FEMALE SEXUAL AROUSAL DISORDER SUBTYPES:
DIFFERENTIATION VIA LABORATORY-INDUCED HYPERVENTILATION

by

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Abstract

The effects of heightened sympathetic nervous system (SNS) activity via laboratory-induced hyperventilation (LIH) on subjective and physiological sexual arousal were examined in a group of women with undifferentiated Female Sexual Arousal Disorder (FSAD), as well as across subtypes of FSAD, in comparison to a control group of women without sexual difficulties. Participants were asked to take part in LIH immediately prior to viewing one of two videos containing erotic stimuli. Physiological arousal was measured via the vaginal photoplethysmograph and subjective arousal was measured via self-report questionnaires. LIH differentiated women with FSAD from controls, with LIH increasing physiological arousal in the control group but having no significant effect in the undifferentiated FSAD group. However, when subtypes of FSAD were examined, LIH differentiated women with genital and subjective subtypes of FSAD from women with combined FSAD and women without sexual difficulties. Results have implications for the conceptualization, diagnosis, and treatment of FSAD.
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Chapter 1: Introduction

Masters and Johnson’s proposed human sexual response cycle conceptualized the stages of the cycle – excitement (now termed ‘arousal’), plateau, orgasm, and resolution – in terms of physiological changes (Masters & Johnson, 1966). This conceptualization has carried on to the current classification system of sexual dysfunctions. Both the current versions of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR; American Psychiatric Association, 2000) and the International Classification of Diseases (ICD-10; World Health Organization, 1999) define the majority of the sexual dysfunctions solely in terms of physiological difficulties.

Female Sexual Arousal Disorder (FSAD) is a sexual dysfunction which occurs in approximately 12 – 21% of women in the United States (Fugl-Meyer & Sjogren Fugl-Meyer, 1999; Laumann, Paik, & Rosen, 1999), with even higher prevalence rates found in Asia and Southeast Asia (Laumann, Nicolosi, Glasser, Paik, Gingell, Moreira, et al., 2005). This disorder, defined according to both the DSM-IV-TR (American Psychiatric Association, 2000) and the ICD-10 (World Health Organization, 1999) as “persistent or recurrent inability to attain, or to maintain until completion of the sexual activity an adequate lubrication, swelling response of sexual excitement,” has further been estimated to occur in as many as 75% of women seeking routine care in the gynecologic setting (Nusbaum, Gamble, Skinner, & Heiman, 2000).

However, the current definition and classification of FSAD has recently led to dissatisfaction among clinicians as it stands in contrast with the difficulties that many women actually present within the clinical setting (Basson, 2001b; 2002a; 2002b). Specifically, the current definition lacks any mention or recognition of the more subjective aspects of sexual arousal. While some women presenting to clinicians with sexual arousal difficulties do complain of a lack of (or decrease in) lubrication or genital swelling, particularly among women with
diabetes, in women after pelvic surgeries, and in women with spinal cord injuries (Schreiner-Engel, Schiavi, Vietorisz, & Smith, 1987; Sipski, Alexander, & Rosen, 2001; West, Vinikoor, & Zolnour, 2004), more common is the complaint of not feeling 'subjectively' aroused.

Paralleling what is seen in the clinical setting, a differentiation between physiological and subjective sexual arousal in women has also become apparent in laboratory research over the last several decades. Research studies employing physiological measures of arousal (most commonly the vaginal photoplethysmograph) as well as subjective measures of arousal (most commonly self-report questionnaires) have often found discordance (also termed 'desynchrony') between subjective and physiological indices of arousal when a woman is exposed to arousing stimuli in the laboratory setting. While a few studies have found a positive correlation between the two indices, the majority have found either a negative relationship or, more commonly, no relationship at all (Laan & Everaerd, 1995; Laan, Everaerd, van der Velde, & Geer, 1995; Morokoff & Heiman, 1980; Palace & Gorzalka, 1992; Rosen & Beck, 1988; Steinman, Wincze, Sakheim, Barlow, & Mavissakalian, 1981; Wincze, Hoon, & Hoon, 1976). There is also evidence that type of statistical approach, such as using within subjects analyses (Merrit, Janssen, & Graham, 2001) or using Hierarchical Linear Regression (Rellini, McCall, & Meston, 2005) significantly affects the degree of synchrony between subjective and physiological measures. Pharmacological agents given to enhance physiological arousal in women have similarly not produced significant increases in subjective arousal (Laan, van Lunsen, Everaerd, Riley, Scott, & Boolell, 2002; Meston & Heiman, 1998; Meston & Worcel, 2002). This stands in contrast to the consistent, positive correlation between physiological and subjective measures of arousal seen in men in which genital responses correspond to subjectively stated arousal including gender and

The growing dissatisfaction around the lack of inclusion of subjective arousal in the DSM-IV-TR (American Psychiatric Association, 2000), based on both the above mentioned research findings of desynchrony as well as the sexual arousal difficulties expressed by women presenting to clinicians, resulted in two consensus conferences made up of panels of international experts in the field of female sexuality to discuss issues with the current nosology of female sexual dysfunctions (Basson, Berman, Burnett, Derogatis, Ferguson, Fourcroy, et al., 2000; Basson, Leiblum, Brotto, Derogatis, Fourcroy, Fugl-Meyer, et al., 2003). These consensus conferences led, among other things, to a new proposed definition and classification of FSAD. Rather than classifying FSAD solely on the basis of physiological arousal impairments, the new classification system called for the delineation of three specific subtypes of FSAD. The first subtype, Genital Sexual Arousal Disorder, refers to the current DSM-IV-TR (American Psychiatric Association, 2000) and ICD-10 (World Health Organization, 1999) definitions of FSAD in which there is absent or impaired physiological arousal (in the form of minimal lubrication or swelling) but unimpaired subjective arousal. The second subtype, Subjective Sexual Arousal Disorder, refers to an absence or marked decrease in subjective feelings of sexual excitement and pleasure in response to sexual stimulation but genital lubrication or swelling remain intact. The third subtype, Combined Sexual Arousal Disorder, refers to absent or markedly diminished subjective and physiological arousal in response to sexual stimulation. Although these new definitions are not officially recognized by the DSM classification system, they are recognized in sexual medicine to be an improved set of criteria that are gaining widespread implementation in the clinical and research settings (Basson, Leiblum, Brotto,
Derogatis, Fourcroy, Fugl-Meyer, et al., 2004). Epidemiological data on the prevalence of the different subtypes does not exist, and data collection is only beginning in an attempt to understand the pathophysiology and characteristics of these separate subtypes.

Brotto, Basson, and Gorzalka (2004) conducted the only study to date providing empirical evidence for these three FSAD subtypes. They found that when women with the three subtypes of FSAD were combined into a single, undifferentiated FSAD group and compared to women with no sexual difficulties (the control group), no differences in either physiological or subjective arousal emerged when women were exposed to audiovisual erotic stimuli in a laboratory. The findings were more complex when subtypes of FSAD were differentiated. Specifically, women reporting arousal difficulties congruent with the Genital Sexual Arousal Disorder subtype showed impaired physiological arousal, while those with the Subjective and Combined Sexual Arousal Disorder subtypes showed similar physiological arousal profiles to the control group. Further, in looking at differences in perceived genital arousal, they found that those with Genital and Combined Sexual Arousal Disorder had lower scores than women in the Subjective Arousal Disorder and control groups.

The delineation of these clinical subtypes has important implications for both research and treatment of FSAD. The fact that Brotto et al. (2004) found no differences between the undifferentiated FSAD and the control group, but did find differences when the subtypes were examined suggests that collapsing FSAD subtypes into one heterogeneous group could result in inaccurate and misleading findings. Moreover, it is likely that the different subtypes will have different etiologies, different pathophysologies, and thus, require different treatment approaches. There is evidence from prior research of this potential undesirable outcome in that past research on interventions for female sexual dysfunction tended to group women with heterogeneous
complaints into one large group (e.g., Morokoff & Heiman, 1980; Palace & Gorzalka, 1992; Wincze, Hoon, & Hoon, 1976) and it is only recently that studies have begun to recognize that the qualitative differences between the various sexual dysfunctions require that they each be examined separately.

It is possible that inappropriate differentiation of sexual dysfunction subtypes is responsible for the disappointing results of the effects of various interventions investigated in women with FSAD. Although there are no published investigations on the effects of psychotherapy for FSAD, pharmacotherapy trials for this condition have been inconclusive. For example, with regard to the effects of sildenafil citrate (Viagra) on FSAD, a study by Basson, McInnes, Smith, Hodgson, & Koppiker (2002) found this vasoactive agent to be completely ineffective. However, women in this study comprised a heterogeneous group of women with different sexual complaints. Basson and Brotto (2003) conducted a subsequent study on the effects of sildenafil in a more diagnostically homogeneous group of women with Genital FSAD but again found that sildenafil improved neither arousal nor orgasm. However, when Basson and Brotto (2003) separated those with low physiological arousal levels in response to an erotic film from those with high physiological arousal levels (in essence, separating the group by physiological profile which may, in fact, reflect the subtypes of FSAD found by Brotto et al. [2004]), significant differences emerged. Specifically, those with low physiological arousal levels exhibited a positive response to sildenafil citrate (Brotto & Basson, 2003).

Research over the past decade on the effects of the sympathetic nervous system (SNS) suggest that it is a promising approach for the treatment of sexual concerns. In women with no sexual difficulties, there is an accumulating body of literature supporting the role of SNS activation on sexual arousal. For example, plasma norepinephrine has been shown to correlate
with increases in female sexual arousal, reaching a peak during orgasm (Exton, Bindert, Kruger, Scheller, Hartmann, & Schedlowski, 1999). In studies that have employed the vaginal photoplethysmograph, there are facilitatory effects on female sexual arousal of both pharmacological agents (Meston & Heiman, 1998; Rosen, Phillips, Gendrano, & Ferguson, 1999; Rubio-Aurioles, Lopez, Lipezker, Lara, Ramirez, Rampazzo, et al., 2002) and non-pharmacological interventions (Brotto & Gorzalka, 2002; Meston & Gorzalka, 1995; 1996a; 1996b; Palace & Gorzalka, 1990) which increase SNS activity. Meston, Gorzalka, and Wright (1997) found that clonidine, a selective $\alpha_2$-adrenergic agonist which inhibits SNS activity, inhibited genital arousal. The photoplethysmograph has been shown to be sensitive to the vasocongestive effects of these treatments, even in cases where subjective sexual arousal is not significantly affected (e.g., Meston & Heiman, 1998; Meston & Gorzalka, 1995; 1996a; 1996b; Meston & Worcel, 2002; Palace & Gorzalka, 1990).

In the few studies which have looked at the effects of SNS activation in women with sexual difficulties, the results parallel those with sildenafil citrate: studies have involved heterogenous groups of women with varying sexual difficulties and the results have been inconsistent. For example, Palace and Gorzalka (1990) found that Vaginal Blood Volume (VBV), but not Vaginal Pulse Amplitude (VPA), significantly increased after visual sexual stimulation following exposure to an anxiety-eliciting film in a sample of women with mixed sexual dysfunction. They hypothesized that anxiety-eliciting film stimuli, by facilitating SNS activity, may play a role in restoring genital arousal in women with sexual dysfunction. However, their inclusion of four different types of sexual dysfunction renders the findings inconclusive.
In contrast, the only study to differentiate sexual dysfunction subtypes found differential effects of SNS activation. Using physical exercise as a method of eliciting SNS activity, Meston and Gorzalka (1996a) found that sexually healthy women and those with sexual complaints that did not include orgasmic difficulty responded with increased VPA after the exercise manipulation whereas anorgasmic women had an impaired VPA. These authors speculated that impairments in autonomic nervous system functioning may play an etiological role in female orgasmic disorders (Meston & Gorzalka, 1996a).

There have been three studies that have investigated SNS-enhancing agents specifically in women with FSAD. A small pilot study found support for a facilitatory effect of phentolamine mesylate, a non-selective α1- and α2-adrenergic antagonist, on VPA in women with subjective and physiological arousal dysfunction (Rosen et al., 1999). In a larger sample of women with FSAD, Rubio-Aurioles et al. found a significant facilitatory effect of phentolamine mesylate on VPA in postmenopausal women receiving hormone replacement (Rubio-Aurioles et al., 2002). However, Meston and Worcel failed to find an effect of the α2-adrenergic antagonist, yohimbine, on genital or subjective sexual arousal in a sample of women with specifically genital FSAD, but when yohimbine was combined with the vasodilator, L-arginine glutamate, this SNS-enhancing drug significantly facilitated VPA (Meston & Worcel, 2002). Given that there was no group receiving L-arginine glutamate only, and that a mixed sample of women both receiving and not receiving hormone replacement were included, it is difficult to make implications with respect to the role of the SNS in this study.

Taken together, these studies point to the need for a further delineation of FSAD subtypes in order to reconcile the contradictory findings. Based on the literature that (1) heightened SNS activity significantly facilitates genital arousal in sexually healthy premenopausal women (Brotto
& Gorzalka, 2002; Meston & Gorzalka, 1995; 1996b) and in premenopausal women with low
desire (Meston & Gorzalka, 1996a), (2) pharmacotherapeutic agents which enhance SNS activity
increase genital arousal in women with FSAD (Rosen et al., 1999; Rubio-Aurioles et al., 2002),
and (3) agents which decrease SNS activity result in inhibited genital arousal in sexually healthy
women (Meston et al., 1997), there appears to be support for exploring the effects of heightened
SNS activity on genital arousal in women with different subtypes of FSAD. Therefore, this
study is designed to examine the effects of heightened SNS activity in both an undifferentiated
group of women with FSAD, as well as across the different subtypes of FSAD in comparison to
a normal control group in order to clarify the prior inconsistencies in the literature.

Laboratory-induced hyperventilation (LIH), a technique that reliably facilitates
sympathetic nervous system activity (George et al., 1989; Olsen et al., 1998; St. Croix et al.,
1999) will be used to enhance SNS activity. It has previously been found to significantly
facilitate genital sexual arousal in sexually healthy premenopausal women (Brotto & Gorzalka,
2002).
Chapter 2: Method

Participants

One hundred and two women between the ages of 18-45 participated in this experiment. All participants were recruited through two separate advertisements (one recruiting women with, and the other recruiting women without, sexual difficulties) posted throughout a university and community, and in the local newspapers of a large metropolitan city in the Northwest.

A detailed telephone screen was used to assess interested participants for eligibility. Specifically, women were assessed either for a complete lack of sexual difficulties (sexually healthy control group), or for sexual difficulties meeting the FSAD subtype criteria discussed above and proposed and defined by Basson, Leiblum, et al. (2003). Forty-two women met criteria for the sexually healthy control group while the remaining 60 women met criteria for one of the FSAD subtypes, resulting in 16 women with Genital Sexual Arousal Disorder (absent or impaired genital sexual arousal although subjective sexual excitement still occurs from non-genital sexual stimuli), 16 women with Subjective Sexual Arousal Disorder (absence of, or markedly diminished, feelings of sexual arousal from any type of sexual stimulation, although vaginal lubrication or other signs of physical response still occur), and 28 women with Combined Genital and Subjective Arousal Disorder (absence of, or markedly diminished, feelings of sexual arousal from any type of sexual stimulation in addition to complaints of absent or impaired genital sexual arousal). These classifications into FSAD subtypes or the control group were later validated in person with a clinical interview at the beginning of the first session. Only women who reported that their arousal difficulties were acquired (i.e., had a new onset) and generalized (i.e., present regardless of contextual setting or menstrual phase) were included.
Many women also indicated orgasmic difficulties due to their low or absent arousal levels and this is consistent with what is seen in the clinical setting.

Participants were also screened for a number of exclusion criteria. These included current use of medications or presence of any medical conditions known to affect sexual functioning (such as antidepressants, anti-hypertensive medications, diabetes, and hypertension). In addition, women were excluded if they self-reported any current, untreated psychopathology, were surgically or naturally menopausal, were not exclusively heterosexual, and had never had sexual intercourse.

Demographic characteristics were similar across participants in both the control group and the group of women with sexual difficulties. The mean age of women in the control group was 23.74 ($SD = 5.37$) and the mean age of women reporting sexual arousal difficulties was 26.32 ($SD = 7.34$). The majority of participants in both groups were Caucasian, (control = 61.9%; FSAD = 75.0%) and single (control = 81.0%; FSAD = 71.7%). The mean number of years of education in both groups was approximately 2 years of post-secondary education (control = 14.63 total mean years of education, $SD = 1.86$; FSAD = 14.97 total mean years of education, $SD = 1.46$). There were no significant differences in these demographic variables between the control group and women with FSAD.

**Apparatus and Materials**

*Film Stimuli.* Film stimuli consisted of two 7-minute films which included a 1-minute display of the word “relax” followed by a 3-minute video clip containing neutral material (either a clip from a documentary about Stonehenge or glaciers), followed by a 3-minute video clip containing erotic material involving a nude, heterosexual couple engaging in foreplay and sexual intercourse. The content of both erotic video clips was matched on the number, order, type, and
duration of sexual activities and has previously been found to reliably increase genital and subjective sexual arousal in women (Brotto & Basson, 2003). The different segments of the films were professionally spliced together to form one continuous, videotape with audio accompaniment.

*Vaginal Photoplethysmograph.* The vaginal photoplethysmograph (Sintchak & Geer, 1975) was used to measure physiological sexual arousal. The vaginal photoplethysmograph is a small, tampon-shaped device which is self-inserted into the vagina. The device measures vaginal vasocongestion, which is an indirect measure of sexual arousal. In this study we used Vaginal Pulse Amplitude (VPA) rather than Vaginal Blood Volume (VBV), as VPA has been shown to be a more sensitive measure of sexual arousal (Laan et al., 1995).

The photoplethysmograph was turned on 30 minutes prior to use in order to minimize potential light history and temperature sensitivity effects. In addition, once inserted a 5-minute adaptation period occurred prior to onset of the experimental stimuli. Psychophysiological data were continuously recorded during presentation of the film clips using Acqknowledge III, Version 3.5 (BIOPAC Systems Inc., Santa Barbara, CA), a Model MP100WSW data acquisition unit (BIOPAC Systems Inc.), and an HP Vectra Celeron personal computer. As in similar past research (e.g., Brotto et al., 2004; Brotto & Gorzalka, 2002), data were analyzed in 30-second segments and then averaged separately over the neutral and erotic film segments in order to derive two data points per participant in each of the no-LIH and LIH conditions.

*Subjective Measurement of Arousal.* Before and after each of the film sequences, a subjective measurement of arousal was taken via a one-page self-report questionnaire containing 33-items. This scale is adapted from Heiman and Rowland (1983) and assesses six domains: mental arousal (1 item), perceptions of physical arousal (4 items), autonomic arousal (5 items),
anxiety (1 item), and positive and negative affect (11 items each). Items are rated on a 7-point Likert scale from not at all (1) to intensely (7). The scale has been found to be a valid and sensitive measure of emotional reactions to erotic stimuli (Heiman, 1980; Heiman & Rowland, 1983; Heiman & Hatch, 1980).

**Clinical Interview.** In order to verify the classification of participants into the control and FSAD subtype groups made during the telephone screen, a detailed semi-structured interview assessing sexual arousal was conducted during the first session. We used criteria from the new definitions to verify correct subtyping of women with FSAD (Basson et al., 2003). Women were also administered the detailed assessment of sexual arousal (Basson & Brotto, 2003) and this instrument has been found to reliably differentiate FSAD subtypes (Brotto & Basson, 2003). Scores are recorded on a 7-point Likert-scale and derived by summing responses in each section and dividing by the number of items endorsed.

**Self-Report Measures.** A battery of self-report measures was given to participants assessing a number of domains related to sexual functioning and mood. The Derogatis Sexual Functioning Inventory (DSFI; Derogatis & Melisaratos, 1979) was administered to assess different aspects of current sexual functioning. This measure is comprised of ten distinct subtests including one diagnostic subtest, the Brief Symptoms Inventory (BSI) which is an independent measure of psychopathology. A total Sexual Functioning Index (SFI) score is derived by summing the eight subscale scores. This index reflects the overall quality of current sexual functioning. In addition, a single-item Global Sexual Satisfaction Index (GSSI) provides information on the respondent’s self-reported quality of sexual functioning. Higher scores on this measure indicate increased sexual functioning. The DSFI has been shown to be a valid and
reliable measure for differentiating sexually functional and dysfunctional women (Derogatis & Melisaratos, 1979; Derogatis & Meyer, 1979).

The Female Sexual Functioning Index (FSFI; Rosen, Brown, Heiman, Leiblum, Meston, Shabsigh, et al., 2000), is a 19-item measure assessing desire, subjective arousal, lubrication, orgasm, satisfaction, and pain during sexual activity over the past month. As with the DSFI, higher scores on this measure indicate higher levels of sexual functioning. The FSFI has been shown to be a valid measure for differentiating women with FSAD from sexually healthy women (Rosen et al., 2000).

The Beck Anxiety Inventory (BAI; Beck, Epstein, Brown, & Steer, 1988) is a commonly used, 21-item self-report measure of the extent to which one has been experiencing symptoms of anxiety in the past week. Higher scores on this measure indicate higher levels of anxiety. The measure was included in order to assess levels of anxiety in participants, particularly to confirm that women experiencing sexual difficulties were not significantly more anxious than women in the sexually healthy control group.

Similarly, the Fear of Negative Evaluation questionnaire (FNE; Watson & Friend, 1969) was administered as a measure of social-evaluative anxiety to try to tap into the extent to which participants may be inclined to respond in a certain fashion out of a fear of negative evaluation. The measure is a 30-item true/false questionnaire.

These self-report questionnaires (DSFI, FSFI, BAI, FNE), as well as the subjective measurements of arousal, were incorporated into the protocol part-way into the study. As a result, only a subsample of the total sample completed these measures ($N = 44$; sexually healthy control group $n = 12$; Genital FSAD $n = 9$; Subjective FSAD $n = 8$; Combined FSAD $n = 14$).

Procedure
Each woman participated in two 1-hour sessions which were identical for control and FSAD participants. A between-within repeated measures design allowed for the comparison between FSAD and control groups, between baseline and heightened SNS conditions, and also within groups to compare the effects of the erotic stimulus with a neutral stimulus.

The sessions were conducted by one of three female researchers. The first session began with a researcher restating the procedures of the study, orienting the participant to the laboratory and the laboratory equipment, obtaining written consent, and answering any questions about the study protocol. Following this, the clinical interview was conducted to verify the original FSAD classification derived from the telephone screen. Finally, participants were left alone to complete the battery of self-report questionnaires assessing demographics, current sexual functioning, and mood.

The second session was booked approximately one week after the first session. In the second session, participants were seated comfortably in a reclining chair in the privacy of an internally-locked room adjacent to the experimenter’s room, and asked to insert the vaginal photoplethysmograph with the aid of diagrammed instructions, after the female researcher had left the room. Participants remained fully clothed and were able to cover themselves with a light blanket at all times. Standard placement of the vaginal probe was used such that it was unnoticed by the participant. The plethysmograph was sterilized in Cidex (long-life activated dialdehyde solution) between uses.

Once the plethysmograph was comfortably in place, participants were asked to sit back quietly in order to allow for a 5-minute adaptation period. Following this, participants were shown the first of two videos, presented in a randomized, counterbalanced fashion on a colour television monitor positioned where subjects could sit comfortably in a recliner with full view of
the screen. As described above, videos began with the word “relax” presented on the screen for one minute, followed by 3 minutes of neutral footage and then 3 minutes of erotic material. During presentation of the films, physiological arousal was measured with the photoplethysmograph. Between presentation of the two videos, participants were given a 15-minute rest period in order to allow arousal levels to return to baseline. Before and after each video, participants completed the one-page questionnaires measuring their subjective assessments of arousal. Participants were randomized to receive LIH prior to watching either the first or the second set of film stimuli.

The LIH protocol employed in the current experiment was identical to that used by Brotto and Gorzalka (2002), and involved two minutes of rapid, deep breathing at a rate of 30 breaths/minute. Subjects breathed along with a pre-recorded audiocassette of paced respiration and were asked to breathe in and out as deeply as possible. The female researcher remained in the room with the participant during the LIH procedure to ensure similar breathing across participants, and left the room prior to the onset of the film. Apart from when the researcher was in the room with the participant to complete the LIH, all communication throughout the second session was carried out over a voice-activated intercom between the participant’s and experimenter’s room.

At the completion of the second session, participants were debriefed and informed of the study hypotheses. All participants were paid a $40.00 honorarium and, additionally, were given a copy of their personal psychophysiological assessment with an accompanying description of the findings. All procedures were approved by the university’s Clinical Research Ethics Board.

Data analyses
All analyses were conducted initially between control versus the three combined FSAD groups (undifferentiated FSAD), and subsequently the three FSAD groups were compared to each other and to the control group. Analyses of variance for repeated measures were used for these comparisons to examine the effects of the erotic stimulus, diagnostic group, and heightened SNS activity on physiological and subjective sexual arousal. Simple effects analyses were conducted in cases of a significant interaction. Within group follow-up analyses were conducted for psychophysiological data given the lack of an absolute metric in this instrument. Between group follow-up analyses were run at each condition on self-report data. Cohen's $d$ was calculated as a measure of effect size. Pearson product moment correlation coefficients were used to investigate the degree of synchrony between physiological and subjective sexual arousal during baseline and heightened SNS conditions across groups of women. Correlations were assessed by correlating percent increase scores in VPA from neutral to erotic conditions with difference scores in subjective arousal from neutral to erotic conditions. Difference scores for subjective measures were computed by subtracting neutral values from erotic values for each subjective measure. Percent increase scores for VPA, calculated by subtracting the mean VPA response during the neutral film from the mean VPA response during the erotic film, dividing by the mean VPA response from the neutral film, and then multiplying by 100%, were used to examine the effects of heightened SNS activity. In all cases a $p$ level of less than .05 was deemed statistically significant.
Chapter 3: Results

Sexual and Affective Characteristics of the Samples via Questionnaire Data

Scores from the DSFI, FSFI, BAI, and FNE were analyzed in order to compare differences between women with (FSAD) and without (control) sexual dysfunction on measures of current sexual functioning and to ensure the absence of significant differences on measures of anxiety and fear of negative evaluation. Table 3.1 shows that, on the DSFI, women with sexual dysfunction attained significantly lower GSSI and Sexual Satisfaction scores than women in the control group. Specifically, women with sexual dysfunction were significantly less satisfied with their sexual behavior than women without sexual dysfunction. In addition, women with sexual dysfunction endorsed feeling significantly less positive emotions on the Affect subscale. Finally, scores on the Drive subscale were marginally significant, again showing women with sexual dysfunction as indicating a lower sexual drive than women without sexual dysfunction. Scores on the Information, Experience, Attitude, Symptoms, Fantasy, and Body Image subscales were not significantly different between groups, indicating that participants had similar knowledge of sexual functioning, ranges of sexual experiences, attitudes toward sexuality, self-reported levels of psychopathology, sexual fantasies, and body images, irrespective of sexual function status.

Table 3.2 shows the comparison of scores from the FSFI between FSAD and control participants. Significant differences between the two groups emerged on the Desire, Arousal, Lubrication, and Orgasm subscales, as well as the Full Scale FSFI score, with women with sexual dysfunction indicating lower scores (i.e., more sexual difficulties) on each of these indices. There were no significant differences on the Satisfaction and Pain subscales between the two groups.
In BAI scores, a significant difference was found between FSAD ($M = 11.52$, $SD = 8.28$) and control ($M = 4.62$, $SD = 3.20$) participants, $F(1,42) = 8.40, p < .05$, with FSAD participants showing significantly higher levels of anxiety. No significant difference was found between the FSAD ($M = 16.90$, $SD = 7.87$) and control ($M = 13.31$, $SD = 8.08$) groups on the FNE, $F(1,42) = 1.88, p > .05$.

Effects of Erotic Stimuli and FSAD on Physiological Sexual Arousal During No Sympathetic Nervous System Activity Condition

There was a significant main effect of film, $F(1,100) = 30.59, p < .001, d = 0.53$, indicating that, as a whole group, women displayed increased VPA after presentation of the erotic film during the no SNS activity condition, as illustrated in Figure 3.1. The interaction between group (control vs. undifferentiated FSAD) and film (neutral vs. erotic) was not significant for VPA, $F(1,100) = 2.99, p > .05$. Group main effects were not analyzed because of the absence of an absolute metric in photoplethysmography, thus necessitating within-subject and interaction analyses only.

Subsequently, analyses were repeated looking at the different FSAD subtypes in comparison to sexually healthy women. There was a significant interaction between FSAD subtype and film, $F(3,98) = 4.51, p < .01$, indicating that the percent increase in VPA between neutral and erotic stimulus conditions varied by group, as indicated in Figure 2. Follow up simple effects analyses revealed that women with Subjective Sexual Arousal Disorder had the largest increase in VPA response from neutral to erotic stimulus conditions compared to the
other three groups ($p < .05$, Control Group $d = 0.38$; Genital FSAD $d = 0.39$; Subjective FSAD $d = 0.85$; Combined FSAD $d = 0.69$), as illustrated in Figure 3.2.

[INSERT FIGURE 3.2 ABOUT HERE]

Effects of Erotic Stimuli and FSAD on Self-Report Measures During the No Sympathetic Nervous System Activity Condition

Self-report data during the no sympathetic nervous system condition are presented in Figure 3.3. The main effect of film was significant for autonomic arousal, $F(1,42) = 19.58, p < .001, d = 0.83$; perception of genital arousal, $F(1,42) = 38.73, p < .001, d = 1.23$; mental sexual arousal, $F(1,42) = 32.94, p < .001, d = 0.92$; and positive affect, $F(1,42) = 23.06, p < .001, d = 0.80$. As illustrated in Figure 3, each of these self-report scores increased in response to the erotic stimulus. The main effect of film on anxiety, $F(1,42) = 1.68, p > .05, d = 0.26$; and negative affect, $F(1,42) = 1.39, p > .05, d = 0.06$; was not significant. No significant interactions between film and FSAD emerged. Identical analyses repeated to look at the different FSAD subtypes in comparison to sexually healthy control women similarly showed no interaction between film and FSAD subtypes.

[INSERT FIGURE 3.3 ABOUT HERE.]

Effects of Heightened Sympathetic Nervous System Activity on Physiological Sexual Arousal

There was a significant interaction between sympathetic nervous system activity (baseline and heightened conditions) and group (control and undifferentiated FSAD) on VPA, $F(1,100) = 7.57, p < .01$, as illustrated in Figure 3.4. Planned dependent t-tests run on each group revealed that LIH significantly increased VPA in the control group, $t(41) = -2.418, p < .05, d = 0.45$, but not in the undifferentiated FSAD group, $t(59) = 1.005, p > .05, d = 0.15$.

[INSERT FIGURE 3.4 ABOUT HERE]
Reanalysis of these data employing the separate subtypes of FSAD with repeated measures analysis of variance also revealed a significant SNS by FSAD subtype interaction, $F(3, 98) = 5.60, p < .01$, as shown in Figure 3.5. Subsequent simple effects analyses performed on each FSAD subtype, across baseline and heightened SNS conditions, revealed significant increases in VPA percent change in the control, $p < .05, d = 0.44$, and the Combined Sexual Arousal Disorder groups, $p < .05, d = 0.49$, and a significant reduction in VPA percent change among women with Genital, $p < .05, d = 0.56$ and Subjective Sexual Arousal Disorder, $p < .05, d = 0.47$.

[INSERT FIGURE 3.5 ABOUT HERE]

**Effects of heightened sympathetic nervous system activity on self-report measures**

The main effect of group (undifferentiated FSAD versus control group) was not significant for any of the self-report measures including: autonomic arousal, $F(1,42) = 0.41, d = 0.21$; perception of genital arousal, $F(1,42) = 0.14, d = 0.13$; mental sexual arousal, $F(1,42) = 1.02, d = 0.36$; anxiety, $F(1,42) = 3.03, d = 0.47$; positive affect, $F(1,42) = 1.11, d = 0.39$; or negative affect, $F(1,42) = 0.17, d = 0.31$, all $p$'s $> .05$ throughout. Similarly, the main effect of heightened SNS activity was not significant for any subjective measure including: autonomic arousal, $F(1,42) = 3.22, d = 0.29$; perception of genital arousal, $F(1,42) = 1.00, d = 0.22$; mental sexual arousal, $F(1,42) = 0.17, d = 0.08$; anxiety, $F(1,42) = 0.09, d = 0.05$; positive affect, $F(1,42) = 0.10, d = 0.01$; or negative affect, $F(1,42) = 0.00, d = 0.16, p > .05$, throughout. There were no significant interactions between FSAD and SNS on any self-report measures; autonomic arousal, $F(1,42) = 0.18$; perception of genital arousal, $F(1,42) = 0.78$; mental sexual arousal, $F(1,42) = 0.00$; anxiety, $F(1,42) = 0.01$; positive affect, $F(1,42) = 0.16$; and negative affect, $F(1,42) = 0.81, p > .05$ throughout, as indicated in Table 3.3.
Reanalysis of the data based on FSAD subtype failed to reveal significant interactions between FSAD subtype and SNS for autonomic arousal, $F(3,40) = 1.41$; perception of genital activity, $F(3,40) = 1.80$; mental sexual arousal, $F(3,40) = 0.16$; anxiety, $F(3,40) = 0.52$; positive affect, $F(3,40) = 0.70$; or negative affect, $F(3,40) = 1.39$, $p > .05$ throughout. Similarly, there were no significant main effects for group or SNS condition.

**Correlations Between Physiological and Subjective Sexual Arousal During the Heightened SNS Activity Condition**

Correlations were conducted on women in the control group and women with undifferentiated FSAD separately. Among sexually healthy women, there was no significant correlation between percent change in VPA and difference in mental sexual arousal between neutral and erotic stimuli ($r = 0.403, p > .05$), or between percent change in VPA and perceptions of genital arousal between neutral and erotic stimuli ($r = 0.399, p > .05$). Genital-subjective correlations were subsequently conducted for women with undifferentiated FSAD. As with sexually healthy women, there was no significant correlation between percent change in VPA and difference in mental sexual arousal between neutral and erotic stimuli ($r = 0.271, p > .05$), or between percent change in VPA and perceptions of genital arousal between neutral and erotic stimuli ($r = 0.165, p > .05$).

Correlations were subsequently analyzed by FSAD subtype. Among women with Genital Sexual Arousal Disorder, correlations between difference in mental sexual arousal and percent change in VPA ($r = 0.294, p > .05$), as well as correlations between difference in perceptions of genital arousal and percent change in VPA ($r = .249, p > .05$) were not statistically significant. Among women with Subjective Sexual Arousal Disorder, the correlations between percent
change in VPA and difference in mental sexual arousal \( (r = 0.644, p > .05) \) and between percent change in VPA and difference in perceptions of genital arousal \( (r = .497, p > .05) \) were similarly not statistically significant. There were also no significant correlations among women with Combined Sexual Arousal Disorder for either difference in mental arousal \( (r = .155, p > .05) \) or difference in perceptions of genital arousal \( (r = .055, p > .05) \) with percent change in VPA.
Table 3.1
Group Differences on Scores from the Derogatis Sexual Functioning Inventory (DSFI)

<table>
<thead>
<tr>
<th>Subtest</th>
<th>DSFI Mean</th>
<th>Undifferentiated Group Mean</th>
<th>Control Group Mean</th>
<th>F(1,30)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Information</td>
<td>49.58</td>
<td>47.85</td>
<td>0.48</td>
<td>.492</td>
<td></td>
</tr>
<tr>
<td>Experience</td>
<td>49.84</td>
<td>50.77</td>
<td>0.12</td>
<td>.736</td>
<td></td>
</tr>
<tr>
<td>Drive</td>
<td>54.51</td>
<td>60.92</td>
<td>4.01</td>
<td>.052</td>
<td></td>
</tr>
<tr>
<td>Attitude</td>
<td>53.48*</td>
<td>53.92</td>
<td>0.02</td>
<td>.888</td>
<td></td>
</tr>
<tr>
<td>Symptoms (BSI)</td>
<td>41.32</td>
<td>46.62</td>
<td>2.19</td>
<td>.146</td>
<td></td>
</tr>
<tr>
<td>Affects*</td>
<td>45.10</td>
<td>53.00</td>
<td>4.23</td>
<td>.046</td>
<td></td>
</tr>
<tr>
<td>Fantasy</td>
<td>53.55</td>
<td>53.15</td>
<td>0.11</td>
<td>.918</td>
<td></td>
</tr>
<tr>
<td>Body Image</td>
<td>35.84</td>
<td>40.69</td>
<td>1.75</td>
<td>.194</td>
<td></td>
</tr>
<tr>
<td>Satisfaction**</td>
<td>40.81</td>
<td>52.54</td>
<td>16.30</td>
<td>.000</td>
<td></td>
</tr>
<tr>
<td>Global Score</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sexual Functioning Index (SFI)</td>
<td>474.35</td>
<td>506.38</td>
<td>3.21</td>
<td>.080</td>
<td></td>
</tr>
<tr>
<td>Global Sexual Satisfaction Index (GSSI)**</td>
<td>4.08</td>
<td>6.28</td>
<td>40.54</td>
<td>.000</td>
<td></td>
</tr>
</tbody>
</table>

Note. Means are based on raw scores that were converted to established percentile rankings (T scores).
BSI = Brief Symptom Inventory
* p < .05
** p < .001
Table 3.2

*Group Differences on Scores from the Female Sexual Functioning Index (FSFI)*

<table>
<thead>
<tr>
<th>Subtest</th>
<th>FSFI Mean</th>
<th>Undifferentiated Group Mean</th>
<th>Control Group Mean</th>
<th>F(1,30)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Desire**</td>
<td>3.23</td>
<td>4.57</td>
<td>14.83</td>
<td>.000</td>
<td></td>
</tr>
<tr>
<td>Arousal**</td>
<td>3.27</td>
<td>4.94</td>
<td>18.20</td>
<td>.000</td>
<td></td>
</tr>
<tr>
<td>Lubrication*</td>
<td>3.92</td>
<td>5.01</td>
<td>6.20</td>
<td>.017</td>
<td></td>
</tr>
<tr>
<td>Orgasm**</td>
<td>2.34</td>
<td>4.74</td>
<td>22.96</td>
<td>.000</td>
<td></td>
</tr>
<tr>
<td>Satisfaction</td>
<td>3.28</td>
<td>4.12</td>
<td>2.55</td>
<td>.118</td>
<td></td>
</tr>
<tr>
<td>Pain</td>
<td>3.93</td>
<td>4.28</td>
<td>0.24</td>
<td>.631</td>
<td></td>
</tr>
<tr>
<td>Full Scale Score**</td>
<td>19.95</td>
<td>27.65</td>
<td>14.86</td>
<td>.000</td>
<td></td>
</tr>
</tbody>
</table>

* p < .05
** p < .001
Table 3.3
Effect of heightened sympathetic nervous system activity on self-report measures of autonomic arousal, perception of genital arousal, mental sexual arousal, anxiety, positive affect, and negative affect, in a control group and in women with undifferentiated FSAD. Data represent mean differences (± standard error).

<table>
<thead>
<tr>
<th></th>
<th>Undifferentiated FSAD Group (N = 31)</th>
<th>Control Group (N = 13)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Autonomic Arousal</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline SNS</td>
<td>3.16 (0.82)</td>
<td>3.69 (1.38)</td>
</tr>
<tr>
<td>Heightened SNS</td>
<td>4.16 (0.73)</td>
<td>5.31 (1.45)</td>
</tr>
<tr>
<td>Perception of Genital Arousal</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline SNS</td>
<td>6.29 (1.09)</td>
<td>6.23 (1.70)</td>
</tr>
<tr>
<td>Heightened SNS</td>
<td>6.39 (1.03)</td>
<td>7.77 (1.55)</td>
</tr>
<tr>
<td>Mental Sexual Arousal</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline SNS</td>
<td>5.35 (1.18)</td>
<td>7.46 (1.99)</td>
</tr>
<tr>
<td>Heightened SNS</td>
<td>5.87 (1.26)</td>
<td>7.85 (2.01)</td>
</tr>
<tr>
<td>Anxiety</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline SNS</td>
<td>-0.44 (0.17)</td>
<td>0.00 (0.32)</td>
</tr>
<tr>
<td>Heightened SNS</td>
<td>-0.48 (0.18)</td>
<td>0.08 (0.18)</td>
</tr>
<tr>
<td>Positive Affect</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline SNS</td>
<td>3.92 (1.16)</td>
<td>6.23 (1.73)</td>
</tr>
<tr>
<td>Heightened SNS</td>
<td>4.00 (1.02)</td>
<td>5.54 (1.99)</td>
</tr>
<tr>
<td>Negative Affect</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline SNS</td>
<td>-0.40 (0.47)</td>
<td>-0.62 (0.71)</td>
</tr>
<tr>
<td>Heightened SNS</td>
<td>-0.97 (0.72)</td>
<td>-0.08 (0.40)</td>
</tr>
</tbody>
</table>
Figure 3.1. Effects of erotic stimulus on vaginal pulse amplitude during baseline sympathetic nervous system activity in a control group (n = 42) and women with undifferentiated FSAD (n = 60). Data represent means (in millivolts) ± standard error of the mean.

*p < .001, significant main effect of erotic film
Figure 3.2. Effects of erotic stimulus on vaginal pulse amplitude (VPA) during the no sympathetic nervous system activation condition in the control group \((n = 42)\), in women with Genital Sexual Arousal Disorder \((n = 16)\), Subjective Sexual Arousal Disorder \((n = 16)\), and women with Combined Sexual Arousal Disorder \((n = 28)\). Data represent the mean percent increase in VPA from the neutral to the erotic stimulus conditions ± standard error of the mean. *\(p < .01\), women with Subjective Sexual Arousal Disorder exhibited a significantly greater increase in VPA than women with Genital and Combined Sexual Arousal Disorder and the Control Group.
Figure 3.3. Effects of erotic stimuli on A) autonomic arousal, B) perception of genital arousal, C) mental sexual arousal, D) anxiety, E) positive affect, and F) negative affect in a control group and women with undifferentiated FSAD during baseline SNS activity. Data represent means ± standard error of the mean.

*p < .001, significant main effect of film (neutral vs. erotic)
Figure 3.4. Effects of laboratory-induced hyperventilation on vaginal pulse amplitude (VPA) percent increase scores (neutral to erotic) in a control group and women with undifferentiated FSAD. Data represent means ± standard error of the mean.

* $p < .05$, significant interaction between group and sympathetic nervous system condition

** $p < .05$, significant effect of LIH
Figure 3.5. Effects of laboratory-induced hyperventilation on vaginal pulse amplitude (VPA) percent increase scores (neutral to erotic) in a control group, in women with Genital Sexual Arousal Disorder, Subjective Sexual Arousal Disorder, and women with Combined Genital and Subjective Sexual Arousal Disorder. Data represent means ± standard error of the mean.

*p < .05, significant effect of laboratory-induced hyperventilation
Chapter 4: Discussion

Overall the findings from this study support a sub-classification of FSAD into Genital, Subjective, and Combined Sexual Arousal Disorder and extend the findings of Brotto, Basson, & Gorzalka (2004) by showing that there are not only differences in physiological profiles across subtypes, but also that these subtypes exhibit differential responses to an intervention. Specifically, the findings suggest that heightened sympathetic nervous system activity effectively facilitates physiological sexual arousal in sexually healthy women as well as in women complaining of Combined Sexual Arousal Disorder, but has a detrimental effect on genital vasocongestion in women with Genital and Subjective Sexual Arousal Disorder.

Significant differences in VPA percent increase from neutral to erotic stimuli were found when the effects of the video stimuli on physiological arousal in the no-LIH condition were examined by FSAD subtype, consistent with the findings of Brotto, Basson, & Gorzalka (2004). However, the findings revealed a different physiological profile than those found by Brotto and colleagues; while their study found that women with Genital Sexual Arousal Disorder did not exhibit a significant percent increase in VPA with erotic film exposure, the current findings showed that all groups experienced a significant percent increase in VPA but that this effect was greatest, and significantly different from the other three groups, in women with Subjective Sexual Arousal Disorder. These findings are interesting and somewhat unexpected as they indicate that women with Genital and Combined Sexual Arousal Disorder, although subjectively complaining of decreased genital arousal, do not show objectively lower levels of genital arousal than sexually healthy (control) women. However, these findings are in line with a recent study which found that women carefully assessed for Genital Sexual Arousal Disorder displayed similar physiological profiles as controls (Laan & van Lunsen, as cited in Brotto, Basson, &
Gorzalka, 2004). As pointed out by Brotto et al. (2004), a lack of objective physiological arousal in the presence of complaints of low arousal may indicate that these women are not attending to a healthy genital response.

With respect to the effects of heightened SNS activity on VPA, sexually healthy women showed enhanced VPA to heightened SNS activity, which is in line with past studies which have found that SNS activity has a facilitatory effect on physiological arousal in women with no sexual difficulties (Brotto & Gorzalka, 2002; Meston & Gorzalka, 1995; 1996a; 1996b; Meston & Heiman, 1998; Meston & Worcel, 2002; Palace & Gorzalka, 1990; 1992; Rosen et al., 2002; Rubio-Aurioles et al., 2002). These findings in sexually healthy women suggest that laboratory-induced hyperventilation, by significantly enhancing SNS activity, potentiates the genital arousal response to erotic stimuli. Heightened SNS activity did not increase VPA overall, but rather exerted its effects only after exposure to the erotic stimulus. This is consistent with the findings of Meston and colleagues (Meston and Gorzalka, 1995; 1996a; 1996b; Meston et al., 1997; Meston and Heiman, 1998) who used acute, intense exercise as a method of enhancing SNS activity. These studies found that exercise significantly facilitated VPA only in the presence of erotic stimuli and had no effect on its own.

In contrast, heightened SNS activity had no effect on physiological arousal in women with undifferentiated FSAD. These results are not surprising, however, given the lack of conclusive findings and inconsistencies in the literature on the effects of SNS activity in women with sexual dysfunction when specific subtypes are not delineated (Meston & Gorzalka, 1996a; Meston & Worcel, 2002; Palace & Gorzalka, 1990; Rosen et al., 2002; Rubio-Aurioles et al., 2002), and in fact lend further support to the rationale of looking at specific subtypes of FSAD in order to reconcile the effects of SNS activity on sexual arousal in women.
Significant differences in VPA in response to heightened SNS activity were found when
the specific subtypes of FSAD were examined. Specifically, women with Combined Sexual
Arousal Disorder exhibited similar results as control participants, showing significant increases
in VPA to the erotic stimulus with heightened SNS activity, while women with Genital and
Subjective Sexual Arousal Disorder displayed the opposite effect; rather than enhancing VPA,
heightened SNS activity appeared to have a negative impact on physiological arousal in response
to erotic stimuli, thereby significantly decreasing VPA in these women. These results are in stark
contrast to the facilitatory effects of the erotic stimuli on arousal during baseline levels of SNS
activity in these women. Heightened SNS activity had no effect on subjective arousal in any of
the groups of women, suggesting that the effects of SNS manipulation occur at a physiological,
and not a cognitive, level.

These contrasting findings among subtypes of FSAD are reminiscent of the findings of
Meston and Gorzalka (1996a) who found that women with orgasmic difficulty experienced a
decreased physiological response to exercise-induced increases in SNS activity, whereas
orgasmic women showed increased physiological patterns similar to the sexually healthy control
and the Subjective Sexual Arousal Disorder groups in the current experiment. These authors
suggested that perhaps the physiological events taking place during heightened SNS activity are
detrimental to the sexual response in anorgasmic women. This explanation may be extended to
the current sample of women with either Genital or Subjective Sexual Arousal Disorder.
Accumulating clinical experience with women complaining of subjective sexual arousal suggests
that this subgroup would not benefit from vasoactive medication designed to increase genital
vasocongestion (Basson, 2002a; 2002b), given that there is no evidence of impaired genital
vasocongestion in this group. The current findings that enhanced SNS activity reduced VPA
supports this view, and implies that agents designed to increase SNS activity may in fact impair genital arousal. Interestingly, the current results also indicate that increasing SNS activity may be unhelpful, and may even result in a further hindrance in arousal, in women complaining of Genital Female Sexual Arousal Disorder.

It is possible that cognitive distraction, related to heightened SNS activity, may contribute to impaired physiological sexual arousal in the Genital and Subjective Sexual Arousal Disorder groups. Excitation transfer theory posits that individuals experiencing heightened activity followed by a sexual stimulus will report significantly lower levels of sexual arousal if they perceive residual effects from the prior activity (Cantor, Zillmann, & Bryant, 1975), and techniques that draw attention away from genital excitement indeed have been shown to significantly reduce the actual level of genital sexual arousal (Sakheim, Barlow, Beck, & Abrahamson, 1984). Barlow's (1986) Cognitive Interference model of sexual dysfunction posits that psychogenic sexual dysfunction may result when a cognitive interference process interacts with anxiety. Instead, sexually healthy subjects may benefit from the arousal-enhancing effects of anxiety or any other technique designed to facilitate SNS activity, since they process and focus on erotic cues without difficulty. Still another possibility, for which there is no empirical evidence as yet, is that the Genital and Subjective Sexual Arousal Disorder subtypes may already be functioning at a higher level of SNS activity than the other FSAD groups and that it is possible that the addition of the SNS manipulation functioned to reduce VPA. According to the Yerkes-Dodson Law of arousal and performance (Yerkes & Dodson, 1908), there is an optimal level of arousal below and above which performance is significantly impaired. If the facilitatory effect of SNS activity on VPA indeed follows such an inverted-U pattern, this would provide
support for this speculation. Future studies should aim to directly assess levels of SNS activity during baseline and following SNS manipulations in order to test this hypothesis.

It is also possible that the current findings are due to an inability of laboratory-induced hyperventilation to evoke sufficient levels of SNS activity in women with Genital and Subjective FSAD that would subsequently facilitate genital sexual arousal. Meston and Gorzalka (1996b) employed intense, acute exercise which has been found to lead to significantly elevated levels of SNS activity for up to 40 minutes following cessation of exercise and to lead to changes in levels of testosterone, cortisol, and prolactin. In an effort to employ a technique that did not preclude the participation of women who are not physically capable of intense exercise for 20 minutes, we used laboratory-induced hyperventilation which has been shown to lead to SNS predominance for 7 minutes (Achenbach-Ng, et al., 1994). It is possible that the use of exercise instead of hyperventilation would have led to different patterns of VPA response in the different FSAD subtypes. It is also possible that a pharmacological intervention (e.g., phentolamine mesylate) would have been more effective at enhancing VPA, though the potential side effects of such a manipulation must be considered.

The lack of statistical significance and effect size in the current study with respect to the effects of enhanced SNS activity on subjective arousal may be attributable to a number of factors. It is plausible that increased SNS activity is not registered at the conscious, subjective level - that participants were unaware of the general physiological changes on the body caused by increased SNS activity as well as the specific effects on genital arousal. It is also plausible that the method of measuring subjective arousal was ineffective. Specifically, the current study used discrete measurements of subjective arousal through self-report questionnaires completed immediately after viewing of the erotic stimuli. This method of measurement was based on the
findings by Steinman, Wincze, Sakheim, Barlow & Mavissakalian (1981) of no significant differences between results obtained by discrete versus continuous subjective measurement. However, since then a study by Rellini et al. (2005) has been published providing new evidence that continuous measurement may in fact be more sensitive than discrete measurement. Finally, the lack of significance due to the small sample sizes cannot be excluded, although the small effect sizes make this conclusion less likely. Nevertheless, future studies should aim to investigate the potential effects of SNS activity on both physiological and subjective measures of arousal in larger samples of women with the various FSAD subtypes.

Synchrony between genital and subjective sexual arousal during the condition of heightened SNS activity was not apparent in either sexually healthy women or women with sexual difficulties. These results are in line with past studies which have found dysynchrony in both sexually healthy women and in women with sexual dysfunction (Laan & Everaerd, 1995; Laan et al., 1995; Morokoff & Heiman, 1980; Palace & Gorzalka, 1990; 1992; Rosen & Beck, 1988; Steinman et al., 1981; Wincze et al., 1976). Numerous explanations have been put forth for the desynchrony seen in women. Palace and Gorzalka (1990) hypothesized that women may not express subjective arousal as a result of social dictates with regard to sexual etiquette that discourage women from attending to, or acknowledging, their arousal. Heiman (1977) postulated that, in contrast to men, women lack an obvious bodily cue (i.e., erection) to give them feedback about their arousal, which can result in discordance between physiological and reported subjective arousal. And Rellini and colleagues suggested that the apparent desynchrony seen in women may in fact be due to methodological and statistical issues which mask the actual concordance between the two indices (Rellini, McCall, Randall, & Meston, 2005).
In contrast, Chivers and Bailey (2005) proposed that physiological and subjective arousal are two separate components of overall sexual arousal. Specifically, based on research in which they found that women - but not men - showed a physiological vasocongestive response to sexual stimuli involving non-human primates (while denying any subjective arousal), they proposed that women’s genital vasocongestion is provoked by exposure to nonspecific sexual features, independent of what those features are. Further, they proposed that this response may be an evolved, protective adaptation which protects women from physical harm or infection by preparing the vagina for sexual activity. In essence, according to this hypothesis women become physiologically aroused in the presence of non-specific sexual stimuli but then make a cognitive appraisal of the stimuli as either subjectively arousing or not.

The finding that women with Combined Sexual Arousal Disorder are more similar to controls than they are to women with Genital or Subjective Arousal Disorder, despite complaining of genital and subjective impairments, suggests that this subtype of FSAD is qualitatively different in some respect and that Combined Sexual Arousal Disorder is not just a sexual dysfunction involving a combination of both Genital Sexual Arousal Disorder and Subjective Sexual Arousal Disorder.

The results from the self-report questionnaires on sexual functioning and psychopathology support the categorization of participants into either sexually healthy (control) or sexual problem (FSAD) groups. As expected, FSAD participants had significantly lower scores on the GSSI and Sexual Satisfaction subtests of the DSFI, indicating that they felt less satisfied with their current sexual functioning than sexually healthy control participants. Significant differences on the DSFI Affect subscale, in which FSAD participants endorsed feeling less positive and more negative emotions than control participants, are in line with
reported decreased satisfaction with sexual function. Lower scores on the DSFI drive subtest in FSAD participants are also in line with decreased sexual functioning and satisfaction. The finding that FSAD and control participants had similar scores on the remaining DSFI subscales (Information, Experience, Attitude, Symptoms, Fantasy, and Body Image) indicates that apart from sexual difficulties, participants were similar in their knowledge, experiences, attitudes, etc. relating to sexual behaviour and also had similarly low rates of self-reported psychopathology. Due to the similarities on these latter subtests between FSAD and control participants, it is not surprising that scores on the SFI, derived by adding scores on the DSFI subtests and generally considered to be a reflection of the overall quality of sexual functioning, were not significantly different between the two groups.

Significant differences were found between FSAD and control participants on the FSFI sexual functioning subscales, with FSAD participants endorsing significantly more difficulties with desire, arousal, lubrication, and orgasm on the FSFI. An unexpected finding was the significant difference between the FSAD and control groups on the BAI. However, although FSAD participants indicated having experienced significantly higher levels of anxiety symptomology in the past week than controls, scores for both groups were in the mild severity range (the lowest range possible) on this measure, indicating that neither group had *clinically* significant levels of anxiety. No significant differences on the FNE were found between the two groups, which is important as significant differences could point to the possibility of one group having higher motivations for responding in a socially desirable manner than the other, potentially bringing into question the validity of the results.

Results pertaining to the effects of the video stimuli on physiological and subjective arousal indicated that the erotic film significantly increased VPA and self-report measures,
including perception of genital activity, mental sexual arousal, autonomic activity, and positive affect, in all participants during the no sympathetic nervous system activity condition. These findings indicate that the film stimuli were in fact effective in increasing both physiological and subjective arousal in all participants, independent of group status, and once again demonstrate that female-made, female-focused erotic stimuli effectively increase sexual arousal and positive affect in women.

The data from this study have important implications for the search for effective treatments for FSAD. The fact that no effect of heightened SNS activity was seen when subtypes of FSAD were combined into one, undifferentiated FSAD group, but that significant differences emerged when subtypes were examined may clarify the past inconsistencies in the literature on the effects of various interventions with FSAD in which subtypes were not delineated, and may lead to a renewed and more successful search for effective interventions for this distressing sexual dysfunction. More specifically, the current results suggest that SNS-enhancing drugs, such as phentolamine mesylate, may be effective specifically for women with Combined FSAD but not women with Genital and Subjective FSAD. However, given that no self-report measure of sexual arousal or affect was influenced by the current SNS manipulation, the current findings suggest that interventions which increase SNS activity will likely only have an affect at the physiological, and not at the cognitive, level. Therefore, it is unclear to what extent these interventions will help women feel subjective improvements in sexual functioning, and in fact, may actually interfere with arousal in women reporting exclusively impairments with subjective arousal. Instead, psychotherapy may be a more effective treatment, either on its own or in combination with a physiological intervention, in increasing subjective feelings of sexual arousal and excitement.
The current findings also have implications for the diagnostic category of FSAD. Considerable research has demonstrated the elusive nature and complexity of FSAD (Basson 2000, 2001a, 2001b, 2002a, 2002b) and has argued against the current DSM-IV-TR classification which necessitates lubrication difficulties in order for a diagnosis of FSAD to be met. The current findings indicate that not only can women with FSAD be subtyped according to genital versus subjective versus combined sexual arousal impairments, but this classification is further supported by their differential responses to heightened SNS activity. Future studies should aim to further elucidate the role of the SNS in the development and maintenance of FSAD and its subtypes.
Chapter 5: References


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