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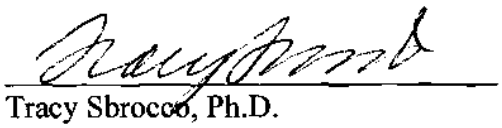

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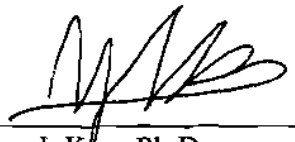

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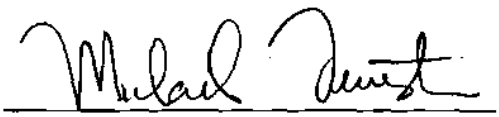

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ABSTRACT

Title of Thesis:	"Heart Rate Variability in Male Sexual Arousal and Erectile Dysfunction"
Author:	Robert D. Clark, Master of Science, 2007
Thesis directed by:	Tracy Sbrocco, Ph.D., Professor Department of Medical and Clinical Psychology

The life-time prevalence of a male sexual dysfunction ranges from 31-52%. Autonomic nervous system (ANS) differences between men with and without erectile dysfunction (ED) may have clinical relevance to the treatment of ED. Parasympathetic nervous system (PNS) activation is necessary to produce erections. ED may be associated with reduced PNS activation, consequently limiting erection capacity. The purpose of this study was to compare the ANS responding of sexually functional and dysfunctional men. Potentially, differences could explain the development and maintenance of ED. Heterosexual men, ages 18-60 with and without psychogenic ED were monitored for tumescence and heart rate variability (HRV) in response to visually erotic video clips. PNS activity (hf-HRV) increased in both groups during erotic stimuli. However, ED participants did not have attenuated PNS increases as compared to functional participants. Anxiety did not moderate the relationship between sexual arousal and changes in hf-HRV. Additionally, PNS activation did not predict changes in tumescence. In this study, PNS activation for men with and without ED was similar and was not associated with other factors known to impact tumescence.

Heart Rate Variability in Male Sexual Arousal and Erectile Dysfunction

by

Robert D. Clark

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LIST OF ACRONYMS

Term	Acronym
Erectile Dysfunction	ED
Autonomic Nervous System	ANS
Parasympathetic Nervous System	PNS
Sympathetic Nervous System	SNS
Heart Rate Variability	HRV
Massachusetts Male Aging Study	MMAS
National Health and Social Life Survey	NHSLS
High Density Lipoprotein	HDL
Body Mass Index	BMI
Waist-to-Hip Ratio	WHR
Selective Serotonin Reuptake Inhibitor	SSRI
Electrocardiogram	ECG
Sleep Related Painful Erections	SRPE

INTRODUCTION

Approximately 31-52% of men within the United States have experienced a sexual dysfunction at some point in their lifetimes with rates increasing during aging (Feldman, Goldstein, Hatzichristou, Krane, & McKinlay, 1994; Laumann, Paik, & Rosen, 1999). Although sexual dysfunctions, including erectile dysfunction (ED), may vary in type, duration, and origin, most adversely affect intimate relationships, quality of life, and self-esteem (Heiman, 2002). Despite the negative impact and the high rates of male sexual dysfunctions, little is known about their physiological mechanisms, consequently limiting diagnoses and treatment. It is well known that the autonomic nervous system (ANS) plays an integral role in achieving and maintaining erections (Brindley, 1992; Giuliano & Rampin, 2004; Guay et al., 2003; Masters, Johnson, & Kolodny, 1982; Miller, 2001; Monga & Hellstrom, 1999), but there is sparse knowledge about the relative ANS activation during human sexual arousal or sexual activity. Therefore, additional research is needed to determine ANS related processes in relation to sexual arousal and sexual dysfunctions.

The Autonomic Nervous System and Erectile Physiology

Masters and Johnson (1966, 1982) pioneered the field of human sexuality and much of today's work is based on their observations. They defined the sexual response in four stages: excitement, plateau, orgasm, and resolution. During excitement and plateau, blood is shunted throughout the body in response to sexual stimuli, particularly to the genitals. In males, the penis becomes erect (tumescence) as penile smooth muscles relax allowing an increased amount of blood to move through the penis and other erogenous areas. The nipples, abdomen, and rectum also become increasingly sensitive to touch.

Orgasm is characterized by muscle contractions throughout the body, particularly the pelvic muscles. In most cases, male ejaculation occurs simultaneously with orgasm and refers to contractions of prostate and pelvic muscles around the base of the penis to release semen. After orgasm, penile smooth muscles contract forcing the blood to drain from the penis causing flaccidity (detumescence).

At the time of their first seminal book, *Human Sexual Response*, Masters and Johnson (1966) had limited technology to explore internal physiological sexual responses. By the time their second book, *Sex and Human Loving* (Masters, Johnson, & Kolodny, 1982), was published, sexuality research was expanding especially in relation to the physiological, hormonal, neurochemical, and cardiovascular changes during sexual arousal and activity. New tools and methodologies, such as vaginal photoplethysmography and penile plethysmograph, became available to examine the ANS during the human sexual response (deGroat & Booth, 1980; Hart, 1974; Zuckerman, 1971). The knowledge gained from these advancements allowed researchers and physicians to predict and study how the various internal physiological systems respond during sexual arousal and activity. Throughout the 1980's and 1990's scientists used and expanded these technologies to understand how disruptions in physiological and psychological systems alter sexual responding.

Despite these advancements and the increase in sexuality research, currently there is a paucity of information about how the body's various physiological systems, especially the autonomic nervous and the cardiovascular systems, are activated during human sexual arousal and activity. In addition, how activation of these physiological systems might differ in those with sexual dysfunctions is unclear. Most research on the

ANS during sexual responses has been, and continues to be, performed on laboratory animals, on cadavers, or requires the use of invasive procedures. There is a necessity for real time research using non-invasive tools, such as heart rate variability (HRV), to measure physiological responses before, during, and after sexual arousal and activity on humans. These tools can provide quantitative data to add to the current knowledge base of both male and female human sexuality and may be used to increase the accuracy of sexual dysfunction diagnoses and treatment.

The Autonomic Nervous System

The ANS is comprised of two pathways: the parasympathetic and the sympathetic nervous systems (Figure 1). The parasympathetic nervous system (PNS) slows metabolism to conserve energy, slows heart rate, constricts blood vessels, and increases reproductive responses (tumescence, vaginal lubrication). Conversely, the sympathetic nervous system (SNS) increases metabolism to mobilize energy, increases heart rate, dilates blood vessels, and decreases reproductive responses (detumescence). Generally, the body is under PNS control and activates the SNS during physical and psychological stressful circumstances. Therefore, the ANS is in constant flux depending on the various situations of daily life, all of which can influence erectile functioning (Goldstein, 2001, pp. 24-38).

To achieve and maintain an erection relies on a delicate balance between the PNS and SNS, which coordinate adrenergic, cholinergic, and non-adrenergic/non-cholinergic (NANC) pathways (Brindley, 1992; Giuliano & Rampin, 2004; Guay et al., 2003; Miller, 2001; Monga & Hellstrom, 1999). Adrenergic sympathetic tone, arising from the thoracic and lumbar (TH12-L2) regions in the spinal cord, maintains flaccidity by

releasing the potent vasoconstrictors norepinephrine and endothelin-1. This tone constricts the blood vessels and penile smooth muscles, consequently limiting blood flow through the penis. During sexual stimuli, arousal, and activity in males, PNS activation of cholinergic and NANC pathways from the sacral (S2-S4) regions in the spinal cord releases acetylcholine and nitric oxide (NO), which are involved in producing erections. NO is the primary mediator of penile smooth muscle relaxation, vasodilation, and increased blood flow to the penis to achieve and maintain erections (Kakiailatu, 2000). During and after orgasm, the SNS once again activates the adrenergic pathway, resulting in ejaculation and detumescence.

A shift in the ANS to become under more PNS control is necessary to produce erections. Any disruption in the control of the ANS or blood flow to the penis, such as increased SNS activity, decreased PNS activity, inadequate hormone or neurotransmitter levels, or physiological damage, can adversely influence the sexual response and produce ED. Therefore, it is imperative that researchers, clinicians, and physicians comprehend how the ANS affects the various physiological systems that influence erectile functioning when diagnosing sexual dysfunctions.

Summary

The ANS controls reproductive functioning including the ability to have and maintain erections. In the absence of injury, the PNS increases tumescence, whereas the SNS is associated with ejaculation, detumescence, and the maintenance of penile flaccidity. Disruption of the ANS, especially to become SNS mediated, can decrease the ability to produce and/or maintain erections.

Erectile Dysfunction (ED)

DSM-IV-TR criteria for a diagnosis of ED include marked distress from difficulty achieving or maintaining an erection for satisfactory sexual intercourse (APA, 2000; NIH consensus conference. Impotence, 1993). Causes of ED generally are divided into organic and psychogenic causes. Despite this dichotomous division, it is likely that in most cases of ED there is a combination of organic and psychogenic causes. To make a diagnosis of ED caused by purely psychological factors, all organic causes must be ruled out. If medical conditions or substance use along with psychological factors impact erectile functioning, then the ED is diagnosed as arising from combined factors. Purely organic ED is difficult to diagnose because various psychological states, such as depression, can be both a cause as well as a symptom of ED.

In addition to a formal diagnosis of ED, there are defined subtypes of ED (APA, 2000). If the ED has been present since the onset of sexual functioning, then it is considered to be life-long. Other individuals may have developed ED after a period of active sexual function, termed acquired ED. The context of sexual activity also influences erectile functioning such that a man could have ED in all sexual situations (generalized ED) or in specific sexual situations (situational ED).

ED from Organic Causes

The Massachusetts Male Aging Study (MMAS; Feldman, et al., 1994) and the National Health and Social Life Survey (NHSLS; Laumann, Paik, & Rosen, 1999) are major epidemiological studies that assessed organic and psychogenic risk factors of ED. In the MMAS study, men had fewer erections as they aged such that approximately 40% of men at 40 years old and 67% of men at 70 years old had ED (Araujo, Mohr, &

McKinlay, 2004; Feldman et al., 1994). When Laumann, Paik, and Rosen (1999) examined the NHSLS data, they found a 3-fold increase in erectile problems in the oldest cohort of participants (ages 50-59) as compared to the youngest cohort (ages 18-29). Additionally, each year of life increases the probability of developing ED by 1.3% (Rowland, Thornton, & Burnett, 2005) and as men age, the frequency of daytime and nocturnal erections decreases (Moore, Strauss, Herman, & Donatucci, 2003). Therefore, ED might be a consequence of aging. In addition, the increase of comorbid diseases during the aging process also may influence the formation of erectile problems.

ED was positively associated with hypertension, heart disease, and diabetes in the MMAS study (Feldman, et al., 1994). Similarly, researchers using NHSLS data found that the risk of ED increases as health deteriorates (Laumann, Paik, & Rosen, 1999). Other researchers have found that cardiovascular risk factors such as ischemia (Roose, 2003), coronary heart disease (Wabrek & Burchell, 1980), peripheral vascular disease (Blumentals, Gomez-Caminero, Joo, & Vannappagari, 2003), and decreased high density lipoprotein (HDL) levels (Feldman, et al., 1994) are associated with ED. The neuropathy caused by diabetes mellitus also greatly increases the risk of developing ED (Ellenberg, 1980; Hecht, Neundorfer, Kiesewetter, & Hilz, 2001). Low testosterone levels can reduce a man's sex drive enough to limit production of erections (Motofei & Rowland, 2005) and physical damage to the vasculature or smooth muscle can directly effect penile functioning by limiting blood flow to the penis (NIH consensus conference. Impotence, 1993).

Lifestyle choices greatly affect the health of any individual, including sexual health. Saigal (2004) suggested that overweight and obese individuals are at risk for

developing a myriad of health conditions, all of which can decrease erectile function. Obesity has been found to be negatively associated with erectile function such that there is approximately a 1.7 risk ratio for developing ED in obese men (Shiri, et al., 2004). Esposito et al., (2004) performed a randomized controlled trial examining the effects of lifestyle changes on ED in obese men. They found that erectile functioning was negatively associated with body mass index (BMI) (r = -0.37, p = 0.02), weight (r = -0.45, p = 0.01), and waist to hip ratio (WHR) (r = -0.49, p = 0.007) and erectile dysfunction was positively associated with average blood pressure (r = 0.28, p = 0.03). Erectile functioning improved as the intervention participants decreased their BMI, WHR, and blood pressure. Although obesity is positively associated with ED, it may be the other health behaviors and individual characteristics that co-occur in obese individuals that may further promote the development of ED.

Prescription medications can increase the risk of ED. Anti-hypertensive medications such as β-blockers, diuretics, and thiazides have been implicated in numerous studies as reducing erectile functioning. In the MMAS, some participants taking antihypertensives (14%), vasodilators (36%), and other cardiac drugs (28%) demonstrated complete ED (Feldman, et al., 1994). Antidepressants, particularly selective serotonin reuptake inhibitors (SSRIs), have been shown to reduce sex drive and increase ED (Derby, Barbour, Hume, & McKinlay, 2001; Dusing, 2005; Keene & Davies, 1999; Nusbaum, 2002). Often, switching to medications with less sexual side effects or ceasing the regimen will return erectile function.

The use of licit or illicit substances can influence sexual functioning. Both alcohol and smoking can impair the ability of a man to achieve or maintain an erection.

Excessive alcohol consumption and smoking were found to be associated with increased risk of ED in the MMAS (Feldman, et al., 1994). Shiri et al., (2004) found similar results smoking (risk ratio = 1.5, 95% CI: 0.9-2.2) and noted that smokers that had no current sexual problems were 1.3 times more likely (95% CI: 0.8-2.1) to develop ED as compared to non-smokers. Other researchers documented that participants who smoked were 34% more likely to have ED (Rowland, Thornton, & Burnett, 2005). The use of illicit drugs such as heroin, cocaine, and barbiturates may interrupt the sexual response, consequently decreasing erectile functioning (Johnson, Phelps, & Cottler, 2004; Keene & Davies, 1999; NIH consensus conference. Impotence, 1993). For reviews of drug-induced male sexual dysfunction, including ED, see Keene and Davies (1999) and Wein and Van Arsdalen (1988).

ED from Psychogenic Causes

Psychogenic ED may arise from many areas of life. The majority of researchers examining psychogenic ED have focused on cognitive interference, specifically anxiety (Hedon, 2003; Norton & Jehu, 1984). Although anxiety can influence ED, it has been suggested that any cognition that draws the erotic focus away from the sexual stimuli can affect erectile functioning (Barlow, 1986). This interference can arise from psychiatric disorders, relationship problems, stressors, distress, anxiety, fear of failure, performance demand, negative outcome expectancies, or numerous other areas (Barlow, 1986; Farre, Fora, & Lasheras, 2004; NIH consensus conference. Impotence, 1993). Although beyond the scope of this paper, some medicinal treatments for ED such as sildenafil and tadalafil may improve some of these risk factors by increasing sexual self-confidence, spontaneity of sexual encounters, sexual desire, overall sexual satisfaction (Dean, Hackett, Gentile,

Pirozzi-Farina, Rosen, Zhao, Warner, & Beardsworth, 2006; Rosen, Janssen, Wiegel, Bancroft, Althof, Wincze, Segraves, & Barlow, 2006).

Depression is one of the largest psychological risk factors for ED and was found to be positively related to ED in the MMAS (Feldman et al., 1994; Moore, Strauss, Herman, & Donatucci, 2003). However, men with ED also have been found to endorse depressive symptoms (Roose, 2003). Therefore, it is difficult to determine how depression and ED influence each other.

Psychological stressors such as work, family, and relationship difficulties as well as anxiety can be important factors in psychogenic ED. Laumann, Paik, and Rosen (1999) found that emotional distress was a risk factor for developing sexual dysfunctions, potentially increasing the risk of ED by 18% in those with moderate psychological problems (Rowland, Thornton, & Burnett, 2005). Additionally, anxiety that invoke cognitions of failure or negative outcome expectancies during sexual situations increases the risk of developing ED. During stressful and anxious times, individuals become highly focused on relieving their stress and anxiety that they may be distracted from sexual cues and not become easily sexually aroused (Smith, 1988). For additional reviews of the causes of erectile dysfunction see Nusbaum (2002) and Smith (1988).

Summary

The development and maintenance of ED is likely caused by many variables, such as organic and/or psychogenic factors. Organic causes such as vascular damage, direct injury, Peyronies disease, heart disease, and diabetes can decrease blood flow to the penis, consequently limiting tumescence. In addition, psychogenic causes such as relationship difficulties, psychiatric illness, stressors, emotional distress, anxiety, and

performance demands all may act as cognitive distracters which can disrupt the PNS response to produce erections during sexual stimuli. In most cases, ED is influenced by a combination of organic and psychogenic causes, which can make diagnoses and treatment challenging.

Model of Psychogenic ED

Sbrocco and Barlow (1996; Figure 2) proposed that task engagement and self-focused attention are necessary to produce an erection during sexually arousing stimuli. If a man believes that he is unable to fulfill the specific sexual activity, then he may perceive the activity as a threat. For example, a man who is physically able (i.e., no organic cause of ED) to have intercourse but does not believe he can perform this activity, has created a discrepancy between his actual and subjective performance capabilities. Such discrepancies may arise when an individual has skill deficits, unrealistic sexual schemas, or negative outcome expectancies. Therefore, during task engagement and discrepancy adjustment the individual must assess whether he can achieve the task (positive outcome expectancy) or cannot achieve the task (negative outcome expectancy). Positive outcome expectancies generally produce functional performance, whereas negative outcome expectancies may produce task disengagement and dysfunctional performance.

How cognitive distracters influence the physiological sexual responses is an area of ongoing research. It is well known that stressors, emotional distress, and anxiety shift the ANS balance towards PNS withdrawal and/or SNS activation (Hoehn-Saric & McLeod, 1988). This shift clearly is observed in individuals with generalized anxiety

disorder (Thayer, Friedman, & Borkovec, 1996) and panic disorder (Friedman & Thayer, 1998). When the PNS is withdrawn or the SNS is activated, erectile response is limited.

If a man perceives that he is able to complete a sexual activity, then activation of the PNS is likely to occur followed by functional sexual performance. Conversely, in the absence of perceived ability to complete a sexual activity, dysfunctional performance may ensue because cognitive distracters such as stressors, anxiety, or negative outcome expectancies result in PNS withdrawal and/or activation of the SNS. In the absence of organic causes of ED, management of cognitions purportedly mediate the activation of the PNS and SNS during sexual activity, as such determines or psychogenic dysfunctional performance. Currently, it is known that psychological factors such as psychiatric conditions, anxiety, emotional distress, stressors, and self-focus may impair sexual functioning, but the physiological mechanisms that cause such impairment are unclear.

Heart Rate Variability

Heart rate does not beat in an exactly regular fashion (Task Force of the European Society of Cardiology & The North American Society of Pacing and Electrophysiology, 1996). Internal control mechanisms such as respiration, blood pressure and temperature regulation, the renin-angiotensin system, and circadian rhythms, and external influences, such as physical activity, exercise, and physical or psychological stress changes the variability of the heart. These irregularities are what researchers have termed heart rate variability or HRV, which is determined by analyzing the intervals between sinus beats or R-R intervals (Task Force, 1996). It is believed that the more variability in the heart, the better the cardiovascular system can adapt to stress. Exercise and cardiovascular fitness

generally are associated with increased HRV, whereas many disease states are associated with reduced HRV (Cohen, Matar, Kaplan, & Kotler, 1999; Pieper & Hammill, 1995; Stein & Kleiger, 1999; Task Force, 1996). HRV allows physicians and researchers to assess the functioning of the heart and the balance of the ANS (PNS & SNS) in a myriad of healthy and diseased states.

HRV is based off of recorded electrocardiograms (ECG), which are recorded using non-invasive means in the laboratory or by portable Holter monitors. The two main approaches to analyzing HRV are time domain and frequency domain analyses (Table 1). Time domain analyses assess the quantity of heart rate variability by examining the variation between R-R intervals (e.g., changes over time in the length between R peaks). Frequency domain analyses assess the rhythms of the heart rate variability using spectral density analyses or how much of given frequency appears over a range of frequencies. Both approaches are used to examine PNS tone, but frequency domain is able to separate the relative frequency of activation between the PNS and the SNS (Cohen, Matar, Kaplan, & Kotler, 1999; Pieper & Hammill, 1995; Stein, Bosner, Kleiger, & Conger, 1994; Task Force, 1996).

Time Domain Analyses of HRV

There are two methods of time domain analysis (Cohen et al., 1999; Pieper & Hammill, 1995; Stein et al., 1994; Task Force, 1996). The first method consists of statistical analyses of the interbeat intervals' standard deviations over a 24-hour (or shorter duration) ECG recording to determine HRV. A variety of algorithms are employed to calculate HRV using interbeat intervals: standard deviation from all normal R-R intervals (SDNN), the average standard deviation calculated from all normal R-R

intervals for 5-minute sections (SDNN index), and others. The second method of time domain analysis compares the lengths between adjacent cycles. Methods that compare lengths between adjacent cycles include analyzing the percent of adjacent cycles that are less than 50 msec apart (pNN50) and taking the square root of the sum of the squared differences between normal R-R intervals (rMSSD) (Table 1).

Frequency Domain Analyses of HRV

The variance of the heart rate caused by periodic fluctuations is used in frequency domain analyses. Different variances are separated into frequencies, called power spectral density (PSD) or spectral analysis (McCraty, Atkinson, Tiller, Rein, & Watkins, 1995). The Task Force (1996) has determined two sets of frequency bands based on the duration of the ECG recording that provide different information about the activation of the ANS. There are three bands of frequencies for short-term recordings: high frequency power (hf-HRV), low frequency power (lf-HRV), and very low frequency power (vlf-HRV). Long-term recordings include the HF, LF and VLF, but add ultra low frequency power (lf-HRV). The total power (tp-HRV) is the sum of all the frequencies and consists of the total variance for the entire ECG recording. Another analysis for short-term recordings includes the lf/hf ratio which is used to determine the relative activation of the SNS as compared to the PNS.

The four bands have distinct frequencies ranging from 0 to 0.4 Hz. The power for the individual components is calculated by examining the area underneath the curve that corresponds with the frequency range of each component (Appel, Berger, Saul, Smith, & Cohen, 1989; Cohen et al., 1999; Pomeranz et al., 1985; Stein et al., 1994; Task Force, 1996). hf-HRV (0.15-0.40 Hz) represents PNS and respiration activity as demonstrated.

To demonstrate that hf-HRV is a measure of PNS activation, researchers have performed experiments using electrical vagal stimulation (Akselrod, Gordon, Ubel, Shannon, Barger, & Cohen, 1981) and muscarinic receptor blockade (Pomeranz, et al., 1985). Electrical vagal stimulation increases PNS activation and the hf-HRV component. Pomeranz, et al. (1985) examined HRV changes using frequency domain analyses from supine (PNS) and standing (SNS) positions in humans. Using a muscarinic receptor blockade which increases SNS activation and decrease PNS activation, they demonstrated that PNS activity mediates all frequencies between 0.024 and 1.0Hz, whereas SNS specifically mediated frequencies between 0.04 and 0.12 Hz. Pagani, et al. (1986) also found that hf-HRV predominated during the supine position, in which the PNS is activated, and lf-HRV increased with an injection of nitroglycerin, an agent known to activate the SNS. Therefore, the evidence support that hf-HRV is an analogue of PNS activation is well supported.

The notion that lf-HRV represents purely SNS activation is less supported by evidence. There is no single frequency for sympathetic tone, but lf-HRV (.04Hz -.15Hz) is believed to originate from a combination of PNS and SNS activation along with baroreceptor activity. As found by Pomeranz, et al. (1985), PNS activation is involved in lf-HRV (0.024-0.12 Hz) and therefore, lf-HRV generally is not considered a pure measure of SNS activation. The precise mechanisms underlying vlf-HRV are yet to be determined and some researchers will either separate or combine vlf-HRV and ulf-HRV. vlf-HRV (0.0033-0.04 Hz) and ULF (1.15×10^{-5}-0.0033 Hz) may be associated with thermoregulation, the renin-angiotensin system, or vasomotor activity. ulf-HRV also may reflect circadian rhythms. Despite the lack of pure measure of lf-HRV, some

researchers consider the lf/hf ratio a measure of SNS/PNS balance (Lombardi, Malliani, Pagani, & Cerutti, 1996; Malliani, Lombardi, & Pagani, 1994). The multiple statistical algorithms used to analyze HRV can be found in Table 1 and in review articles (Cohen et al., 1999; Pieper & Hammill, 1995; Stein et al., 1994; Task Force, 1996).

Health, Disease, and HRV

HRV has evolved into measuring ANS activity in a variety of healthy and disease states (Kristal-Boneh, Raifel, Froom, & Ribak, 1995). HRV can be used clinically to determine cardiac tone, autonomic activity, and vulnerability from disease (Kautzner & Camm, 1997). During the aging process HRV decreases and it has been suggested that this decrease is a result of reduced PNS tone to the heart (Reardon & Malik, 1996). Applying this research to erectile functioning, it follows that as men age parasympathetic tone decreases, consequently making it more difficult for erections to be achieved, which is consistent with current ED data.

Moderate aerobic exercise is generally associated with decreased mortality and mortality, but results from research examining aerobic exercise have been inconclusive in determining changes in HRV. Some researchers have found that aerobic exercise increases HRV, consequently making the body better suited to adapt to stress (Uusitalo, Laitinen, Vaisanen, Lansimies, & Rauramaa, 2002; Yataco, Fleisher, & Katzel, 1997) whereas other researchers have found that aerobic exercise does not change HRV (Uusitalo, Laitinen, Vaisanen, Lansimies, & Rauramaa, 2004). In a review of the literature, Kristal-Boneh et al. (1995) state that the majority of studies reported that HRV increases in response to aerobic exercise.

Conversely, abnormal HRV patterns have been implicated in many diseases and even have become markers for mortality. Researchers examining HRV and heart disease, such as congestive heart failure (Jiang, Hathaway, McNulty, Larsen, Hansley, Zhang, & O'Connor, 1997), ischemia (Kop, Verdino, Gottdiener, O'Leary, Bairey Merz, & Krantz, 2001), hypertension (Singh, Larson, Tsuji, Evans, O'Donnell, & Levy, 1998), and myocardial infarction (Craelius, Akay, & Tangella, 1992), have found that reduced HRV is a significant predictor of mortality and other adverse cardiovascular outcomes in these disease states. Other diseases that interfere with cardiovascular process such as diabetes (Flynn, Jelinek, & Smith, 2005), depression (Agelink, Box, Ullrich, & Andrich, 2002), and sleep apnea (Vanninen, Tuunainen, Kansanen, Uusitupa, & Lansimies, 1996) are all associated with abnormal HRV patterns. For a review of HRV in health and disease see Kristal-Boneh, Raifel, Froom, and Ribak (1995).

Because ED and cardiovascular disease are related, it has been suggested that ED might be an early predictor of cardiovascular problems (Russell, Khandheria, & Nehra, 2004). HRV may be a beneficial tool to use in both research and practice to assess cardiac autonomic tone with patients presenting with ED symptoms. However, HRV patterns during sexual arousal and activity must be defined as well as how HRV patterns may differ in individuals with sexual dysfunctions. Until standards of HRV during sexual arousal and activity are formulated, it is difficult to predict whether HRV would be a useful tool in diagnosing cardiovascular problems in ED patients.

Summary

HRV is a non-invasive technique that measures ANS tone to the heart, which is presumed to reflect ANS tone throughout the body. Different statistical techniques are

used to separate the heart rate into frequencies, standard deviations, or lengths between R-R intervals. Generally, decreased HRV is a significant predictor of mortality and morbidity in many diseased sates including ischemia, atherosclerosis, diabetes, and cardiovascular disease. Conversely, moderate aerobic exercise can be a cardioprotective factor by increasing HRV. If HRV differences between sexually functional individuals and individuals with ED exist, then HRV could be valuable tool in assessing and treating sexual dysfunctions.

HRV and Sexuality Research

HRV assessment before, during, or after sexual arousal has rarely been employed in sexuality studies. Consequently, little is known about the relative HRV-derived measure of ANS activation during the different phases of sexual arousal. In addition, few researchers have examined how ANS activity might differ in those individuals with sexual dysfunctions. Of the studies that have examined HRV during sexual activity or arousal, few have controlled for age, medical conditions, or other factors that influence HRV. Therefore, conclusions from these studies should be made with caution. The lack of information about the role of the ANS during sexual arousal and activity leaves a large gap in the sexuality research. Knowledge gained from such research can impact how sexual dysfunctions, like ED, are diagnosed and treated.

HRV and Sexual Functioning

Three studies have been performed examining the influence of sexual activities on HRV. In a preliminary study assessing HRV, sexual intercourse frequency, and cohabitation status in healthy participants, Brody, Veit, and Rau (2000) found that increased frequency of self-reported penile-vaginal intercourse, but not masturbation or

other sexual activities, was associated with greater levels of HRV among cohabitating individuals [$F(1,38) = 8.4$, $p < 0.01$]. Brody and Preut (2003) replicated this study and found similar results [$F(1,100) = 5.8$, $p < 0.02$] and concluded that the frequency of penile-vaginal intercourse among cohabitating individuals correlated positively with HRV. If sexual intercourse is considered an aerobic activity and aerobic activity increases HRV, then it follows that increasing sexual intercourse frequency would increase HRV. Generalizing these data, it might be assumed that individuals with ED, who are less likely to engage in sexual intercourse, would have decreased HRV. However, the studies were cross sectional precluding causal interpretations, used healthy participants, and negative cognitions that may influence sexual performance were not measured.

In another study, Yeragani, Pohl, and Balon (2004) analyzed HRV during sexual activity. All participants were healthy, wore 24-hour Holter monitors, and kept an activity diary. The authors matched ECG readings with corresponding sexual activity. They found that the lf/hf ratio increased during orgasm [before orgasm: $M = 10.3$, $SD = 4.5$; during orgasm: $M = 21.4$, $SD = 7.3$; $F(2,10) = 15.1$, $p < 0.001$], caused by an increase in lf-HRV [before orgasm: $M = 22$, $SD = 11$; during orgasm: $M = 37$, $SD = 23$] and no change in hf-HRV [before orgasm: $M = 6.6$, $SD = 7.3$; during orgasm: $M = 6.1$, $SD = 7.7$]. These results are consistent with current knowledge that orgasm is associated with an increase in SNS activity.

HRV and ED

Hypertension and mental stress have been implicated as risk factors for ED (Pagani, 2000). In a review of the ED and HRV literature, Pagani (2000) noted that ED

is likely to co-occur in individuals with hypertension. He found that hypertension increases and erectile functioning decreases in response to mental stress. In addition, HRV is greatly decreased during mental stress and also in individuals with hypertension. Therefore, he suggested that hypertension and mental stress purportedly alter the ANS balance towards PNS deactivation and/or SNS activation consequently adversely impacting erections.

HRV has been used to investigate how the cardiovascular system may be differentially affected by the different causes of ED. Ferini-Strambi, et al (1996) compared HRV of men with sleep related painful erections (SRPE) to controls who did not have SRPE. Individuals with SRPE had reduced PNS activity (assessed by the sleep/wakefulness ratio; Rs/w) during both non-REM (Rs/w; 1.08 ± 0.10 vs. 1.15 ± 0.08, $p = 0.03$) and REM (Rs/w; 1.04 ± 0.13 vs. 1.14 ± 0.08, $p = 0.01$) sleep as compared to controls. They suggested that a dysfunction in the β-adrenergic system decreases parasympathetic activity in the etiology of SRPE.

Hecht, et al. (2001) assessed HRV in participants with diabetic ED. Because ED might be one of the first symptoms of diabetes, researchers were interested in understanding how diabetes altered penile functioning. Participants were into three groups: diabetic ED, neuropathic ED, and other ED. HRV abnormalities were slightly more frequent, but not significantly so, in individuals with diabetic ED (39%) as compared to neuropathic ED (25%). Therefore, it was concluded the neuropathy was present in diabetic participants, possibly being the cause ED in the diabetic men. However, normative HRV values were based on a sample of diabetic individuals with neuropathy rather than normative HRV values based on the larger samples representative

of the population. Additionally, there was no proper control group (i.e., participants with no ED) to compare HRV values across groups.

Lavie, Shlitner, and Nave (1999) compared nocturnal penile tumescence of men with psychogenic ED and organic ED. Participants with psychogenic ED had lower lf/hf ratios [$F(1,34) = 6.77$, $p < 0.01$], higher hf-HRV areas [$F(1,34) = 9.53$, $p < 0.004$], and lower lf-HRV areas [$F(1,34) = 9.53$, $p < 0.004$] across all stages of sleep (non-REM & REM) as compared to participants with organic ED. Both groups of participants demonstrated increases in the lf/hf ratio [$F(1,34) = 10.58$, $p < 0.003$] and decreases in hf-HRV [$F(1,34) = 30.53$, $p < 0.00001$] during REM sleep as compared to non-REM sleep, with a trend for participants with psychogenic ED having larger decreases in the hf-HRV area [$F(1,34) = 3.32$, $p < 0.08$]. However, participants with organic ED had decreases in lf-HRV whereas participants with psychogenic ED had increases in lf-HRV in REM sleep as compared to non-REM sleep [$F(1,34) = 7.58$, $p < 0.009$]. The evidence supports the notion that men with organic ED may have different patterns of HRV, particularly lower total hf-HRV, higher total lf-HRV and decreases in lf-HRV during REM sleep, during sleep as compared to men with psychogenic ED, which can reduce nocturnal erections in men with organic ED.

In a similar study, Ferini-Strambi and colleagues (1992) compared HRV patterns of participants with organic and psychogenic ED during sleep. Body movement ratio (Rbm) was significantly decreased in participants with organic ED as compared to participants with psychogenic ED during both non-REM (1.35 ± 0.10 vs. 1.51 ± 0.15, $p < 0.001$, respectively) and REM sleep (1.48 ± 0.12 vs. 1.59 ± 0.18, $p < 0.05$, respectively). This information suggests that participants with organic ED had increased sympathetic

activity during sleep as compared to participants with psychogenic ED. The authors proposed that these results were possibly caused by a β-adrenergic dysfunction in participants with organic ED. However, negative or distracting cognitions that could affect ANS activity were not accounted for in either study because participants were tested while asleep.

Daffertshofer, et al. (1994) examined how HRV and sympathetic skin response (SSR) might vary between individuals with and without ED. Men with ED presenting to a urologic clinic were compared to controls without ED. The ED participants were separated into three subgroups: men with neurological deficits with both marked and moderate ED (e.g., diabetic neuropathy, spinal cord lesions), men with another organic disease (e.g., cardiovascular disease, arteriosclerosis, painful urinary stenosis), and men with "psychiatric" disturbances (i.e., no verified organic cause). Controls and participants with a "psychiatric" disturbance demonstrated similar HRV patterns and SSR responses at the penis, hand, and leg. However, some participants with neurological deficits (53%) and organic disease (43%) presented with HRV patterns that differed from controls. In addition, some neurological deficit participants demonstrated no SSR responses at any of the three measurement points, whereas all organic disease participants presented with SSR responses similar to controls.

There are methodological flaws that limit generalization of these results to men with psychogenic ED. First, as noted by the authors, these participants were diagnosed with psychiatric disturbance by ruling out organic causes and not by DSM-IV criteria for psychogenic ED. Second, it is unclear whether the participants had psychogenic ED or other psychiatric disturbances that may have impacted erectile functioning. Third, there

were only six participants in the psychiatric group. Given these limitations the data may not be relevant to men with a formal DSM-IV diagnosis of ED.

Summary

Few studies have examined HRV in relation to sexual arousal and activity. The evidence from these studies has predominantly focused on organic ED participants, which have been shown to have HRV patterns that differ from controls or men with other types of ED. On the contrary, psychiatric ED participants generally do not display HRV changes that differ from controls. Many of the published studies have methodological flaws that prevent direct comparisons of sexually functional participants and participants with psychogenic ED. The present study avoids these limitations which will allow HRV comparisons of sexually functional men and men with DSM-IV diagnosed psychogenic ED.

Rationale

It is well established that activation of the PNS is necessary to produce erections. In addition, researchers have suggested that men with ED have many negative cognitions, cognitive distracters, and anxiety during sexual stimuli. These factors can reduce the activation of the PNS, consequently limiting erection capabilities. The intention of this study was to explore such ANS differences in men with and without psychogenic ED which could explain the formation and maintenance of ED. The information gained from this study can have significant implications for sexual dysfunction research, diagnoses, and treatment.

This study aims to explore three hypotheses (Figure 3). Because PNS activation is necessary for tumescence, increases in PNS activity are expected during the erotic

stimulus for both sexually functional and psychogenic ED men (Hypothesis 1a). This study further examines ANS differences between sexually functional men versus men with psychogenic ED (Hypothesis 1b), which has never been examined. Anxiety (Hypothesis 2a & 2b) is proposed to moderate the relationship between exposure to sexual stimuli and HRV changes such that PNS activity in ED and high anxiety men was expected to be attenuated as compared to sexually functional and low anxiety men, respectively. The key issue for clinical practice is whether the ANS disturbances predicted in Hypotheses 2 limit tumescence or whether tumescence limitations are independent of ANS alterations. It is expected that high anxiety men will have attenuated increases in tumescence during the erotic film clip as compared to low anxiety participants and that ANS disturbances partially account for this association (Hypothesis 3). Support for Hypothesis 2 and 3 would bolster the notion that anxiety alters the ANS towards PNS deactivation and/or SNS activation, which in turn, limits tumescence increases during sexual arousal. Therefore, to examine the effects of sexual stimuli on the ANS and tumescence of sexually functional and men with psychogenic ED, the following hypotheses are posited:

Hypotheses

Hypothesis 1 – Increases in hf-HRV during sexual arousal

a) An erotic stimulus will result in elevated hf-HRV in sexually functional men and men with ED, signaling increases in PNS activity necessary for tumescence.
b) Men with ED will have attenuated increases in hf-HRV during the erotic stimulus as compared to sexually functional men.

Hypothesis 2 – The association between anxiety and HRV

a) State anxiety will be associated with attenuated hf-HRV increases to the erotic stimulus.
b) The association between state anxiety and attenuated hf-HRV increase will be greater in men with ED as compared to sexually functional men.

Hypothesis 3 – The association between HRV and anxiety with tumescence

a) The magnitude of hf-HRV increases to the erotic stimulus will be associated with the magnitude of tumescence increases for both sexually functional men and men with ED.
b) Reduced hf-HRV during the erotic stimulus among men with ED will be associated with attenuated tumescence increases.
c) State anxiety will partially account for the relationship between attenuated hf-HRV increases and reduced tumescence in men with ED.

RESEARCH DESIGN AND METHODOLOGY

Measures – Interview

Structured Clinical Interview for Axis I DSM-IV Disorders

Participants were administered the Structured Clinical Interview for Axis I DSM-IV Disorders (SCID) – Patient Edition to screen for mood, anxiety, and psychiatric disorders (First, Spitzer, Gibbon, & Williams, 1994). Current contact with a psychotherapist for behavioral and/or emotional disturbance, other than erectile difficulties for participants with ED also was assessed using this instrument. In addition, participants were asked whether they had any previous psychiatric hospitalizations.

Participants were excluded from the study if they had been or currently were diagnosed with a DSM-IV Axis I disorder assessment.

Sexual Dysfunction Interview

Participants were administered the Sexual Dysfunction Interview (SDI) to assess sexual functioning (Sbrocco, Weisberg, & Barlow, 1995; Appendix E). This one-hour interview asks participants about sexual history, experiences, attitudes, and difficulties and aids the interviewer in formulating a DSM-IV diagnosis of psychogenic ED.

Objection to View Erotic Materials

Participants were asked their experiences with and their emotional reactions to erotic materials such as literature, film, and photographs. Participants expressing concern about viewing erotic materials were excluded from the study.

Measures – Self-Report

Beck Depression Inventory

Screening for depression was performed using the Beck Depression Inventory (BDI-II), a 21-item self-report instrument that assesses cognitive, affective, and psychological aspects of depression (Beck & Steer, 1987; Beck, Steer, & Brown, 1996; Appendix F). On each item, participants chose from one of four responses that increase in severity based on their current feelings. A normal depression score is between 0-9, mild to moderate score between 10-18, moderate to severe score between 19-29, and severe range between 30-63. Beck and Steer (1984) found that the internal consistency of the BDI was good (Chronbach's alpha = 0.88). Because erectile dysfunction and depression are highly correlated, this screening was necessary to account for significant depressive symptoms.

Beck Anxiety Inventory

The Beck Anxiety Inventory (BAI) is a 21-item self-report instrument that was used to assess subjective, somatic, and panic-related aspects of anxiety (Beck & Steer, 1990; Appendix G). This instrument asked participants to rate common anxiety symptoms they might have had within the past week on a scale of 0 ("Not at all") to 3 ("Severely"). A normal anxiety score is between 0-9, mild to moderate score between 10-18, moderate to severe score between 19-29, and severe range between 30-63. Non-disordered populations have been found to report an average score of 6 with a standard deviation of 8 (Gillis, Haaga, & Ford, 1995). In addition, the internal consistency coefficient (Cronbach's alpha) of the BAI is good (0.92; Beck, Epstein, Brown, & Steer, 1988). Anxiety was assessed to account for possible cognitive processes influencing psychogenic erectile dysfunction.

Sexual Arousal Questionnaire

The Sexual Arousal Questionnaire (SAQ; Appendix H) is an 11-item questionnaire developed for this study that was given after viewing the film to assess sexual arousal, anxiety, confidence, attention, control, negative thoughts, and cognitive interference. Visual Analogue Scales (VAS, 10 cm) were used for all ratings with some items having two anchors (e.g. no negative thoughts – lots of negative thoughts) and others having three anchors (e.g. no control – medium control – maximum control). Because this questionnaire was developed specifically for this study, the reliability and validity are unknown.

Measures – Medical Information

Medical Information Form

Participants were mailed and completed a medical history form developed for this study (Appendix I). This form was reviewed with them for relevant medical complications, such as diabetes, genital surgery, and prostatitis, during the interview and by the project physician.

Organicity/Psychogenic Rating Scale

Participants with ED were rated by the study physician on two rating scales: an organicity rating scale and a psychogenic rating scale. *Psychogenic* causes were rated using a 0-5 rating scale (0 = no psychogenic origins to the sexual dysfunction, 5 = psychogenic factors were a major influence on the sexual dysfunction, possibly in a causative relationship) (Appendix J). The project physician reviewed the participants' medical records to form an organicity rating using a 0-5 rating scale (0 = no pathology found, 5 = significant evidence for pathology directly relating to the sexual dysfunction) (Appendix K). Participants scoring a 2 or greater on the organicity rating were excluded from the study.

Measures – Physiological

Penile Plethysmograph

Before each experimental session, the polygraph was calibrated to a range of 40 mm by using a Plexiglas calibrating cone. This process was performed to ensure linear output of penile tumescence changes. A Stretchistor mercury-in-rubber strain gauge 5-10 mm smaller than the circumference of the participant's penis was used to record changes in penile tumescence (D.M. Davis, Inc., Teaneck, NJ). This device consists of a hollow

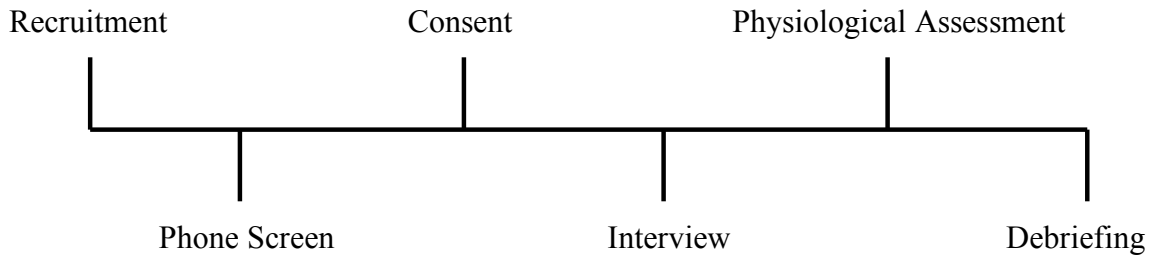
rubber tube filled with mercury that is sealed at the ends. The sealed ends contain platinum electrodes that are inserted into the mercury to form a bridge circuit. Changes in the cross-sectional area of the strain gauge alter the resistance of the mercury, which reflect changes in the circumference of the penis. The bridge circuit relayed these changes to a Grass Instruments Dual Mercury Gauge Adapter with a pre-amplifier (Model F-70DMGAC; Grass Instruments, Quincy, MA). A Grass Instruments 78g polygraph equipped with a 7p122H amplifier and a 7DAK driver amplifier recorded the pre-amplifier output onto polygraph chart paper that moved at a rate of 50 mm/sec. Tumescence data was only gathered during the 5 minute erotic film clip.

Heart Rate

Heart rate was measured using a portable Marquette Series 8500 7-lead ECG Holter monitor (GE-Marquette Medical Systems Information Technologies, Milwaukee, WI) coupled with 3M red dot electrodes (3M, London, Ontario). Continuous heart rate measurement was recorded at a rate of 10 mm/1 mV on cassette tapes during the experimental session (pre-film, film, post-film). Data was uploaded onto a Dell computer with Microsoft Windows for reduction and analysis using the MARS PC software provided by GE-Marquette Medical Systems Information Technologies (Milwaukee, WI, 2003).

Procedure

Procedure Timeline



Participant Recruitment

Sexually functional heterosexual 18-60 year-old males were recruited by newspaper and television advertisements throughout the Washington D.C. Metropolitan area (Appendix A). In addition, all potential participants with ED were referred by local urologists. Urologists were mailed a letter containing the purpose of the study and the types of participants needed. Participants referred by urologists signed a release of information form (Appendix C) to release their medical records to the experimenter.

Summary of Inclusion/Exclusion Criteria

Inclusion Criteria
1) Heterosexual males ages 18-60

Exclusion Criteria
1) Major psychological disturbance
2) Refusal to view sexually explicit materials and to participate in the study
3) A psychogenic rating of < 3 for participants with ED
4) An organicity rating of > 1 for both groups
5) Poor health - current emotional problems, substance abuse, history of heart disease, treated or untreated hypertension, diabetes, kidney problems, or other medical conditions that may influence erectile functioning

Phone Screen

Potential participants were screened by phone using the Phone Screen Form (Appendix B) by a clinical psychology graduate student at the Uniformed Services University in the Department of Medical and Clinical Psychology (MPS), Major Jay Stone, who was under the direction of Tracy Sbrocco, Ph.D. (MPS), and Eveyln Lewis, M.D., Department of Family Medicine. The telephone screen consisted of a semi-structured interview that assessed general information about medical, sexual, and psychiatric histories. Participants not meeting inclusion criteria were excluded from the study. If participants were not excluded from the study during the phone screen, then

they were asked to come to the Uniformed Services University for a screening interview and physiological measurement session.

Informed Consent

Before the screening interview, the participant was informed of nature of the study and asked to sign an informed consent form (Appendix D). Confidentiality was explained to the participant by informing him that the data were coded and that their names would not appear on any records besides the original data files, which would be kept in a locked file cabinet.

Interview

During the screening interview, the participant was administered the SCID and the SDI, was asked to complete the BDI and the BAI, and was asked whether he had objections to viewing erotic materials, which usually took between 1-2 hours. In addition, the medical information form was reviewed.

Physiological Assessment

The experimenter carefully outlined the procedure to the participant, including the use of the penile strain gauge and the Holter monitor, before the physiological assessment began. The participant was told, "You will watch a five-minute videotape showing a man and a woman having sex while we collect all the information we need. Do you have any questions before we proceed?" The participant then was informed that he could choose not to participate without consequence.

The participant was guided to the sound attenuated room and was instructed on how to take the mid-shaft circumference of his penis using a paper strip. The experimenter exited the room while the participant disrobed from the waist down and

took this measurement. Each participant measured the circumference of his flaccid penis by wrapping a strip of paper around the mid-shaft of the penis and marking where the end meets with a pencil. The participant was told to contact the experimenter in the adjacent control room using the intercom when he had the measurement and was fully clothed. When the experimenter returned to the room, the participant was instructed to remove his shirt for the Holter monitor electrode placement. A technician proceeded to attach the seven electrodes to the participant's chest while he sat in a paper lined recliner (see Figure 4 for electrode placement). During that time the experimenter returned to the control room to measure the distance (in mm) of the mark to the end of the paper strip used to measure the participant's flaccid penis. This measurement was used to choose a strain gauge that was 5-10 mm smaller than the participant's flaccid penis. Before the experimenter returned to the room, the strain gauge was calibrated using the Plexiglas calibration cone.

The experimenter returned to the room with a strain gauge and instructed the participant on how to attach it around the mid-shaft of his penis. The experimenter then exited the room while the participant undressed from the waist down, attached the strain gauge, and sat on the paper-lined recliner. The experimenter once again returned to the room to visually inspect that that strain gauge had been attached properly to the mid-shaft of the penis with no twists or bends in it. If the strain gauge had been attached improperly, then the experimenter re-explained how to attach the strain gauge and asked the participant to adjust it accordingly. A medical grade cotton absorbent cloth was place over the participant's lap to prevent him from seeing or touching his penis during the experiment.

The participant was informed that the video clip would begin on the monitor and continue for five minutes. He was instructed to imagine himself involved in the action which he saw on the monitor. In addition, he was asked not to remove the cloth covering his lap or to touch his genitals. The experimenter asked the participant whether he had any questions, dimmed the lights, and left the room. The experimenter returned to the control room to operate the equipment (polygraph and VCR) and to monitor the participant via the intercom.

After the film, the experimenter allowed some time for the participant's penile circumference to return to baseline flaccidity. At this point, the experimenter returned to the room, raised the lights, and asked the participant to complete a Sexual Arousal Questionnaire (Appendix I). Upon completion of the Sexual Arousal Questionnaire, the participant was instructed to remove the strain gauge and Holter monitor electrodes and get dressed. In accordance with manufacturer recommendations (Johnson & Johnson, 1994), the re-useable mercury-in-rubber strain gauges were sterilized after each use by immersing them in Cidex activated dialdehyde solution for 10 minutes followed by three rinsings in clean water. The paper lining on the recliner was disposed.

Debriefing Session

The debriefing session began after the participant was dressed. The experimenter that collected the physiological data informed the participant of the purpose of the study. Participants with ED also were explained how their performance during the experimental session was related to the sexual dysfunction and how the information gained from this experiment could be used to develop treatments. Following debriefing, the experimenter thanked the participant and informed him to call should he have any questions or

problems. Functional participants were paid $40 for their participation and participants with ED were offered treatment for their erectile dysfunction.

DATA ANAYSES

Statistical Software

SPSS (v. 12.0.1; SPSS Inc., Chicago, IL), a statistical software package designed for social science research, was used for data analyses and SigmaPlot 2000 (v. 6.00; SPSS Inc., Chicago, IL) was used for formulating graphs.

Exclusions Based on Missing Data

HRV, demographic, and tumescence data were reviewed to ensure adequacy. A total of 12 participants were excluded from overall data analyses (n = 1 no demographic data, n = 6 sexually functional, n = 5 ED). One participant was excluded because there were no original paper files or demographic data in the original electronic data file. Therefore, this participant's group assignment, demographics, and questionnaire information could not be determined. Seven participants were excluded from the original study because their sexual functioning status was ambiguous (n = 2 sexually functional, n = 5 ED). Four participants (sexually functional) were excluded because they had no pre-film HRV data, consequently limiting HRV analyses. Therefore, this study used data from 33 sexually functional participants and 22 ED participants, for a total of 55 participants. Participant characteristics are presented in Table 2.

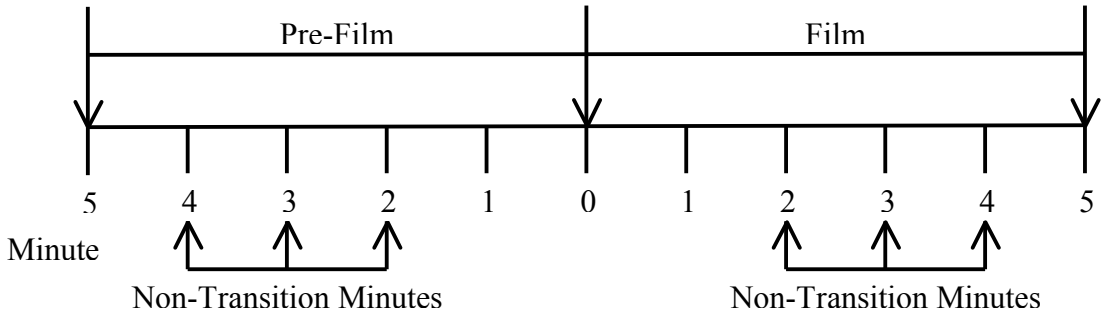
Data Reduction

Heart Rate and HRV

During the experimental session, continuous ECG measurements were recorded on cassette tapes. Data from the tapes were uploaded onto a Dell computer with

Microsoft Windows for reduction and analysis using the MARS PC 6.01 software provided by GE-Marquette Medical Systems Information Technologies (Milwaukee, WI, 2003). Extreme values (ectopic, ventricular, and supraventricular beats) were removed from analyses. Frequency domain parameters were used to assess HRV changes. The software uses a Fast Fourier Transform to separate R-R intervals into high (hf-HRV; 0.15 – 0.40 Hz) and low (lf-HRV; 0.04 – 0.15 Hz) frequency bands expressed in ln (ms^2). The lf/hf ratio also was calculated as an additional index of SNS to PNS balance.

To determine HRV on a per minute basis, the window of analysis was moved in 1-minute increments. The software uses a 1-minute section for HRV trending on each side of the window, which means that to calculate each HRV minute, the minute before, the actual minute, and the minute after are involved in the calculation. Consequently, for the HRV data, transition minutes (the first and last minute of each phase) may skew averages and minute-by-minute changes by reflecting ANS alterations of both baseline and film exposure phases. Therefore, transition minutes were removed and only non-transitional minutes (Pre-Film 4, 3, 2 and Film 2, 3, 4) and their 3-minute averages were used for statistical analyses of HRV. Average HR also was calculated similarly to HRV (i.e., in 1-minute intervals and 3 minute means).



Anxiety

SAQ Question #2 (SAQ#2), which assessed state anxiety after the film, was used as a measure of filmed induced state anxiety. Because the distributions of SAQ#2 for both groups were non-normally distributed, the square root of SAQ#2 was computed and used for data analyses.

Tumescence

Each participant's raw data, expressed in changes in millimeters of penile tumescence, was reduced to the average millimeters of change. The first epoch of penile response was subtracted from subsequent epochs for each film. For each five minute film, penile responses for each participant were divided into 50 time segments/epochs of 6 seconds. The average and maximum tumescence during the film were calculated for each participant.

Analytic Strategies

Hypotheses 1

Hypothesis 1 was analyzed using a 2 x 2 repeated-measures mixed model ANOVA. The between-subjects variable was sexual functioning status (functional, ED) and the within-subjects variable was time (pre-film, film). The dependent variables were the 3-minute pre-film and film hf-HRV averages. Additional repeated measures analyses were performed to examine the minute-to-minute variations in HRV before and during the erotic film.

Hypothesis 2

A hierarchical linear regression was used to examine the effects of sexual functioning status and anxiety on hf-HRV. Three models were tested using the following

predictor variables: Model 1 – sexual functioning status (functional, ED); Model 2 – sexual functioning status and baseline hf-HRV (pre-film 3-minute average); Model 3 – sexual functioning status, baseline hf-HRV, and SAQ#2. This order was chosen to assess how much film hf-HRV variance anxiety accounted for above and beyond differences in sexual functioning status and baseline hf-HRV.

To obtain a single data point for the hf-HRV for each participant, the 3-minute film hf-HRV average was subtracted from the 3-minute pre-film hf-HRV for difference score. This score was entered into regression analyses as the dependent variable.

Hypothesis 3

A hierarchical linear regression was used to examine the effects of sexual functioning status, anxiety, and hf-HRV on average and maximum tumescence. Three models were tested using the following predictor variables: Model 1 – the difference score of hf-HRV; Model 2 – the difference score of hf-HRV and sexual functioning status (functional, ED); Model 3 – the difference score of hf-HRV, sexual functioning status, and SAQ#2. This order was chosen to first examine the role of hf-HRV on tumescence then to examine how much variance is accounted for by sexual functioning status and anxiety.

Sample Size and Power Considerations

Previous studies were used to estimate the effect sizes for the current investigation. However, of the published studies comparing normal controls to men with ED, none used HRV data for their statistical analyses. Instead, they compared the number of participants with abnormal HRV patterns. Therefore, two studies comparing normal controls to diseased participants (depression and diabetes) were chosen to

estimate an effect size. All power analyses were performed using nQuery power analysis software (Statistical Solutions; Saugus, MA).

Agelink, Boz, Ullrich, and Andrich (2002) found that the LF/HF ratio of controls was 2.33 ± 2.07 and the LF/HF ratio of the severely depressed population was 3.86 ± 3.22. In addition, the HF power for normal controls was 22.5 ms^2 ± 13.5 and was 15.7 ms^2 ± 10.8 for severely depressed participants. These results suggest that the ANS of severely depressed participants is sympathetically dominated. Although these results do not exactly parallel the current study, they suggest that the depressed participants have PNS withdrawal. The hypotheses of the current study are that participants with ED will have less PNS activation as compared to normal controls. Both these results yield an effect size (*d)* of approximately 0.6.

Flynn, Jelinek, and Smith (2005) compared normal controls to participants with diabetes. Control participants had a DFA α_1 (a measure of HRV) of 1.12 ± 0.18 and diabetic participants had a DFA α_1 of 1.00 ± 0.22. Similarly to the Agelink, Boz, Ullrich, and Andrich (2002) study, these results suggest a sympathetically dominated ANS in diabetic participants. An approximate effect size (*d*) of 0.60 was calculated using this result.

Power analyses were based on a two-sample *t*-test design because the primary aim was to examine differences between participants with and without ED. Based on prior studies, 29 participants per group are needed to detect an effect size (Cohen's d) of 0.75 (equivalent to $\Delta^2 = 0.14$) with a power of 80% at $\alpha = 0.05$. Regression analyses were powered to detect an increase in R^2 of 0.10 for the added variable under investigation, assuming an explained variance (R^2) of 0.30 accounted for by the

covariates already in the model (examined as one set). Fifty participants are needed to detect an increment of 0.10 in R-squared. The HRV analyses were conducted as part of a larger project, and the available sample size (functional = 39, ED = 28) is therefore sufficient to detect the aformenentioned effect-sizes; however the study is underpowered to detect smaller effects (see discussion).

RESULTS

Demographics

Independent samples *t*-tests and χ^2 analyses were used to examine potential demographic differences between functional and ED participants (Table 2). No significant differences for age [$t(53) = 0.08$, *ns*], ethnicity [$\chi^2(3) = 2.46$, *ns*], relationships status [$\chi^2(4) = 2.85$, *ns*], or education level [$\chi^2(4) = 5.87$, *ns*] were found when comparing functional to ED participants.

Comparisons also were performed on variables that could potentially influence the results of the analyses, such as depression, anxiety, and average and maximum tumescence (Table 3). There were no differences between the sexual functioning groups on the BDI [$t(52) = 0.82$, *ns*], the BAI [$t(52) = -0.24$, *ns*], or average [$t(53) = -0.40$, *ns*] or maximum [$t(53) = -0.26$, *ns*] tumescence. ED participants reported more state anxiety (SAQ#2) than the functional participants [$t(53) = 2.23$, $p = 0.03$].

Preliminary Analyses

Age, HRV, and Tumescence

Because age was likely to be related to HRV and tumescence, the interrelationships among these variables were examined using correlation analyses and are depicted in Table 4.

Age was negatively correlated with the 3-minute pre-film and film hf-HRV averages for all participants [pre-film (r = -0.50); film (r = -0.47)], for sexual functional [pre-film (r = -0.50); film (r = -0.40)], and ED participants [pre-film (r = -0.55); film (r = -0.58)]. Age also was negatively correlated with average (r = -0.37) and maximum (r = -0.31) tumescence for both groups. Age remained negatively correlated with average (r = -0.46) and maximum tumescence (r = -0.49) for ED participants. Age approached significance with average tumescence (r = -0.30), but was not significantly correlated maximum tumescence for functional participants (r = -0.21).

Heart Rate

Changes in heart rate (HR) were examined over the entire experimental session. All available data [functional (N = 38); ED (N = 27)] were used for these analyses. A 2 (group) x 2 [pre-film, film (3 minute means)] repeated measures ANOVA was used to examine changes in HR (Figure 5). There was a trend for interaction between the variables [$F(1,57) = 2.88$; $p = 0.10$], with ED participants decreasing faster [pre-film (M = 80.79, SD = 13.20); film (M = 73.95, SD = 14.04)] faster than functional participants [pre-film (M = 83.50, SD = 14.48); film (M = 79.50, SD = 16.41)]. There also was a significant main effect for time [$F(1,57) = 41.47$, $p < 0.001$] with both groups decreasing HR during the film. Adding age as a covariate did not affect the interaction [$F(1,56) = 3.06$, $p = 0.085$], but reduced the main effect of time [$F(1,56) = 4.08$, $p < 0.05$].

Figure 6 depicts changes in HR by minute for functional and ED participants. Paired samples t-tests revealed a trend for an increase in HR between pre-film 4 (M = 76.00, SD = 11.75) and pre-film 2 [M = 78.93, SD = 12.73; $t(14) = 2.00$, $p = 0.07$], a trend for an increase in HR between pre-film 3 (M = 79.96, SD = 14.25) and pre-film 2

[M = 82.88, SD = 13.59; $t(23) = 1.93$, $p = 0.07$], and a decrease in hf-HRV between pre-film 2 (M = 82.88, SD = 13.31) and film 2 [M = 73.88, SD = 13.58; $t(24) = 5.97$, $p < 0.001$] for the ED participants. Functional participants demonstrated a decrease in HR between pre-film 2 (M = 83.71, SD = 16.22) and film 2 [M = 79.09, SD = 16.02; $t(33) = 3.75$, $p < 0.001$].

Hypotheses

Hypothesis 1 – Increases in hf-HRV during sexual arousal

A 2 (group) x 2 [pre-film, film (3 minute means)] repeated measures ANOVA was used to examine changes in hf-HRV. There was no interaction between time and functioning group (Hypothesis 1b) [$F(1,52) = 1.99$, $p = 0.16$]. However, hf-HRV increased from pre-film (M = 4.89, SD = 1.29) to film (Hypothesis 1a) [M = 5.26, SD = 1.26; $F(1,52) = 15.67$, $p < 0.001$]. Although the interaction was not significant (p = 0.16), exploratory analyses were performed because the study may be underpowered to detect an interaction and must be interpreted with caution. Within group analyses also revealed an increase in hf-HRV from pre-film (M = 4.75, SD =1.49) to film [M = 5.33, SD = 1.12; $F(1,32) = 15.85$, $p < 0.001$] for the functional participants and a trend for an increase in hf-HRV from pre-film (M = 5.11, SD = 1.38) to film [M = 5.33, SD = 1.49; $F(1,20) = 3.64$, $p = 0.07$] for the ED participants (Figure 7).

Figure 8 depicts changes in hf-HRV by minute for functional and ED participants. Paired samples t-tests revealed an increase in hf-HRV between pre-film 2 (M = 5.10, SD = 1.38) and film 2 [M = 5.43, SD = 1.50; $t(20) = 2.87$, $p < 0.01$] and a decrease in hf-HRV between film 3 (M = 5.39, SD = 1.46) and film 4 [M = 5.26, SD = 1.49; $t(21) = 2.21$, $p < 0.05$] for the ED participants. Functional participants demonstrated an increase

in hf-HRV between pre-film 2 ($M = 4.77$, $SD = 1.31$) and film 2 [$M = 5.35$, $SD = 1.14$; $t(32) = 4.75$, $p < 0.001$], a decrease between film 2 and 3 [$M = 5.24$, $SD = 1.11$; $t(32) = 2.60$, $p < 0.05$], and a decrease between film 3 and 4 [$M = 5.05$, $SD = 1.16$; $t(32) = 3.65$, $p < 0.001$]. These findings indicate that during the erotic film, functional participants had significant attenuation of hf-HRV that occurred later in the ED participants. However, when examining a 2 (group) by 3 (film hf-HRV), no significant interaction was found [$F(2) = 0.97$, $p = 0.38$].

Although age was significantly correlated with HRV, controlling for age in these analyses did not improve results.

Hypothesis 1 – Responders vs. Non-responders

Responders were defined as participants who had a maximum of 4 mm of tumescence change and averaged at least 1 mm tumescence change over the entire experimental session. Participants that did not meet these criteria were defined as non-responders. A series of 2 (group) x 2 (responder vs. non-responder) univariate ANOVAs were used to examine changes in baseline, change, average, and peak hf-HRV. Overall, there were no significant main effects or interactions for all analyses. Therefore, responders and non-responders had similar changes in hf-HRV, which suggest that hf-HRV may not be a sensitive measure for tumescence studies and may not be a good physiological correlate of changes in tumescence.

Hypothesis 2 – The association between anxiety and HRV

It was expected that anxiety would affect HRV (Hypothesis 2a and 2b). Therefore, a correlation analysis was run between SAQ#2 and, hf-HRV pre-film and film

averages. SAQ#2 was not correlated with hf-HRV pre-film (r = -0.14) and film (r = -0.21) averages for all participants and for both sexual functioning groups (Table 4).

A hierarchical linear regression with three models was used to examine the association of sexual functioning status and anxiety on hf-HRV response (Table 5). Model 1 (difference score of hf-HRV) was not significant such that sexual functioning status accounted for 3.7% of the variance in hf-HRV response. Adding baseline hf-HRV into Model 2 (difference score of hf-HRV and baseline hf-HRV) accounted for significant amount of variance of hf-HRV [p = 0.047] and the overall model (Model 2) approached significance [p = 0.053]. Anxiety was added to Model 3 (difference score of hf-HRV, baseline hf-HRV, and SAQ#2) and was not predicative of hf-HRV. The overall model with all three predictors approached significance [p = 0.082] accounting for approximately 12.4% of the variance of hf-HRV, with baseline hf-HRV being the only significant predictor of film hf-HRV.

To examine the effects of anxiety on hf-HRV for sexually functional and ED participants separately, sexual functioning status was removed as a predictor and the data set was split by sexual functioning status (Table 5). Baseline hf-HRV and anxiety were not predictors of hf-HRV for the ED participants, accounting for a total of 2% of the variance. However, for the functional participants, baseline hf-HRV was predicative [p = 0.012] and anxiety was not predicative of hf-HRV during the film. The overall model with baseline hf-HRV and anxiety for functional participants was significant [p = 0.033] accounting for 20.3% of the variance of hf-HRV.

Hypothesis 3 - The association of HRV and anxiety with tumescence

It was expected that anxiety would affect tumescence (Hypothesis 3a and 3b) through alterations in hf-HRV (Hypothesis 2). SAQ#2 was not correlated with tumescence average or maximum for all (r = -0.11), functional (r = -0.01), and ED participants (r = -0.28). However, the correlation between maximum tumescence and SAQ#2 was significant for ED participants (r = -.47), but not for functional (r = -0.22) and all participants (r = -0.02) (Table 4).

A hierarchical linear regression with three models was used to examine the association of sexual functioning status and anxiety on tumescence response. For the entire sample, changes in hf-HRV did not predict changes in average (Table 6) or maximum (Table 7) tumescence. Adding sexual functioning status and anxiety also did not predict changes in tumescence.

To examine the effects of hf-HRV and anxiety on tumescence for functional and ED participants separately, sexual functioning status was removed as a predictor and the data set was split by functioning group. hf-HRV was placed into the model first followed by anxiety as predictors for average and maximum tumescence. For average tumescence, there were no significant predictors in either group. There were no significant predictors of maximum tumescence for the functional participants. However, for the ED participants anxiety was a significant predictor of maximum tumescence [p = 0.029]. There was a trend for the overall model which included both hf-HRV and anxiety as predictors of maximum tumescence [p = 0.080] accounting for approximately 25% of the variance with anxiety accounting for 24% of the variance in tumescence.

DISCUSSION

The primary purpose of this study was to determine whether men with ED have reduced PNS activation that may reduce blood flow to the penis and restrict tumescence increases during sexual arousal. Secondly, this study investigated the influence of anxiety on the ANS and tumescence. Anxiety was predicted to limit PNS activation, which in turn would limit tumescence increases. If anxiety attenuated HRV, and HRV predicted tumescence, then a case could be made for the theory that anxiety influences the ANS to limit PNS activation and blood flow to the penis, causing and maintaining ED.

Hypothesis 1a was supported by the observation that hf-HRV increased during the film, indicating that PNS activation (hf-HRV) occurs during sexual arousal. PNS activation is further evidenced by the reduction in HR for both groups during the erotic film. Additionally, lf/hf-HRV decreased during the film, but lf-HRV did not decrease during the film, demonstrating that changes in the lf/hf-HRV were predominantly influenced by increasing PNS activity. These findings are in agreement with the current anatomical, cardiovascular, and hormonal literature (Brindley, 1992; Giuliano & Rampin, 2004; Guay et al., 2003; Miller, 2001; Monga & Hellstrom, 1999) such that PNS activation is necessary to produce erections in both sexually functional and men with ED. However, increases in hf-HRV were not related to increases in tumescence (Hypothesis 3a), which is potentially contradictory to the physiological model of erections. PNS activation did occur in both sexually functional and men with ED, but these changes did not differentially affect tumescence increases.

Contrary to Hypothesis 1b, men with ED did not have different ANS responses than controls. PNS increases during sexual arousal were not attenuated among men with ED as compared to sexually functional men. For this study, it does not appear that there was attenuated PNS activation in men with ED that could limit tumescence increases and affect their ED. It could have there was a ceiling effect because that men with ED had higher pre-film hf-HRV than functional men. The results of this study support the work of others (Daffertshofer et al., 1994; Ferini-Strambi et al., 1992) demonstrating that men with psychogenic or psychiatric ED do not have ANS activation during sexual arousal that differs from men without ED. The results that men with psychogenic ED have a ANS response similar to men with psychogenic ED suggests that there must be another mechanism that accounts for reduced abilities to achieve and maintain erections in this population.

Anxiety was predicted to moderate changes in the ANS during sexual arousal (Hypothesis 2a). However, anxiety was not correlated to HRV during sexual arousal. Although the overall model approached significance, the only significant predictor of hf-HRV was baseline hf-HRV, which was primarily accounted for by the sexually functional men. The finding that anxiety did not moderate changes in HRV is contradictory to previous research. HRV has been shown to decrease in individuals with panic anxiety (Friedman & Thayer, 1998) and dental anxiety (Johnsen, Thayer, Laberg, Wormnes, Raadal, Skaret, Kvale, & Berg, 2003). However, these observations were based on individuals with extreme forms of anxiety, which did not occur in the present study.

Because of the inclusion/exclusion criteria participants with high trait anxiety may have been excluded, as demonstrated by the low BAI scores. Individuals with low levels of trait anxiety do not have HRV responses that differ from individuals without such anxiety. In physically fit men and women, trait anxiety is not associated with reductions in HRV (Dishman, Nakamura, Garcia, Thompson, Dunn, & Blair, 2000). In an adult sample, anxiety also is not related to reductions in HRV, but results in decreases in the baroreceptor reflex, (Virtanen, Jula, Salminen, Voipio-Pulkki, Helenius, Kuusela, & Airaksinen, 2003). Therefore, anxiety may only produce reductions in HRV for individuals who have extreme forms of trait anxiety but state anxiety does not seem to be associated with reductions in HRV. The finding that anxiety did not moderate changes in hf-HRV could have been influenced by the inclusion of individuals with low trait anxiety.

Given that this study was part of a larger study, it allowed comparisons of men who were asked to wear Holter monitors ($N = 74$) to those who were not asked to wear Holter monitors ($N = 69$). An interesting additional finding in support of the Sbrocco-Barlow model (1996) is that sexually functional men ($N = 19$) who were attached Holter monitors demonstrated less average [$M = 8.10$, $SD = 7.19$ vs. $M = 13.14$, $SD = 9.14$; $t(56) = 2.29$, $p = 0.03$] and maximum [$M = 13.90$, $SD = 10.56$ vs. $M = 20.05$, $SD = 12.15$; $t(56) = 1.98$, $p = 0.05$] tumescence changes as compared sexually functional men ($N = 39$) not attached to Holter monitors. Men with ED ($N = 33$) who were attached Holter monitors demonstrated no differences in average [$M = 7.80$, $SD = 7.89$ vs. $M = 4.96$, $SD = 6.54$; $t(68) = 1.35$, $p = 0.18$] and maximum [$M = 13.24$, $SD = 11.09$ vs. $M = 9.30$, $SD = 9.60$; $t(68) = 1.60$, $p = 0.12$] tumescence changes as compared men with ED ($N = 37$) not attached to Holter monitors. Sexually functional men could have been distracted away

from erotic cues because of the attached electrodes. Sexually ED men potentially entered the study distracted from sexual cues, which is why the Holter monitors had no effect on their tumescence changes.

Limitations and Future Directions

The results of this study must be considered in light of some limitations. The content of the sexual stimuli may not have appealed to some men such that they may not have been fully sexually aroused. Without full sexual arousal, erections of some men may have been limited, which is supported by the evidence that some men did not produce erections to the film. In order for men to become maximally aroused, it may be important for them to choose the types of erotica they find sexually arousing rather than a standardized erotic segment. Additionally, similar to Brody and colleagues (2000, 2003), HRV could be examined during "real-world" sexual arousal, activity, and dysfunction by having men keep journals of their sexual encounters. By measuring HRV during such encounters, men are choosing the sexual stimuli that arouse them and can choose whether or not to engage in sexual activity. Although these types of studies introduce many potential confounding factors such as recall bias or inaccurate recording of sexual activity and length of activity, the external validity may prove to be more clinically relevant than laboratory based studies.

Other limitations included the film length and assessment period. Some men may have needed a longer erotic segment to become maximally sexually aroused and to produce erections. The choice for this segment length was chosen because previous researchers used similar paradigms (Abrahamson, Barlow, & Abrahamson, 1989). Additionally, HRV was assessed prior to the film segment, but tumescence measurement

did not begin until the film started with the assumption that the men would not produce erections before the film. Therefore, obtaining pre-film correlation or regression data on HRV and tumescence could not be performed. Future research could rectify these issues by potentially having a longer film segment and by measuring both HRV and tumescence continuously throughout the entire experimental session, including baseline and post-film.

Because of the relative small sample size, there was not enough power to see some effects, especially for Hypothesis 3. For Hypothesis 1a, there was a large effect size (partial $\eta^2 = 0.23$), but hypothesis 1b revealed a small effect size (partial $\eta^2 = 0.04$). Additionally, artifacts in the HRV data and missing data caused many men to be excluded from data analyses. Oversampling of participants should be performed in future research to ensure adequate sample size to achieve good power and effect sizes. Additionally, artifacts from HRV and tumescence recordings could be limited by standardization of procedures and ensuring the physiological equipment is functioning properly, calibrated, and placed on participants correctly.

There may be serious limitations with using HRV as a correlational measure in tumescence studies. In this study, there was no correlation between HRV and tumescence changes. Adding further support to this evidence, it was found that there were no differences in average, baseline, or changes in HRV between tumescence responders and non-responders. The lack of significance may be influenced by the use of a difference score (hf-HRV film minus hf-HRV pre-film) to obtain a single hf-HRV number. Loss of reliability and an increased chance of regression to the mean are

byproducts of difference scores that can negatively influence significance. Therefore, HRV as a measure of ANS changes in relation to tumescence changes may be inaccurate.

Sbrocco and Barlow's (1996) and Barlow's (1986) models of sexual dysfunction both assume that cognitive distracters are the largest factor in determining psychogenic sexual dysfunction in men. Despite measuring trait anxiety, state anxiety, attention to film, and attention to body, there may have been other cognitive distracters, including life stressors, dislike of the erotica, or the laboratory setting, that attenuated tumescence increases of some participants. One of these unmeasured factors, Holter versus absence of Holter equipment, seemed to distract sexually functional participants from erotic cues, consequently decreasing average and maximum tumescence. Such factors should either be studied in future research or controlled for by statistically.

The measures of anxiety that were used may have been limited in their predictability of changes in hf-HRV. The BAI scores were of limited use because the majority of participants scored low [94% $\leq$ score of 10] on trait anxiety. This result may partially be from the extensive screening criteria that excluded individuals with any signs of psychopathology. The SAQ#2 was a single post-film question that was created for this study and has not been shown to be reliable or valid. Better measures of anxiety may be useful to detect associations between HRV and anxiety. Some support for the validity of the presently used anxiety measures is found in the significant correlation between anxiety and lower tumescence.

Much of the research on male sexual dysfunction has focused on cognitive distracters, including this study, but has little research has been performed using imaging techniques to understand how central mechanisms of men with and without ED differ.

For instance, men are known to demonstrate increase activation in the thalamus and hypothalamus areas as compared to women (Karama, Lecours, Leroux, Bourgouin, Beaudoin, Joubert, & Beauregard, 2004). Additionally, the hypothalamus has been found to be part of the sexual arousal circuit in men along with the anterior cingulate, insula, amygdala, putamen, and secondary somatosensory cortices (Arnow, Desmond, Banner, Glover, Solomon, Polan, Lue, & Atlas, 2002; Ferretti, et al., 2005). A comparison of men with and without ED examining these brain circuits is necessary to comprehend whether cognitive distracters alter sexual arousal brain circuitry to decrease the ability of obtaining erections. For example, although their sample was healthy, Kim, et al. (2006) found limited activation of the thalamus and hypothalamus in middle-aged men. They suggested that these two brain structures may be responsible for decreasing physiological arousal as a man ages. Additionally, these brain areas may be underactivated in men with sexual dysfunctions, including ED.

This study was performed in a heteronormative manner meaning that all the participants were heterosexual men viewing heterosexual erotica for the purposes of inducing sexual arousal and tumescence. It cannot be assumed that men of other sexual orientations report ED in the same manner as heterosexual men. Additionally, the causes and maintenance of ED in men of other sexual orientations may be different than those of heterosexual men (Sandfort & de Keizer, 2001). In order to begin to address these topics, ED in non-heterosexual men may need to be examined first before physiological studies are considered. The majority of research exploring sexual dysfunctions in homo/bisexual men with HIV has been in the area of sexual dysfunction resulting from antiretroviral treatments and low T-cell count (Cove & Petrak, 2004; Lamba, Goldmeier, Mackie, &

Scullard, 2004). Additionally, homosexual HIV positive men also have been shown to report high rates of erectile difficulties as compared to homosexual HIV negative men (Bancroft, Carnes, & Janssen, 2005). However, many researchers have assumed that ED in non-heterosexual men is similar to ED in heterosexual men, when there is little evidence to support this assumption.

The results of this study also cannot be generalized to females. Currently, there is little literature published concerning the role of the ANS during sexual arousal or dysfunction in women. Yeragani, Pohl, and Balon (2004) found that orgasm in women and men is associated with an increase in sympathetic tone suggesting relative SNS activation, PNS deactivation, or a combination thereof. Additionally, Brody and colleagues (2000, 2003) demonstrated that HRV increases in women and men as the frequency of vaginal intercourse increases. However, these studies did not specifically examine the role of the ANS in women or women with sexual dysfunctions. Further studies of female sexual dysfunctions are needed examining HRV in a broad range of ethnicities, ages, and sexual orientations.

Summary

This study documented PNS activity increases during erotic film exposure in both sexually functional men and men with psychogenic ED, lending support to the notion that PNS activation occurs during sexual arousal and is necessary for producing erections. However, there were no differences between men with and without ED in the magnitude of PNS responses to the erotic film clip, and PNS activity did not predict changes in tumescence. This study adds to the literature that men with psychogenic ED do not display reduced PNS. Anxiety also did not moderate the relationship between exposure

to sexual stimuli and changes in hf-HRV stemming from low trait anxiety. Therefore, it is concluded that PNS activation increases during an erotic film clip for sexually functional and ED men, men with psychogenic ED have ANS activation similar to sexually functional men during erotic stimuli, state anxiety does not moderate changes in PNS activation, and PNS activation does not predict changes in tumescence.

REFERENCES

Abrahamson, D. J., Barlow, D. H., & Abrahamson, L. S. (1989). Differential effects of performance demand and distraction on sexually functional and dysfunctional males. *J Abnorm Psychol, 98*(3), 241-247.

American Psychological Association. (2000). Diagnostic and statistical manual of mental disorders (4th Ed. text revision). Washington, D.C.: Author.

Agelink, M. W., Boz, C., Ullrich, H., & Andrich, J. (2002). Relationship between major depression and heart rate variability. Clinical consequences and implications for antidepressive treatment. *Psychiatry Res, 113*(1-2), 139-149.

Appel, M. L., Berger, R. D., Saul, J. P., Smith, J. M., & Cohen, R. J. (1989). Beat to beat variability in cardiovascular variables: Noise or music? *J Am Coll Cardiol, 14*(5), 1139-1148.

Araujo, A. B., Mohr, B. A., & McKinlay, J. B. (2004). Changes in sexual function in middle-aged and older men: Longitudinal data from the Massachusetts male aging study. *J Am Geriatr Soc, 52*(9), 1502-1509.

Arnow, B. A., Desmond, J. E., Banner, L. L., Glover, G. H., Solomon, A., Polan, M. L., et al. (2002). Brain activation and sexual arousal in healthy, heterosexual males. *Brain, 125*(Pt 5), 1014-1023.

Barlow, D. H. (1986). Causes of sexual dysfunction: The role of anxiety and cognitive interference. *J Consult Clin Psychol, 54*(2), 140-148.

Beck, A. T., & Steer, R. A. (1987). BDI, Beck Depression Inventory: Manual. San Antonio, TX: Psychological Corporation.

Beck, A. T., Epstein, N., Brown, G., & Steer, R. A. (1988). An inventory for measuring clinical anxiety: Psychometric properties. *Journal of Consulting and Clinical Psychology, 56,* 893-897.

Beck, A .T., Steer, R. A., & Brown, G. K. (1996). Beck Depression Inventory (2nd ed. Manual). San Antonio, TX: Psychological Corporation.

Beck, A. T., & Steer, R. A. (1990). The Beck Anxiety Inventory. Austin, TX: The Psychological Corporation.

Beck, A. T., & Steer, R. A. (1984). Internal consistencies of the original and revised beck depression inventory. *J Clin Psychol., 40*(6), 1365-1367.

Blumentals, W. A., Gomez-Caminero, A., Joo, S., & Vannappagari, V. (2003). Is erectile dysfunction predictive of peripheral vascular disease? *Aging Male, 6*(4), 217-221.

Brindley, G. S. (1992). Neurophysiology. In R. S. S. Kirby, G. D. Webster, C. Carson, (Eds). *Impotence: Diagnosis and Management of Male Erectile Dysfunction.* St. Louis: Elsevier.

Brody, S., & Preut, R. (2003). Vaginal intercourse frequency and heart rate variability. *J Sex Marital Ther, 29*(5), 371-380.

Brody, S., Veit, R., & Rau, H. (2000). A preliminary report relating frequency of vaginal intercourse to heart rate variability, valsalva ratio, blood pressure, and cohabitation status. *Biol Psychol, 52*(3), 251-257.

Cohen, H., Matar, M. A., Kaplan, Z., & Kotler, M. (1999). Power spectral analysis of heart rate variability in psychiatry. *Psychother Psychosom, 68*(2), 59-66.

Cove, J., & Petrak, J. (2004). Factors associated with sexual problems in hiv-positive gay men. *Int J STD AIDS, 15*(11), 732-736.

Craelius, W., Akay, M., & Tangella, M. (1992). Heart rate variability as an index of autonomic imbalance in patients with recent myocardial infarction. *Med Biol Eng Comput, 30*(4), 385-388.

Daffertshofer, M., Linden, D., Syren, M., Junemann, K. P., & Berlit, P. (1994). Assessment of local sympathetic function in patients with erectile dysfunction. *Int J Impot Res, 6*(4), 213-225.

Dean, J., Hackett, G. I., Gentile, V., Pirozzi-Farina, F., Rosen, R. C., Zhao, Y., et al. (2006). Psychosocial outcomes and drug attributes affecting treatment choice in men receiving sildenafil citrate and tadalafil for the treatment of erectile dysfunction: Results of a multicenter, randomized, open-label, crossover study. *J Sex Med, 3*(4), 650-661.

deGroat, W. C., & Booth, A. M. (1980). Physiology of male sexual function. *Ann Intern Med, 92*(2 Pt 2), 329-331.

Derby, C. A., Barbour, M. M., Hume, A. L., & McKinlay, J. B. (2001). Drug therapy and prevalence of erectile dysfunction in the Massachusetts male aging study cohort. *Pharmacotherapy, 21*(6), 676-683.

Dishman, R. K., Nakamura, Y., Garcia, M. E., Thompson, R. W., Dunn, A. L., & Blair, S. N. (2000). Heart rate variability, trait anxiety, and perceived stress among physically fit men and women. *Int J Psychophysiol, 37*(2), 121-133.

Dusing, R. (2005). Sexual dysfunction in male patients with hypertension: Influence of antihypertensive drugs. *Drugs, 65*(6), 773-786.

Ellenberg, M. (1980). Sexual function in diabetic patients. *Ann Intern Med, 92*(2 Pt 2), 331-333.

Esposito, K., Giugliano, F., Di Palo, C., Giugliano, G., Marfella, R., D'Andrea, F., et al. (2004). Effect of lifestyle changes on erectile dysfunction in obese men: A randomized controlled trial. *JAMA, 291*(24), 2978-2984.

Farre, J. M., Fora, F., & Lasheras, M. G. (2004). Specific aspects of erectile dysfunction in psychiatry. *Int J Impot Res, 16 Suppl 2*, S46-49.

Feldman, H. A., Goldstein, I., Hatzichristou, D. G., Krane, R. J., & McKinlay, J. B. (1994). Impotence and its medical and psychosocial correlates: Results of the Massachusetts male aging study. *J Urol, 151*(1), 54-61.

Ferini-Strambi, L., Zucconi, M., Rigatti, P., Grasso, M., Montorsi, F., & Smirne, S. (1992). Male impotence: A possible beta-adrenergic dysfunction in some patients. *Eur Urol, 21*(4), 332-334.

Ferini-Strambi, L., Montorsi, F., Zucconi, M., Oldani, A., Smirne, S., & Rigatti, P. (1996). Cardiac autonomic nervous activity in sleep-related painful erections. *Sleep, 19*(2), 136-138.

Ferretti, A., Caulo, M., Del Gratta, C., Di Matteo, R., Merla, A., Montorsi, F., et al. (2005). Dynamics of male sexual arousal: Distinct components of brain activation revealed by fmri. *Neuroimage, 26*(4), 1086-1096.

First, M. B., Spitzer, R. L., Gibbon, M., and Williams, J. B.W. (1994) *Structured Clinical Interview for DSM-IV Personality Disorders, (SCID)*. Washington, D.C.: American Psychiatric Press, Inc.

Flynn, A. C., Jelinek, H. F., & Smith, M. (2005). Heart rate variability analysis: A useful assessment tool for diabetes associated cardiac dysfunction in rural and remote areas. *Aust J Rural Health, 13*(2), 77-82.

Freeman, S. (2004). *Biological Science* 2nd Ed., Prentice Hall, Inc.

Friedman, B. H., & Thayer, J. F. (1998). Anxiety and autonomic flexibility: A cardiovascular approach. *Biol Psychol, 49*(3), 303-323.

Friedman, B. H., & Thayer, J. F. (1998). Autonomic balance revisited: Panic anxiety and heart rate variability. *J Psychosom Res, 44*(1), 133-151.

Gillis, M. M., Haaga, D. A. F., & Ford, G. T. (1995). Normative values for the beck anxiety inventory, fear questionnaire, penn state worry questionnaire, and social phobia and anxiety inventory. *Psychol Assess, 7*(4), 450-455.

Giuliano, F., & Rampin, O. (2004). Neural control of erection. *Physiol Behav, 83*(2), 189-201.

Goldstein, D. S. (2004). *The autonomic nervous system*. (pp. 24-38). New York: Marcel Dekker, Inc.

Guay, A. T., Spark, R. F., Bansal, S., Cunningham, G. R., Goodman, N. F., Nankin, H. R., et al. (2003). American association of clinical endocrinologists medical guidelines for clinical practice for the evaluation and treatment of male sexual dysfunction: A couple's problem--2003 update. *Endocr Pract, 9*(1), 77-95.

Hart, B. L. (1974). Physiology of sexual function. *Vet Clin North Am, 4*(3), 557-571.

Hecht, M. J., Neundorfer, B., Kiesewetter, F., & Hilz, M. J. (2001). Neuropathy is a major contributing factor to diabetic erectile dysfunction. *Neurol Res, 23*(6), 651-654.

Hedon, F. (2003). Anxiety and erectile dysfunction: A global approach to ED enhances results and quality of life. *Int J Impot Res, 15 Suppl 2*, S16-19.

Heiman, J. R. (2002). Sexual dysfunction: Overview of prevalence, etiological factors, and treatments. *J Sex Res, 39*(1), 73-78.

Hoehn-Saric, R., & McLeod, D. R. (1988). The peripheral sympathetic nervous system. Its role in normal and pathologic anxiety. *Psychiatr Clin North Am, 11*(2), 375-386.

Jiang, W., Hathaway, W. R., McNulty, S., Larsen, R. L., Hansley, K. L., Zhang, Y., et al. (1997). Ability of heart rate variability to predict prognosis in patients with advanced congestive heart failure. *Am J Cardiol, 80*(6), 808-811.

Johnsen, B. H., Thayer, J. F., Laberg, J. C., Wormnes, B., Raadal, M., Skaret, E., et al. (2003). Attentional and physiological characteristics of patients with dental anxiety. *J Anxiety Disord, 17*(1), 75-87.

Johnson, S. D., Phelps, D. L., & Cottler, L. B. (2004). The association of sexual dysfunction and substance use among a community epidemiological sample. *Arch Sex Behav, 33*(1), 55-63.

Kakiailatu, F. A. (2000). The role of nitric oxide in the mechanism of penile erection. *Clin Hemorheol Microcirc, 23*(2-4), 283-286.

Karama, S., Lecours, A. R., Leroux, J. M., Bourgouin, P., Beaudoin, G., Joubert, S., et al. (2002). Areas of brain activation in males and females during viewing of erotic film excerpts. *Hum Brain Mapp, 16*(1), 1-13.

Kautzner, J., & Camm, A. J. (1997). Clinical relevance of heart rate variability. *Clin Cardiol, 20*(2), 162-168.

Keene, L. C., & Davies, P. H. (1999). Drug-related erectile dysfunction. *Adverse Drug React Toxicol Rev, 18*(1), 5-24.

Kim, S. W., Sohn, D. W., Cho, Y. H., Yang, W. S., Lee, K. U., Juh, R., et al. (2006). Brain activation by visual erotic stimuli in healthy middle aged males. *Int J Impot Res, 18*(5), 452-457.

Kop, W. J., Verdino, R. J., Gottdiener, J. S., O'Leary, S. T., Bairey Merz, C. N., & Krantz, D. S. (2001). Changes in heart rate and heart rate variability before ambulatory ischemic events(1). *J Am Coll Cardiol, 38*(3), 742-749.

Kristal-Boneh, E., Raifel, M., Froom, P., & Ribak, J. (1995). Heart rate variability in health and disease. *Scand J Work Environ Health, 21*(2), 85-95.

Lamba, H., Goldmeier, D., Mackie, N. E., & Scullard, G. (2004). Antiretroviral therapy is associated with sexual dysfunction and with increased serum oestradiol levels in men. *Int J STD AIDS, 15*(4), 234-237.

Laumann, E. O., Paik, A., & Rosen, R. C. (1999). Sexual dysfunction in the united states: Prevalence and predictors. JAMA*, 281*(6), 537-544.

Lavie, P., Shlitner, A., & Nave, R. (1999). Cardiac autonomic function during sleep in psychogenic and organic erectile dysfunction. *J Sleep Res, 8*(2), 135-142.

Lombardi, F., Malliani, A., Pagani, M., & Cerutti, S. (1996). Heart rate variability and its sympatho-vagal modulation. *Cardiovasc Res, 32*(2), 208-216.

Malliani, A., Lombardi, F., & Pagani, M. (1994). Power spectrum analysis of heart rate variability: A tool to explore neural regulatory mechanisms. *Br Heart J, 71*(1), 1-2.

Masters, W. H., & Johnson, V. E. (1966). Human sexual response. London: J. & A. Churchill Ltd.

Masters, W. H., Johnson, V. E., & Kolodny, R. C. (1982). Masters and Johnson on sex and human loving. New York: Little, Brown, and Company.

McCraty, R., Atkinson, M., Tiller, W. A., Rein, G., & Watkins, A. D. (1995). The effects of emotions on short-term power spectrum analysis of heart rate variability. *Am J Cardiol, 76*(14), 1089-1093.

Miller, M. (2001). Physical aspects of the male sexual response. In R. Skrine & H. Montford (Eds.), *Psychosexual Medicine*. (pp. 14-24). New York: Arnold.

Monga, M. & Hellstrom, W. J. G. (1999). Male Sexual Dysfunction (Chapter 15). In C. Wang (Ed.), *Male Reproductive Function*, (pp. 280-283). Boston: Kluwer.

Moore, T. M., Strauss, J. L., Herman, S., & Donatucci, C. F. (2003). Erectile dysfunction in early, middle, and late adulthood: Symptom patterns and psychosocial correlates. *J Sex Marital Ther, 29*(5), 381-399.

Motofei, I. G., & Rowland, D. L. (2005). The physiological basis of human sexual arousal: Neuroendocrine sexual asymmetry. Int J Androl, 28(2), 78-87.

NIH consensus conference. Impotence. *NIH consensus development panel on impotence.* (1993). *JAMA, 270*(1), 83-90.

Norton, G. R., & Jehu, D. (1984). The role of anxiety in sexual dysfunctions: A review. *Arch Sex Behav, 13*(2), 165-183.

Nusbaum, M. R. (2002). Erectile dysfunction: Prevalence, etiology, and major risk factors. *J Am Osteopath Assoc, 102*(12 Suppl 4), S1-6.

Pagani, M., Lombardi, F., Guzzetti, S., Rimoldi, O., Furlan, R., Pizzinelli, P., et al. (1986). Power spectral analysis of heart rate and arterial pressure variabilities as a

marker of sympatho-vagal interaction in man and conscious dog. *Circ Res, 59*(2), 178-193.

Pagani, M. (2000). Hypertension, stress and erectile dysfunction: Potential insights from the analysis of heart rate variability. *Curr Med Res Opin, 16 Suppl 1*, s3-8.

Pieper, S. J., & Hammill, S. C. (1995). Heart rate variability: Technique and investigational applications in cardiovascular medicine. *Mayo Clin Proc, 70*(10), 955-964.

Pomeranz, B., Macaulay, R. J., Caudill, M. A., Kutz, I., Adam, D., Gordon, D., et al. (1985). Assessment of autonomic function in humans by heart rate spectral analysis. *Am J Physiol, 248*(1 Pt 2), H151-153.

Reardon, M., & Malik, M. (1996). Changes in heart rate variability with age. *Pacing Clin Electrophysiol, 19*(11 Pt 2), 1863-1866.

Rosen, R., Janssen, E., Wiegel, M., Bancroft, J., Althof, S., Wincze, J., et al. (2006). Psychological and interpersonal correlates in men with erectile dysfunction and their partners: A pilot study of treatment outcome with sildenafil. *J Sex Marital Ther, 32*(3), 215-234.

Roose, S. P. (2003). Depression: Links with ischemic heart disease and erectile dysfunction. J *Clin Psychiatry, 64* Suppl 10, 26-30.

Rowland, D. L., Thornton, J. A., & Burnett, A. L. (2005). Recognizing the risk of erectile dysfunction in a urology clinic practice. *BJU Int, 95*(7), 1034-1038.

Russell, S. T., Khandheria, B. K., & Nehra, A. (2004). Erectile dysfunction and cardiovascular disease. *Mayo Clin Proc, 79*(6), 782-794.

Saigal, C. S. (2004). Obesity and erectile dysfunction: Common problems, common solution? *JAMA, 291*(24), 3011-3012.

Sandfort, T. G., & de Keizer, M. (2001). Sexual problems in gay men: An overview of empirical research. *Annu Rev Sex Res, 12*, 93-120.

Sbrocco, T., Weisberg, R., & Barlow, D. H. (1992). Sexual dysfunction inventory. The University at Albany: State University of New York; Albany, NY.

Sbrocco, T. & Barlow, D.H. (1996). Conceptualizing the cognitive component of sexual arousal: Implications for sexuality research and treatment. In P.M. Salkovskis (Ed.), *Frontiers of cognitive therapy*. (pp. 419-449). NY: Guilford Publications.

Shiri, R., Koskimaki, J., Hakama, M., Hakkinen, J., Huhtala, H., Tammela, T. L., et al. (2004). Effect of life-style factors on incidence of erectile dysfunction. *Int J Impot Res, 16*(5), 389-394.

Singh, J. P., Larson, M. G., Tsuji, H., Evans, J. C., O'Donnell, C. J., & Levy, D. (1998). Reduced heart rate variability and new-onset hypertension: Insights into pathogenesis of hypertension: The Framingham heart study. *Hypertension, 32*(2), 293-297.

Smith, A. D. (1988). Psychologic factors in the multidisciplinary evaluation and treatment of erectile dysfunction. *Urol Clin North Am, 15*(1), 41-51.

Stein, P. K., Bosner, M. S., Kleiger, R. E., & Conger, B. M. (1994). Heart rate variability: A measure of cardiac autonomic tone. *Am Heart J, 127*(5), 1376-1381.

Stein, P. K., & Kleiger, R. E. (1999). Insights from the study of heart rate variability. *Annu Rev Med, 50*, 249-261.

Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology. (1996). Heart rate variability. Standards of measurement, physiological interpretation, and clinical use.. *Eur Heart J, 17*(3), 354-381.

Thayer, J. F., Friedman, B. H., & Borkovec, T. D. (1996). Autonomic characteristics of generalized anxiety disorder and worry. *Biol Psychiatry, 39*(4), 255-266.

Uusitalo, A. L., Laitinen, T., Vaisanen, S. B., Lansimies, E., & Rauramaa, R. (2002). Effects of endurance training on heart rate and blood pressure variability. *Clin Physiol Funct Imaging, 22*(3), 173-179.

Uusitalo, A. L., Laitinen, T., Vaisanen, S. B., Lansimies, E., & Rauramaa, R. (2004). Physical training and heart rate and blood pressure variability: A 5-yr randomized trial. *Am J Physiol Heart Circ Physiol, 286*(5), H1821-1826.

Vanninen, E., Tuunainen, A., Kansanen, M., Uusitupa, M., & Lansimies, E. (1996). Cardiac sympathovagal balance during sleep apnea episodes. *Clin Physiol, 16*(3), 209-216.

Virtanen, R., Jula, A., Salminen, J. K., Voipio-Pulkki, L. M., Helenius, H., Kuusela, T., et al. (2003). Anxiety and hostility are associated with reduced baroreflex sensitivity and increased beat-to-beat blood pressure variability. *Psychosom Med, 65*(5), 751-756.

Wabrek, A. J., & Burchell, R. C. (1980). Male sexual dysfunction associated with coronary heart disease. *Arch Sex Behav, 9*(1), 69-75.

Wein, A. J., & Van Arsdalen, K. N. (1988). Drug-induced male sexual dysfunction. *Urol Clin North Am, 15*(1), 23-31.

Yataco, A. R., Fleisher, L. A., & Katzel, L. I. (1997). Heart rate variability and cardiovascular fitness in senior athletes. *Am J Cardiol, 80*(10), 1389-1391.

Yeragani, V. K., Pohl, R., & Balon, R. (2004). Complex demodulation of cardiac interbeat intervals: Increased cardiac sympathovagal interaction during human sexual activity. *Arch Sex Behav, 33*(1), 65-69.

Zuckerman, M. (1971). Physiological measures of sexual arousal in the human. *Psychol Bull, 75*(5), 297-329.

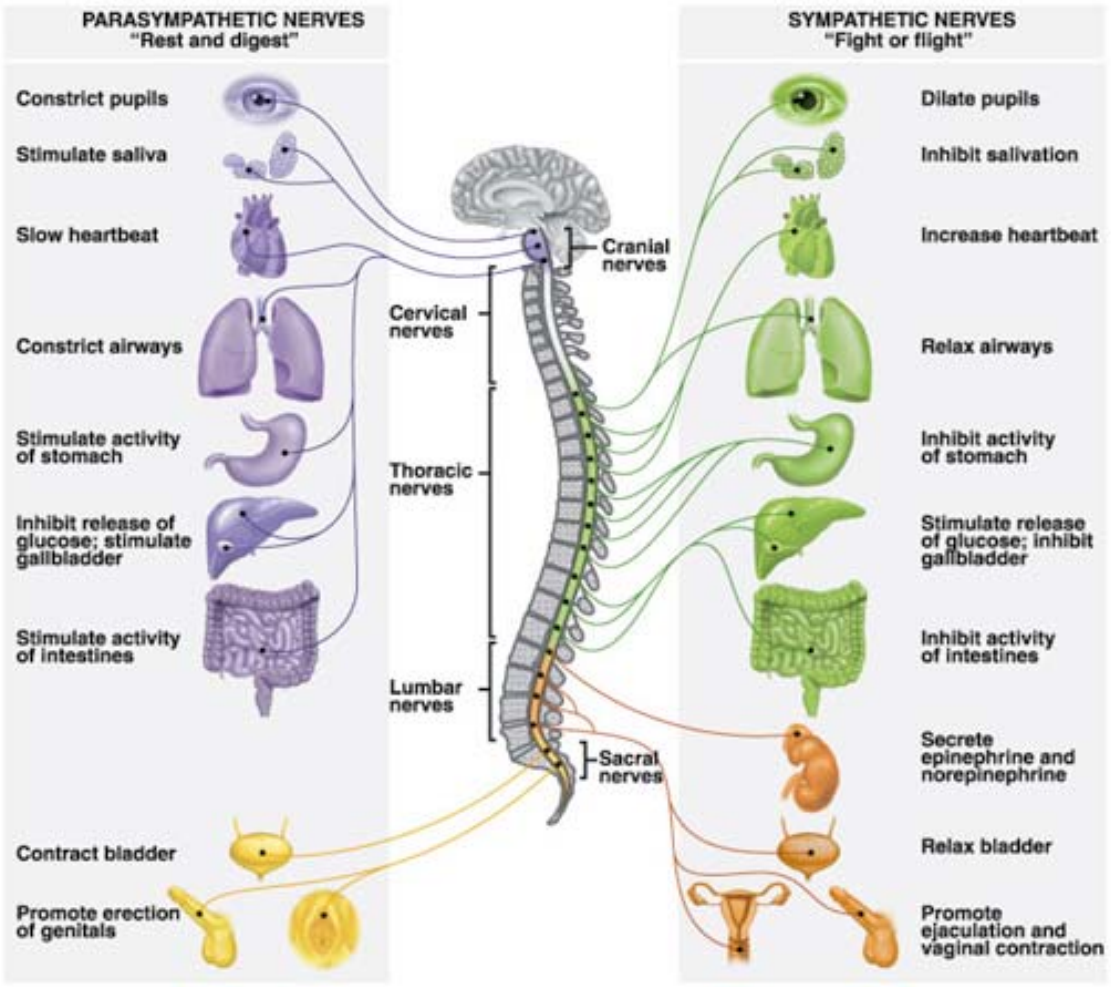


Figure 1. The autonomic nervous system (ANS) and its two parts: the parasympathetic nervous system (PNS) and sympathetic nervous system (SNS). The PNS controls erection capabilities from nerves in the sacral region of the spine, whereas the SNS controls detumescence and ejaculation from nerves in the lumbar region of the spine. In S. Freeman, *Biological Science*, 2nd Ed. Prentice Hall, Copyright, 2003 by Prentice Hall, Inc.

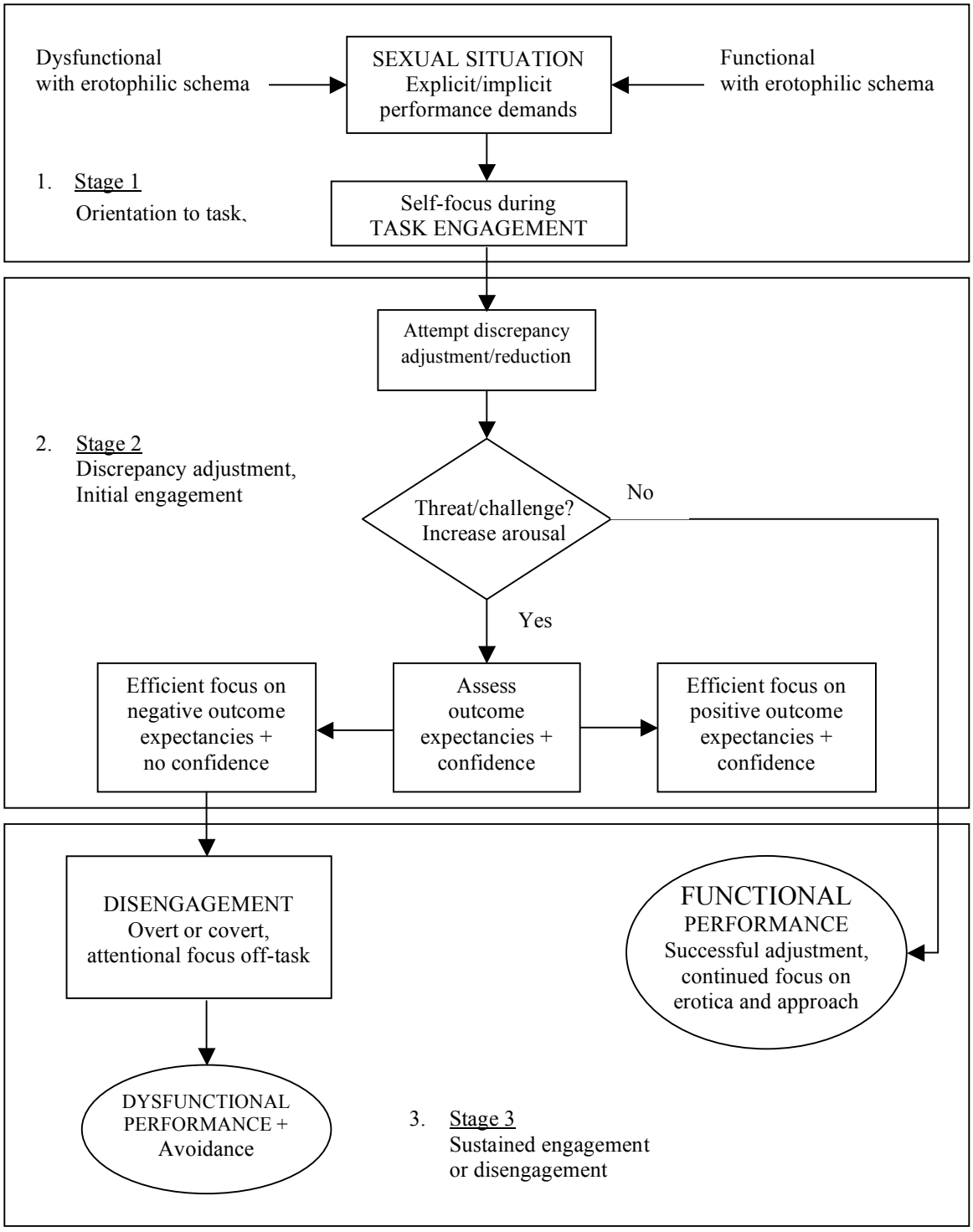


*Figure 2***.** A model of sexual dysfunction: implications for examining attentional processes. From "Conceptualizing the Cognitive Component of Sexual Arousal: Implications for Sexuality Research and Treatment," by T. Sbrocco, and D. H. Barlow, 1996, in P. Sulkouskis (Ed.), *Frontiers of Cognitive Therapy*, p. 440, Guilford. Copyright 1996 by Guilford Press.

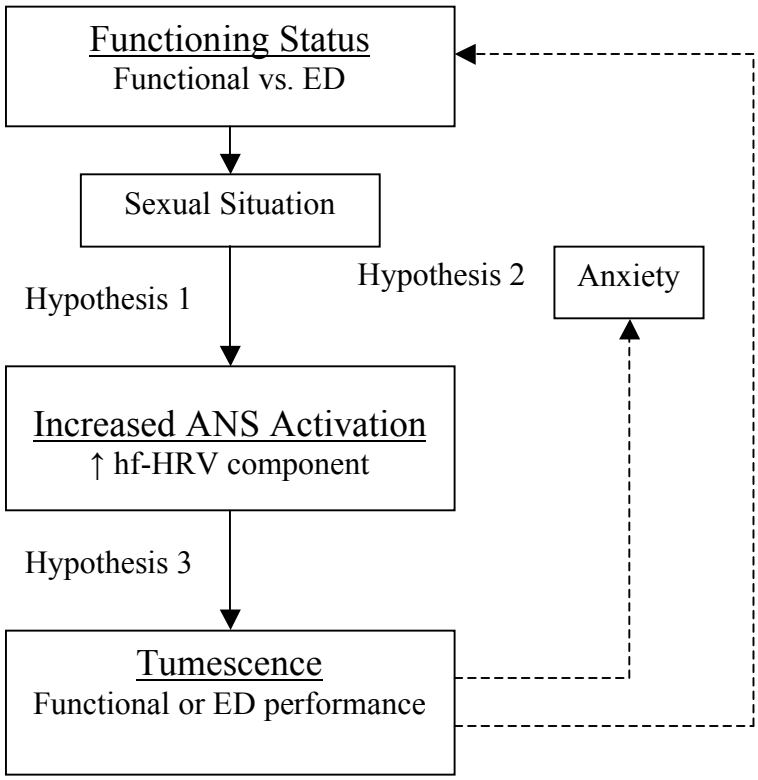
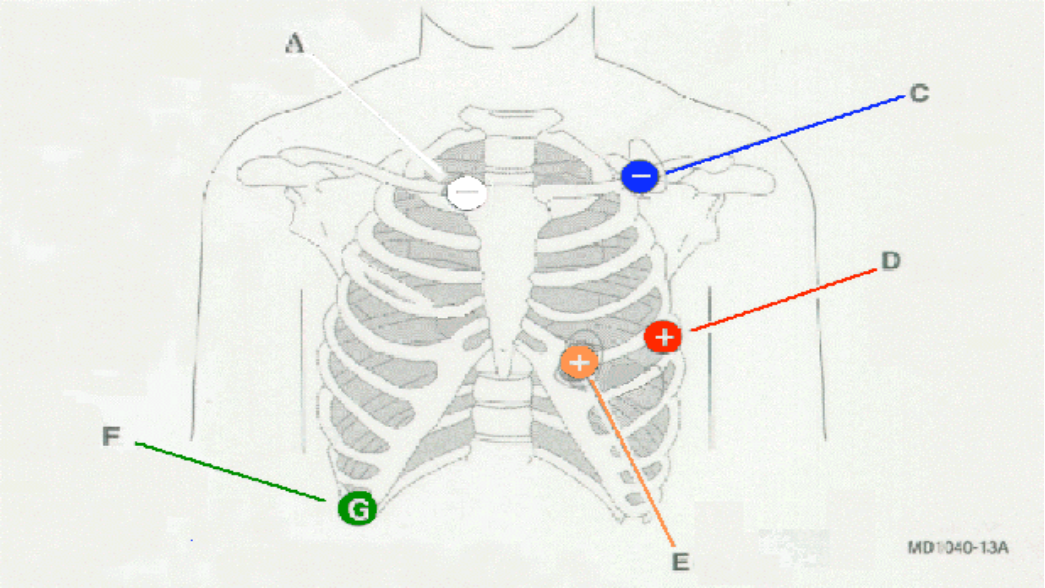


Figure 3. Hypothetical model of how sexual arousal impacts the autonomic nervous system and tumescence. Sexual arousal causes an increase in hf-HRV (increase in parasympathetic tone), which causes increase in tumescence. Anxiety potentially moderates the changes in hf-HRV induced by sexual arousal.



Item	Color	Channel	Lead	Location
A	White	CH 1 (--)	mV5 (–)	Right clavicle, just lateral to sternum
C	Blue	CH 3 (–)	aVF (–)	Left clavicle, at the mid-clavicular line
D	Red	CH 1 (+)	mV5 (+)	Fifth intercostal space at the left axillary line
E	Orange	CH 3 (+)	aVF (+)	Sixth rib, at the left mid-clavicular line
F	Green	----------	Ground	Lower right chest wall.

Figure 4. Holter electrode lead placement**.** Series 8500 Ambulatory Tape Recorder Operators Manual, Rev. C, Section 3 page 6. Copyright Marquette Medical Systems, Inc. 1996,1997.

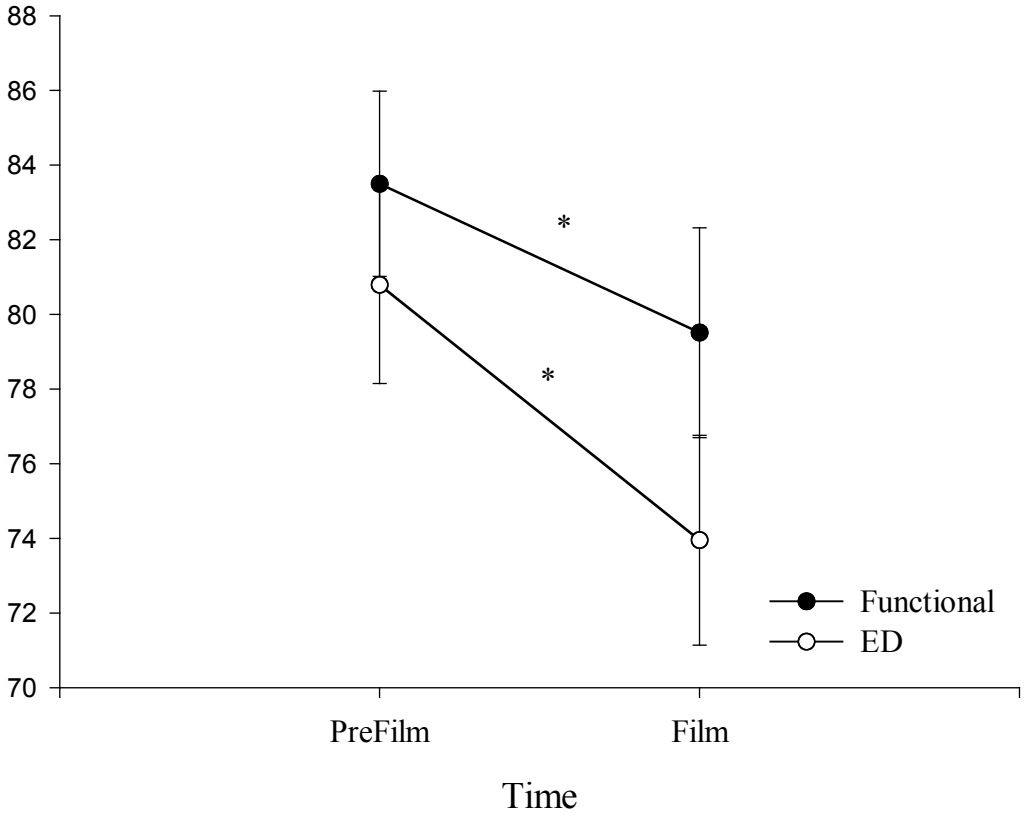


Figure 5. 3-minute means of HR before and after the erotic film for functional and ED participants.
* $p < 0.05$ significant decrease in HR from prefilm to film.

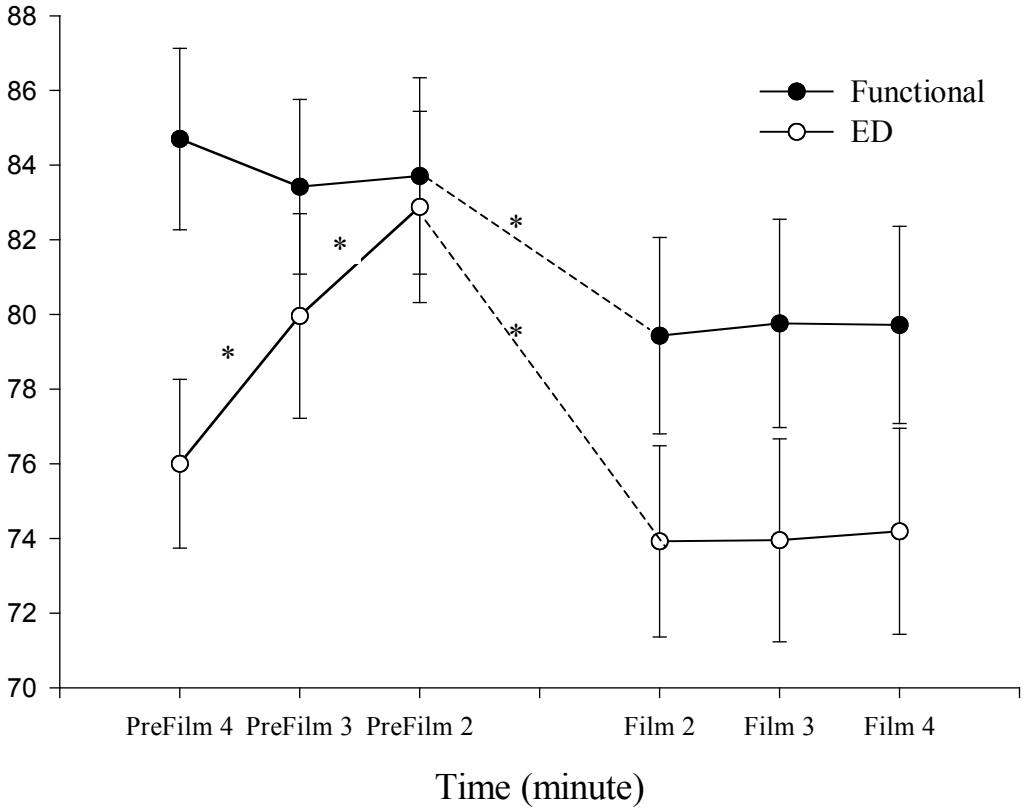


Figure 6. Minute HR before and after the erotic film for functional and ED participants. * $p < 0.05$ significant change in HR.

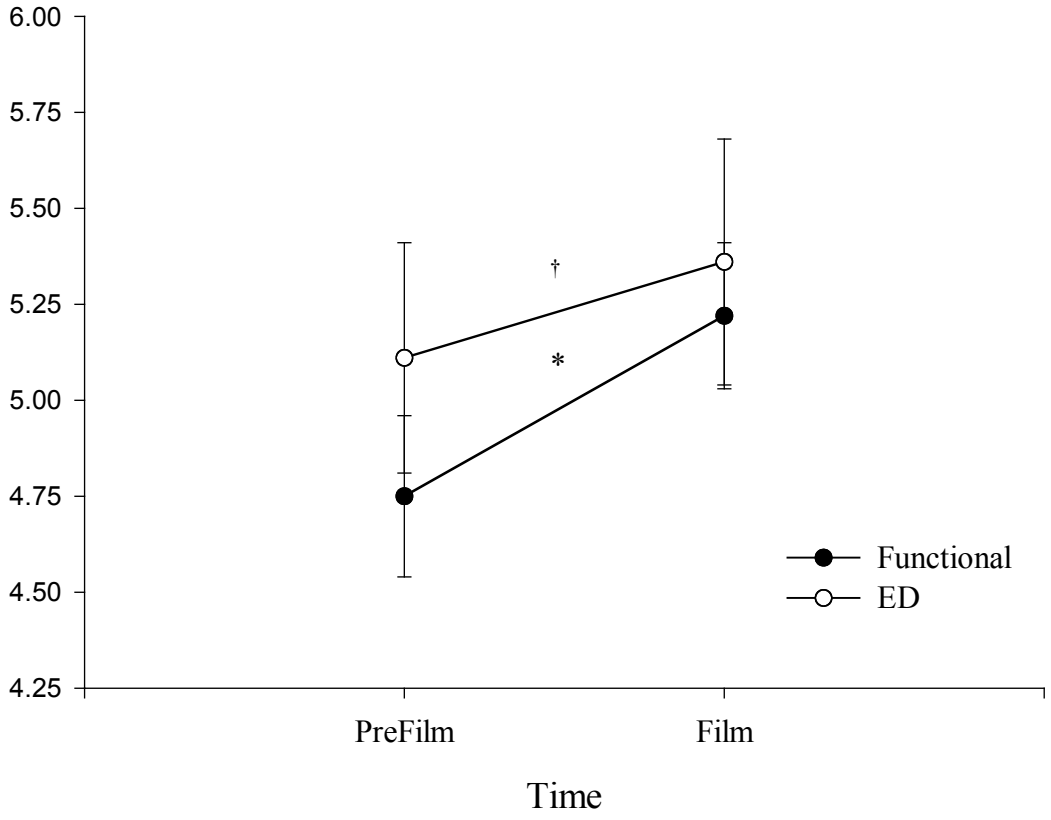


Figure 7. 3-minute means of hf-HRV before and after the erotic film for functional and ED participants.
† $p < 0.10$ trend for increase in hf-HRV from prefilm to film.
* $p < 0.05$ significant increase in hf-HRV from prefilm to film.

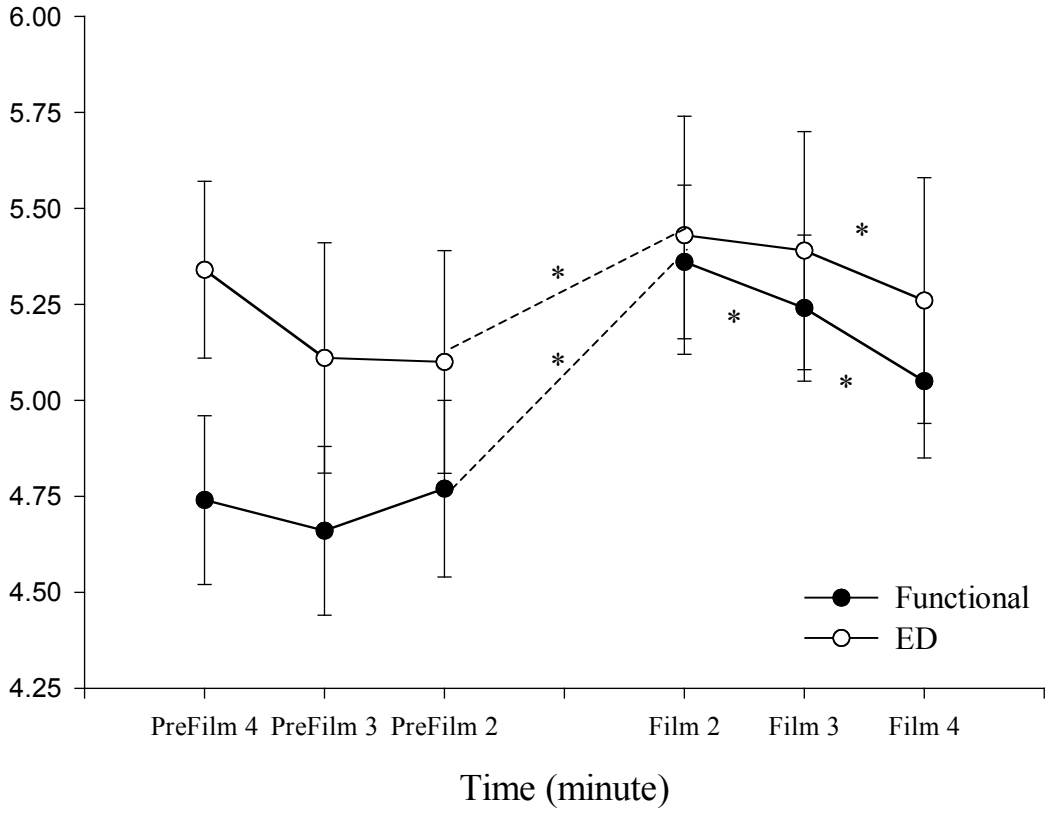


Figure 8. Minute hf-HRV before and after the erotic film for functional and ED participants.
* $p < 0.05$ significant change in hf-HRV.

APPENDIX A

Newspaper Advertisement for Recruitment of Subjects

Men Earn $40 in Laboratory Study of Factors Impacting Sexual Arousal

University study seeks healthy men, 18-60, for 3-hr laboratory assessment. The purpose of the study is to gain a better understanding of factors that affect sexual functioning. We are looking for 2 types of volunteers: men with erection problems and men without any sexual problems. If you are interested, call Jay Stone at (301) 295-3672 for more information.

APPENDIX B

Phone Screen (Sexually Functional)

INTRODUCTION (READ TO CALLER)

"Based on experimental data collected over the past decade, men who have erection problems are known to differ from men who don't in several areas. One of these important areas includes how feedback about their sexual performance affects erection size. The purpose of this study is to determine how the performance of sexually functional and dysfunctional men is affected by receiving feedback about their erection size while viewing sexually explicit videotapes. Sexually functional and dysfunctional men meeting certain criteria will be asked to participate in a sexual functioning study.

The study will consist of three phases. During the first phase, we will be collecting information on your physical health, sexual functioning, and psychological health. This phase will take approximately one hour to complete. The second phase will also take approximately one hour to complete and involves the physiological assessment of your erection while viewing sexually explicit videotapes. You will be asked to wear a thin rubber tube around your penis to collect information about your erection while you view erotic movies in the privacy of a small room in one of our laboratories. During the second phase, you will also be filling out questionnaires asking you about your sexual performance and making predictions about your performance. During the third phase you will be explained the results of your participation in the study. This phase will take approximately 30 minutes. The interview and physiological assessment are conducted by doctoral students in clinical psychology who are supervised by a licensed clinical psychologist. All information collected during the study is coded and your name will not appear on any records. You will be paid $40 for your participation in the three phases of the study. Do you have any questions? If you are interested in participating in this study I now need to ask you a series of questions to determine if you are the type of person we are looking for: Are you interested?" (If yes, get the following information. If no, thank the caller and discontinue the screening.)

DATE _____

NAME _____

ADDRESS _____

_____

1. HOME PHONE _____
2. WORK PHONE _____
3. AGE _____
4. RACE _____
5. HEIGHT _____
6. WEIGHT _____

7. DO YOU SMOKE? YES NO

8. WHAT IS YOUR MARITAL STATUS?

_____

9. ARE YOU EMPLOYED? YES NO

10. ARE YOU IN THE MILITARY OR A MILITARY DEPENDENT? YES NO

11. HAVE YOU EVER BEEN TOLD BY A PHYSICIAN THAT YOU HAD:

A.	HEART DISEASE	YES NO
B.	HIGH BLOOD PRESSURE	YES NO
C.	KIDNEY DISEASE	YES NO
D.	DIABETES	YES NO
E.	SEXUAL PROBLEMS	YES NO
F.	PROSTATE PROBLEMS	YES NO
G.	BACK INJURY	YES NO

12. ARE YOU CURRENTLY ON ANY MEDICATION? YES NO

IF YES, WHAT ARE YOU TAKING?

_____

13. DO YOU HAVE ANY PROBLEMS WITH YOUR SEXUAL FUNCTIONING?

YES NO

SPECIFICALLY, DO YOU HAVE ANY PROBLEMS:

OBTAINING ERECTIONS? YES NO

MAINTAINING/KEEPING ERECTIONS YES NO

EJACULATING/CUMMING TOO QUICKLY? YES NO

HAVE YOU EVER HAD ANY PROBLEMS WITH YOUR SEXUAL FUNCTIONING? YES NO

HAVE YOU EVER HAD ANY PROBLEMS:

OBTAINING ERECTIONS? YES NO

MAINTAINING/KEEPING ERECTIONS YES NO

EJACULATING/CUMMING TOO QUICKLY? YES NO

14. ARE YOU HETEROSEXUAL? YES NO

15. HAVE YOU EVER RECEIVED MENTAL HEALTH COUNSELING? YES NO

IF YES, CAN YOU TELL ME ABOUT THAT?

_____

16. WOULD YOU BE ABLE TO COME IN FOR A 3 HOUR SESSION AS PART OF THIS STUDY? YES NO

17. WOULD YOU BE WILLING TO ANSWER QUESTIONS ABOUT YOUR PHYSICAL AND MENTAL HEALTH AND YOUR SEXUAL FUNCTIONING? YES NO

18. WOULD YOU BE WILLING TO WATCH EROTIC VIDEOTAPES WHILE WE MEASURE YOUR ERECTION? YES NO

19. WHEN CAN YOU COME IN FOR A 3 HOUR SESSION FOR YOUR PARTICIPATION IN THIS STUDY?

DATE _____ TIME _____

Phone Screen (ED)

DATE _____

NAME _____

ADDRESS _____

_____

1. HOME PHONE _____
2. WORK PHONE _____
3. AGE _____
4. RACE _____
5. HEIGHT _____
6. WEIGHT _____
7. DO YOU SMOKE? YES NO
8. WHAT IS YOUR MARITAL STATUS?
   _____
9. DO YOU HAVE A REGULAR PARTNER? YES NO
10. ARE YOU EMPLOYED? YES NO
11. ARE YOU IN THE MILITARY OR A MILITARY DEPENDENT? YES NO
12. WHO REFERRED YOU TO THIS STUDY?

    NAME _____

    ADDRESS _____

    _____

    PHONE _____
13. HAVE YOU EVER BEEN TOLD BY A PHYSICIAN THAT YOU HAD:

    A. HEART DISEASE YES NO
    B. HIGH BLOOD PRESSURE YES NO
    C. KIDNEY DISEASE YES NO
    D. DIABETES YES NO
    E. PROSTATE PROBLEMS YES NO

F. BACK INJURY YES NO

14. ARE YOU CURRENTLY ON ANY MEDICATION? YES NO

IF YES, WHAT ARE YOU TAKING?

_____

15. DO YOU HAVE ANY PROBLEMS WITH YOUR SEXUAL FUNCTIONING?

YES NO

16. SPECIFICALLY, DO YOU HAVE ANY PROBLEMS:

OBTAINING ERECTIONS? YES NO WHEN BEGAN? _____

MAINTAINING/KEEPING ERECTIONS YES NO WHEN BEGAN?

_____

EJACULATING/CUMMING TOO QUICKLY? YES NO WHEN BEGAN?

_____

17. HAVE YOU EVER HAD ANY PROBLEMS:

OBTAINING ERECTIONS? YES NO WHEN? _____

MAINTAINING/KEEPING ERECTIONS YES NO WHEN?

_____

EJACULATING/CUMMING TOO QUICKLY? YES NO WHEN?

_____

18. SOMETIMES PEOPLE HAVE HAD A VARIETY OF TESTS TO EVALUATE THEIR SEXUAL FUNCTIONING. HAVE YOU EVER HAD ANY?

BLOOD TESTS? YES NO

TEST OF HORMONE LEVELS? YES NO

MEASUREMENT OF BLOOD FLOW IN YOUR PENIS (DOPPLER STUDIES)?

YES NO

19. HAS YOUR DOCTOR TRIED ANY MEDICATION? YES NO

IF YES, WHAT?

_____

20. HAS YOUR DOCTOR INJECTED YOUR PENIS? YES NO

IF YES, WITH WHAT?

_____

21. ARE YOU HETEROSEXUAL? YES NO

22. HAVE YOU EVER RECEIVED MENTAL HEALTH COUNSELING? YES NO

IF YES, CAN YOU TELL ME ABOUT THAT? _____

23. RATIONALE: WE PROVIDE PSYCHOPHYSIOLOGICAL ASSESSMENTS OF SEXUAL FUNCTIONING. THIS MEANS WE TAKE VERY DETAILED INFORMATION ABOUT YOUR SEXUAL AND PSYCHOLOGICAL FUNCTIONING AND MEASURE YOUR ABILITY TO GET AN ERECTION WHILE VIEWING AN EROTIC VIDEOTAPE. THIS TAKES APPROXIMATELY 3 HOURS. BECAUSE THIS IS A RESEARCH STUDY, WE DO NOT CHARGE FOR THESE ASSESSMENTS AND TESTS. AT THE END OF THE ASSESSMENT WE PROVIDE YOU WITH THE RESULTS OF YOUR ASSESSMENT AND GIVE YOUR DOCTOR A REPORT.

24. WHEN CAN YOU COME IN FOR A 3 HOUR ASSESSMENT?

DATE _____ TIME _____

APPENDIX C

Authorization for Exchange of Information

SEXUALITY ASSESSMENT AND TREATMENT PROGRAM
Tracy Sbrocco, Ph.D., Director
Uniformed Services University of the Health Sciences
Department of Medical and Clinical Psychology
4301 Jones Bridge Road
Bethesda, Maryland 20814-4799
301-295-3270

AUTHORIZATION FOR EXCHANGE OF INFORMATION

I hereby authorize the Sexuality Assessment and Treatment Program and

(Doctor/Agency) _____

(Address) _____

_____

to share with each other any and all information in their possession acquired in the course of evaluation and/or treatment of _____.
(Name of Client)

You may accept a photocopy of this authorization.

DATE: _____ SIGNED: _____

WITNESS: _____

CLIENT'S NAME: _____
(Please Print)

ADDRESS: _____

_____

BIRTH DATE: _____

SOCIAL SECURITY #: _____

APPENDIX D

Informed Consent Form (Sexually Functional)
Research Study

Title of Project: Effect of Physiological Feedback on Arousal
Principal Investigator: Jay M. Stone, M.F.S.

Name of Volunteer: _____
(Please Print)

TO PERSONS WHO AGREE TO PARTICIPATE IN THIS STUDY:

You are being asked to take part in a research study. Before you decide to be a part of this research study, you need to understand the risks and benefits so that you can make an informed decision. This is known as informed consent.

This consent form provides information about the research study which has been explained to you. Once you understand the study and the tests it requires, you will be asked to sign this form if you want to take part in the study. Your decision to take part in the study is voluntary. This means that you are free to choose if you will take part in the study.

If, during the course of the study you should have any questions about the study, your participation in it or about your rights as a research subject, you may contact:

a. **Jay M. Stone, M.F.S.**, at 301-295-3672 (Principal Investigator)
Department of Medical & Clinical Psychology, USUHS, Bethesda, MD 29814-4799

b. **Tracy Sbrocco, Ph.D.**, at 301-295-9674 (Academic Advisor)
Department of Medical & Clinical Psychology, USUHS, Bethesda, MD 29814-4799

c. **Research Administration**, at 301-295-3303, USUHS, Bethesda, MD 29814-4799

1. INDICATED BELOW ARE THE FOLLOWING:

a. THE PURPOSE OF THIS STUDY
b. THE PROCEDURES TO BE FOLLOWED
c. THE APPROXIMATE DURATION OF THE STUDY

1.a. THE PURPOSE OF THIS STUDY:

The Department of Medical and Clinical Psychology of The Uniformed Services University of the Health Sciences is carrying out a research study to find out what effect getting feedback about men's erections has on their ability to maintain the erections. Problems with erections are common, affecting approximately 10% of the male population. Great strides have been made in the treatment of psychologically-based erection problems, yet little is known about how it can best be treated. This is because of a limited understanding of the cause and maintenance of the problem. It is now known that erection difficulties are normal in the sense that they are commonly experienced.

Yet only a percentage of men develop a problem significant enough to require treatment. Based on experimental data collected over the past decade, dysfunctional men and functional men are known to differ in several areas. Two of these important areas include how feedback about their sexual performance affects their continued and future performance, and where their attention is focused during sexual performance. The first purpose of this study is to determine how the performance of sexually functional and dysfunctional men is affected by receiving physiological (body-based) feedback about their erections while viewing erotic videotapes. The tapes involve consensual heterosexual sex and do not involve violence of any type. The second purpose of this study is to determine where the attention of sexually functional and dysfunctional men is focused when they receive erection feedback while viewing erotic videotapes. This study is being conducted by Jay M. Stone, M.F.S., a doctoral student in the Department of Medical and Clinical Psychology, for fulfillment of his degree requirements.

1.b. THE PROCEDURES TO BE FOLLOWED:

Phase 1. Initial Information Collection (1 hour)

During the first phase, you will be interviewed about your physical, sexual, and psychological history. The interviewer will use a standard format so that each participant will be asked the same questions. You will also be asked to complete a number of self-report measures that ask you about your medical history, and your sexual and psychosocial functioning.

Phase 2. Physiological Assessment (1 hour)

The second phase will also take approximately one hour to complete and involves a physiological assessment of your erection. Your erection will be monitored while you view an erotic videotape. You will be asked to partially undress, sit in a recliner, and place a strain gauge on your penis. A strain gauge is a small rubber tube while is placed around the shaft of your penis. It measures changes in penis size by having an electric current pass through it. The strain gauge is attached to a polygraph machine that receives and prints the information. You will not feel the electric current and the procedure is not dangerous. We will also monitor your heart rate using several sensors attached to your chest.

After the monitoring equipment is in place, you will be asked to watch three 5-minute videotapes showing a man and woman having sex. We will monitor your erection and heart rate. Before and after each video segment we will ask you to make some ratings about such things as how aroused and how nervous you are.

During the video, some men will receive feedback on their erection. We know men use all kinds of information to evaluate their performance such as how big their erection appears to be and how aroused their partner is. In this study, some participants will see an erection score on the video screen. The erection score is based on the size of your erection. We are interested in finding out how this information affects sexual responding and confidence and predictions about future sexual performance. Some participants will not be shown their erection score. This way we can compare the results

of men who see their erection score with men who do not. The men who do not see their score will be chosen at random. This means we will use a procedure like "drawing a number out of a hat" to assign you to a group. The interviewer will inform you what group you are in.

All material and equipment that comes in contact with participants is either sterilized or disposed of after use.

Phase 3. Post-Session (30 min)

At the end of the videotape sessions (after you get dressed) the interviewer will meet with you and describe and explain your results from Phase 2. Any questions or concerns you have will be discussed. This phase will take approximately ½ hour to complete. The interviewer will also call you in a week to see if you have any additional questions.

1.c. DURATION OF THE STUDY:

The study will take approximately 2 ½ hours to complete.

2. THIS STUDY IS BEING DONE SOLELY FOR THE PURPOSES OF RESEARCH.

3. DISCOMFORTS, INCONVENIENCES, AND/OR RISKS THAT CAN BE REASONABLY EXPECTED ARE:

a. The risks associated with this study are minor. You may find that the interviews and the physiological assessment may make you uncomfortable. You will be asked detailed questions about your sexual functioning and activities. You will also be asked to partially undress and put a strain gauge on your penis while viewing erotic videotapes. You will NOT be forced to do anything you do not want to do. You may decline to participate at any time and/or withdraw your participation at any time.

b. You may feel upset or distressed if your erection score or your erection is lower than you predicted or expected. The interviewer will meet with you following the physiological assessment to address any concerns you may have. Your results will be presented and discussed with you. Your questions and concerns will be addressed.

c. You will probably experience sexual arousal during the physiological assessment phase of the study. This response is normal and expected. Questions and concerns you may have about your response will be addressed during the post session.

d. The study involves a small time commitment that you may find inconvenient. You will be asked to come to the university for one 2 ½ hour appointment.

4. POSSIBLE BENEFITS TO YOU THAT MAY BE REASONABLY EXPECTED ARE:

a. You will earn $40 for your participation in this study.
b. You may learn information about your sexual functioning that is helpful to you.

5. THE BENEFITS TO SCIENCE AND TO HUMANKIND THAT ARE SOUGHT IN THIS STUDY ARE:

You will be providing information that will be helpful in expanding scientific knowledge about sexual behavior. The results of this study will help us gain a better understanding of how physiologic feedback affects sexual functioning. The results will also tell us how the attention of sexually functional men compares to sexually dysfunctional men. This knowledge could have important treatment and prevention implications.

6. ALTERNATE PROCEDURES THAT MAY BE ADVANTAGEOUS:

Not applicable.

7. COSTS

There are no costs to you for participating in this study.

8. YOUR RIGHTS, WELFARE, AND PRIVACY WILL BE PROTECTED IN THE FOLLOWING MANNER:

(1) All data obtained about you during the course of this study will be kept confidential and accessible only to the principal investigator, his academic advisor, and their assistants on this project. In addition, the Institutional Review Board at The Uniformed Services University of the Health Sciences may see your records.

(2) Your name will not be associated with the information you provide. You will be assigned a subject number.

(3) Should the results of this project be published, you will be referred to only by number.

(4) Confidentiality is protected to the best extent provided under law.

9. RIGHT TO WITHDRAW FROM THE STUDY

You may decide to stop this study at any time. Your care and relations with the faculty, staff and administration at USUHS will not be changed in any way if you decide to stop the study. You should let the investigator in charge of the study know if you decide to stop the study.

10. RECOURSE IN THE EVENT OF INJURY:

This study should not entail any physical or mental risk beyond those described above. We do not expect complications to occur, but if, for any reason, you feel that continuing this study would constitute a hardship for you, we will end your participation in the study.

The Department of Defense will provide medical care at government facilities for DoD eligible members (active duty, dependents, and retired military) for physical injury or illness resulting from participation in this research. Such care may not be available to other research participants, except in the event of an emergency. Compensation may be available through judicial avenues to non-active duty research participants if they are injured through the negligence (fault) of the government.

If at any time you believe you have suffered an injury or illness as a result of participating in this research project, you should contact the Office of Research at the Uniformed Services University of the Health Sciences, Bethesda, Maryland 20814-4799 at (301) 295-3303. This office can review the matter with you, can provide information about your rights as a subject, and may be able to identify resources available to you. Information about judicial avenues of compensation is available from the University's General Counsel at (301) 295-3028.

11. QUESTIONS

If you have any questions at any time about the study you may contact the principal investigator, Jay Stone, M.F.S., at the Department of Medical and Clinical Psychology, Uniformed Services University, at (301) 295-3672, or his academic advisor, Dr. Tracy Sbrocco, at (301) 295-9674. If you have questions about your rights as a research subject, you should call the Director of Research Programs, in The Office of Research at the Uniformed Services University of the Health Sciences (301) 295-3303. This person is your representative and has no connection to the investigators conducting this study.

12. STATEMENT AND SIGNATURE OF VOLUNTEER

I have read this consent form and I understand the procedures to be used in this study and the possible risks, inconveniences, and/or discomforts that may be involved. All of my questions have been answered. I freely and voluntarily choose to participate. I understand I may withdraw at any time. My signature also indicates that I have received a copy of this consent form for my information.

Signature of Volunteer: _____

Printed Name of Volunteer: _____

Date: _____

13. STATEMENT AND SIGNATURE OF INVESTIGATOR

I certify that the research study has been explained to the above individual, by me or my research staff, and that the individual understands the nature and purpose, the possible risks and benefits associated with taking part in this research study. Any questions have been raised, have been answered.

Signature of Witness: _____

Signature of Investigator: _____

Printed Name, Rank, and Title of Investigator: _____

Date: _____

Informed Consent Form (ED)
Research Study

Title of Project: Effect of Physiological Feedback on Arousal
Principal Investigator: Jay M. Stone, M.F.S.

Name of Volunteer: _____
(Please Print)

TO PERSONS WHO AGREE TO PARTICIPATE IN THIS STUDY:

You are being asked to take part in a research study. Before you decide to be a part of this research study, you need to understand the risks and benefits so that you can make an informed decision. This is known as informed consent.

This consent form provides information about the research study which has been explained to you. Once you understand the study and the tests it requires, you will be asked to sign this form if you want to take part in the study. Your decision to take part in the study is voluntary. This means that you are free to choose if you will take part in the study.

If, during the course of the study you should have any questions about the study, your participation in it or about your rights as a research subject, you may contact:

a. **Jay M. Stone, M.F.S.**, at 301-295-3672 (Principal Investigator)
Department of Medical & Clinical Psychology, USUHS, Bethesda, MD 29814-4799

b. **Tracy Sbrocco, Ph.D.**, at 301-295-9674 (Academic Advisor)
Department of Medical & Clinical Psychology, USUHS, Bethesda, MD 29814-4799

c. **Research Administration**, at 301-295-3303, USUHS, Bethesda, MD 29814-4799

1. INDICATED BELOW ARE THE FOLLOWING:

a. THE PURPOSE OF THIS STUDY
b. THE PROCEDURES TO BE FOLLOWED
c. THE APPROXIMATE DURATION OF THE STUDY

1.a. THE PURPOSE OF THIS STUDY:

The Department of Medical and Clinical Psychology of The Uniformed Services University of the Health Sciences is carrying out a research study to find out what affect getting feedback about men's erections has on their ability to maintain the erections. Problems with erections are common, affecting approximately 10% of the male population. Great strides have been made in the treatment of psychologically-based erection problems, yet little is known about how it can best be treated. This is because of a limited understanding of the cause and maintenance of the problem. It is now known that erection difficulties are normal in the sense that they are commonly experienced. Yet only a percentage of men develop a problem significant enough to require treatment. Based on experimental data collected over the past decade, dysfunctional men and

functional men are known to differ in several areas. Two of these important areas include how feedback about their sexual performance affects their continued and future performance, and where their attention is focused during sexual performance. The first purpose of this study is to determine how the performance of sexually functional and dysfunctional men is affected by receiving physiological (body-based) feedback about their erections while viewing erotic videotapes. The tapes involve consensual heterosexual sex and do not involve violence of any type. The second purpose of this study is to determine where the attention of sexually functional and dysfunctional men is focused when they receive erection feedback while viewing erotic videotapes. This study is being conducted by Jay M. Stone, M.F.S., a doctoral student in the Department of Medical and Clinical Psychology, for fulfillment of his degree requirements.

1.b. THE PROCEDURES TO BE FOLLOWED:

Phase 1. Initial Information Collection (1 ½ hour)

During the first phase, you will be interviewed about your physical, sexual, and psychological history. The interviewer will use a standard format so that each participant will be asked the same questions. You will also be asked to complete a number of self-report measures that ask you about your medical history, and your sexual and psychosocial functioning.

Phase 2. Physiological Assessment (1 hour)

The second phase will also take approximately one hour to complete and involves a physiological assessment of your erection. Your erection will be monitored while you view an erotic videotape. You will be asked to partially undress, sit in a recliner, and place a strain gauge on your penis. A strain gauge is a small rubber tube while is placed around the shaft of your penis. It measures changes in penis size by having an electric current pass through it. The strain gauge is attached to a polygraph machine that receives and prints the information. You will not feel the electric current and the procedure is not dangerous. We will also monitor your heart rate using several sensors attached to your chest.

After the monitoring equipment is in place, you will be asked to watch three 5-minute videotapes showing a man and woman having sex. We will monitor your erection and heart rate. Before and after each video segment we will ask you to make some ratings about such things as how aroused and how nervous you are.

During the video, some men will receive feedback on their erection. We know men use all kinds of information to evaluate their performance such as how big their erection appears to be and how aroused their partner is. In this study, some participants will see an erection score on the video screen. The erection score is based on the size of your erection. We are interested in finding out how this information affects sexual responding and confidence and predictions about future sexual performance. Some participants will not be shown their erection score. This way we can compare the results of men who see their erection score with men who do not. The men who do not see their score will be chosen at random. This means we will use a procedure like “drawing a

number out of a hat" to assign you to a group. The interviewer will inform you what group you are in.

All material and equipment that comes in contact with participants is either sterilized or disposed of after use.

Phase 3. Post-Session (30 min)

At the end of the videotape sessions (after you get dressed) the interviewer will meet with you and describe and explain your results from Phase 2. Any questions or concerns you have will be discussed. This phase will take approximately ½ hour to complete. The interviewer will also call you in a week to see if you have any additional questions.

1.c. DURATION OF THE STUDY:

The study will take approximately 3 hours to complete.

2. THIS STUDY IS BEING DONE PRIMARILY FOR THE PURPOSES OF RESEARCH.

The results will be explained to you and a written copy will be provided to your referring physician. This information may be helpful in gaining a better understanding of your problem.

3. DISCOMFORTS, INCONVENIENCES, AND/OR RISKS THAT CAN BE REASONABLY EXPECTED ARE:

a. The risks associated with this study are minor. You may find that the interviews and the physiological assessment may make you uncomfortable. You will be asked detailed questions about your sexual functioning and activities. You will also be asked to partially undress and put a strain gauge on your penis while viewing erotic videotapes. You will NOT be forced to do anything you do not want to do. You may decline to participate at any time and/or withdraw your participation at any time.

b. You may feel upset or distressed if your erection score or your erection is lower than you predicted or expected. The interviewer will meet with you following the physiological assessment to address any concerns you may have. Your results will be presented and discussed with you. Your questions and concerns will be addressed.

c. You will probably experience sexual arousal during the physiological assessment phase of the study. This response is normal and expected. Questions and concerns you may have about your response will be addressed during the post session.

d. The study involves a small time commitment that you may find inconvenient. You will be asked to come to the university for one 3 hour appointment.

4. POSSIBLE BENEFITS TO YOU THAT MAY BE REASONABLY EXPECTED ARE:

a. You will receive an extensive psychophysiological assessment of your sexual functioning. This information will be explained to you. A report will be provided to your referring physician and this information may be helpful in recommending treatment for your difficulties.

b. If it seems you may benefit from psychological treatment for sexual functioning, you will be offered treatment free of charge to treat your erection difficulty.

c. You will earn $40 for your participation in this study.

5. THE BENEFITS TO SCIENCE AND TO HUMANKIND THAT ARE SOUGHT IN THIS STUDY ARE:

You will be providing information that will be helpful in expanding scientific knowledge about sexual behavior. The results of this study will help us gain a better understanding of how physiologic feedback affects sexual functioning. The results will also tell us how the attention of sexually functional men compares to sexually dysfunctional men. This knowledge could have important treatment and prevention implications.

6. ALTERNATE PROCEDURES THAT MAY BE ADVANTAGEOUS:

You may obtain similar psychological and physiological assessments elsewhere. Should you decide not to participate in this study we will still provide you with a psychophysiological assessment of your sexual functioning, a written report will be sent to your referring physician, and if it seems you may benefit from psychological treatment for sexual functioning, you will be offered treatment free of charge to treat your erection difficulty. If, on the other hand, you would prefer a referral for assessment and/or treatment, a community referral will be provided.

7. COSTS

There are no costs to you for participating in this study.

8. YOUR RIGHTS, WELFARE, AND PRIVACY WILL BE PROTECTED IN THE FOLLOWING MANNER:

(1) All data obtained about you during the course of this study will be kept confidential and accessible only to the principal investigator, his academic advisor, and their assistants on this project. In addition, the Institutional Review Board at The Uniformed Services University of the Health Sciences may see your records.

(2) Your name will not be associated with the information you provide. You will be assigned a subject number.

(3) Should the results of this project be published, you will be referred to only by number.

(4) Confidentiality is protected to the best extent provided under law.

9. RIGHT TO WITHDRAW FROM THE STUDY

You may decide to stop this study at any time. Your care and relations with the faculty, staff and administration at USUHS will not be changed in any way if you decide to stop the study. You should let the investigator in charge of the study know if you decide to stop the study.

10. RECOURSE IN THE EVENT OF INJURY:

This study should not entail any physical or mental risk beyond those described above. We do not expect complications to occur, but if, for any reason, you feel that continuing this study would constitute a hardship for you, we will end your participation in the study.

The Department of Defense will provide medical care at government facilities for DoD eligible members (active duty, dependents, and retired military) for physical injury or illness resulting from participation in this research. Such care may not be available to other research participants, except in the event of an emergency. Compensation may be available through judicial avenues to non-active duty research participants if they are injured through the negligence (fault) of the government.

If at any time you believe you have suffered an injury or illness as a result of participating in this research project, you should contact the Office of Research at the Uniformed Services University of the Health Sciences, Bethesda, Maryland 20814-4799 at (301) 295-3303. This office can review the matter with you, can provide information about your rights as a subject, and may be able to identify resources available to you. Information about judicial avenues of compensation is available from the University's General Counsel at (301) 295-3028.

11. QUESTIONS

If you have any questions at any time about the study you may contact the principal investigator, Jay Stone, M.F.S., at the Department of Medical and Clinical Psychology, Uniformed Services University, at (301) 295-3672, or his academic advisor, Dr. Tracy Sbrocco, at (301) 295-9674. If you have questions about your rights as a research subject, you should call the Director of Research Programs, in The Office of Research at the Uniformed Services University of the Health Sciences (301) 295-3303.

This person is your representative and has no connection to the investigators conducting this study.

12. STATEMENT AND SIGNATURE OF VOLUNTEER

I have read this consent form and I understand the procedures to be used in this study and the possible risks, inconveniences, and/or discomforts that may be involved. All of my questions have been answered. I freely and voluntarily choose to participate. I understand I may withdraw at any time. My signature also indicates that I have received a copy of this consent form for my information.

Signature of Volunteer: _____

Printed Name of Volunteer: _____

Date: _____

13. STATEMENT AND SIGNATURE OF INVESTIGATOR

I certify that the research study has been explained to the above individual, by me or my research staff, and that the individual understands the nature and purpose, the possible risks and benefits associated with taking part in this research study. Any questions have been raised, have been answered.

Signature of Witness: _____

Signature of Investigator: _____

Printed Name, Rank, and Title of Investigator: _____

Date: _____

APPENDIX E

SEXUAL DYSFUNCTION INTERVIEW-revised

◆◆◆

Tracy Sbrocco, Ph.D.,
Risa Weisberg, B.A.,
&
David H. Barlow, Ph.D.

Albany, NY: 1995

◆◆◆

SEXUAL DYSFUNCTION INVENTORY

Client Name:

Address:

Home Ph:

Work Ph:

Referral Source:

Address:

Release of Information Obtained? **Yes** **No**

Date referral received:

I. Life Situation

Let me begin by getting some basic information:

1. DOB/Age

2. Ethnicity
   1 = Caucasian, Non-Hispanic
   2 = Black, Non-Hispanic
   3 = Hispanic
   4 = Asian
   5 = Other

3. Current Relationship Status
   1 = Never Married
   2 = Divorced
   3 = Separated
   4 = Widowed
   5 = Married
   6 = Living Together

4. Duration of Marital/Relationship Status (# of years)
   99= Missing or Not Applicable

5. Years of Education

a. Less than High School Degree
b. High School Degree
c. Partial College
d. College Degree
e. Graduate or Professional School

6. Occupation (Present or Previous)

1 = High Level Executive, Professional (M.D., Ph.D., Attorney)
2 = Business Manager, Lesser Professional (Nurse, Teacher, Social Worker)
3 = Administrator, minor professional [legal secretary, small business owner (e.g. bakery, clothing)]
4 = Clerical or sales worker, technician
5 = Skilled manual employee
6 = Machine operator, semi-skilled employee
7=Unskilled Employee (laborer, messenger)

7. Employment Status
1 = Retired
2 = Full-Time
3 = Part-Time
4 = Disabled
5 = Unemployed

8. Length of time at current job _____

II. **Presenting Problem(s)**
I **know about your sexual problem(s)** from what you said on the phone/what your M.D. said, etc.
Is that correct?

I will get back to your sexual functioning, but first I would like to ask you some questions pertaining to your overall psychological functioning. Many of the questions may not apply to you and some will.

Psychiatric Diagnostic Interview, revised (PDI-R)

Begin with **Alcoholism,** come back to Organic Brain Syndrome only if it seems necessary at the end of the interview.

Pay close attention to signs and symptoms of **Major Depression.** MDE can have a profound affect on sexual functioning. When in doubt, continue questioning.

Diagnoses:
NONE

FREQUENCY OF SEXUAL BEHAVIOR

1. How often to you engage in intercourse with your/a partner?

2. What is your ideal frequency of intercourse?

3. How often do you engage in mutual cuddling/stimulation without intercourse?

4. I'd like to ask you some questions about masturbation/self-stimulation. I want to assure you that we consider it to be a normal, healthy activity. We are aware that not everybody feels this way...
How often do you engage in self-stimulation/masturbation?

SEXUAL DYSFUNCTIONS

I. SEXUAL DESIRE DISORDERS

A. Hypoactive Sexual Desire Disorder

1. How would you describe your interest in sex?

a. (If client describes problems) Has your interest changed or is your current interest pretty typical for you?

b. How long have you felt this way?

c. If change occurred, What was associated with or caused this change? i. personal stress/emotional problems ii. illness iii. marital problems iv. partner stress/emotional problems v. partner illness vi. sexual problem vii. medication

2. Do you have sexual fantasies

a. during intercourse? YES NO % time

b. during masturbation? YES NO % time

c. at other times? YES NO % time

3. Do you always feel this way or are there times or situations when you have a strong interest/desire in sex?
If client **is currently depressed (or has another Axis I) disorder OR **If the client has a medical problem(s) that may be related to his/her sexual functioning: ASK 0. 4. otherwise SKIP to 5.

4. Was **your interest/desire in sex low before your problem(s)** with _____ began?

5. Have you ever **been sexually abused,** raped, or **had a very** negative experience associated with sex?

If yes, what affect did this experience have on your sexual behavior?

a. avoid all/most sexual behavior
b. relationship difficulties; trust
c. pain
d. OTHER

SEXUAL AVERSION DISORDER

6. Do you avoid engaging in sexual behavior with your/a partner?

7. If in a relationship: Who usually intiates sexual activity in your relationship?

8. Do you experience anxiety or worry when you think about
engaging in sexual behavior with your/a partner?

If yes, what types of things do you say to yourself?

-performance self statements
-failure self statements
-concern about pleasing partner
-concern/worry about sexually transmitted diseases
-more general cognitive interference

9. **Do you fear engaging in sex?**

****If client is currently depressed** (or has **another Axis I such as I OCD disorder)** I or **If the client has a medical problem(s) that may be related to **his/her sexual functioning: (ASK** Q.10)

10. Did you avoid/fear sex before your problems with began?

DX:

Hypoactive Sexual Desire Disorder

Sexual Aversion Disorder

Specify: psychogenic only psychogenic and biogenic (biogenic only record on Axis III)

lifelong or acquired generalized or situational

II. MALE ERECTILE DIS0RDER

1. Do you have problems attaining/getting an erection?

2. Do you have problems maintaining/keeping an erection?

3. When did these problems begin? month and year

a. Did the problem come on gradually?

b. Is there a specific event associated with the start of the difficulty?

i. personal stress/emotional problems
ii. illness iii. marital problems
iv. partner stress/emotional problems
v. partner illness
vi. drinking/alcohol
vii. medication
viii. loss of partner

Classify event - Medical or Psychological

4. What percentage of the time is this a problem?

5. Using a 1 to 100 scale, where 1 is no erection and 100 is the best erection You've ever had ...

a. What percent of an erection do you **typically** obtain? _____

b. What percent describes the best erection you can get?_____

6. Do you have a problem with erections during foreplay?

7. Do you have a problem with erections when attempting penetration?

8. Are you able to penetrate?

What percent of the time? _____

9. Do you have problems maintaining your erection, that is, do you lose your erection?

What do you do when you lose your erection?
a. quit/give up
b. try to get it back - successful or not?

l0. Do you have problems obtaining or maintaining an erection during masturbation?

**If individual describes problems obtaining or maintaining an erection:

11. Do you notice your ability to get an erection depends on the situation?

Partner
Masturbation vs Partner
Stress
Fatigue/Tired

12. Do you have morning erections?

13. Do you ejaculate with intercourse? with stimulation by partner? with self-stimulation?

Subjective pleasure/excitement:

14. How do you feel during sexual activity?

15. Do you experience an orgasm?

Dx:

Male Erectile Disorder

Onset

Specify: psychogenic only, psychogenic, and biogenic (biogenic only record on Axis III)

lifelong or acquired generalized or situational

III. ORGASM DISORDER

1. Do you ever have problems reaching orgasm during sexual behavior?

If yes, Does this difficulty occur with

a. masturbation
b. intercourse
c. foreplay/partner stimulation

2. Does it seem like you are aroused before experiencing this difficulty?

3. When did this difficulty begin?

Is onset associated with events such as

a. Relationship change or length of relationship

b. Change in the pattern of sexual behavior (for example, being used to multiple partners and now has one partner)

c. Stress

d. Medical problem

Dx:

Inhibited Male Orgasm

Onset

Specify: psychogenic only **psychogenic and biogenic (biogenic only record on Axis III)**

lifelong or acquired generalized or situational

IV. PREMATURE EJACULATION

1. Do you ever experience problems ejaculating/coming before you are ready to?

Percent of the time:_____

2. Do you consider this a problems?

If client describes a problems ASK q.3, otherwise SKIP to DYSPAREUNIA

3. When did this begin?

4. If client indicates he does not ejaculate, inquire about prostate surgery.

5. Do you sometimes ejaculate before penetration, when you do not want to?

Does this occur after stimulation or is thinking about sex enough?

6. Do you ejaculate immediately after penetrating or in a shorter time than you wish?

Estimate the length of time _____

7. How long before you ejaculate during masturbation?

8. Do you have problems controlling your erections, that is having erections when you do not want to generally because it is embarrassing?

9. Do you notice that changes in the situation make a difference, for example:

a. partner attractiveness b. novelty of the situation c. length of time since last sexual experience or ejaculation d. oral sex e. what other factors influence latency to ejaculation (increase or decrease)?

10. Have you tried any of the following to delay ejaculation:

a. alcohol percent of time used

b. drugs

c. numbing cremes/ointments

d. thinking of un-arousing things

e. withdrawal/ceasing stimulation
DX:

Premature Ejaculation

Onset

Specify: psychogenic only psychogenic and biogenic (biogenic only record on Axis III)

lifelong or acquired generalized or situational

V. DYSPAREUNIA

1. Do you ever experience pain associated with sexual activity?

2. Does this occur before, during, after sexual activity?

Describe problem:

3. When did this problem begin?

4. Does this occur across all situations?

5. Assess whether this is due to lack of partner's **lubrication or** difficulty penetrating due to vaginismus.

DX:

Dyspareunia

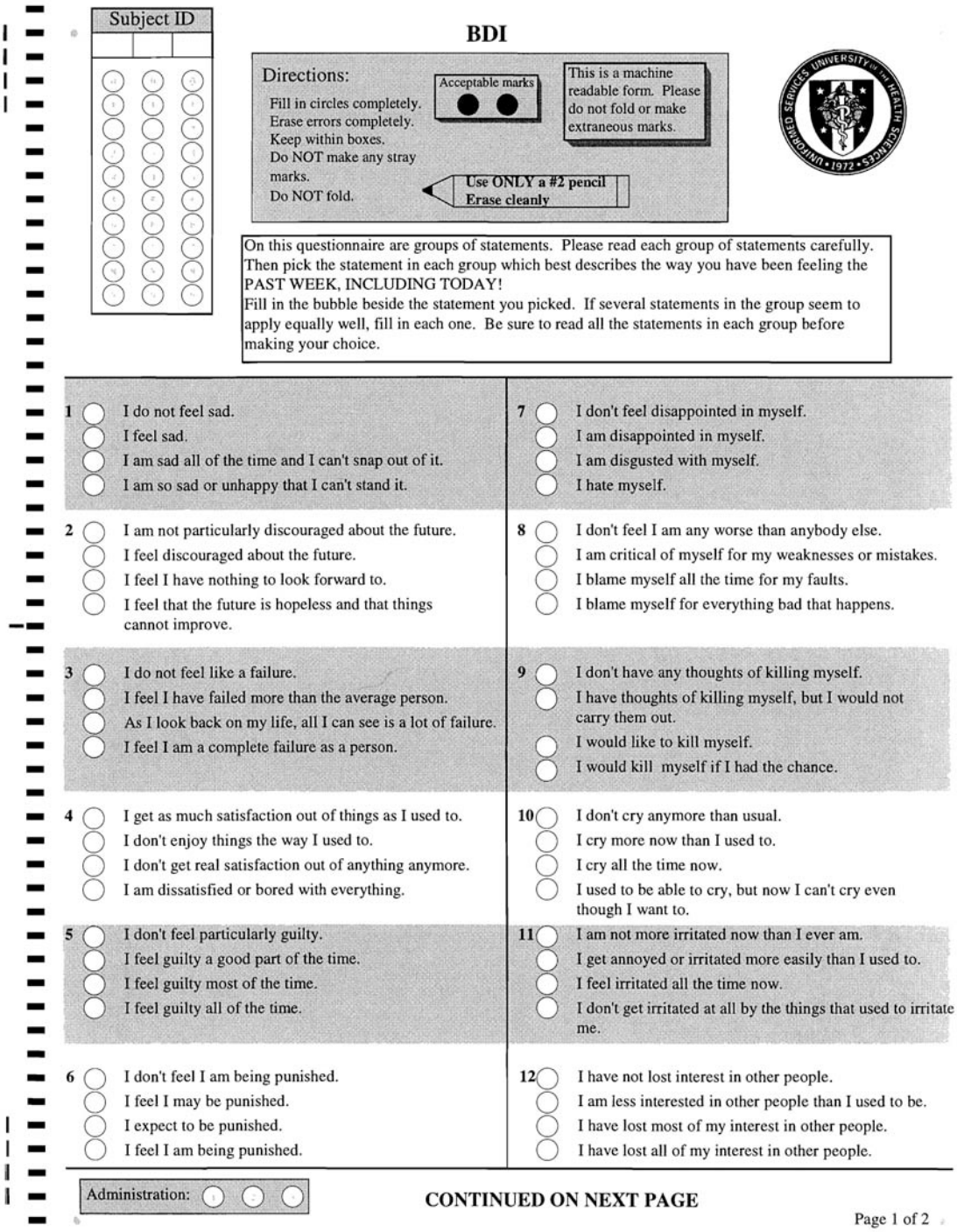
Onset

Specify: psychogenic only psychogenic and biogenic (biogenic only record on Axis III)

lifelong or acquired generalized or situational

COMMENTS:

APPENDIX F



Subject ID

BDI

Directions:

Fill in circles completely.
Erase errors completely.
Keep within boxes.
Do NOT make any stray marks.
Do NOT fold.

Acceptable marks

This is a machine readable form. Please do not fold or make extraneous marks.

Use ONLY a #2 pencil
Erase cleanly

On this questionnaire are groups of statements. Please read each group of statements carefully. Then pick the statement in each group which best describes the way you have been feeling the PAST WEEK, INCLUDING TODAY!
Fill in the bubble beside the statement you picked. If several statements in the group seem to apply equally well, fill in each one. Be sure to read all the statements in each group before making your choice.

1	I do not feel sad.	7	I don't feel disappointed in myself.
	I feel sad.		I am disappointed in myself.
	I am sad all of the time and I can't snap out of it.		I am disgusted with myself.
	I am so sad or unhappy that I can't stand it.		I hate myself.
2	I am not particularly discouraged about the future.	8	I don't feel I am any worse than anybody else.
	I feel discouraged about the future.		I am critical of myself for my weaknesses or mistakes.
	I feel I have nothing to look forward to.		I blame myself all the time for my faults.
	I feel that the future is hopeless and that things cannot improve.		I blame myself for everything bad that happens.
3	I do not feel like a failure.	9	I don't have any thoughts of killing myself.
	I feel I have failed more than the average person.		I have thoughts of killing myself, but I would not carry them out.
	As I look back on my life, all I can see is a lot of failure.		I would like to kill myself.
	I feel I am a complete failure as a person.		I would kill myself if I had the chance.
4	I get as much satisfaction out of things as I used to.	10	I don't cry anymore than usual.
	I don't enjoy things the way I used to.		I cry more now than I used to.
	I don't get real satisfaction out of anything anymore.		I cry all the time now.
	I am dissatisfied or bored with everything.		I used to be able to cry, but now I can't cry even though I want to.
5	I don't feel particularly guilty.	11	I am not more irritated now than I ever am.
	I feel guilty a good part of the time.		I get annoyed or irritated more easily than I used to.
	I feel guilty most of the time.		I feel irritated all the time now.
	I feel guilty all of the time.		I don't get irritated at all by the things that used to irritate me.
6	I don't feel I am being punished.	12	I have not lost interest in other people.
	I feel I may be punished.		I am less interested in other people than I used to be.
	I expect to be punished.		I have lost most of my interest in other people.
	I feel I am being punished.		I have lost all of my interest in other people.

Administration:

CONTINUED ON NEXT PAGE

Page 1 of 2

Subject ID

BDI Continued

Directions:

Fill in circles completely.
Erase errors completely.
Keep within boxes.
Do NOT make any stray marks.
Do NOT fold.

Acceptable marks

This is a machine readable form. Please do not fold or make extraneous marks.

Use ONLY a #2 pencil
Erase cleanly

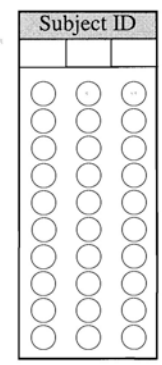
13 ◯ I make decisions about as well as I ever could. ◯ I put off making decisions more than I used to. ◯ I have greater difficulty in making decisions than before. ◯ I can't make decisions at all anymore.	18 ◯ My appetite is no worse than usual. ◯ My appetite is not as good as it used to be. ◯ My appetite is much worse now. ◯ I have no appetite at all anymore.
14 ◯ I don't feel I look any worse than I used to. ◯ I am worried that I am looking old or unattractive. ◯ I feel that there are permanent changes in my appearance that make me look unattractive. ◯ I believe that I look ugly.	19 ◯ I haven't lost much weight, if any lately. ◯ I have lost more than 5 pounds. ◯ I have lost more than 10 pounds. ◯ I have lost more than 15 pounds. I am purposely trying to lose weight by eating less. ◯ ◯
15 ◯ I can work as well as before. ◯ It takes an extra effort to get started at doing something. ◯ I have to push myself very hard to do anything. ◯ I can't do any work at all.	20 ◯ I am no more worried about my health than usual. ◯ I am worried about physical problems such as aches and pains, or upset stomach, or constipation. ◯ I am very worried about physical problems, and it's hard to think of much else. ◯ I am so worried about my physical problems, that I cannot think about anything else.
16 ◯ I can sleep as well as usual. ◯ I don't sleep as well as I used to. ◯ I wake up 1-2 hours earlier than usual and find it hard to get back to sleep. ◯ I wake up several hours earlier than I used to and cannot get back to sleep.	21 ◯ I have not noticed any recent change in my interest in sex. ◯ I am less interested in sex than I used to be. ◯ I am much less interested in sex than I used to be. ◯ I have lost interest in sex completely.
17 ◯ I don't get more tired than usual. ◯ I get tired more easily than I used to. ◯ I get tired from doing almsot anything. ◯ I am too tired to do anything.	

Administration: ◯ ◯ ◯

APPENDIX G

BAI

Subject ID



Directions:

Fill in circles completely.
Erase errors completely.
Keep within boxes.
Do NOT make any stray marks.
Do NOT fold.

Acceptable marks

This is a machine readable form. Please do not fold or make extraneous marks.

Use ONLY a #2 pencil
Erase cleanly

Below is a list of common symptoms of anxiety. Please carefully read each item in the list. Indicate how much you have been bothered by each symptom during the PAST WEEK, INCLUDING TODAY, by filling in the appropriate bubble corresponding to each symptom.

	NOT AT ALL	MILDLY It did not bother me much	MODERATELY It was very unpleasant, but I could stand it	SEVERELY I could barely stand it
1. Numbness or tingling.	○	○	○	○
2. Feeling hot.	○	○	○	○
3. Wobbliness in legs.	○	○	○	○
4. Unable to relax.	○	○	○	○
5. Fear of the worst happening.	○	○	○	○
6. Dizzy or lightheaded.	○	○	○	○
7. Heart pounding or racing.	○	○	○	○
8. Unsteady.	○	○	○	○
9. Terrified.	○	○	○	○
10. Nervous.	○	○	○	○
11. Feelings of choking.	○	○	○	○
12. Hands trembling.	○	○	○	○
13. Shaky.	○	○	○	○
14. Fear of losing control.	○	○	○	○
15. Difficulty breathing.	○	○	○	○
16. Fear of dying.	○	○	○	○
17. Scared.	○	○	○	○
18. Indigestion or discomfort in adbomen.	○	○	○	○
19. Faint.	○	○	○	○
20. Face flushed.	○	○	○	○
21. Sweating (not due to heat).	○	○	○	○

Administration: ○ ○ ○

Page 1 of 1

APPENDIX H

Subject:_____

Sexual Arousal Questionnaire

1. Mark on the line how sexually aroused you felt during the film you just watched:

|_____|

no arousal medium arousal maximum arousal

2. Mark on the line how anxious, tense, or nervous you felt during the film you just watched:

|_____|

no anxiety medium anxiety maximum anxiety

3. Mark on the line how much confidence you had in your ability to maintain an erection during the film you just watched:

|_____|

no confidence medium confidence maximum confidence

4. Mark on the line the maximum size of your erection during the film you just watched:

|_____|

no erection half erection full erection

5. Mark on the line your level of attention to the film you just watched:

|_____|

no attention medium attention maximum attention

6. Mark on the line your level of attention to your body during the film you just watched:

|_____|

no attention medium attention maximum attention

7. Mark on the line how much control you had over your erection:

|_____|

no control medium control maximum control

8. Mark on the line how many negative-type thoughts you had during the film you just watched:

|_____|

no negative thoughts lots of negative thoughts

9. Mark on the line how much your thoughts interfered with your ability to maintain your erection:

|_____|

no interference medium interference maximum interference

10. Mark on the line how similar your response was (for example: erection, thoughts, arousal) during this lab experience compared to actual sexual situations:

|_____|

not at all similar very similar

11. List the thoughts you had during the film you just watched:

1. _____

2. _____

3. _____

4. _____

5. _____

6. _____

7. _____

8. _____

9. _____

10. _____

11. _____

12. _____

13. _____

APPENDIX I

Medical Information Form

A. **Identifying Data:**

Name: _____

Home phone: ( ) _____

Address: _____

Marital Status: _____

Date of Birth: _____

Occupation: _____

Work phone: ( )_____

Married	____Yes
Single, never married	____Yes
Divorced	____Yes
Widowed	____Yes

B.

1. Do you receive regular medical care from a physician or clinic? ☐ No ☐ Yes

If yes, please provide the following information:

Name of Physician or Clinic: _____

2. Have you been evaluated by a urologist? ☐ No ☐ Yes

If yes, please provide the following information:

Name of Physician or Clinic: _____

3. Have you ever had to be hospitalized? ☐ No ☐ Yes

If yes, complete the following:

Year	Doctor's Name	Name of Hospital	Reason
_____	_____	_____	_____
_____	_____	_____	_____

4. Have you ever had surgery, or been advised to have surgery? ☐ No ☐ Yes

If yes, complete the following:

Year	Doctor's Name	Name of Hospital	Reason
_____	_____	_____	_____
_____	_____	_____	_____

No. _____

C. Personal Medical History:

1. Have you ever been told you had any of the following medical conditions?

	NO	YES	When/ Explain	If yes, are you currently being treated or followed for these problems
Heart Disease				
High Blood				
Diabetes or				
Cancer				
Thyroid				
Depression				
Alcoholism				
High				
Low				
Other				
Prostate				
Anxiety or				
Spinal cord,				
Back problems				
Drug				
Gall Bladder				
Digestive				
Kidney				
Peptic Ulcers				
Colitis				
Meningitis or				
Tuberculosis				
Stroke				
Rheumatic				
Asthma				
Birth Defects				
Gout				

(a) Have you ever had any other disease? ☐ No ☐ Yes

If yes, explain:

_____

_____

_____

(b) What is your current weight?_____ lbs. __estimate __actual

(c) What is the most you have ever weighed? _____ lbs.

When? _____

(d) Have you recently lost or gained any weight? ☐ No ☐ Yes

(e) Can you explain any recent weight loss or gain?

_____

2. Have you recently had any of the following tests?

	No	**Yes**	**When**	**Results**
Physical Exam				
Blood Tests				
Hormone Levels				
Electrocardiogram (EKG)				
Blood Flow in penis (Doppler Study)				
Penis Injection (Papaverine)				
Nocturnal penile tumescence				
Other				

3. Are you in the habit of using any of the following?

	Amount Currently Using	**Most Ever Used**	**When Stopped Using**
Coffee (cups/day)			
Cigarettes (packs/day)			
Alcohol (amount and types of alcohol used daily)			
Vitamins			
Sleeping Pills			
Aspirin			
Laxatives			
Diet Pills			

4. Are you currently on any medication? ☐ No ☐ Yes

If yes, please give name and dosage:

_____

_____

_____

5. Have you ever used any of the following medications for your mood, nerves, sleep. pain, or energy level?

(Circle the ones used.)

	No	**Yes**	**When/ How Long**	**How Much/ Reason**
Dilantin, Tegretol, L-Dopa, Cogentin, Artane				
Medication for anxiety , stress or nerves (Xanax, Valium, Librium, Serax, Dalmane. Tranxene, Ativan, etc.)				
Medication for depression (Prozac, Wellbutrin, Elavil, etc.)				
Lithium				
Thorazine, Mellaril, Stelazine, Navane, Haldol, Prolixin Injection, Loxitane, Moban, Serentil				
Phenobarbital, Seconal, Tuinal, Other barbiturates				
Amphetamines, Ritalin, Other stimulants				
Codeine, Methadone, Percodan, Dilaudid, Talwin, Darvon, Demerol, other prescription pain killers				
Other				

6. What type(s) of treatment have you tried for your sexual difficulties?

	No	Yes	When	How successful/helpful? (Please rate from 0-5, 0=no change) Please describe
Testosterone Injections				
Testosterone Patch				
Other hormone Replacement (Specify):				
Vacuum Pump (ErecAid)				
Penis Injection (Papaverine)				
MUSE				
Medication(s) (Specify):				
Surgery or Penile Implant				
Self-help books/videos				
Creams/Ointments				
Psychological Treatment (Sex Therapy, Marital Therapy)				
Other (Please Specify):				

D. Personal Psychiatric History:

1. Have you ever received any previous psychiatric or psychological evaluation or treatment? ☐ No ☐ Yes

If yes, complete the following:

Year	Reason	Medication Used (if any)
_____	_____	_____
_____	_____	_____
_____	_____	_____

2. Have you ever attempted suicide in the past? ☐ No ☐ Yes

If yes, complete the following:

Year	How did you attempt suicide?	What happened?
_____	_____	_____
_____	_____	_____
_____	_____	_____

E. Review of Your Current Health:

1. Do you have? or **Have you ever had?**	**No**	**Yes**		**No**	**Yes**
Lumps anywhere			Unusual excessive thirst		
Double vision or poor vision			Urine problems, blood in urine		
Difficulty hearing			Indigestion, gas, heartburn		
Fainting spells, blackout spells			Stomach pain or stomach ulcer		
Hernia			Groin or Penis Injury		
Sexually Transmitted Disease/HIV			Joint pain		
Convulsion			Diarrhea		
Paralysis			Constipation		
Dizziness			Vomiting, vomiting blood		
Headaches			Blood in stool		
Thyroid problem, goiter			Change in appetite or eating habits		
Skin problem			Trouble sleeping		
Cough or wheeze			Sexual problems		
Chest pain			Weight loss or weight gain		
Spitting up blood			Depression		
Shortness of breath at night or with exercise			Problems with memory, thinking, concentration		
Palpitation or heart fluttering			Suicidal thoughts		
Swelling of hands or feet			Weakness or tiredness		
Visual hallucinations			Other		

Please describe or explain any of the positive answers above

_____

_____

_____

_____

APPENDIX J

Organicity Rating Scale

0 = No pathology found; normal

1 = Some deviation from normal but significance unknown; probably not significant

2 = One or more deviations from normal; might be significant

3 = Deviation probably significant or of sufficient magnitude to be important

4 = Significant deviation which is probably a contributory factor in erectile disorder

5 = Definite evidence of pathology directly related to erectile disorder

EXAMPLES:

5 = Marked atherosclerosis with decreased penile flow and no bc reflex

4 = Decreased penile flow; many medications

3 = Atherosclerosis and hypertension

2 = Some medications

1 = Overweight, hypertensive, endomorph

APPENDIX K

Psychogenic Rating Scale

0 = Psychogenic factors do not appear to be involved (i.e., no psychogenic factors found or possible presence of one or two minor factors that have no temporal or other relationship to problem onset).

1 = Psychogenic factors are probably not significant or significance is unknown (i.e., one or more minor factors coupled with positive sexual functioning factors).

2 = Psychogenic factors might be significant (i.e., multiple minor factors, with at least one showing clear temporal or other relationship to problem onset; or presence of one major factor that doesn't clearly relate to onset coupled with numerous positive functioning factors).

3 = Psychogenic factors are probably significant or of sufficient magnitude to be important (i.e., presence of one clear major factor that doesn't directly relate to problem onset; not a significant number of positive functioning factors).

4 = Psychogenic factors are significant and probably at least a contributing factor (i.e., presence of one major or many minor factors that either don't directly relate to problem onset or are in the presence of positive functioning factors that directly lessen their impact).

5 = Psychogenic factors are definitely involved and appear to be the causative or maintaining factor in the dysfunction (i.e., presence of a clear major contributing factor with no positive sexual functioning factors that would directly lessen this; a clear relationship of the major factor to problem onset).