treatment rewarming following cold challenge Page 176

Source	Sum of Squares	df	Mean Square	F-ratio	P value
Corrected Model	2354253.1	3	784751.03	3.644	0.026
Intercept	2496928.4	1	249628.4	11.593	0.002
Covariates:					
Room Temperature	1300806.6	1	1300806.6	6.040	0.021
Finger Starting Temp	154261.49	1	154261.49	0.716	0.405
Main Effects					
Experimental Groups Vs Controls	64610.896	1	64610.896	0.300	0.589
Error	5384517.6	25	215380.70		
Total	30038250.0	29			
Corrected Total	7738770.7	28			

Table 2.b.I.ii: An analysis of covariance comparing the pooled experimental groups and the Controls in rewarming following post-treatment cold challenge Page 176

Source	Sum of Squares	df	Mean Square	F-ratio	P value
Corrected Model	2780732.3	3	926910.77	3.666	0.026
Intercept	2601577.0	1	2601577.0	10.288	0.004
Covariates:					
Room Temperature	1482758.2		1482758.2	5.864	0.023
Finger Starting Temp	1009859.3	1	1009859.3	3.994	0.057
Main Effects					
Experimental Groups Vs Controls	81028.835	1	81028.835	0.320	0.577
Error	6068948.9	24	252872.87		
Total	32742475.0	28			
Corrected Total	8849681.3	27			

Table 2.b.I.iii: An analysis of covariance comparing the pooled experimental groups and the Controls in cold challenge rewarming at follow up. Page 177

2.b.II: The Biofeedback (ATT, ATE, ART, ARE) versus the No Biofeedback (AT, AR) Experimental groups

Source	Type III Sum of Squares	DF	Mean Square	F - ratio	P-value
Corrected Model	1671378.8	3	557126.27	4.601	0.011
Intercept	621617.49	1	621617.49	5.134	0.032
Covariates					
Room Temperature	109716.35		109716.35	0.906	0.350
Finger starting temperature	893368.43	1	893368.43	7.378	0.012
Main Effects					
Biofeedback versus no Biofeedback	260534.15	1	260534.15	2.152	0.155
Error	3027002.2	25	121080.09		
Total	20467975	29			
Corrected Total	4698381.0	28			

Table 2.b.II.i: An analysis of covariance comparing the Biofeedback (ATT, ATE, ART, ARE) and the No Biofeedback (AT, AR) experimental groups' rewarming times following Cold Challenge during the pre-treatment assessment session. Page 177

Source	Type III Sum of Squares	DF	Mean Square	F - ratio	P-value
Corrected Model	2343979.1	3	781326.36	3.042	0.057
Intercept	2323605.9	1	2323605.9	9.046	0.008
Covariates					
Room Temperature	1444926.8	1	1444926.8	5.625	0.030
Finger Starting Temperature	89817.275	1	89817.275	0.350	0.562
Main Effects Biofeedback versus no Biofeedback	476467.52	1	476467.52	1.855	0.191
Error	4366751.9	17	256867.76		
Total	23684750	21			
Corrected Total	6710731.0	20			

Table 1.b.II.ii: An analysis of covariance comparing the Biofeedback (ATT, ATE, ART, ARE) and the No Biofeedback (AT, AR) experimental groups' rewarming times following Cold Challenge during the post-treatment assessment session. Page178

Source	Type III Sum of Squares	DF	Mean Square	F - ratio	P-value
Corrected Model	3767102.8	3	1255700.9	6.730	0.003
Intercept	2470179.2	1	2470179.2	13.240	0.002
Covariates					
Room Temperature	1416719.6	1	1416719.6	7.593	0.014
Finger Starting Temperature	1360873.1	1	1360873.1	7.794	0.015
Main Effects Biofeedback versus no Biofeedback	35269.118	1	35269.118	0.189	0.669
Error	3171754.3	17	186573.78		
Total	24383600	21			
Corrected Total	6938857.1	20			

Table 1.b.II.iii: An analysis of covariance comparing the Biofeedback (ATT, ATE, ART, ARE) and the No Biofeedback (AT, AR) experimental groups' rewarming times following Cold Challenge during the follow up assessment session. Page 178

2.b.III The Controls versus the Individual Experimental Groups

	AT v. Controls	ATT v. Controls	ATE v. Controls	AR v. Controls	ART v. Controls	ARE v. Controls
Contrast Estimate	-252.891	-86.880	-167.365	-345.624	-167.808	-66.548
Hypothesised value	0	0	0	0	0	0
Difference (Estimate - Hypothesised)	-252.891	-86.880	-167.365	-345.624	-167.808	-66.548
Std. Error	246.117	234.522	228.675	236.419	229.678	231.157
P-value	0.312	0.714	0.470	0.154	0.471	0.775
95% CI	-755.5,249.8	-565.8,392.1	-634.4,299.7	-828.5,137.2	-636.9,301.3	-538.6,405.5

Table2.b.III.i: Comparisons of the Individual Treatment groups with the Control group in rewarming following Cold Challenge at the pre-treatment assessment session. Pages179 - 183

	AT v. Controls	ATT v. Controls	ATE v. Controls	AR v. Controls	ART v. Controls	ARE v. Controls
ontrast stimate	369.078	88.738	-146.952	-643.571	-10.812	-56.054
lypothesised alue	0	0	0	0	0	0
)ifference Estimate - lypothesised)	369.078	88.738	-146.952	-643.571	-10.812	-56.054
td. Error	385.567	292.526	250.267	297.825	315.295	277.479
-value	0.350	0.765	0.564	0.043	0.973	0.842
5% CI	-435.2,1173.4	-521.4,698.9	-669.0,375.1	-1264.8,-22.3	-668.5,646.9	-634.9,522.8

Table 2.b.III.ii: Comparisons of the Individual Treatment groups with the Control group in rewarming following Cold Challenge at the post-treatment assessment session. Pages 179-183

	AT v. Controls	ATT v. Controls	ATE v. Controls	AR v. Controls	ART v. Controls	ARE v. Controls
'ontrast stimate	-87.263	-509.127	-69.768	-521.505	217.355	-417.466
[ypothesised alue	0	0	0	0	0	0
iifference Estimate - ypothesised)	-87.263	-509.127	-69.768	-521.505	217.355	-417.466
td. Error	364.356	439.426	329.485	324.786	320.005	383.093
-value	0.813	0.261	0.835	0.125	0.505	0.289
5% CI	-849.9,675.4	-1428.8,410.6	-759.4,619.9	-1201.3,158.3	-452.4,887.1	-1219.2,384.4

Table 2.b.III.iii: Comparisons of the Individual Treatment groups with the Control group in rewarming following Cold Challenge at the follow-up assessment session. Pages179-183

3. ***AMBULATORY MONITORING*** (pages 184-198)

a. **Main Analysis**: incomplete blocks design split plot analysis of co-variance with two empty cells

Source of Variation		**DF**	**Seq SS**	**MS**	**F-ratio**	**P-value**
Covariates						
Ambient Temperature.		1	269.930	269.930	15.949	<0.001
Main Effects						
Biofeedback		2	13.079	6.9395	0.410	NS
Relaxation		2	82.495	41.2475	2.437	NS
Time (pre/post/followup)		2	79.042	39.521	6.654	<0.01
Interaction Effects						
Biofeedback by Relaxation		2	149.763	74.8815	4.424	<0.025
Biofeedback by Time		4	120.459	30.1148	5.0706	<0.01
Relaxation by Time		4	13.437	3.359	0.5655	NS
Biofeedback by Relaxation by Time		4	17.453	4.363	0.734	NS
Error Terms						
Between Subjects error term		30	507.710	16.9236		
Within Subjects error term		37	219.778	5.939		
Total		88	1473.148			

Table 3.a.I: Comparison of middle finger skin temperature during Ambulatory Monitoring across **Time of Testing** (pre-treatment, post-treatment, follow-up sessions), **Relaxation group** (Autogenic Training, applied Relaxation, No Relaxation), and **Biofeedback group** (No Biofeedback, Temperature biofeedback, EMG biofeedback) covarying ambient temperature Pages 184-187

Source of Variation		DF	Seq SS	MS	F-ratio	P-value
Covariates						
Ambient Temperature.		1	269.930	269.930	15.949	<0.001
Main Effects						
Relaxation		2	33.395	16.6975	0.9866	NS
Biofeedback		2	62.179	31.0895	1.837	NS
Time (pre/post/followup)		2	79.042	39.521	6.654	<0.01
Interaction Effects						
Relaxation by Biofeedback		2	149.763	74.8815	4.424	<0.025
Relaxation by Time		4	14.675	3.6687	0.6177	NS
Biofeedback by Time		4	119.220	29.805	5.018	<0.01
Biofeedback by Relaxation by Time		4	17.453	4.363	0.734	NS
Error Terms						
Between Subjects error term		30	507.710	16.9236		
Within Subjects error term		37	219.778	5.939		
Total		88	1473.148			

Table 3.a.II: Comparison of middle finger skin temperature during Ambulatory Monitoring across **Time of Testing** (pre-treatment, post-treatment, follow-up sessions), **Biofeedback group** (No Biofeedback, Temperature biofeedback, EMG biofeedback), and **Relaxation group** (Autogenic Training, Applied Relaxation, No Relaxation) covarying ambient temperature Pages 184-187

Covariate	B	Beta	S.E	t-value	sig of t	95% CI for Diff
Ambient temperature	0.54240	0.43324	0.124	4.376	0.000	0.295, 0.790

Table 3.a.III: Regression analysis for the within and residual error term: The role of the covariate (ambient temperature) on the middle finger skin temperature difference during the cold challenge test Page 185

b. **The *A priori* Analyses**: pair wise comparisons (pages188-195)

3.b.I: The Controls versus the Pooled Experimental Groups

Source	Sum of Squares	df	Mean Square	F-ratio	P value
Corrected Model	130.985	2	65.492	5.498	0.009
Intercept	52.933	1	52.933	4.444	0.043
Covariates: Ambient Temperature	130.605	1	130.605	10.965	0.002
Main Effects Experimental Groups Vs Controls	9.389	1	9.389	0.788	0.381
Error	393.081	33	11.912		
Total	19795.613	36			
Corrected Total	524.065	35			

Table 3.b.I.i: An analysis of covariance comparing the pooled experimental groups' and the Controls' pre-treatment middle finger skin temperature during ambulatory monitoring Page 188

Source	Sum of Squares	df	Mean Square	F-ratio	P value
Corrected Model	47.001	2	23.500	1.522	0.237
Intercept	247.560	1	247.560	16.029	0.000
Covariates: Ambient Temperature	46.269	1	46.269	2.996	0.095
Main Effects Experimental Groups Vs Controls	2.352	1	2.352	0.152	0.700
Error	401.559	26	15.445		
Total	19638.407	29			
Corrected Total	448.560	28			

Table 3.b.I.ii: An analysis of covariance comparing the pooled experimental groups' and the Controls' post-treatment middle finger skin temperature during ambulatory monitoring Page 188

Source	Sum of Squares	df	Mean Square	F-ratio	P value
Corrected Model	53.592	2	26.796	1.987	0.162
Intercept	29.236	1	29.236	2.168	0.156
Covariates: Ambient Temperature	48.779	1	48.779	3.617	0.071
Main Effects Experimental Groups Vs Controls	9.716	1	9.716	0.720	0.406
Error	283.181	21	13.485		
Total	16664.262	24			
Corrected Total	336.773	23			

Table 3.b.I.iii: An analysis of covariance comparing the pooled experimental groups' and the Controls' follow up middle finger skin temperature during ambulatory monitoring Page 188

3.b.II: The Biofeedback (ATT, ATE, ART, ARE) versus the No Biofeedback (AT, AR) Experimental groups

Source	Sum of Squares	df	Mean Square	F-ratio	P value
Corrected Model	88.574	2	44.287	3.198	0.059
Intercept	68.245	1	68.245	4.928	0.036
Covariates: Ambient Temperature	62.979	1	62.979	4.547	0.043
Main Effects Experimental Groups Vs Controls	26.711	1	26.711	1.929	0.178
Error	332.394	24	13.850		
Total	14948.808	27			
Corrected Total	420.968	26			

Table 3.b.II.i: An analysis of covariance comparing the Biofeedback (ATT, ATE, ART, ARE) and the No Biofeedback groups' pre-treatment middle finger skin temperature during ambulatory monitoring Page 189

Source	Sum of Squares	df	Mean Square	F-ratio	P value
Corrected Model	161.601	2	80.800	6.266	0.008
Intercept	245.525	1	245.525	19.041	0.000
Covariates: Ambient Temperature	78.570	1	78.570	6.093	0.023
Main Effects Experimental Groups Vs Controls	106.275	1	106.275	8.242	0.009
Error	257.889	20	12.894		
Total	15543.142	23			
Corrected Total	419.490	22			

Table 3.b.II.ii: An analysis of covariance comparing the Biofeedback (ATT, ATE, ART, ARE) and the No Biofeedback groups' post-treatment middle finger skin temperature during ambulatory monitoring Page 189

Source	Sum of Squares	df	Mean Square	F-ratio	P value
Corrected Model	60.767	2	30.384	2.185	0.145
Intercept	23.413	1	23.413	1.684	0.213
Covariates: Ambient Temperature	44.476	1	44.476	3.198	0.093
Main Effects Experimental Groups Vs Controls	5.999	1	5.999	0.431	0.521
Error	222.501	16	13.906		
Total	13437.901	19			
Corrected Total	283.269	18			

Table 3.b.II.iii: An analysis of covariance comparing the Biofeedback (ATT, ATE, ART, ARE) and the No Biofeedback groups' follow up middle finger skin temperature during ambulatory monitoring Page 189

3.b.III The Controls versus the Individual Experimental Groups

	AT v. Controls	ATT v. Controls	ATE v. Controls	AR v. Controls	ART v. Controls	ARE v. Controls
Contrast Estimate	2.501	2.287	-2.208	2.777	-0.277	3.036
Hypothesised value	0	0	0	0	0	0
Difference (Estimate - Hypothesised)	2.501	2.287	-2.208	2.777	-0.277	3.036
Std. Error	1.935	2.126	1.781	1.805	1.857	1.818
P-value	0.207	0.291	0.225	0.135	0.883	0.106
95% CI	-1.463,6.466	-2.068,6.642	-5.857,1.441	-0.921,6.474	-4.081,3.527	-0.688,6.761

Table 3.b.III.i: Comparisons of the Individual Treatment groups with the Control group in terms of finger skin temperature during ambulatory monitoring at the pre-treatment assessment session. Pages 190-194

	AT v. Controls	ATT v. Controls	ATE v. Controls	AR v. Controls	ART v. Controls	ARE v. Controls
Contrast Estimate	3.435	2.949	-2.136	4.802	-2.593	1.277
Hypothesised value	0	0	0	0	0	0
Difference (Estimate - Hypothesised)	3.435	2.949	-2.136	4.802	-2.593	1.277
Std. Error	2.294	2.279	1.983	2.292	2.164	2.167
P-value	0.149	0.210	0.293	0.048	0.244	0.562
95% CI	-1.335,8.205	-1.790,7.689	-6.260,1.987	0.036,9.567	-7.094,1.909	-3.229,5.783

Table 3.b.III.ii: Comparisons of the Individual Treatment groups with the Control group in terms of finger skin temperature during ambulatory monitoring at the pos-treatment assessment session. Pages 190-194

	AT v. Controls	ATT v. Controls	ATE v. Controls	AR v. Controls	ART v. Controls	ARE v. Controls
Contrast Estimate	0.03835	5.471	1.990	1.323	0.318	2.101
Hypothesised value	0	0	0	0	0	0
Difference (Estimate - Hypothesised)	0.03835	5.471	1.990	1.323	0.318	2.101
Std. Error	2.944	3.232	2.597	2.604	2.594	3.271
P-value	0.990	0.110	0.455	0.618	0.904	0.530
95% CI	-6.202,6.278	-1.381,12.32	-3.516,7.495	-4.196,6.843	-5.181,5.817	-4.833,9.035

Table 3.b.III.iii: Comparisons of the Individual Treatment groups with the Control group in terms of finger skin temperature during ambulatory monitoring at the follow up assessment session. Pages 190-195

c. **The Post Hoc Analyses**: Scheffé multiple range tests (pages 196-198)

3.C.I.a: The interaction between Biofeedback and Relaxation Group: Comparison 1

Biofeedback Combinations where relaxation group is Autogenic Training	Mean Difference	Std. Error	P-value	95% Confidence Interval	
				Lower Bound	Upper Bound
No Biofeedback x Temperature Biofeedback	-0.3818	0.4841	0.740	-1.7684	1.0049
No Biofeedback x EMG Biofeedback	0.1010	0.4192	0.971	-1.0998	1.3018
Temperature Biofeedback x No Biofeedback	0.3818	0.4841	0.740	-1.0049	1.7684
Temperature Biofeedback x EMG Biofeedback	0.4827	0.4841	0.622	-0.9039	1.8693
EMG Biofeedback x No Biofeedback	-0.1010	0.4192	0.971	-1.3018	1.0998
EMG Biofeedback x Temperature Biofeedback	-0.4827	0.4841	0.622	-1.8693	0.9039

Table 3.c.I: A univariate analysis comparison of the Biofeedback groups' mean residual scores for difference in finger skin temperature during ambulatory monitoring where relaxation group is Autogenic Training and scores are pooled over time Page196

3.C.I.b: The interaction between Time and Biofeedback Group: Comparisons 1 and 2

Biofeedback Combinations at Post-treatment assessment	Mean Difference	Std. Error	P-value	95% Confidence Interval	
				Lower Bound	Upper Bound
No Biofeedback x Temperature Biofeedback	0.7900	0.4135	0.186	-0.2988	1.8789
No Biofeedback x EMG Biofeedback	1.0868	0.3633	0.024	0.1302	2.0434
Temperature Biofeedback x No Biofeedback	-0.7900	0.4135	0.186	-1.8789	0.2988
Temperature Biofeedback x EMG Biofeedback	0.2968	0.4416	0.800	-0.8659	1.4596
EMG Biofeedback x No Biofeedback	-1.0868	0.3633	0.024	-2.0434	-0.1302
EMG Biofeedback x Temperature Biofeedback	-0.2968	0.4416	0.800	-1.4596	0.8659

Table 3.c.II: A univariate analysis comparison of the Biofeedback groups' mean residual scores for difference in finger skin temperature during post-treatment ambulatory monitoring Page 196-197

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