Effects of relaxation and diaphragmatic breathing on respiratory sinus arrhythmia: Implications for cardiovascular disease.

EFFECTS OF RELAXATION AND DIAPHRAGMATIC BREATHING ON RESPIRATORY SINUS ARRHYTHMIA: IMPLICATIONS FOR CARDIOVASCULAR DISEASE

by

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As members of the Final Examination Committee, we certify that we have read the dissertation prepared by Lynette Heslet entitled Effects of Relaxation and Diaphragmatic Breathing on Respiratory Sinus Arrhythmia: Implications for cardiovascular diseases and recommend that it be accepted as fulfilling the dissertation requirement for the Degree of Doctor of Philosophy

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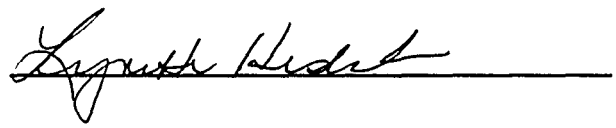
SIGNED: 

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ABSTRACT

Respiratory sinus arrhythmia (RSA) has been shown to be a sensitive noninvasive index of parasympathetic cardiac control with empirical evidence supporting its utility in the study of numerous cardiac problems including: cardiac arrhythmias, sudden cardiac death and as a prognostic indicator of recovery after myocardial infarction. Respiratory modulation of vagal tone causes a reduction in heart period during inspiration and an increase in cardiac parasympathetic functioning during expiration. This pattern of change would be expected to increase if individuals were trained in diaphragmatic breathing. Spectral analysis of the respiratory frequency band was used to determine if the relaxation techniques studied, diaphragmatic breathing and autogenic training, had a differential effect on cardiac autonomic nervous system functioning.

The current study evaluated the respiratory sinus arrhythmia in college students (N=160) under the four experimental conditions (baseline, diaphragmatic breathing, modified autogenic training technique, and concentration task). Spectral analysis of RSA was employed to assess the relationship between vagal tone, diaphragmatic breathing and modified autogenic training. As hypothesized, diaphragmatic breathing consistently increased vagal tone when compared to baseline, modified autogenic training, and a concentration task. The modified autogenic training effect evidenced no

increase in vagal tone. Indeed, the mean area under the respiratory band for this technique was less than baseline the results.

The results of this study suggest that specificity of psychophysiological interventions is necessary to produce effects that improve cardiac functioning. A second implication is that the limited effects relaxation methods have had in the past in the treatment of cardiovascular problems (i.e., hypertension) may be secondary to lack of specificity of the techniques employed. Finally, bringing cardiac parasympathetic functioning under increased voluntary control may increase our ability to treat cardiovascular disease processes in a safe and cost effective way.

Introduction

Sudden cardiac death remains a major unresolved clinical and public health problem in the United States (Myerburg, Kessler, & Castellanos, 1992). Although sudden cardiac death can occur in individuals without heart disease, it is well known that individuals with coronary disease are at a greater risk, especially in the subset of patients with a history of recent myocardial damage (Lown, B. and Verrier, R.L., 1977; Lown, B. and Verrier, R.L. 1978; Multicenter Post-Infarction Group, 1988). In 1994, approximately 1.25 million people experienced an acute myocardial infarction (MI), resulting in nearly 500,000 deaths (U.S. Department of Health and Human Services, 1994). Disturbances in the autonomic control of the cardiovascular function can contribute significantly to cardiac mortality resulting from fatal irregular heart beats (arrhythmias).

Most of the progress made in reducing the number of deaths due to cardiovascular disease has been in secondary prevention among patients who have survived potentially fatal arrhythmias or who are identified as high risk because of a recent cardiovascular event or specific clinical risk factors (Myerburg et al., 1992). Increased heart rate variability (respiratory sinus arrhythmia) has recently been linked to increased survival in patients with recent myocardial infarction (Cripps, Malik, Farrell, & Camm, 1991; Hull et al., 1990; Klieger, Miller, Bigger, Moss, &

Multicenter Post-MI Research Group, 1987; Martin et al., 1987).

Respiratory sinus arrhythmia (RSA) is the mechanism that is thought to accurately monitor neurally mediated cardiac vagal tone (Porges, 1992). Thus, changes in cardiac rhythm, autonomic nervous system function, brain mechanisms, and psychological stress that could lead to sudden cardiac death can be altered with potentially positive clinical outcomes by increasing RSA.

The purpose of the present study is to determine if relaxation techniques, diaphragmatic breathing (DB) and autogenic training (AT), have a differential effect on heart rate variability (HRV). If changes in RSA accurately reflects physiological status of the heart, as hypothesized by other researchers, then a non-invasive means exists that would monitor the effectiveness of treatments and the progression of disease over time. Early detection of cardiovascular disease could facilitate effective treatment interventions before irreversible organ damage occurs.

Respiratory sinus arrhythmia (RSA) is a respiratory influence on heart period that phasically modulates vagal influences on cardiac control (Jewett, 1964; Katona, Poitras, Barnett, & Terry, 1970; Kuntze, 1972). At rest the heart rate increases on inspiration and decreases on expiration. Cardiac vagal tone is reflected in the amplitude of the heart rate rhythm associated with the

frequency of spontaneous breathing (Porges, 1992). Figure 1 (De Meersman, 1993) illustrates RSA pictorially.

Insert Figure 1 about here

Contemporary research has provided empirical evidence that the amplitude of RSA accurately maps the efferent influences of the vagus nerve on the heart. Heart rate variability may index health status of the individual's capacity to organize physiological resources to respond appropriately (Levy, 1984; Porges, 1992). Thus, measurement of cardiac vagal tone has been proposed as a method to assess both stress response and the vulnerability to stress (Porges, 1992). The RSA phenomenon is therefore relevant to physiologists studying neural control of the heart, to physicians assessing medical risks, and to psychophysiologists interested in stress and cognitive processes (Berntson, Cacioppo, & Quigley, 1993; Byne & Porges, 1992; Porges, 1992).

Historically, periodic fluctuations in the heart beat were identified over two hundred years ago. Hales, in 1733, noted that changes in blood pressure and pulse were related to rhythmic respiratory patterns in a horse (Hales, 1733). Cardiopulmonary physiologists have been studying RSA since the middle of the 19th century (Hirsch & Bishop, 1981). In 1846, Ludwig invented the kymograph which allowed his

observations of the rhythmic variations in heart rate associated with respiration in the dog (Hrushesky, 1992). Hering, in 1910, proposed a relationship between phasic changes in heart rate and vagal tone. He stated that "it is known with breathing that a demonstrable lowering of heart rate...is indicative of the function of the vagi" (Hering, 1910). Later, in 1966, Levy and co-workers found positive correlations between the amplitude of the RSA phenomenon and vagal tone (Levy, DeGeest, & Zieske, 1966).

Present day research has identified determinants of respiratory sinus arrhythmia and yielded information about underlying mechanisms of control (Berntson et al., 1993; Loewy & Spyer, 1990, p. 1168-1188). Recent studies have focused on the utility of RSA as a non-invasive, selective index of cardiac autonomic activity (Akselrod et al., 1981; Berntson et al., 1993; Bigger et al., 1988; McCabe, Yongue, Porges, & Ackles, 1984; Odemuyiwa et al., 1991; Porges, McCabe, & Yongue, 1982). Over the past decade, different disciplines have converged on RSA as an index of vagal control of the heart (Berntson et al., 1993).

In a recent review of the literature Berntson et al. (1993) found that RSA measures have been employed in 1) behavioral studies of stress, attention, learning, and cognitive effort; 2) physiological studies of exercise, diurnal rhythms, and central autonomic control; and 3) clinical studies of infants at risk, attentional

dysfunctions, and cardiovascular disease. Because rhythmic variations in the cardiac rate convey important and reliable information regarding the neural control of the heart (Porges, 1986) the utility of RSA as a sensitive and accessible clinical tool assumes special importance. RSA is readily determined from electrocardiographs and techniques for quantifying RSA are well developed (Porges, 1992).

Autonomic Nervous System

The basic function of the nervous system is internal communication that permits the functioning of the various systems to be integrated. The anatomy of the nervous system has an hierarchical arrangement, with two major divisions (central nervous system, peripheral nervous system) and subdivisions. The peripheral nervous system and its component branches provide information about processes interrupting homeostasis. Historically, the autonomic nervous system (ANS) was conceptualized as a branch of the peripheral nervous system subject to reciprocal central control--autonomic reciprocity (Porges, 1992) with increases in the sympathetic (SNS) division associated with decreases in the parasympathetic branch (PNS) (Berntson, Cacioppo, & Quigley, 1991; Cannon, 1939). Simply stated, the ANS regulates homeostatic function. It continuously monitors visceral afferents in an attempt to maintain internal stability.

The PNS and SNS represent neural networks that

originate in the brainstem and contribute to the regulation of a variety of target organs, including: tear ducts, eyes, salivary glands, stomach, pancreas, bladder, external genitalia, sweat glands, trachea, blood vessels, lungs, and heart. The PNS promotes the functions associated with growth and restoration-anabolic activities concerned with conservation of energy and resting of vital organs. The principle function of the SNS is to increase metabolic output. The SNS is the system that prepares the organism for "fight or flight" situations.

In contrast to the Doctrine of Autonomic Reciprocity, it is now firmly established that the two divisions of the ANS may covary reciprocally, independently, or nonreciprocally, as evidenced by co-activation or co-inhibition of both division (Berntson et al., 1991). Berntson et al. (1991) present an extensive review of autonomic modes of control and elaborates on multideterminism. The multiple modes of autonomic control reflect differential central states, and thus may offer a more refined perspective on the relationship between cognitive/behavioral variables and physiological functions (Berntson et al., 1991; Berntson et al., 1993). This perspective is problematic in dually innervated organs (i.e., the heart) since the functional state may be ambiguous with regard to autonomic origins (Berntson et al., 1993). Because RSA phenomenon has been shown to be

primarily neurally mediated, and since it is easily quantifiable, the phenomenon holds considerable promise as a non-invasive index of vagal tone.

Anatomy of Cardiac Innervation

The heart is continuously influenced by direct innervation of the autonomic nervous systems (ANS) via its two branches: the sympathetic (SNS) and parasympathetic nervous system (PNS). Cardiac function is greatly influenced by changes in both the sympathetic and parasympathetic aspects of the autonomic nervous system. In general, parasympathetic influences on the heart are primarily mediated by the tenth cranial nerve, the vagus. Changing vagal influences on the sinoatrial node control most of the observed rapid changes in heart rate (chronotropic changes). Sympathetic innervation is mainly responsible for inotropic effects and mediates changes in contractility of the heart muscle (Porges, 1992). Based on Berntson et al., 1993, Figure 2 depicts sympathetic and parasympathetic efferent pathways of the heart.

Insert Figure 2 about here

Sympathetic-parasympathetic interactions exert opposing effects on the chronotropic state of the heart (Verrier & Hagestad, 1985; Berntson et al., 1993). Vagal fibers exert inhibitory effects on myocardial and sino-atrial pacemaker

cells, whereas post synaptic sympathetic fibers produce facilitory effects. The quantity of NE released at postganglionic terminals may be decreased secondary to the influence of ACh released at vagal endings. This process may also inhibit the accelerated rate of cyclic AMP production in myocardial cells.

Sympathetic-parasympathetic interactions are also manifest as reciprocal excitation. True reciprocal excitation is seen when vagal activity evokes cardiostimulatory responses from chromaffin cells. Postganglionic sympathetic nerve terminals in response to ACh released during vagal activity also produce reciprocal excitation while cardiac sympathetic activity can elicit the release of ACh from postganglionic parasympathetic fibers by virtue of the activity of NE at some pre-synaptic receptors.

It is important, however, to point out that the chronotropic effects are mediated through interacting but separate synaptic channels (Levy, 1984). While the primary interactions between sympathetic and parasympathetic tend to be mutually antagonistic, the neurochemical processes associated with these processes are not symmetrical (Berntson et al., 1993). The asymmetry in neurochemical mechanisms create differences in temporal dynamics, neurophysiological properties, and frequency-dependencies of the autonomic innervations of the heart that may be responsible for the frequency-dependent manifestations of

RSA (Berntson et al., 1993).

Physiological Mechanisms of RSA

Because of the close relationship between respiration and cardiovascular processes, mechanical and neurohumoral alterations in one system affects the functioning of the other in order to maintain homeostasis (Grossman, 1983). The phenomena of RSA results from a complex interaction of central and peripheral factors including: cardio-respiratory reflexes, cardiac and pulmonary stretch reflexes, tonic and phasic baroreceptor and chemoreceptor reflexes, as well as local mechanical and metabolic factors (Grossman, 1983; Spyer, 1989; Saul, Rea, Eckberg, Berger, & Cohen, 1990; Berntson et al., 1993). It is also well established that this neural regulatory process is affected systematically by a wide range of stimuli including life threatening events, drugs, and psychological stress (Porges, 1991b). Berntson et al. (1993) report that neural mechanisms far outshadow non-neural determinants of RSA. This is demonstrated in heart transplant recipients. These individuals do not exhibit RSA, the ANS in the heart is denervated during surgery.

Other evidence also exists that indicates RSA is neurally mediated. Early research, and more modern investigations, have found that: 1) RSA is not generally attenuated by sympathectomy, 2) sympathetic contributions are seen only under depressed vagal conditions, 3) when

present, sympathetic contributions are minimal (Anrep, Pascual, & Rossler, 1936). Because there is substantial evidence of respiratory modulation of vagal activity (primary chronotropic influence on the heart), it has been proposed that information regarding vagal control of the heart can be obtained from amplitude fluctuations in heart rate due to breathing (McCabe et al., 1984; Porges et al., 1982). McCabe et al. (1984) states that approximately 90% of the variance in RSA amplitude can be explained by parasympathetic control. Levy et al. (1966) have also supported the hypothesis that vagal tone is parasympathetically controlled.

In 1966, Levy and co-workers, found positive correlations between amplitude of the RSA and vagal tone. Other investigations have demonstrated that RSA amplitude accurately reflects shifts in a wide range of variation of cardiac vagal tone during experiments in which respiratory rate and, sometimes, tidal volume have been controlled (Pomeranz et al., 1985; Porges, 1986). Pharmacological blockade with atropine was utilized to assess PNS effects and correspondence between vagal control and RSA magnitude was uniformly reported.

The amplitude of RSA may be primarily influenced by vagal as opposed to non-neural factors (Chess, Tam, & Calaresu, 1975; Berntson et al., 1993). RSA is determined by the efferent neural influences from the medulla to the

sino-atrial node (the pacemaker of the heart) via the vagus nerve. When vagal influences to the heart are diminished by direct central mechanisms or by neural blockade, the amplitude of RSA is systematically and predictably decreased (Katona & Jih, 1975, Dellinger, Taylor, & Porges, 1987). Thus, the quantification of abnormalities in cardiac autonomic tone may be useful in identifying patients at risk for cardiac problems, including life threatening cardiac rhythms. The clinical value of RSA as a non-invasive index of vagal effects on the heart is especially important because it is an accessible and sensitive sensor of vagal effects on cardiac function. Figure 3 (Berntson et al., 1993) illustrates the complex interaction between the cardiorespiratory generator, vagal motor neurons, and sympathetic motor neurons on cardiac function.

Insert Figure 3 about here

Mechanical Effects of Respiration on Cardiac Function

The pumping action of respiration forces blood into and out of the heart by changes in intrapleural pressure, a pulling force of inspiration on the thoracic veins, and a diaphragmatic compression of the abdominal veins. Each breath serves to increase venous return to the right heart, and depending on inspiratory effort, cardiac output is changed (Grossman, 1983; Johnson, 1980). The resultant

increased right sided venous return enhances left heart contractility and stroke volume (Grossman, 1983).

Baroreceptors and chemical receptors influencing RSA

Baroreceptors and chemical receptors are important determinants of heart rate and heart rate variability (Berntson et al., 1993). Both mechanisms exert powerful excitatory effects on vagal cardiomotor neurons via central relays in the nucleus tractus solitarius (Grossman, 1983; Spyer, 1990; Berntson et al., 1993). Modulation of respiratory sinus arrhythmia occurs when afferent barrages within the central reflex networks are selectively blocked or "gated." Thus, baroreceptor and chemical receptor reflexes are inhibited during inspiration which contributes to the increase in heart rate during the inspiratory phase of respiration (Berntson et al., 1993). Stimulating baroreceptors can yield maximum vagal excitation during expiration, with minimal effects seen during early or mid-inspiration (Berntson et al., 1993; Spyer, 1990).

Chemoreceptors

Chemoreceptors are located in the carotid and aortic bodies, and are sensitive to alterations in blood concentrations of oxygen, carbon dioxide, and hydrogen ion which can be affected by variations in other respiratory parameters such as: respiratory rate and depth of ventilation (Grossman, 1983). Stimulation of carotid chemoreceptors reduces the heart rate and cardiac output.

Dilation of coronary, cerebral and skin vessels also occurs while vessels in the kidneys, skeletal muscles, and splanchnic bed constrict. Opposing vasoconstriction in these different systems is another example of the complex interactions within the human organism that affect homeostatic functioning.

Pulmonary Stretch Receptors

Reflexes from receptors in the lungs and/or thoracic wall may contribute to the phenomenon of RSA but are of secondary importance (Melcher, 1976). Berntson et al. (1993) in a review of the literature report that stretch receptors do not appear to exert any observable effects on either cardio-motor neurons or on the nucleus tractus solitarius. Stretch receptors apparently modulate transmission through interneuronal reflex pathways to cardiomotor neurons. Pulmonary stretch receptors can, however, exert indirect effects on heart rate via reflexive changes in blood pressure, vascular resistance, and other aspects of hemodynamic control.

Central Rhythm Generators as a source of RSA

Respiration heart periodicity (RSA) is evidenced in the absence of peripheral inputs and is hypothesized to result from a central "respiratory generator." The concept of a Central rhythm generator was proposed as early as 1936 when Anrep et al. (1936) recognized that fluctuations in cardiac rate continued at approximately respiratory frequency in the

absence of respiration or after denervation of pulmonary reflexes. Anrep et al. (1936) demonstrated that respiratory periodicity was related to phrenic activity. Variation in CO_2 tension altered the excitability of the central rhythm generator as evidenced by increased central respiratory drive resulting from hypercapnia and an inhibition noted when hypocapnia was present. Other physiological responses have been observed that also support the concept of a central rhythm generator. Fluctuation in heart rate, baroreflex responsiveness, and autonomic outflow have been observed in phase with phrenic nerve activity in paralyzed animals (Daly, 1985) and controlled breathing in humans can also attenuate RSA (C. T. Davies & J. M. M. Neilson, 1967). C. T. Davies and J. M. M. Neilson (1967) demonstrated that controlled breathing increases RSA amplitude and that the effect terminates immediately when normal breathing resumes.

Sympathetic-Parasympathetic Interactions

Beat-to-beat changes in cardiac vagal and sympathetic nerve activity and their effect on cardiac cycle have been studied extensively. RSA has been shown to be produced mainly by the fluctuation in the amount of vagal discharges during the phrenic cycle (Katona & Jih, 1975), as a result of altered excitability in vagal preganglionic neurons interacting with central respiratory function (Spyer & McAllen, 1980). Kiyomi, Naohito, and Kollai (1985) have shown that sympathetic discharges can also contribute to the

"shaping" of sinus arrhythmia. Sympathetic activity may alter RSA through interactions with vagal innervation of the heart (Berntson et al., 1993; Katona & Jih, 1975).

Katona and Jih (1975) found that cooling of the vagus substantially eliminated RSA. Changes in cardiac cycle length before cooling were about 300 ms, indicating strong vagal input. Kiyomi et al. (1985) on the other hand demonstrated that interaction of sympathetic and parasympathetic control could affect cardiac cycle. Sympathetic effect appeared more delayed than vagal effect. Vagal discharges during one cardiac cycle usually prolonged the next cycle. Cycle length varied unless the heart was slowed by baroreceptor stimulation. Kiyomi et al. (1985) report that the discrepancy might be secondary to the fact that naturally occurring vagal discharges are never synchronized to the extent which would increase the rate of transmitter release as rapidly as the synchronized activity evoked by supramaximal stimulus pulses employed in most studies. Vagal activation can inhibit sympathetic actions on the heart by at least two mechanisms. First, vagal activation can oppose post-synaptic processes. Second, vagal activation can suppress release of norepinephrine (NE) from sympathetic terminals (Levy, 1984). Historically, autonomic interactions have been thought to be extensively one-way because NE or its agonists do not appreciably reduce vagal effects (Levy, 1984; Manabe et al., 1991). However,

as more neurotransmitters or co-modulators are identified additional mechanisms of action and the resultant impact on vagal activity will need to be examined.

For example, neuropeptide Y released from sympathetic nerve terminals in the heart, can exert an inhibitory function on the vagal innervation of the heart that yield a decrease of over 50% in the chronotropic effect of vagal activity (Berntson et al., 1993). The neuropeptide Y inhibition is not eliminated by beta blockers, and because of its long time course the effects could continue well after neural or catecholaminergic markers indicate a return to normal sympathetic activity (Berntson et al., 1993; Fuxe et al., 1990).

Relative Importance of Central and Peripheral Mechanisms Of RSA

Anrep et al. (1936) suggested that respiratory generator and pulmonary stretch receptors contribute equally to the phenomenon of respiratory sinus arrhythmia. Based on canine research, Anrep et al. (1936) demonstrated that inhibition of vagal motor neurons can produce vagal silence during inspiration, and that central or peripheral mechanisms can effect the level of inhibition. Thus, the central respiratory generator or pulmonary stretch can sustain RSA. They may not, however, be readily dissociated and relative contributions of the central and peripheral determinants may vary across behavioral conditions (Berntson

et al., 1993).

Cognitive and Behavioral Influences on RSA

Emotional and neurophysiological changes that characterize physiological responses to stress have been associated with changes in the parasympathetic nervous system and alterations in RSA have been seen in a wide variety of emotional states (Adinoff, Mefford, Waxman, & Linnoila, 1992). In a review of the literature, Adinoff et al. (1992) report that several studies support the hypothesis that emotional states influence vagal tone. For instance, George et al. (1989) found a marked decrease in vagal tone among normal subjects following both intravenous administration of lactate and hyperventilation-paradigms that are known to induce panic attacks in susceptible individuals. Researchers have also shown that a decrease in vagal tone occurs during tasks that require sustained attention and mental effort (Allen & Crowell, 1989). Thyer and Curtis (1985) demonstrated that vasovagal responses (increased vagal tone) often follows anxiety provoking stimuli which can lead to symptoms of dizziness or fainting. Porges (1991a) has also shown that the behavioral reactivity of infants to various external stimuli can be correlated with vagal tone integrity.

Cognitive and behavioral influences on RSA may be partially mediated by descending projections, or neurohumoral influences from higher brain systems. These

influences have the ability to modulate RSA by actions at nodal points in the brainstem cardio-respiratory systems, including: 1) direct effects on vagal motor neurons, 2) modulations of the gain vago-excitatory baroreflexes, 3) direct alterations in the pattern of activity of neuronal subsets of the respiratory generator, and 4) secondary changes in pulmonary reflex gating associated with respiration variations (Berntson et al., 1993). Multi loci control suggests that different behavioral or cognitive states can evoke differential patterns of influence on RSA substrates. Therefore, an important basis for more refined differentiation of behavioral effects is available (Berntson et al., 1993).

Current interest in RSA is, in part, focusing on how rostral brain structures exert a regulatory effect on RSA. For example, rostral systems have extensive neural connections with multiple nodal points in the brainstem regions. Direct projections have been found from the amygdala, hypothalamus, and orbitofrontal cortex to the nucleus solitarius, dorsal motor nucleus, nucleus ambiguus, medullary cardio-respiratory networks, and the intermediolateral cell column of the spinal cord which could account for behavioral and neural influences on RSA (Berntson et al., 1993).

Role of the Autonomic Nervous System in Neurally induced Cardiac Arrhythmias

Several physiological processes can produce changes in cardiac function that could lead to irregular heart rhythms (cardiac arrhythmias) with potentially fatal consequences. Among the range of problems seen are coronary artery spasm, variant angina, classical and unstable angina, changes secondary to exertional effects, platelet aggregation, thrombus, and coronary artery occlusion with subsequent myocardial infarction.

Autonomic Nervous System and Mortality

Prognostic Value of Heart Rate Variability In Myocardial Disease

In a review of the literature Cranefield & Aronson (1988) report that the arrhythmias associated with myocardial ischemia and myocardial infarction-ventricular extrasystoles, ventricular tachycardia, and ventricular fibrillation-have been attributed to enhanced normal automaticity, abnormal automaticity, interactions across segments of depressed excitability, after potential-dependent rhythmic activity, and secondary to circus movement of excitation (p. 481). Extensive studies in experimental animal research, and increasing clinical evidence with patients resuscitated from cardiac arrest, indicate a pervasive involvement of the nervous system in the genesis of fatal cardiac arrhythmias (Verrier &

Hagestad, 1985).

Autonomic neural reflexes have been implicated in the initiation of cardiac arrhythmias during early phases of ischemic heart disease and during the first few hours after a myocardial infarct (Webb, Adgey, & Pantridge, 1972). Webb, Adgey, and Pantridge made the important observation that the incidence of autonomic disturbance was related to the site of cardiac damage. Parasympathetic overactivity occurred most often with posterior wall infarctions. Webb, Adgey, and Pantridge (1972) demonstrated that within 30 minutes post infarction only 17% of 89 patients had normal heart rates and arterial blood pressures. 47% of the patients with bradyarrthythmias exhibited systolic blood pressures less than 80mm Hg. AV block was seen in 8% of the post infarction patients. Lombardi, Verrier, and Lown (1983) found that enhanced cardiac sympathetic neural activity contributes to ventricular vulnerability during coronary artery obstruction.

Some evidence also indicates that parasympathetic neural influences directly affect inotropic, chronotropic, and electrophysiological properties of the ventricles. A high degree of heart rate variability has been found in compensated hearts with good function, whereas heart rate variability can be markedly decreased with severe coronary artery disease, congestive heart failure (CHF), aging and diabetic neuropathy (Klieger et al., 1987).

Heart rate variability has been shown to be a prognostic indicator of post-myocardial infarction survival by numerous researchers. Wolf, Vargios, Hunt, and Sloman (1978) found that low HR variability in post infarction patients was a significant predictor of mortality. 73/176 patients with HR variability of less than 32 ms had a significantly higher hospital mortality rate than patients with more variability. Klieger et al. (1987) found that patients whose heart rate variability (HRV) was less than 50 ms have approximately a 4-fold increased risk of dying during follow-up (34%) compared with those with standard deviation of more that 100 ms (9.0%). The study included 808 patients of which 127 died on or before the end of the 4 year followup period. Decreased HRV was associated with ventricular premature contractions (VPC), coupled VPC, repetitive VPC, and runs of ventricular beats.

Heart rate variability also correlates with other measures of post infarction risk such as peak BUN, total duration of hospital stay, and age (Klieger et al. 1987). It relates directly to ejection fraction and exercise capacity, and has an inverse relationship to left ventricular failure or shock during an acute myocardial infarction. Using spectral analysis, Klieger et al. (1987) found that parasympathetic activity was substantially reduced in patients with low HR variability. Individuals with decreased parasympathetic activity were at greater risk

of cardiac death.

Psychological factors influencing sudden cardiac death

In recent years a growing body of evidence has accumulated that suggests psychological factors play an important role in triggering sudden cardiac death (Kamarck & Jenkins, 1991). Retrospective interviews with relatives and witnesses of victims of sudden cardiac death have shown that acute emotional disturbances were evident in 20% of all sudden cardiac death patients within 6 months (Cottingham, Matthews, Talbott, & Kuller, 1980), twenty four hours (Rissanen, Romo, & Siltanen, 1978), and thirty minutes (Meyers & Dewar, 1975) of death. Reich, Desilva, and Lown (1981) reported a similar rate of acute emotional distress 24 hours prior to resuscitation from near-fatal arrhythmias.

Stress has been associated with occurrence of coronary heart disease and has been implicated as a significant factor in recovery from myocardial infarction (Frasure-Smith, 1991; Miller, Garrett, Stoltenberg, McMahon, & Ringel, 1990). In the late 1960's and early 1970's a number of researchers investigated the association between stressful life events and myocardial infarction. Early results did not yield consistent findings. However, a review of the literature suggests that methodological differences may account for many of the inconsistent findings. First, the definition of stress has varied widely across experiments. Stress has been studied both as a cause

of poor health and also as an outcome. Second, measurement of "stress" has often been based on self-report. Numerous researchers have acknowledged that stress has a subjective component that has not been easily quantified. The wide variability in subjective interpretation of what is stressful makes interpretation of individual differences among participants difficult, but it also restricts our ability to make comparisons across studies. Third, it has been difficult to relate current stress to long term physical health consequences.

Frasure-Smith (1991) studied the long term impact of high levels of psychological stress symptoms in the hospital and after an acute myocardial infarction. Her study assessed men who had taken part in a one year randomized, controlled trial of a program of monthly monitoring of psychological stress levels coupled with home nursing visits for highly stressed patients. The program significantly reduced stress scores, had a marginal impact on cardiac mortality over the first post-infarction year, and reduced long-term recurrence of myocardial damage over a five year followup.

Stress research has, however, focused on description of events that are considered stressful and not on the functional impact of stressful events on physiology (Porges, 1992). Because current definitions of stress are often based on stress as a cause and as a response to events,

non-physiological stress-monitoring is problematic and of limited use in medical settings.

Vagal tone: A physiological marker of stress

Parasympathetic nervous system activity (vagal tone) has been proposed as a novel index of stress vulnerability and reactivity with applications in all branches of medicine (Berntson et al., 1993; Porges, 1992). Porges (1992) suggests a new definition of stress based on the physiological function of the autonomic nervous system. Physiologically based measures can be objectively determined within clinical settings with on-line monitoring of stress and stress vulnerability. RSA (vagal tone) reflects rhythmic vagal efferent influences on the sino-atrial node (cardiac pacemaker) modulated by respiratory processes in the medulla. Heart rate patterns, like behavioral processes, are dependent on the status of the ANS and the quality of neural feedback (Porges, 1992). Employing RSA as an indicator of vagal tone would permit real time assessment of the competency of the ANS.

RSA data reflects individual differences in basal cardiac vagal tone. Within-subject changes in RSA amplitude have been thought to reflect corresponding alterations in parasympathetic control. This conclusion has been based on the fact that changes in vagal tone reflect real time alterations in RSA amplitude. This interpretation, however, is faulty because RSA is not mediated solely by vagal

efferents. RSA is clearly multiply determined by tonic (VT) and phasic (VP) processes having different origins, dynamics, and functional outcomes (Berntson et al., 1993). Thus,

$$RSA = f(VT, VP) + e.$$

The tonic and phasic terms in equation 1 are also multiply determined (Berntson et al., 1993). There is a need for future research to refine the interpretations of the autonomic origins and mechanisms underlying RSA; however, biometric issues also need to be included to further enhance the utility of RSA in psychobiological research (Berntson et al., 1993).

Quantification of Vagal Tone

Quantification procedures for estimating respiratory sinus arrhythmia include: 1) spectral analytic techniques, 2) complex detrending approaches removing periodic and aperiodic cardiac variations unrelated to respiration, and 3) a time-domain, peak to valley procedures employing inspiratory and expiratory periods as windows for determining range of cardiac-interval fluctuations associated with respiratory phase. None of the methods listed can claim to be a superior index of RSA, but differences in the degree each correlates with respiratory period do exist and may have clinical significance (Grossman, van Beek, & Wientjes, 1990).

Time-Domain analysis as a measurement of vagal tone was

first used by Katona and Jih (1975) but has been employed by numerous researchers supporting its utility in cardiac vagal tone monitoring. "Peak-to-Valley" quantification is a closely related technique that is also highly sensitive to variations in tonic parasympathetic cardiac control (Grossman et al., 1990). The time-domain type of analysis measures the differences in milliseconds (ms) between the shortest heart period interval accompanying inspiration and the longest interval accompanying expiration (time is represented along a horizontal axis and RSA magnitude is graphed on the vertical axis). Differences are then averaged across the number of breaths occurring during the measurement period. Eric K.Y. Chan, Ph.D., Arrhythmia Research Technology, illustrates time domain analyses in a modified histogram (Figure 4) and in a Delta R-R Times series (Figure 5).

Insert Figure 4 & 5 about here

The major criticism of these techniques is based on the belief that slow periodic and aperiodic changes may be unduly confounded with peak-to-valley estimates (Grossman et al., 1990). RSA is often superimposed upon two slower frequency rhythms in the cardiac signal (occurring at approximately .03 and .10 Hz), as well as upon other slower nonrhythmic shifts in heart rate. To eliminate the concern

that slower fluctuations would leak into and contaminate the peak-valley measures, frequency domain techniques have been developed (frequency is represented on the x axis). Grossman et al. (1990) found that peak-valley RSA shared about 12% more variance with respiration than the Vagal Tone Monitoring detrending method used by Porges. Although this may be a small difference, any clouding of the relationship between RSA amplitude and respiratory period could be a critical issue under conditions in which statistical control for respiratory influences upon RSA may be needed for accurate assessment of cardiac vagal tone.

Frequency Domain approaches decompose the heart period time series into separate components with mutually exclusive, and distinct bandwidths. RSA can be partitioned within the frequency band characterized by respiration rate of the subject and separated from the other slower rhythmic components. Figure 6 is a graphic representation of frequency domain analysis using a power spectrum. B1, B2, and B3 are the bandwidths typically employed in research. These spectral analysis bands are also called the low frequency (thermoregulation) band, Mid Frequency (Mayer Wave) band, and the High frequency Peak (RSA) (S. Akselrod et al, 1981).

Insert Figure 6 about here

Additionally, non-rhythmic aspects of RSA can be treated as linear trends that are statistically removed from the time series by detrending analysis. For the most part, decomposition makes use of Fourier or Fast-Fourier transformations and is referred to as spectral analysis. A limitation of this method is the need for the mean level and variability of the signal to be relatively constant across the assessment period. Spectral estimates of RSA are used frequently in the assessment of pharmacological effects on vagal tone (Grossman et al., 1990).

Complex detrending procedures are applicable to both time and frequency domain analysis and can apparently eliminate complex aperiodic trends in time series and separates RSA from other components of HR variability. The technique moves a polynomial equation of variable length (Bohrer & Porges, 1982) stepwise through the data, and estimates the variance of the remaining, filtered time series of points within the presumed respiratory band (referred to as Vagal Tone Monitoring) Grossman et al. (1990).

Band width analysis, used in the frequency analysis and spectral analysis techniques can also be problematic. Bandwidth determinations have varied from study to study, with some investigators employing narrower widths than others. When a band is chosen to assess RSA on the basis of mean or modal respiratory rate, but without regard to the

actual range of breathing frequencies of the subject, there is a greater risk that respiration is measured beyond the band width chosen for RSA (Grossman et al., 1990). Heart rate fluctuation within bandwidths can also be influenced by cognitive-behavior factors such as mental tasks or muscular activities. An additional problem with complex detrending techniques is related to the potential distortion that could be induced by the specific algorithms employed (Grossman et al., 1990).

Goal of the current Study

The current project has theoretical and clinical perspectives and is based on the following conclusions: 1) heart rate variability reflects vagal tone, 2) RSA is one measurement of heart rate variability and has been shown to be neurally mediated, 3) substantial evidence indicates heart rate variability is a prognostic indicator of survival after myocardial infarctions. The specific goals of this project was to determine if heart rate variability can be mediated by conscious control.

The study evaluated the impact of relaxation therapy in general, and diaphragmatic breathing in particular, on heart rate variability. Participants were asked to participate in breathing exercises while non-invasive physiologic measurements of heart rate and rhythm were obtained. The influence of patterned breathing exercises were compared to the effects of autogenic training (a relaxation imagery

task) and the effects of simply sitting quietly (control condition). It is hypothesized that diaphragmatic breathing and autogenic training exercises will increase heart rate variability. The size of effect is anticipated to be greater for diaphragmatic breathing (Db) compared to autogenic training (AT).

Our knowledge base on the effect of controlled breathing on HR variability is scant and only one study, to this writers knowledge, has been done to evaluate self-regulation of respiratory sinus arrhythmia (Reye del Paso, Godoy, & Vila, 1992). Reyes del Paso et al. (1992) found that self-regulation of RSA amplitude was possible. Reyes del Paso et al. (1992) employed four strategies including: biofeedback of RSA amplitude, biofeedback of RSA amplitude plus respiratory instructions, respiratory biofeedback, and respiratory instructions only to increase RSA amplitude in normal subjects. All four procedures produced a significant increase of RSA amplitude from baseline measurements with respiratory biofeedback and respiratory instructions producing faster results. Figure 7 (Reyes, et al., 1992) illustrates mean RSA amplitude as a function of group membership in the experiment performed by Reyes del Paso, et al.

Insert Figure 7 about here

Most empirical studies, however, have focused on identifying physiological mediators of beat-to-beat changes in heart rate. Effects on RSA of characteristic breathing pattern variations of breathing frequency, depth of ventilation, breath-holding, alveolar carbon dioxide tension, and the relative contribution of ribcage and abdominal muscles have been investigated (Grossman, 1983). The potential significance of conscious respiratory effort influencing cardiovascular functioning with the possibility of decreasing cardiac sudden death appears to be a logical extension of current research.

Effects of breathing on RSA. Respiratory processes exert specific influences upon various aspects of cardiovascular functioning in the normal, alert human. As a result of changes in different parameters of respiration, central, autonomic, mechanical and hemodynamic mechanisms interact, contributing to both tonic and phasic cardiovascular functioning (Grossman, 1983).

Under normal resting conditions the actual pattern of RSA observed depends largely on the rate of breathing (C. T. M. Davies & J. M. M. Neilson, 1966). Hirsch and Bishop (1981) studied the effects of depth and frequency of breathing on RSA and report that at constant tidal volume RSA amplitude was stable for low breathing frequencies. Levy et al. (1966) also report that changes in respiratory frequency consistently show a correlation between

respiratory frequency and the amplitude of the contractility waves of the left ventricle. When tidal volume is increased, the frequency response curve of the RSA at the new volume exhibits the same characteristic slopes and corner frequencies, but RSA amplitude is increased at each breathing frequency (Hirsch & Bishop, 1981). These studies support the hypothesis that conscious control of respiratory rate and depth of respiration could enhance heart rate variability.

Method

Subjects

Volunteer students (N=160) from the University of Arizona subject pool, during the Summer and Fall 1993 enrollment periods, were asked to have their heart monitored while performing certain tasks. Participants were randomly assigned to one of two groups in order to cross validate the results. A table illustrating group size and composition by gender is illustrated below. Tables 2 and 3

Insert Table 1 about here

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illustrate the ethnic composition and marital status of both groups. The mean age of the participants was nineteen.

Insert Table 2 and 3 about here

Participants were told that they were part of an experiment which assessed different types of stimuli (verbal, no stimuli, and a counting task) on relaxation as measured physiologically. Informed consent (Appendix A) was obtained and debriefing occurred at the end of each semester. Volunteers received credit for participation in the research project as directed by their individual instructors and University policy.

Instrumentation

The current project monitored individual's heart rhythm directly and focused on health histories of their families. Although additional personality and coping instruments were administered, they were not specific to the current project and will, therefore, be analyzed at a later time. A copy of the questions used in the present study can be found in Appendix B.

Subjective Experience Questionnaire: Initially the questionnaire was composed of seven questions asking participants to rate their overall subjective experience. The instrument was expanded during the course of the experiment to include 36 items designed to assess individual's subjective response to each of the four experimental conditions. Subjects rated their feelings of calmness, energy, stress, and how pleasant they felt for each condition. They also indicated the percent of time they were able to follow the instructions and rank ordered

their preference for the interventions. General linear equations were used to analyze responses to theses question.

Procedure

The design of the study randomly assigned subjects to one of two groups. Each participant was asked to complete a comprehensive objective questionnaire to determine their general physical well-being, coping strategies, personality traits. These data were not pertinent to the present study, therefore, they will be analyzed at a later time. Participants were also asked to evaluate their subjective experiences. Subjective experiences were analyzed using general linear equations. Copies of this questionnaire can be found in Appendix B.

Subjects were instructed to abstain from ingesting substances known to affect RSA including: alcohol, caffeine, over the counter cold medications, or diet aids for four hours preceding their participation in the experiment. They were also asked not to engage in any strenuous exercise for the same time period.

Participants were asked to engage in four different exercises, presented in a counterbalanced format. All subjects 1) sat quietly with their eye closed, 2) engaged in diaphragmatic breathing, 3) used autogenic relaxation training, and 4) counted their pulse. Participants had their ECG monitored for seven minutes during baseline (sitting quietly), and during each of the

three active participation conditions. In each condition, subjects followed standardized instructions given by the experimenter (Appendix C and D).

Apparatus

The experiment was conducted in a temperature and humidity controlled, quiet room that was shielded from extraneous noise. Subjects were placed on a hospital stretcher to facilitate measurement of RSA using the PREDICTOR HRVECG system. The hardware consisted of the following components: the computer, digitalizing hardware, programmable amplifiers and QRS detect hardware, preamplifiers, an output device (plotter or printer), and the PREDICTOR signal averaging ECG system. Specific signal-averaging or HR variability software was used to facilitate the use of the component parts in an interactive manner.

The data recording equipment permitted real time input acquired from three bipolar body surface ECG leads (an orthogonal X, Y, Z configuration). Analog signals were amplified, filtered, and sampled by the computer. These data were saved in a data file on the computer's floppy or hard disk for subsequent analysis.

Design and Statistical Analysis

Statistical analyses using general linear models was employed. Because a large sample of subjects was obtained, subjects were randomly assigned to two groups in an effort

1) to cross validate the effects of DB and AT on heart rate variability, 2) compare subject's subjective responses to the interventions, 3) explore the relationship between participant's family member's CV health and HRV.

Results

The results obtained 1) reflect how heart rate varied with different interventions, 2) evidence for a differential effect between DB and AT on RSA, 3) review participant's subjective responses to the procedures, and 4) explore the relationship between family history and the heart's physiological response using spectral analysis.

Group comparisons assessing heart rate differences among the four experimental conditions (BL, AT, DB, FOC) are displayed in Table 4. No significant differences in heart rate were obtained between diaphragmatic breathing and autogenic training, nor were differences obtained between the concentration task (FOC) and baseline condition (BL).

Insert Table 4 about here

However, both DB and AT produced heart rates significantly lower from the BL and FOC. These effects replicated in both groups. Heart rate was also found to decrease consistently for both groups from BL through the subsequent interventions, regardless of order (Table 5). The implication of this finding is that some measure of

relaxation, as measured by reduction in heart rate occurred

Insert Table 5 about here

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with both DB and AT. Because no significant decrease in HR occurred during the FOC task, one could infer that changes in the participant's heart rates were not due to participant's concentrating on the tasks at hand per se.

High frequency peaks (AU3) of power spectrum of instantaneous heart rate represent vagal influences on the sinoatrial node around respiratory frequency (RSA). Comparisons between each treatment condition and the third band (AU3) of the spectral analysis data were also made (see table 6). Diaphragmatic breathing was significantly different from all other interventions in the effect it produced on AU3. AT was not significantly different from

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Insert Table 6 about here

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the concentration task (FOC). When comparing the AT-BL, FOC-BL conditions, results evidence group differences. However, in both groups mean AU3 decreased from baseline with AT and FOC treatments, while the AU3 in the DB conditions increased substantially (Table 7). No order effects were found for any of the three active treatment conditions.

Insert Table 7 about here

Area under the curve for the second spectral band (AU2) was also calculated. Again, diaphragmatic breathing produced a significant effect compared to AT (see Table 8). Comparison of mean AU2 for both groups is illustrated in Table 9.

Insert Table 8 and 9 about here

Participants, in both groups, were asked to give subjective responses about their experiences. Table 10 depicts participant's self report of their ability to following the instructions. For all treatment conditions, subjects were able to follow instructions without difficulty.

Insert Table 10 about here

Participant's also rank ordered their preference for the interventions (Table 11) and answered questions related to their feelings about being energized, stressed, sleepy, calm, and pleasant. In general, participant's liked the FOC (counting) task least. AT was preferred by Group 1 (35.3%) more than Group 2 (25.8%). The opposite relationship was evident with DB. Group 2 preferred DB (35.5%) with DB

ranked first by 21.1% of the repondants in Group 1. No significant difference were evident in responses to

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Insert Table 11 about here

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individual questions assessing feelings of calmness, energy level, sleepiness, stress, or pleasant feelings (Table 12).

Insert Table 12 about here

Discussion

The purpose of this dissertation was to examine the relationship between relaxation techniques and respiratory sinus arrhythmia, specifically evaluating the differential effects of diaphragmatic breathing and autogenic training.

The findings demonstrate the usefulness of heart rate variability in understanding how psychological interventions impact on the autonomic nervous system of the heart. The method provides real time assessment of an individual's response to an intervention, demonstrates specificity of relaxation techniques, and suggests hypotheses that would help psychologist understand why past research employing psychological interventions with cardiovascular problems has not been as effective as hoped.

The results support the hypothesis that the two relaxation techniques evaluated in this study, DB and AT,

evidence specificity in their effect on RSA. In the present study, DB produced a marked increase in vagal tone. The effect occurred within a short period of time (7 minutes) and was consistent across subjects. Autogenic training, another popular relaxation techniques, decreased vagal tone, even below baseline findings.

One implication of these results is that a psychological intervention, DB, can be effective in treatment of some cardiac disease. First, some arrhythmias are thought to be vagally mediated. Because DB increases vagal tone the potential exists that DB could be a useful, non-pharmacological intervention for these arrhythmias, the technique is easy to learn. DB could be employed by individuals receiving maximum medical management, by individuals who have adverse reactions to medication, or in emergencies before medications reach effective doses.

A second use of DB in cardiac problems would be with individuals immediately post myocardial infarction. Researchers have already shown that patients with increased vagal tone evidence greater survivability in the immediate post MI period and in the year following. Teaching DB immediately after an infarction may increase an individual's chance of survival. In addition to reducing mortality from the infarction, DB may decrease the risk of complications. Eliminating or reducing cardiac arrhythmias reduces the risk of thrombi. This would lower the incidence of post

infarction complications such as stroke, or anoxic encephalopathy.

Conversely, AT would be counter indicated for use with individuals with a cardiac history, or in individuals at risk for cardiac problems. The technique reduced vagal tone, and theoretically could potentiate the risk for etrasystoles, and myocardial infarction in susceptible individuals.

Historically, researchers have used pulse, respiration, or blood pressure to determine if relaxation techniques have produced an effective. These criteria may not be sensitive enough to reflect the impact of different techniques on the cardiovascular system. Assessing the heart with a more direct technique, HRV, can enhance our knowledge of the relationship between treatment interventions and physiological response. Understanding the effect of currently known techniques on the ANS of the heart can also enhance our understanding of organs and systems that are not as accessible to measurement.

RSA has also been shown to reflect an organisms level of stress and stress response to varying stimuli. The results of this study support the usefulness of RSA monitoring for assessing the utility of psychological interventions in stress management. RSA provides a unique method of studying the effects of different relaxation techniques on the cardiac system. In addition to aiding in

understanding of cardiac disease, and disease progression, RSA measurements can also aid in identification and development of psychological interventions that might reduce the risk of cardiac problems. Techniques with a more direct impact on the ANS within the heart may produce greater, and more lasting control of other cardiovascular problems such as hypertension.

Implications and Recommendation

The extrapolation of the current findings to the clinical setting and the verification of their relevance may not be immediate. Because DB and AT evidence specificity on cardiac ANS function, it seems prudent to investigate how other relaxation techniques affect vagal tone. Research needs to address the utility of DB in prevention and management of cardiac symptoms. Studies will need to be performed to determine if conscious control of HR variability can facilitate clinical management of cardiac problems, especially post infarction survival and reduction of cardiac arrhythmias.

Prospective research would aid in determining when physiologic changes occur, the degree of physiologic changes needed to produce overt symptoms, and increase our knowledge of the relationship of CV risk factors to disease onset. Investigating the link between HRV and other phenomena may also increase our ability to treat other somatic problems with psychological interventions.

It appears evident that enhancing our understanding of the biochemical and physiological mechanisms will lead to behavioral therapies having a greater impact. The challenge is to expand into other areas, develop theories about the mechanism by which behavioral techniques might work, and the test those theories. This would facilitate identification of interventions appropriate for patients at risk for cardiac problems. It would also help psychologist understand why past research, especially in the area of hypertension, may not have been as successful as hoped.

APPENDIX A

Heart Rate Variability

Informed Consent Form

You are being asked to read the following material to ensure that you are informed of the nature of this research study and of how you will participate in it, if you consent to do so. Signing this form will indicate that you have been so informed and that you give your consent. Federal regulations require written informed consent prior to participation in this research study so that you know the nature and risks of your participation and can decide to participate or not participate in a free and informed manner.

Purpose

You are being invited to participate voluntarily in the research project named above. The purpose of this project is to find out if heart rate changes (increased or decreased rhythm), which occur naturally, are affected by different relaxation techniques.

Selection Criteria

You are being invited to participate because you are part of the University of Arizona undergraduate subject pool, and are interested in this study as part of your course work requirement.

Procedure

If you agree to participate, you will be asked to do the following: 1) attend a 1 1/2 hour session in which you will complete questionnaires about general styles of coping, personality characteristics, your health and that of your family, and 2) participate in a second thirty minute session where you will be asked yo perform 3 relaxation exercises (diaphragmatic breathing, imagery, and sitting quietly) while your heart rate is being monitored. Heart monitoring is done by placing electrodes on your chest.

Risks

The risks to participating in this study is that you may feel uncomfortable answering personal questions about your health and your family's health history, or that you may feel uncomfortable having your heart monitored by attaching electrodes to your chest.

Benefits

We expect that most participants will find the experiment interesting and that they will appreciate knowing that their participation may prove to be helpful in the development of an intervention used in the treatment of heart attack patients.

Confidentiality

The data we collect from the questionnaires and the information gained from physiologic monitoring will only have a subject number on it, so that it will be impossible for anyone to link you name to the actual responses you have made or to the tracing of your heart rate variability.

Authorization

In giving my consent by signing this form, I agree that the methods, inconveniences, risks and benefits have been explained to me and my questions have been answered. I understand that I may ask questions at any time and that I am free to withdraw from the project at any time without causing bad feelings. I also understand that the results of this research may be presented or published, but as mentioned above, all participants will remain anonymous. I understand that this consent form will be filed in an area designated by the Human Subjects Committee with access restricted to the principal investigator. I understand that I do not give up any of my legal rights by signing this form. A copy of this signed consent will be given to me.

_____ _____

Signature Date

APPENDIX B

SUBJECTIVE FEELINGS QUESTIONNAIRE

Circle:
1. Baseline
2. Autogenic training
3. Diaphragmatic breathing
4. Focused (counting)

PLEASE CIRCLE THE RESPONSE THAT BEST DESCRIBES HOW YOU FELT IMMEDIATELY AFTER THE CURRENT EXERCISE:

1. How calm were you feeling?

Not calm at all		Neutral		Extremely calm
1	2	3	4	5

2. How energized were you feelings?

Not energized at all		Neutral		Extremely energized
1	2	3	4	5

3. How sleepy were you feeling?

Not sleepy at all		Neutral		Extremely sleepy
1	2	3	4	5

4. How much stress were you feeling?

No stress at all		Neutral		Very stressed
1	2	3	4	5

5. How pleasant were you feeling?

Unpleasant		Neutral		Very Pleasant
1	2	3	4	5

6. What percent of the time were you able to follow the instructions during the session? Pick any value between 0% and 100%.

SUBJECTIVE FEELINGS QUESTIONNAIRE
(overall assessment)

PLEASE CIRCLE THE RESPONSE THAT BEST DESCRIBES HOW YOU FELT DURING THE HEART MONITORING EXERCISES:

1. How calm were you feeling?

Not calm at all		Neutral		Extremely calm
1	2	3	4	5

2. How energized were you feelings?

Not energized at all		Neutral		Extremely energized
1	2	3	4	5

3. How sleepy were you feeling?

Not sleepy at all		Neutral		Extremely sleepy
1	2	3	4	5

4. How much stress were you feeling?

No stress at all		Neutral		Very stressed
1	2	3	4	5

5. How pleasant were you feeling?

Unpleasant		Neutral		Very Pleasant
1	2	3	4	5

6. What percent of the time were you able to follow the instructions during the session? Pick any value between 0% and 100%. _____.

Continued

7. Please rank order the methods by order of preference: 1=liked best, 4=liked least

____ Sitting quietly

____ Diaphragmatic breathing (hands on abdomen)

____ Autogenic training (heavy and warm)

____ Focused relaxation (counting task)

8. Please make a brief comment on why you preferred the method you chose in question #7 (why you liked the method best)

_____

_____

_____

_____

_____

APPENDIX C

Autogenic Training Instructions

You are about to learn a method of deep relaxation called Autogenic Training. You should be a comfortable position, with your arms at you side. Let you body relax.

For the next 7 minutes, I will be asking you to repeat certain phrases to yourself. These phrases may have the effect of making various parts of your body feel heavy and warm. This is a sign that you are beginning to relax. Proper use of these phrases will help you achieve a very deep, pleasant state of relaxation. There are two important rules which you must follow in order for these phrase to work. The first rule is that you maintain ***passive concentration.*** This means that you should not actively try to make anything happen. Instead, you should let the phrases flow effortlessly through your mind, as you let go and passively observe the relation taking over you body. The minute you consciously make an effort to relax, you will interfere with your body, as it naturally and easily relaxes itself. So let go, passively concentrate on your body.

The second rule is that repetition of the phrases should become automatic and conscious. Imagine each phrase as an endless loop which goes round and round in your head. You may either visualize the words of the phrase, or imagine the words being spoken over and over, by yourself or someone else. But make sure that the phrase keeps repeating itself throughout the entire exercise.

You may at times, become distracted by thoughts or sensations such as muscle twitches, itches, or irregular breathing. This is perfectly normal. If distractions should occur, just try to ignore them and return to your passive concentration on the phrase. Concentrate on the repetition of the phrases.

Now, let's begin. Let you attention focus on your right arm and hand. Notice how it rests on the stretcher. Maintain mental contact with your right arm and repeat to yourself "my right arm is heavy...my right arm is heavy...my right arm is heavy.." continue to silently repeat this phrase for the next few seconds (30 sec)... Now, observe the sensations in your right arm. Does it feel heavy> Does it feel relaxed? Tingly? Let the feelings of heaviness and relaxation spread throughout your body. Now focus on your right leg, become aware of your right thigh, knee, lower leg, ankle, foot, and toes. Slowly and silently repeat to yourself: "my right leg is heavy and warm...my right leg is heavy and warm" Continue to repeat this phrase for the next few seconds (30 sec). Notice the sensations in you leg. Describe these feeling silently to yourself and relax.

Gently shift your attention over to your left leg. Let your attention wander from you left thigh to you knee, to your calf, to you ankle and then to your foot. Slowly and silently to yourself begin to repeat the phrase: "my left leg is heavy and warm...(30 sec). Become aware of the sensations in your left leg, notice any sensation of tingling, lightness, heaviness or numbness. Continue to relax.

Now focus on both your legs at the same time. Let yourself experience the sensations in your body, the feelings in your legs. Enjoy the feelings of warmth, heaviness, and relaxation. Then begin saying silently to yourself: "both legs are heavy and warm..both legs are heavy and warm...(30 sec)." Gently notice the sensations in your body, feel the warmth, the heaviness and relaxation for a few seconds.

APPENDIX D

Diaphragmatic Breathing Instructions

The purpose of breathing is to get oxygen into the body and to get carbon monoxide out. Most people breathe by moving the muscles of the chest up and down. Put your hand on your chest and feel your chest movement. With diaphragmatic breathing, air gently flows into and out of the lungs, at the start it may feel awkward. Most people find that they adapt quickly.

What I want you to do is this. First, place your dominant hand on your abdomen. Then, I want you to inhale and simultaneously push out and up on your abdomen. Up and out while you inhale. Then, when you are ready to exhale, let your stomach fall. Try again. As you inhale, let your stomach push out and up. Then exhale and let your stomach fall. Try to keep your chest as still as possible.

Do you feel dizzy? Do you feel light headed? All these states are a sign of more oxygen going into your body; a sign of relaxation.

Continue to breath slowly and deeply, pushing you r stomach up as you inhale. Letting it fall gently as you exhale. Nice slow, deep breathes. Push out and up as you inhale, letting you stomach fall as you exhale....(repeat through exercise).

TABLES

Table 1. Table of Gender by Group membership.

Gender Group

Frequency
Percent
Row percent
Col percent

	1	2	Total
Female	54 33.75 50.94 69.23	52 32.50 49.06 63.41	106 66.25
Males	24 15.00 44.44 30.77	30 18.75 55.56 36.59	54 33.75
Total	78 48.75	82 51.25	

Table 2. Ethnic composition of participants by group.

Ethnic Composition (%)	Group 1	Group 2
Hispanic	16.9	15.9
Caucasian	61.0	69.5
Black	6.5	6.1
Asian/Pacific	9.1	7.3
Native American	6.5	1.2

Table 3. Marital Status of participants by group.

Marital Status (%)	Group 1	Group 2
Single	94.9	97.6
Married	1.3	0.0
Divorced	1.3	0.0
Co-habitating	2.6	2.4
Widowed	0.0	0.0

Table 4. Group Comparison of HR data with interventions.

Group 1

Comparison	df	F	Pr>F	N
Overall	3	15.54	.0001	58
DB-AT	1	1.21	.2759	60
DB-FOC	1	15.86	.0002	61
DB-BL	1	20.42	.0001	61
AT-FOC	1	23.60	.0001	61
AT-BL	1	43.22	.0001	62
FOC-BL	1	0.72	.3990	62

Group 2

Comparison	df	F	Pr>F	N
Overall	3	9.83	.0001	61
DB-AT	1	0.06	.8006	62
DB-FOC	1	6.65	.0124	62
DB-BL	1	15.87	.0002	63
AT-FOC	1	16.74	.0001	61
AT-BL	1	26.52	.0001	62
FOC-BL	1	1.87	.1768	62

*Group 1 & 2 cross validated

Table 5. Comparison of Heart rates for both groups. HR1 represents the mean heart rate for subjects at baseline. HR 2,3 and 4 illustrate the decline in heart rate during subsequent treatment interventions. ATHR, DBHR, FOCHR results show mean heart rates specific for autogenic training, diaphragmatic breathing, and the Focused task respectively.

Comparison	Group 1	Group 2
HR 1 (BL)	68.23	72.25
HR 2	68.07	71.47
HR 3	66.21	70.14
HR 4	65.35	68.80
ATHR	65.44	69.40
DBHR	66.22	69.51
FOCHR	67.93	71.54

Table 6. Comparison of mean area under the curve of the third band of the spectral analysis (AU3) across treatment conditions for both groups.

Group 1

Comparison	df	F	Pr>F	N
Overall	3	6.31	.0004	54
DB-AT	1	7.07	.0101	59
DB-FOC	1	11.53	.0013	58
DB-BL	1	5.44	.0232	59
AT-FOC	1	0.02	.8955	58
AT-BL**	1	0.85	.3631	60
FOC-BL**	1	1.13	.2934	57

Group 2

Comparison	df	F	Pr>F	G-G Epsilon	N
Overall	3	8.86	.0001	.0008	58
DB-AT	1	9.87	.0026		60
DB-FOC	1	13.35	.0006		60
DB-BL	1	4.95	.0300		60
AT-FOC	1	0.19	.6682		60
AT-BL**	1	4.29	.0428		60
FOC-BL**	1	4.29	.0426		61

**Comparisons did not cross validate

Table 7. Mean area under for the third spectral band (AU3) for each treatment condition.

Comparison	Group 1		Group 2	
	Mean	Std dev	Mean	Std dev
Baseline	4792.38	6287.85	4008.88	4698.52
AT	4140.39	5215.37	3343.82	3249.26
DB	6690.17	9971.91	5726.66	6585.60
FOC	4121.83	6532.13	3074.56	4262.76

(group size averaged 60)

Table 8. Results comparing treatment interventions with area under the second spectral band.

Group 1

Comparison	df	F	Pr>F	N
Overall	3	15.38	.0001	54
DB-AT	1	18.05	.0001	59
DB-FOC	1	19.60	.0001	58
DB-BL	1	27.39	.0001	59
AT-FOC	1	0.27	.6053	58
AT-BL	1	0.17	.6846	60
FOC-BL	1	0.56	.4571	57

Group 2

Comparison	df	F	Pr>F	N
Overall	3	22.06	.0001	58
DB-AT	1	25.72	.0001	60
DB-FOC	1	21.74	.0001	60
DB-BL	1	21.96	.0001	60
AT-FOC	1	1.50	.2259	60
AT-BL	1	2.19	.1440	60
FOC-BL	1	0.03	.8652	61

*Results cross validated

Table 9. Group comparisons for mean area under the second, mid-frequency, band of the spectral analysis (AU2).

	Group 1		Group 2	
Condition	Mean	Std dev	Mean	Std dev
Baseline	4537.65	4256.13	3653.70	3704.48
AT	4226.86	6130.31	3226.45	3539.49
DB	8372.52	7774.71	10562.43	11894.98
FOC	3941.63	4684.19	3545.66	3654.57

(both groups utilized 60-62 observations for comparisons)

Table 10. Percent of the time participants reported they were able to follow instructions.

Treatment	Group 1	Group 2
Baseline	96.86	97.16
AT	96.41	94.03
DB	90.88	89.92
FOC	96.35	88.46
Overall rating	94.58	92.66

Table 11. Rank order, by subject's preference, of treatment conditions (1=liked best, 4= liked least).

Baseline

Preference	Group 1 (N=71)	Group 2 (N=68)
1	32.4%	33.8%
2	19.7%	29.4%
3	19.7%	32.4%
4	16.9%	4.4%

DB Condition

Preference	Group 1 (N=45)	Group 2 (N=31)
1	21.1%	35.5%
2	42.4%	29.0%
3	30.3%	32.3%
4	6.1%	3.2%

AT Condition

Preference	Group 1 (N=34)	Group 2 (N=31
1	35.3%	25.8%
2	26.5%	35.5%
3	17.6%	25.8%
4	20.6%	12.9%

FOC Condition

Preference	Group 1 (N=34)	Group 2 (N=31)
1	2.9%	3.2%
2	5.9%	3.2%
3	38.2%	9.7%
4	52.9%	83.9%

Table 12. Subjective Experience Information: 1=not at all, 3=neutral, 5=extremely

Question	Group 1 Mean	Std dev	N	Group 2 Mean	Std dev	N
Baseline Condition						
How calm were you feeling?	4.05	.81	37	3.96	.87	31
How energized were you feeling?	2.40	.89	37	2.19	1.01	31
How sleepy were you feeling?	3.40	.86	37	3.64	.98	31
How much stress were you feeling?	2.05	.97	37	2.32	1.10	31
How pleasant were you feeling?	4.05	.77	37	3.96	.87	31
Autogenic training condition						
How calm were you feeling?	4.05	1.12	34	4.30	1.01	26
How energized were you feeling?	1.19	.96	34	2.03	1.03	26
How sleepy were you feeling?	3.56	1.18	34	3.80	1.16	26
How much stress were you feeling?	1.50	.86	34	1.73	.87	26
How pleasant were you feeling?	4.08	.83	34	4.03	.82	26
DB breathing condition						
How calm were you feeling?	4.02	.90	34	4.12	.78	25
How energized were you feeling?	2.08	.96	34	1.96	.89	25
How sleepy were you feeling?	3.47	1.08	34	3.76	1.09	25
How much stress were you feeling?	1.88	1.06	34	2.04	.97	25
How pleasant were you feeling?	3.88	1.95	34	3.72	1.02	25
Focus Condition						
How calm were you feeling?	3.62	1.26	34	3.58	1.20	26
How energized were you feeling?	2.35	1.12	34	2.42	1.02	26
How sleepy were you feeling?	3.20	1.15	34	3.42	1.30	26
How much stress were you feeling?	2.09	.96	34	2.46	1.36	26
How pleasant were you feeling?	3.56	.86	34	3.46	.95	26
Overall ratings						
How calm were you feeling?	4.34	.82	73	4.18	.81	69
How energized were you feeling?	2.33	1.07	73	2.22	.97	69
How sleepy were you feeling?	3.86	.96	73	3.87	.94	69
How much stress were you feeling?	1.72	.83	73	1.75	.79	69
How pleasant were you feeling?	4.35	.73	72	4.17	.80	69

FIGURE 1

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RESPIRATORY SINUS ARRHYTHMIA

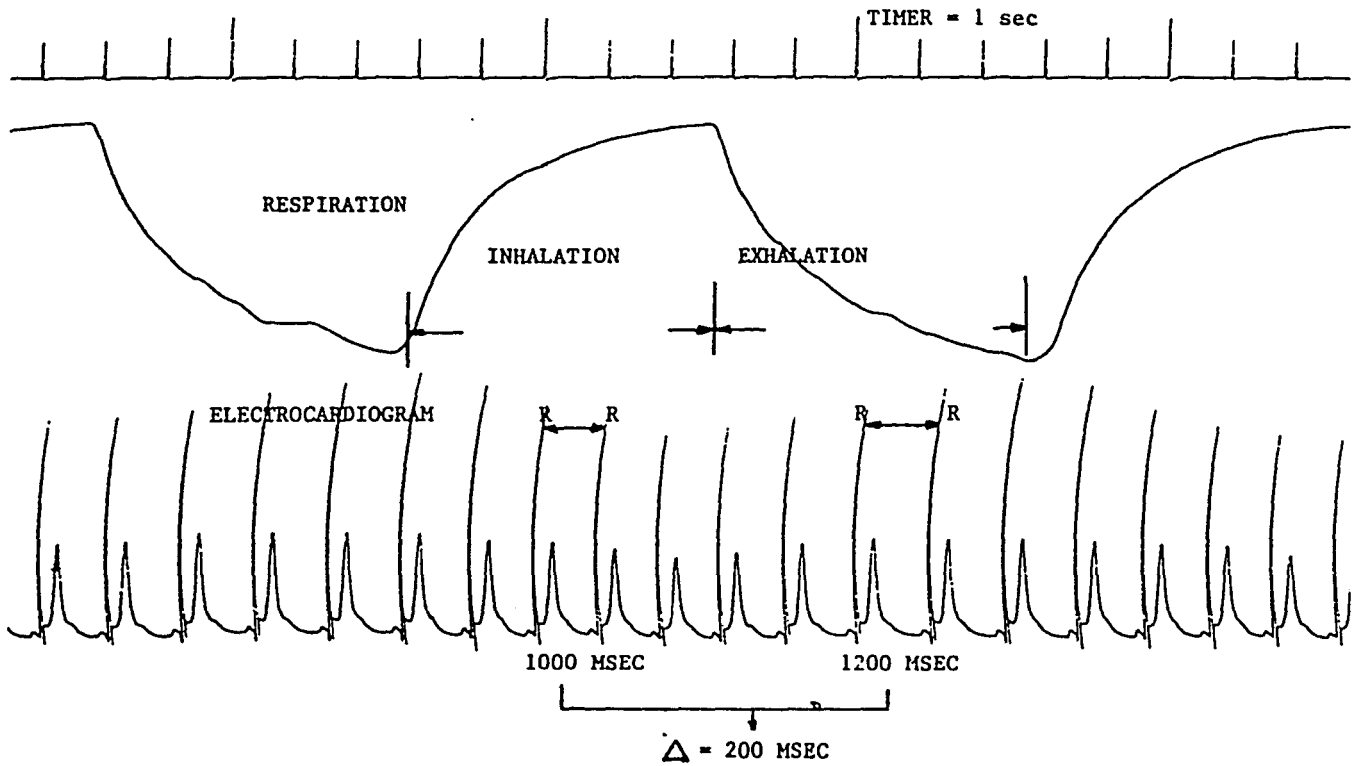
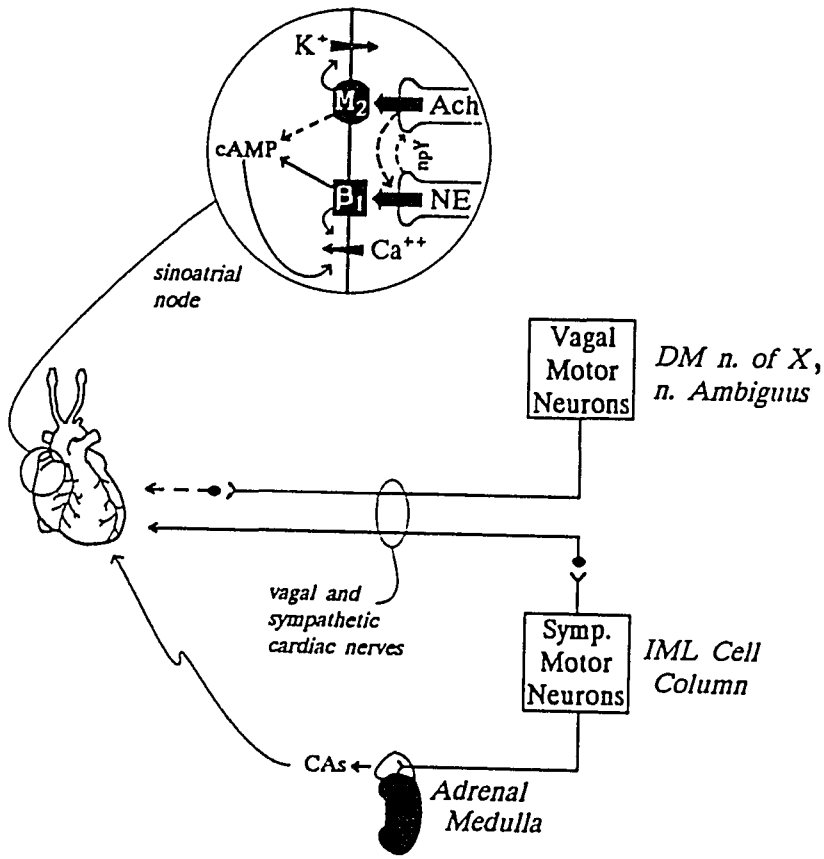


Figure 1. Physiographic recordings depicting, from top to bottom, timer, respirations, electrocardiogram and end-tidal carbon dioxide recordings, respectively. Heart period variability (P-P interval) is expressed in milliseconds. Note variability when comparing exhalation (downward slope) vs inhalation (upward slope).

FIGURE 2

(Reprinted with permission from Psychophysiology, 30 (1993), p.185)

SCHEMATIC DEPICTION OF AUTONOMIC CHRONOTROPIC CONTROL OF THE HEART

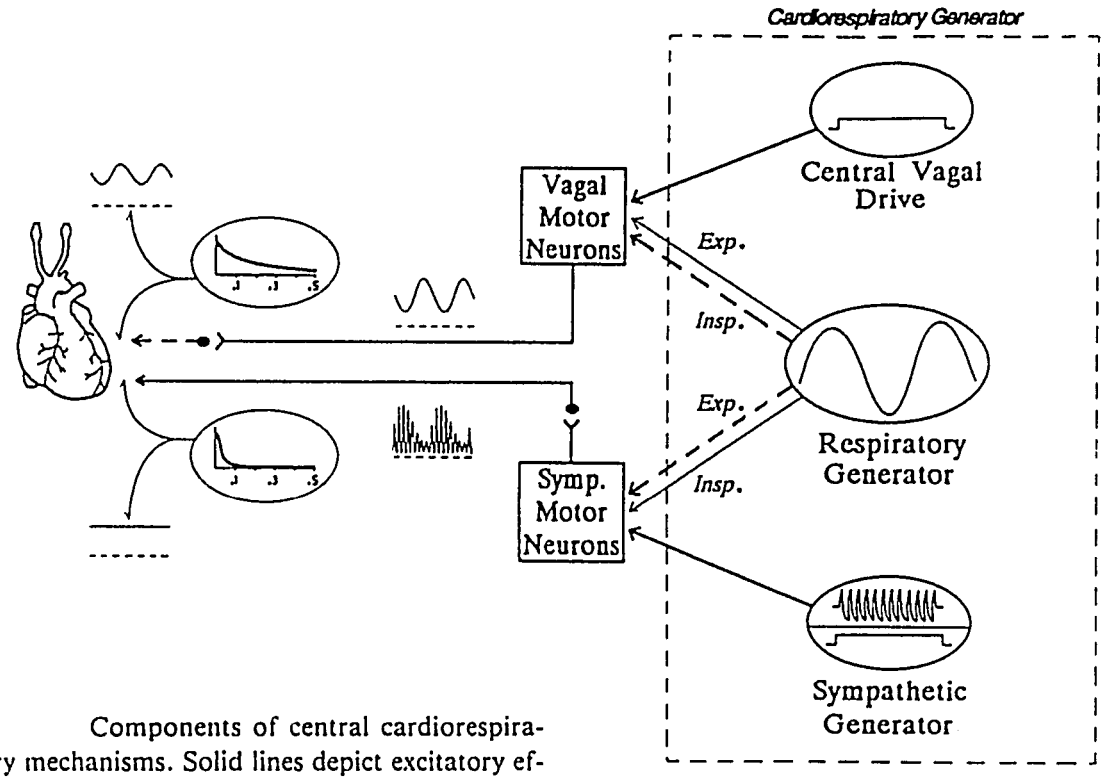


Synaptic interactions are illustrated in the insert. Noradrenergic (beta$_1$) receptor activation leads to calcium (Ca^{++}) mobilization via the second messenger cyclic AMP (cAMP). Muscarinic (M_2) receptor action opposes this action and opens potassium (K^+) channels leading to hyperpolarization. Both innervations mutually inhibit the other via acetylcholine (Ach) released from the vagal terminals and neuropeptide Y released by the sympathetic terminals. Solid arrows depict activational effects and dashed arrows indicate inhibitory effects. DM n. of X = dorsal motor nucleus of the vagus; IML Cell Column = intermediolateral cell column; CAs = catecholamines; npY = neuropeptide Y; M_2 = postganglionic muscarinic cholinergic receptor of the parasympathetic cardiac synapses; β_1 = postganglionic adrenergic receptor of the sympathetic cardiac synapses.

FIGURE 3

(Reprinted with permission from Psychophysiology, 30 (1993), p.186)

COMPONENTS OF CENTRAL CARDIORESPIRATORY MECHANISMS



Components of central cardiorespiratory mechanisms. Solid lines depict excitatory effects; dashed lines represent inhibitory effects. Waveforms illustrate the time varying patterns of activity at the respective site, based on the mechanisms depicted. Graphic inserts depict the frequency-transfer functions of the cardioeffector synapses (see Figure 3), and the associated waveforms illustrate the transformations on the input functions. Tonic drive to vagal motor neurons may be due to basal influences of phasic generators rather than a functionally distinct tonic generator. Consequently, the box depicting central vagal drive is intended to represent the aggregate basal steady-state inputs to motor neurons and does not imply a specific functional entity. Exp. = expiratory phase; Insp. = inspiratory phase.

FIGURE 4

(Reprinted with permission from Eric K.Y. Chan, Ph.D., Arrhythmia Research Technology, Inc.)

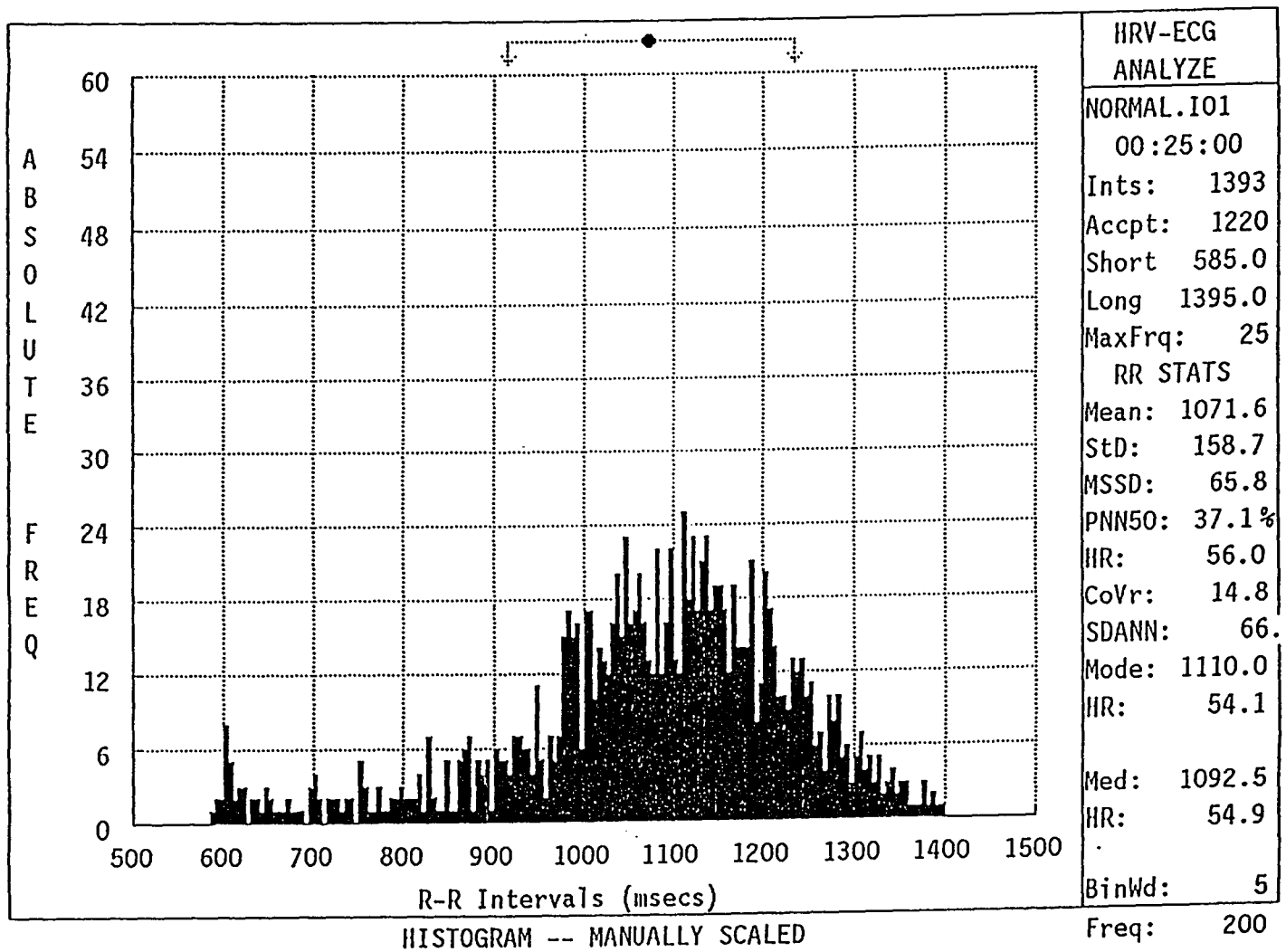
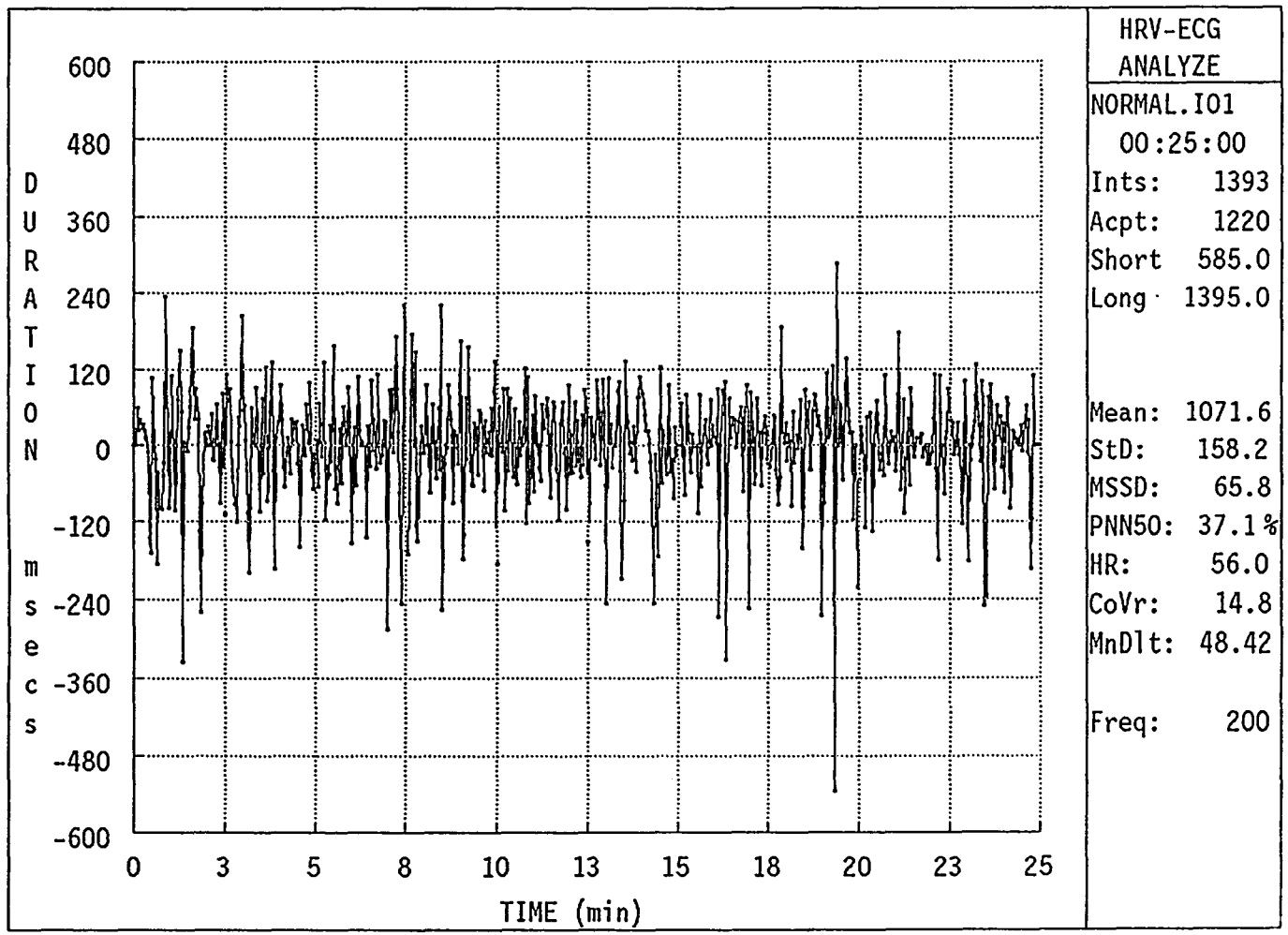


FIGURE 5

(Reprinted with permission from Eric K.Y. Chan, Ph.D., Arrhythmia Research Technology, Inc.)

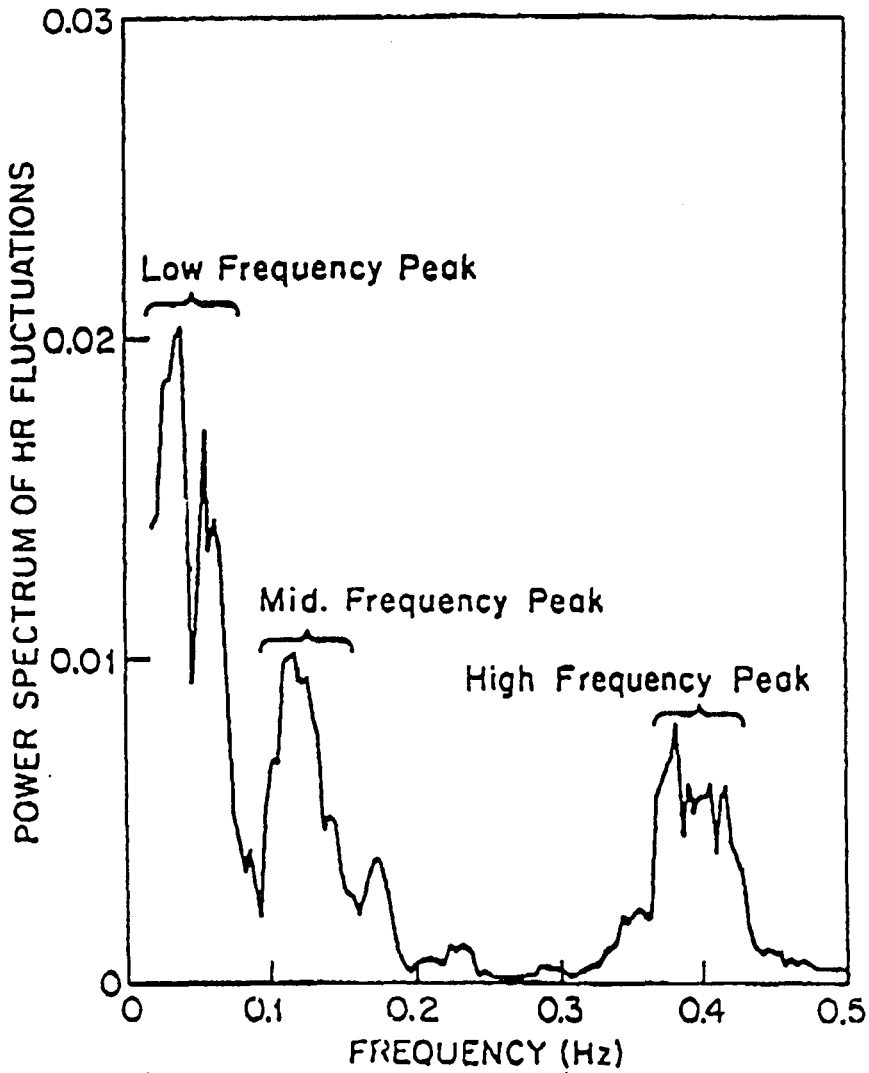


DELTA INTERVAL DURATION vs. TIME -- MANUALLY SCALED

FIGURE 6

(Reprinted with permission from Science, 213, p.220)

POWER SPECTRUM OF INSTANTANEOUS HEART RATE (HR) FLUCTUATIONS

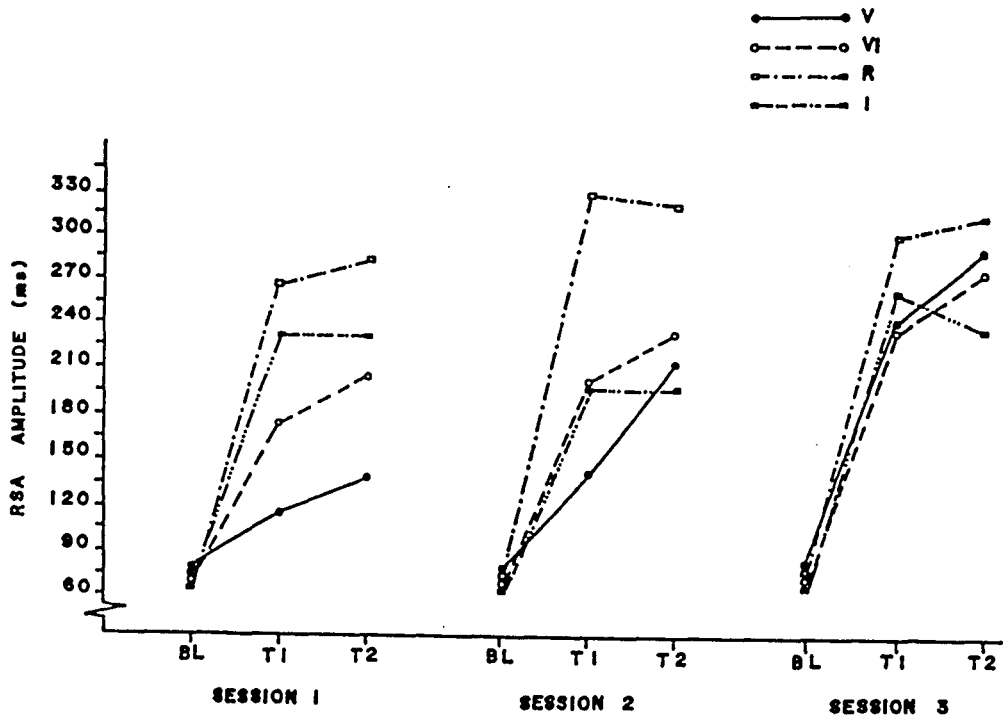


Power spectrum of instantaneous heart rate (HR) fluctuations featuring 3 main peaks: low-frequency peak (from 0.02 to 0.09 Hz), midfrequency peak (from 0.09 to 0.15 Hz), and high-frequency peak (around respiratory frequency). (Reproduced from *Science* 213, p. 220, 1981. S. Akselrod, D. Gordon, F. A. Ubel, D. C. Shannon, A. C. Barger, and R. J. Cohon. Copyright 1981 by AAAS.)

FIGURE 7

(Reprinted with permission from Biofeedback and Self-Regulation, 17(4), p.267)

MEAN RSA AMPLITUDE



Mean RSA amplitude during the different experimental sessions and periods within the session as a function of groups. V, biofeedback of the RSA amplitude without respiratory instructions; VI, biofeedback of the RSA amplitude with respiratory instructions; R, respiratory biofeedback; I, respiratory instructions only; BL, baseline; T, physiological control trial.

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