THE ROLE OF THE SYMPATHETIC NERVOUS SYSTEM IN FEMALE SEXUAL AROUSAL

by

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ABSTRACT

In contrast to the long-held assumption that heightened sympathetic nervous system (SNS) activity inhibits sexual arousal in women, Meston and Gorzalka (1995) recently provided evidence for a facilitatory influence of SNS activation on physiological sexual arousal in women. The present investigation was aimed at further elucidating the role of the SNS in female sexual arousal by examining the time course of the effects of SNS activation on sexual arousal in women, by providing the first empirical test of the effects of SNS inhibition on physiological and subjective sexual arousal in women, and by examining the effects of SNS activation on sexual arousal in sexually dysfunctional women.

In Experiment 1, 36 sexually functional women participated in two experimental sessions in which they viewed a neutral film followed by an erotic film. In one of these sessions, subjects were exposed to 20 min of intense exercise prior to viewing the films. Subjective (self-report) and physiological (photoplethysmograph) sexual arousal were measured at either 5 min, 15 min, or 30 min post-exercise. By measuring the effects of exercise on sexual arousal at these time intervals, Experiment 1 allowed for examination of high, moderate, and low levels of SNS activation on sexual responding. Acute exercise marginally decreased vaginal pulse amplitude (VPA) and had no effect on vaginal blood volume (VBV) responses to an erotic film when measured 5 min post-exercise. At 15 min post-exercise, exercise significantly increased VPA and showed a trend toward increasing VBV responses. At 30 min post-exercise, VBV responses to an erotic film were marginally increased and VPA responses showed a trend toward increasing. Acute exercise had no significant effect on subjective perceptions of sexual arousal in any of the experimental conditions. These findings suggest an optimal level of SNS activation for facilitation of physiological sexual arousal in women.

Experiments 2 and 3 were designed to examine the effects of SNS inhibition, via clonidine administration, on sexual arousal in women. In Experiment 2, the effects of SNS inhibition on sexual arousal were examined following experimentally-induced nervous system
arousal. Fifteen sexually functional women engaged in 20 min of intense exercise during each of two experimental sessions. One hour prior to exercise, subjects received either 0.2 mg clonidine or a placebo. Clonidine significantly decreased VPA, VBV, and subjective sexual responses to the erotic films. In Experiment 3, the effects of SNS inhibition on sexual arousal were examined during baseline arousal, i.e., in the absence of acute exercise. Fifteen sexually functional women participated in two experimental sessions in which they received either 0.2 mg clonidine or a placebo one hour prior to viewing the erotic films. Clonidine marginally decreased subjective ratings of sexual arousal but had no significant effect on VPA or VBV responses. The findings from Experiments 2 and 3 argue against the notion that SNS inhibition facilitates the initial stages of sexual arousal in women.

In Experiment 4, the effects of SNS activation (via acute exercise) on sexual arousal were compared between 12 sexually functional women, 12 women with low sexual drive, and 12 women with either primary or secondary anorgasmia. Acute exercise significantly increased VPA and VBV responses to an erotic film among sexually functional women and women with low sexual drive. Among anorgasmic women, exercise marginally decreased VPA while having no effect on VBV responses to an erotic film. Acute exercise had no significant effect on subjective perceptions of sexual arousal among either sexually functional, low sexual drive, or anorgasmic subjects. The results from Experiment 4 replicate and extend the findings of Meston and Gorzalka (1995) to a sample of women with low sexual drive, and suggest an inhibitory influence of SNS activation on sexual arousal in anorgasmic women. These findings provide the first empirical evidence to suggest neurophysiological differences between women with and without orgasmic dysfunction. Together, Experiments 1 to 4 provide evidence for a primarily facilitatory role of SNS activation, and an inhibitory role of SNS inhibition on sexual arousal in women. These results have implications for deriving an etiological theory of sexual dysfunction in women and for developing new methods of treatment for women with sexual difficulties.
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The Role of The Sympathetic Nervous System in Female Sexual Arousal

It is well known that the sympathetic and parasympathetic branches of the autonomic nervous system do not function independently, and that nerve fibers from both systems innervate many bodily systems involved in the sexual response. It is generally assumed, however, that one or the other of these systems becomes dominant during increasing levels of sexual arousal and serves to maintain further arousal and/or orgasm. It remains open to conjecture which of these two systems better facilitates sexual arousal.

For over 30 years, clinicians, researchers, and theorists in the field of human sexuality have worked largely under the assumption that the sympathetic nervous system (SNS) plays an inhibitory role, and the parasympathetic nervous system (PNS) plays a facilitatory role in initiating and maintaining the early stages of sexual arousal. In men, this assumption is based on neurophysiological studies which reveal PNS mediation of the erectile response, and on an abundance of clinical reports which link generalized anxiety (i.e., SNS activation) and erectile failure. In women, however, this assumption is based primarily on analogies that have been drawn between the erectile response in men and the vasocongestive response in women. Direct, empirical evidence for a PNS-mediated sexual response in women has not yet been offered. Determination of the precise role of the SNS in sexual arousal not only has important implications for deriving an etiological theory of sexual dysfunction, but for developing effective treatments for the alleviation of sexual difficulties.

The present investigation follows from a recent study (Meston and Gorzalka, 1995) which found that intense, acute exercise significantly increased physiological sexual arousal in women. The results of this study challenged the long-held notion that activation of the SNS inhibits sexual arousal in women, and suggested that SNS activation may, in fact, play an opposite, facilitatory role in enhancing the initial stages of sexual arousal in women. The present study aims at further elucidating the role of the SNS in female sexual arousal by (1) examining the temporal boundaries by which exercise facilitates physiological sexual arousal
in women, (2) determining whether the facilitatory effects of exercise on sexual arousal can be attributed to enhanced SNS activity, (3) determining the effects of SNS activation on sexual arousal among sexually dysfunctional women, and (4) providing the first empirical examination of the effects of SNS inhibition on physiological and subjective sexual arousal in women.

Activation of the Sympathetic Nervous System Inhibits Sexual Arousal

Indirect evidence for an inhibitory role of the SNS in sexual arousal is provided by early behavioral observations of the physiological changes which occur during the sexual act. Based on the observation of a higher relative number of PNS versus SNS-mediated physiologic elements reportedly involved in the sexual response (e.g., increased perspiration, salivary secretion, vasodilation), Kinsey, Pomeroy, Martin, and Gebhard (1953) concluded that the sexual response is more dependent on PNS than SNS activity. Early research conducted by Wolpe (1958) led to the same conclusion. In a series of animal experiments, Wolpe demonstrated that anxiety can inhibit behaviors such as eating, sleep, and sexual arousal. Wolpe concluded from these findings that sexual arousal is mutually antagonistic to an anxiety state, and that this was operative at both a behavioral and neural level. That is, sexual arousal is dominated by PNS activity and anxiety by SNS activity. Since Wolpe's reciprocal inhibition theory was originally proposed, there has been only one study aimed directly at testing the assertion that anxiety and sexual arousal are mutually inhibitory (i.e., Hoon, Wincze, & Hoon, 1977). Consistent with Wolpe's theory, this study demonstrated that anxiety exposure following an erotic film decreased physiological sexual arousal in women more rapidly than did a neutral film. Presumably, the SNS influence of anxiety extinguished the PNS influence of the erotic stimulus. Inconsistent with Wolpe's theory however, anxiety stimuli prior to an erotic film enhanced physiological sexual arousal. Wolpe (1978) criticized the Hoon et al. (1977) study on the grounds that the anxiety and sexual stimuli were presented sequentially and there was no evidence that the anxiety response was maintained
during presentation of the erotic film. According to Wolpe, in order for two events to be mutually inhibitory and, therefore, to appropriately test the reciprocal inhibition hypothesis, it is necessary that the anxiety and sexual response be present simultaneously.

Based on physiological studies in men which indicate that penile erection is primarily under sacral PNS control and ejaculation under SNS control, some researchers have postulated that sexual arousal in men and women may be mediated by differential changes in the activity of both branches of the autonomic nervous system. Wenger, Jones, and Jones (1956) proposed that sexual excitement is comprised of three relatively distinct phases, each of which is dominated by alternate branches of the autonomic nervous system. The initial phase, leading to engorgement of tissue, is mediated primarily by the PNS. As emotion intensifies to the point of climax, the SNS becomes dominant and, finally, following orgasm, the PNS again becomes dominant through overcompensation (Wenger et al., 1956). This model is similar to the biphasic model of sexual responding later proposed by Kaplan (1974), which asserts sexual excitement is comprised of an arousal stage mediated by the PNS, and an orgasmic stage dominated by SNS activity. Kaplan's model has been given considerable attention and is currently accepted by numerous researchers, theorists and clinicians. In their remarkably detailed analysis of the human sexual response, Masters and Johnson (1966) described various stages of the human sexual response cycle, but devoted little attention to its neural regulation.

Perhaps the widest support for an inhibitory role of the SNS on sexual arousal comes from clinical reports which indicate that performance anxiety causes erectile failure in men. In a retrospective study of males with erectile dysfunction, Johnson (1965) reported that 48% of the men studied suffered from an anxiety disorder. In a similar study of sexually dysfunctional males, Cooper (1969a) reported that 51% of the subjects suffered from "marked coital anxiety". Coital anxiety was most frequently reported to be a consequence of fear of failure (73%), being regarded as sexually inferior (44%), or being ridiculed (40%). Consistent with
Cooper's findings, Ansari (1975) reported that 66% of the patients studied reported that their erectile dysfunction developed as a reaction to discrete sexual experiences. Taken together, these findings support the connection between anxiety and erectile dysfunction, but also suggest the dysfunction may be more a consequence of negative cognitions associated with specific sexual activities than of anxiety per se. Nevertheless, the pervasiveness of these and similar reports has led a number of researchers and clinicians to label anxiety as one of the leading causes of sexual dysfunction in both men and women (e.g., Jehu, 1979; Kaplan, 1974; 1988; Masters & Johnson, 1970), and has provided the impetus for using anxiety-reduction techniques in the treatment of sexual dysfunction (e.g., Barlow, 1986; Beck & Barlow, 1984).

Anxiety is thought to inhibit sexual function through both cognitive and physiological mechanisms. At a cognitive level, research suggests that anxiety inhibits sexual arousal by disrupting the processing of erotic cues (e.g., Abrahamson, Barlow, Beck, Sakheim, & Kelly, 1985; Beck & Barlow, 1986a, 1986b; Beck, Barlow, Sakheim, & Abrahamson, 1987; Farkas, Sine, & Evans, 1979; Geer & Fuhr, 1976). At a physiological level, anxiety is thought to disrupt sexual arousal by inducing a state of SNS dominance (e.g., Zuckerman, 1971). The precise physiological mechanism for this presumed inhibition, however, is poorly understood. In men, it has been suggested that SNS activity causes erectile impairment by inhibiting arterial inflow to the erectile sinus tissue (Lange, 1979). It is well established that the PNS signal activates penile nerves which cause dilation of the penile arteries and relaxation of muscles in the wall of the sinusoids and arterioles that supply erectile tissue. As a result, blood flow in erectile tissue increases which leads to a rise in intracavernous pressure and expansion of the cavernous spaces to produce extension and rigidity of the penis (e.g., deGroat & Steers, 1990). In women, assertions regarding the nervous regulation of the vasocongestive response are made primarily by analogy to men: "By analogy it is inferred that, like male erection, female lubrication-swelling is under the control of the
parasympathetic division of the autonomic nervous system." (Kaplan, 1974, pp. 25). Hence, as noted by Barlow (1986), etiological assumptions of what facilitates and maintains sexual arousal have been based primarily on clinical inference as opposed to empirical data. This appears to be particularly true with respect to female sexual behavior.

Activation of the Sympathetic Nervous System Facilitates Sexual Arousal

Indirect support for a facilitatory influence of SNS activity on sexual arousal comes from animal studies which have examined the effects of various stress-inducing stimuli, thought to increase nervous system arousal, on copulatory behavior. Beach (1947) was one of the first researchers to report that general activation induced by "batting the animals sharply about the cage" could renew copulatory behavior in sluggish male rats. Other generally arousing events, such as fear, were also shown to facilitate erection and ejaculation in male dogs and chimpanzees (Beach, 1947). Since the original work by Beach, a number of studies have demonstrated that electric shock or tail pinch can increase the number of prepubertal male rats copulating, increase the rate of copulation in experienced males, decrease the length of the postejaculatory period of sexual inactivity, increase the rate of mounting behavior in aging males, and temporarily restore copulatory activity suppressed by various forms of brain damage (for review, see Antelman & Caggiula, 1980). Consistent with these reports, Calhoun (1962) reported increases in sexual activity in both male and female rats following a succession of aggressive encounters.

In humans, indirect support for a facilitatory influence of SNS activity on sexual arousal is provided by clinical and experimental evidence which indicates that anxiety and various forms of aversive stimuli, known to increase nervous system activity, can sometimes enhance sexual arousal in men and women. One early example of such evidence is Ramsey's (1943) finding that 50% of adolescent boys have experienced erections in response to fearful situations such as being in an accident or being chased by the police. Equally as intriguing, is the report by Sarrel and Masters (1982) that men were able to perform sexual intercourse
repeatedly despite direct threats with knives and other weapons if they failed. A similar phenomenon has been noted amongst exhibitionists and voyeurs who are often unable to become aroused without first experiencing the threat of being arrested (Stoller, 1976). Related findings have been noted in women. In the well known Kinsey report on women, Kinsey and associates (1953) point out that many women experience erotic sensations to being bitten. In a sample of 2,200 women, 26% reported "definite and/or frequent erotic responses", 29% reported "some erotic responses" and 45% reported "never" experiencing erotic responses to being bitten. Apparently, humans are not unique in this regard; biting is an integral part of sexual activity in a number of mammalian species including the baboon, various monkeys, mink, marten, ferret, skunk, horse, pig, sheep, lion and shrew (Kinsey et al., 1953).

The results from more recent empirical investigations have also challenged the long-held notion that SNS dominance necessarily interferes with sexual arousal. In men, reports have been made of increased subjective sexual arousal in response to such anxiety-evoking stimuli as crossing a fear-arousing suspension bridge (Dutton & Aron, 1974), and receiving performance demand instructions to maintain an erection (Heiman & Rowland, 1983). With respect to physiological sexual arousal, studies have noted increased penile tumescence among males who were exposed to an anxiety-arousing videotape (Wolchik, Beggs, Wincze, Sakheim, Barlow, & Mavissakalian, 1980), who were given shock threats contingent on the size of their erection (Barlow, Sakheim, & Beck, 1983), and who were given performance demand instructions to demonstrate and maintain sexual arousal (Heiman & Rowland, 1983). The few studies conducted in the female reveal similar increases in physiological sexual arousal with exposure to anxiety-evoking stimuli. Hoon and associates (1977) reported greater physiological (vaginal blood volume; VBV) sexual arousal in women when they viewed an anxiety-producing film prior to an erotic film, than when they viewed a neutral film prior to an erotic film. Using the same preexposure paradigm, Palace and Gorzalka (1990)
replicated Hoon and associates' (1977) findings in both sexually functional and sexually
dysfunctional women. Palace and Gorzalka (1990) suggested that one explanation for the
facilitatory effect of anxiety on sexual arousal, is that the physiological component of anxiety
(i.e., increased blood pressure, heart rate, muscle tension) may serve to "jump start" or
prepare the individual for sexual arousal.

Cognitive Explanations for the Facilitatory Effect of Anxiety on Sexual Arousal

While it is feasible that the increase in physiological sexual arousal reported by Hoon
and associates (1977) and Palace and Gorzalka (1990) is attributable to increased SNS
activation, evidence for an increase in SNS activity was not provided in either study. Hoon
and associates (1977) failed to find significant elevations in heart rate with exposure to the
anxiety films, and Palace and Gorzalka (1990) did not provide any physiological index of
SNS activation. It is therefore impossible to determine whether the increases in VBV
reported by Hoon et al. (1977) and Palace and Gorzalka (1990) are attributable to
physiological factors or to cognitive factors associated with the anxiety stimulus. As
suggested by Wolpe (1978), cessation of the unpleasant film may have left an emotional state
of relief which, in turn, facilitated sexual responding. Wolpe based this explanation, in part,
on the findings from a series of experiments whereby patients reported a feeling of relief at
the cessation of electric shock. Often the patients' reported sense of relief was greatly out of
proportion to the discomfort they had experienced (Wolpe, 1958).

Other cognitive explanations have also been offered to explain the enhancing effects
of anxiety. Social psychologists have described the enhancing effects of anxiety on sexual
arousal as a form of residual arousal which has moved from one emotional experience to
another. For example, Dutton and Aron (1974) and Beggs, Calhoun and Wolchik (1987)
explained the increases in sexual arousal resulting from anxiety exposure within the
conceptual framework of Schacter's two-factor theory of emotion (Schacter, 1964). This
theory posits that, when the context for heightened arousal is not apparent, the individual
relies on the current context for interpretation. Faced with anxiety when viewing the erotic films, subjects may have mislabeled the residual anxiety arousal as sexual arousal. This, in turn, would serve to heighten sexual responding.

Not unrelated to the explanations provided by Schacter's two-factor theory of emotion, are explanations provided by excitation-transfer theory (Zillman, 1972). According to this theory, residual sympathetic excitation from preceding emotional reactions may serve to intensify responses to present stimuli, provided the source of prior arousal is not readily available. In terms of anxiety and sexual arousal, this theory would predict that undecayed arousal from an anxiety film may combine additively with excitation from an erotic film and serve to intensify the experience of sexual arousal. In support of this explanation, Cantor, Zillman, and Bryant (1975) found that subjective sexual arousal was increased in male subjects who viewed an erotic film at 5 min post-exercise, but not when they viewed the film at either 1 min or 9 min post-exercise. Presumably, at 1 min post-exercise, cues from the prior aroused state (i.e., exercise) were still available and hence excitation transfer did not occur, and at 9 min post-exercise, excitation had decayed and, hence, could not occur. Only at 5 min post-exercise, when excitation was still present but the cues unavailable did a transfer of arousal take place.

**Physiological Explanations for the Facilitatory Effect of Anxiety on Sexual Arousal**

In an effort to provide a more direct examination of the effects of SNS activation on sexual arousal, Meston and Gorzalka (1995) examined the effects of acute, intense exercise on subjective and physiological sexual arousal in 35 sexually functional women. Acute, intense exercise was used as a means of activating the SNS based on a number of pharmacological and physiological studies which indicate that activation of the SNS becomes prominent at high intensities of exercise (e.g., Galbo, Holst, & Christensen, 1976; Haggendal, Hartley, & Saltin, 1970; Mazzeo & Marshall, 1989; Robinson, Epstein, Beiser, & Braunwald, 1966). Exercise was also used to enhance SNS activity based on the belief that exercise,
versus preexposure to a film of threatened amputation, would be less likely to influence cognitive processes. Using a repeated-measures design, Meston and Gorzalka (1995) found that exercise, when measured 15 min post-exercise, had a significant facilitatory effect on physiological, but not subjective, sexual arousal. This effect included a significant increase in vaginal pulse amplitude (VPA) and a marginally significant increase in VBV. Heart rate was significantly elevated with exposure to exercise, providing indirect evidence for an increase in SNS activity. Unlike the findings of Palace and Gorzalka (1990), which indicated that preexposure to the anxiety-evoking film increased subjects' ratings of feeling "worried", the findings of Meston and Gorzalka (1995) indicated that exercise had no significant effect on subjective ratings of either positive or negative affect. The fact that exercise significantly increased heart rate but did not alter subjects' mood, suggested that the increase in physiological sexual arousal with exposure to exercise was more likely due to physiological (i.e., increased SNS activity) than cognitive (e.g., anxiety relief) factors.

Because the effects of exercise on plethysmograph indices of sexual arousal had not previously been examined, it was possible that the increases in physiological responses may have represented "nonsexual" cardiovascular and/or hormonal responses to exercise. To investigate this possibility, Meston and Gorzalka (1995) examined the effects of exercise on sexual responses to two consecutive neutral films. The results indicated that, in the absence of an erotic film, exercise had no significant influence on either subjective or physiological indices of sexual arousal. This finding had important implications for interpretation of Meston and Gorzalka's results. First, it suggested that the photoplethysmograph did, as intended, measure only the sexual consequences of acute exercise. Second, and most importantly, it indicated the SNS activation alone does not elicit sexual arousal. It in only in the presence of a cognitively interpreted sexually arousing event that SNS activation facilitates sexual responding.
The results of Meston and Gorzalka (1995) provide support for a facilitatory role of the SNS in female sexual arousal, but are limited by a number of factors. First, the effects of exercise on sexual arousal were measured at only one time point (i.e., 15 min) post-exercise. Research indicates that, at 15 min post-exercise, SNS influences remain significantly elevated, but have declined considerably from intensities during and immediately following the cessation of exercise (Savin, Davidson, & Haskell, 1982). This raises the question of whether exercise would have an even greater facilitatory effect on sexual arousal if measured when SNS activation was at a peak, and whether the sexually facilitatory effects of exercise are in some way directly related to the level of SNS activation.

Experiment 1 of the present investigation was designed to examine the time course of the effects of SNS activation on sexual arousal in women by measuring subjective and physiological (VBV, VPA) sexual arousal at 5 min, 15 min, and 30 min post-exercise. By examining sexual arousal at these time intervals post-exercise, Experiment 1 allowed for comparison of the effects of high, moderate, and low levels of SNS activation on sexual responding. In addition, Experiment 1 helped to elucidate the boundaries by which SNS activation might be successfully integrated into clinical treatment. Based on their finding that exercise facilitated physiological sexual arousal when measured 15 min post-exercise, Meston and Gorzalka (1995) suggested that activation of the SNS could potentially play an important role in the treatment of sexual dysfunction in women. Determination of whether the immediate, delayed, or residual effects of SNS activation are optimal in facilitating sexual arousal is essential before clinical treatments can be successfully derived.

As noted by Meston and Gorzalka (1995), interpretation of the finding that exercise facilitates sexual arousal via activation of the SNS is limited by the fact that exercise, at the intensity and duration used in the present investigations and in the research of Meston and Gorzalka (1995), not only elicits significant increases in SNS activity (e.g., Nakamura, Yamamoto, & Muraoka, 1993; Yamamoto, Hughson, & Nakamura, 1992), but also causes
numerous hormonal changes. Intense, acute exercise has been shown to increase cortisol (Carr et al., 1981), prolactin and growth hormone (Sutton & Lazarus, 1976), norepinephrine and epinephrine (Hartley et al., 1972), and plasma levels of beta-endorphin (Gambert et al., 1981; Farrell, Gates, Maksud, & Morgan, 1982; Fraioli, Moretti, Paolucci, Alicicco, Crescenzi, & Fortunio, 1980). The effects of acute exercise on gonadal hormones are controversial in that testosterone levels have been reported to increase (Guglielmini, Paolini, & Conconi, 1984), decrease (Kuusi, Kostiainen, Vartiainen, Pitkanen, & Ehnholm, 1984), or not change (Bunt, 1985), and estrogen and progesterone either to increase (Bonen, Ling, MacIntyre, Neil, & McGrail, 1979), or not change (Loucks & Horvath, 1984). Conceivably, any number of the above alterations could account, at least in part, for the facilitatory effect of exercise on physiological sexual arousal.

Experiment 2 of the proposed investigation was designed to examine whether the enhancing effects of exercise on sexual arousal in women (Meston & Gorzalka, 1995) can be blocked by inhibiting SNS activity. The study was conducted using a double-blind, placebo-controlled, repeated-measures design in which subjects engaged in 20 min of intense exercise during each of two experimental sessions. In one session they received a placebo, and in one session they received clonidine, a selective SNS blocking agent, one hour prior to engaging in exercise. This experimental design allowed for comparison of the hormonal and SNS influences of exercise on sexual arousal (i.e., Placebo condition) with the hormonal influences alone (i.e., Clonidine condition). If exercise were to facilitate physiological sexual arousal in both the Clonidine and Placebo conditions, and there were significant differences in arousal levels between conditions, this would provide evidence that exercise facilitates sexual arousal via hormonal processes. On the other hand, if physiological sexual arousal were inhibited when exercising subjects received clonidine, the findings would support the notion that exercise facilitates sexual arousal via activation of the SNS.
Evidence that exercise facilitates physiological sexual arousal via SNS activation would cast doubt on the previously held assumption that the initial stages of sexual arousal in women are dominated by PNS activity and inhibited by SNS influences (e.g., Kaplan, 1974; Kinsey et al., 1953; Wolpe, 1958). In addition, the possibility for a facilitatory role of the SNS on sexual arousal in women would challenge the use of anxiety-reduction techniques in the treatment of female sexual dysfunction. A number of techniques used in the treatment of sexual dysfunction focus on altering negative cognitions while inducing a state of relaxation (e.g., systematic desensitization, sensate focus). Presumably, these techniques enhance sexual arousal by facilitating cognitive change, and by decreasing SNS and increasing PNS influences. The assumption that decreasing SNS and increasing PNS activity facilitates sexual arousal in women, however, has not been tested empirically. Outcome studies have shown techniques such as systematic desensitization and sensate focus to be successful in decreasing sexual anxiety and increasing coital frequency, but largely unsuccessful in facilitating orgasmic ability (for review, see Andersen, 1983). By contrast, directed masturbation, a technique which does not focus on inducing a state of relaxation, has proven extremely effective in treating orgasmic dysfunction (for review, see Nairne & Hemsley, 1983). It is possible that the relaxation component of anxiety-reduction techniques may be productive at a cognitive level (i.e., changing negative thoughts), but counterproductive at a physiological level (i.e., decreasing SNS activity). Perhaps, in order to attain levels of sexual arousal intense enough to achieve orgasm, significant activation of the SNS is necessary.

The purpose of Experiment 3 and an additional purpose of Experiment 2, is to examine the effects of SNS inhibition on physiological and subjective sexual arousal in women. In both of these experiments, clonidine, an antihypertensive medication shown to selectively block peripheral sympathetic activity (e.g., Engelman et al., 1989; Flacke et al., 1987), was used as a means of inhibiting the SNS. In Experiment 2, the effects of SNS inhibition on sexual arousal were measured during high levels of autonomic arousal (e.g.,
increased heart rate, perspiration, muscle tension) experimentally induced using 20 min of intense exercise. In Experiment 3, the effects of SNS inhibition on sexual arousal were measured during baseline levels of autonomic arousal.

Evidence in support of an inhibitory role of SNS inhibition on sexual responding comes from animal studies which have shown that both moderate and high doses of clonidine inhibit sexual behavior in female rats (Davis & Kohl, 1977; Meston, Moe, & Gorzalka, 1995). This effect includes decreases in both receptive (lordosis, a spinal reflex in response to male attempts to mate) (Davis & Kohl, 1977; Meston et al., 1995) and proceptive (ear wiggling) (Meston et al., 1995) behaviors, and increases in rejection behaviors (kicking, boxing, squealing) (Meston et al., 1995). In humans, the effects of clonidine and other antihypertensive agents on sexual function have been studied almost exclusively in men (e.g., Aldridge, 1982; Buffum, 1982; Moss & Procci, 1982; Rosen, Kostis, Jekelis, & Taska, 1994; Smith & Talbert, 1986; Wein & van Arsdalen, 1988). Reported side effects of antihypertensive drugs on male sexual function include: decreased libido, impotence, delayed or retrograde ejaculation, priapism, and gynecomastia (Stevenson & Umstead, 1984; Weinberger, 1989). The few articles which refer to female sexual function secondary to antihypertensive treatment refer primarily to menstrual abnormalities, decreased vaginal lubrication, and galactorrhea (Arze, Ramos, Rashid, & Kerr, 1981; Loriaux, 1976), although a few anecdotal reports of decreased libido and anorgasmia have appeared (Editorial, 1977; Stevenson & Umstead, 1984). To the author's knowledge, only one study has empirically examined the effects of antihypertensive agents on female sexual responding (Hodge, Harward, Stewart-West, Krongaard-Demong, & Kowal-Neeley, 1991). In this study, self-administered daily diaries of sexual arousal, desire, and orgasmic function were examined in 10 premenopausal and eight postmenopausal women with mild hypertension and unimpaired sexual function. Over a 24 week period, subjects received either placebo, clonidine, or prazosin in alternating 4-8 week drug intervals. Clonidine showed a nonsignificant trend
toward decreasing sexual desire (measured as fewer positive responses to the following questions: Did you wish your partner would approach you?, Did you approach your partner?, Did you have sexual daydreams today?, Did you think about lovemaking or masturbating?, Were you receptive to your partner when approached?). Clonidine had no significant effect on subjective ratings of either the number, strength, or latency of orgasms. The interpretation of these findings is confounded by the use of both pre- and post-menopausal women, the use of women with hypertension, and the exclusive reliance upon subjective measures. The present investigation extends this methodology by using premenopausal, sexually functional women with no history of high blood pressure, and by measuring sexual arousal both subjectively and physiologically.

Experiments 1 to 3 of the present investigation, as well as the investigation of Meston and Gorzalka (1995), examined sexual responding only in sexually functional women. Consequently, the results of these studies are limited in their generalizability to a sexually dysfunctional population. Experiment 4 of the present investigation was designed to provide the first empirical examination of the effects of SNS activation, via acute exercise, on sexual arousal in women with sexual difficulties. Experiment 4 was also designed to examine potential differences in sexual responding between sexually dysfunctional women with and without orgasmic dysfunction. Subjects in Experiment 4 were divided into those who are either sexually functional, or who meet criteria for low sexual drive, or either primary or secondary anorgasmia.

To date, differences in vaginal responding between sexually functional and dysfunctional women have been investigated in only four studies. In a sample of six nonclinical women and six women seeking treatment for various sexual dysfunctions, Wincze, Hoon, and Hoon (1976) examined the effects of an erotic film on subjective and physiological (VBV) measures of sexual arousal. The authors reported significantly higher levels of physiological (VBV), but not subjective, sexual arousal in the functional women. Using a
similar experimental paradigm, Palace and Gorzalka (1992) reported significantly greater increases in both VBV and subjective ratings of sexual arousal among 16 sexually functional women than 16 women with either sexual desire, drive, or orgasm difficulties. In contrast, Morokoff and Heiman (1980) reported no significant difference in physiological (VPA) sexual arousal between 11 nonclinical women and 11 women with low arousal and anorgasmia, but significantly lower subjective ratings of sexual arousal among the sexually dysfunctional versus functional women. Palace and Gorzalka (1990) compared the sexual responses of 16 sexually functional and 16 dysfunctional women to an erotic film preceded by either a neutral or anxiety-evoking preexposure stimulus. The results revealed that preexposure to an anxiety-evoking film enhanced physiological (VBV), but not subjective, sexual arousal in both groups of women, but sexually functional women attained higher levels of VBV in both the neutral and anxiety preexposure conditions. It should be noted that, in these studies, differences reported between sexually functional and dysfunctional women were all quantitative, as opposed to potential qualitative differences in sexual response patterns. Experiment 4 aims to uncover whether orgasmic and anorgasmic women differ qualitatively in their sexual response patterns at a neurophysiological level.

In summary, the proposed investigation is aimed at further elucidating the role of the SNS in female sexual arousal by examining the following six questions:

1. What is the relationship between level of SNS activity and level of subjective and physiological sexual arousal in sexually functional women?

2. Can the enhancing effects of acute exercise on physiological sexual arousal in women be blocked by inhibiting the SNS?

3. Does SNS inhibition during heightened nervous system arousal inhibit sexual arousal in sexually functional women?

4. Does SNS inhibition during baseline nervous system arousal inhibit sexual arousal in sexually functional women?
5. Does SNS activation have a differential effect on sexual arousal among sexually functional women, women with low sexual drive, and women with orgasm difficulties?

6. Can the findings of Meston and Gorzalka (1995), which indicate that SNS activation facilitates physiological sexual arousal, be extended to a group of sexually dysfunctional women?

In addition to providing further insight into the role of the SNS in female sexual function, the present investigation was designed to extend the methodology of previous research on sexual arousal in women in several respects. First, in order to provide more valid and reliable physiological data, both VBV and VPA were used as indices of physiological sexual arousal. Vaginal blood volume, the dc signal, reflects slow changes in the pooling of blood in the vaginal tissue (Hatch, 1979). Vaginal pulse amplitude, the ac signal, reflects short-term changes in engorgement (Rosen & Beck, 1988). With the exception of the Meston and Gorzalka (1995) study, previous studies on nervous system activation and sexual arousal in women have examined only one or the other of these two physiological indicators of sexual responding.

Second, to verify that exercise has a significant effect on nervous system activity, heart rate was measured throughout the stimulus exposure during all experimental conditions. Past researchers (e.g., Palace & Gorzalka, 1990) have speculated that the recently reported facilitatory effects of anxiety on sexual arousal may be due to increased SNS activation, but have not provided any physiological index to support their assertion.

Third, in each experiment, information on subjects' past and predicted levels of sexual arousal was obtained. This allowed for examination of the possibility that a ceiling exists on the level of sexual arousal that can be experienced in a laboratory setting. Researchers have generally found a desynchrony between subjective and physiological sexual responses in women (e.g., Meston & Gorzalka, 1995; Morokoff & Heiman, 1980; Palace & Gorzalka, 1990, 1992; Steinman, Wincze, Sakheim, Barlow, & Mavissakalian, 1981), and have
explained their findings in terms of an indirect feedback system between components of the sexual response (Heiman, 1977), or social standards which prevent women from admitting to being sexually aroused (Palace & Gorzalka, 1990). This investigation entertains the notion that the levels of subjective sexual arousal reported in this, and past, research may represent the highest levels that can be expected when using erotic films as sexual stimuli, and an experimental laboratory as a sexual setting. If subjects' reported levels of sexual arousal are statistically similar to their predicted levels of sexual arousal to an erotic film, support for a ceiling effect on subjective measures will be provided. In addition, if subjects report having previously experienced higher levels of sexual arousal than that which they experience in the present study, evidence against the notion that subjective reports of sexual arousal in women are routinely lowered because of a need for socially desirable responding will be provided.

Finally, to examine potential differences in sexual responding between women with various sexual difficulties, in Experiment 4, sexually dysfunctional women were divided into those with primarily sexual drive difficulties, and those with primarily orgasmic difficulties. This classification differs from previous research (e.g., Morokoff & Heiman, 1980; Palace & Gorzalka, 1990; Wincze et al., 1976) which has combined women with a variety of sexual difficulties, including low sexual desire, anorgasmia, and dyspareunia, into one heterogeneous experimental group.

EXPERIMENT 1

The Effects of Immediate, Delayed, and Residual Sympathetic Activation on Physiological and Subjective Sexual Arousal in Women

Experiment 1 was designed to examine the time course of the effects of acute exercise on sexual arousal in women. The purpose is to examine whether a relationship exists between level of SNS activity and level of sexual arousal in women. Subjective (self-report) and physiological (photoplethysmograph) sexual arousal were measured in response to erotic
films at either 5 min, 15 min, or 30 min post-exercise. There are several possible outcomes which would suggest a relationship between level of SNS activation and level of sexual arousal. One such possibility is that exercise will cause a significantly greater increase in sexual arousal at 5 min post-exercise, when SNS activation is at the highest post-exercise level, compared to 15 and 30 min post-exercise, when SNS activity has declined to consecutively lower levels. If this is the case, support for a positive, linear relationship between level of SNS activation and sexual arousal will be provided. Equally likely is the possibility that exercise will have a greater facilitatory influence on sexual arousal at 30 min versus 15 or 5 min post-exercise, in which case support for a negative, linear relationship between level of SNS activation and sexual arousal will be provided. Finally, if exercise causes either a significantly greater or lesser increase in sexual arousal at 15 min post-exercise, compared with levels measured at 5 and 30 min post-exercise, the findings will suggest that moderate rather than high or low levels of SNS activation are optimal in facilitating sexual arousal in women. The notion of a curvilinear relationship between SNS activation and sexual arousal was suggested by Jupp and McCabe (1989) who found that moderate, versus low or high, levels of self-reported, general, physiological arousability were optimal in facilitating sexual function.

The level of exercise used in the present investigation was chosen based on a number of pharmacological and physiological studies which indicate that activation of the SNS becomes prominent at moderate (e.g., 60% HR$_{\text{max}}$) to high intensities of exercise. For example, an early study on the effects of heart rate response to exercise during pharmacological blockade (Robinson et al., 1966) demonstrated that, while the increase in heart rate with exposure to low or moderate intensities of exercise is mediated primarily by PNS withdrawal, the increase in heart rate at higher levels of work is mediated primarily by SNS stimulation. Numerous studies which have reported increases in plasma norepinephrine and epinephrine to moderate and heavy exercise also support the idea that intense exercise
activates the SNS (e.g., Galbo et al., 1976; Haggendal et al., 1970; Mazzeo & Marshall, 1989). More recently, studies using the technique of spectral analysis of heart rate variability have shown that PNS withdrawal occurs primarily up to moderate levels of exercise, and SNS activity becomes prominent during moderate to heavy exercise (Nakamura et al., 1993; Yamamoto et al., 1992). The effects of exercise on sexual arousal are measured at 5, 15, and 30 min post-exercise based on research which indicates that the SNS remains significantly elevated at 30 min post-exercise (e.g., Cleroux, Peronnet, & Champlain, 1985; Kraemer et al., 1991; Strobel, Hack, Kinscherf, & Weicker, 1993).

**Method**

**Subjects**

Thirty-six sexually functional women (M age = 25.6 years, range = 18-45) participated in one of three experimental conditions: Immediate (n=12), Delayed (n=12), and Residual (n=12) sympathetic activation. Mean ages of subjects by experimental condition were 23.83, 29.42, 22.58, for Immediate, Delayed, and Residual conditions, respectively. The subjects were recruited through a psychology department undergraduate research participant pool or via local newspaper advertisements requesting volunteers for the current experiment. Three subjects were undergraduate students in psychology, 15 subjects were undergraduate or graduate students in disciplines other than psychology, and the remaining 18 subjects were employed in various professions outside the university. Because of reported ethnic and racial differences in sexual activity (e.g., Meston, Trapnell, & Gorzalka, 1995), subject background information was recorded. Racial background of the subjects was: Caucasian (35), and Asian (1). All subjects were currently involved in sexual relationships; two of the subjects were married. Subjects were paid $15.00 for their participation. Initial telephone screening criteria were: between the ages of 18-45 years, no use of medications known to affect vascular or sexual functioning, no history of treatment for sexual dysfunction, no medical condition that may put the subject at risk when exercising, and current involvement in a heterosexual
relationship. Further inclusion criteria, based on subject information from the Derogatis Sexual Functioning Inventory (DSFI; Derogatis, 1978), the Orgasmic Functioning Questionnaire (OFQ; Meston, Jung, Hanson, & Gorzalka, 1993) and the Physical Readiness Exam for Fitness Test (developed by the British Columbia Ministry of Health) included: absence of general psychopathology, absence of sexual dysfunction, absence of anorgasmia, within the normative range of sexual experience, no history of heart disease or cardiovascular dysfunction, no history of dizzy spells or "light-headedness", and no bone or joint problem that might be aggravated by 20 min of cycling.

Profile descriptions of all subjects were obtained via the DSFI, and the OFQ. The DSFI is a standardized self-report multidimensional inventory comprised of 10 distinct subtests designed to measure current levels of sexual functioning. The DSFI subtest scores are summed to provide an overall measure of sexual functioning (Sexual Functioning Index; SFI). Responses to the single item "How satisfying is your sexual relationship? (0=could not be worse, to 8=could not be better) provide a global subjective rating of sexual functioning (Global Sexual Satisfaction Index; GSSI). The SFI, GSSI, and the Drive subscale of the DSFI were used to screen for absence of sexual dysfunction. All subjects employed in the study scored greater than or equal to the 30th percentile (i.e., within two standard deviations of the normative mean) on the SFI (M = 46.6, range = 40-64), the GSSI (M = 54.4, range = 40-70), and the Drive subscale (M = 59.6, range = 40-71). In addition, the Brief Symptom Inventory (BSI; Derogatis, 1975) subtest of the DSFI was used to screen for absence of general psychopathology. The BSI is a distinct psychometric diagnostic instrument, empirically validated as an independent measure of psychopathology. All subjects employed in the study scored greater than or equal to the 30th percentile (i.e., within two standard deviations of the normative mean) on the BSI (M = 42.0, range = 30-61). Data from the Experience subtest of the DSFI were used to ensure that all subjects were within the normative range of sexual experience. The Experience subtest assesses the range of
hierarchically scaled sexual behaviors experienced by the individual, progressing from fundamental (e.g., clothed embrace) to relatively advanced (e.g., mutual oral stimulation of the genitals). All subjects scored above the 30th percentile on the Experience subtest (M = 51.4, range = 35-64). Data from the OFQ was used to screen for absence of orgasmic dysfunction. The OFQ is a self-report inventory of one's ability to achieve orgasm in response to a wide variety of sexual activities (see Appendix for complete item list). All subjects employed in the study were able to achieve orgasm by some means (e.g., intercourse, oral sex, masturbation) on at least 50 percent of the attempted trials (M percent = 95: on average, subjects were able to achieve orgasm by some means on 95% of the attempted trials). Mean scores by experimental condition for the DSFI and the OFQ are presented in Table I.

One subject was eliminated from the study because she scored below the cutoff criterion for general psychopathology, and one subject was eliminated because she scored above the cutoff criterion for sexual experience. Four subjects were eliminated from the study because they were considered sexually dysfunctional according to the SFI criteria, and four subjects were eliminated because they were considered anorgasmic (never achieved an orgasm by any means) according to the OFQ criteria. Data from three subjects were eliminated because of technical difficulties during physiological recording. Thirty-six subjects met all inclusion criteria and served as subjects in the study.

Design and Procedure

The procedure consisted of three sessions: a 1-hr orientation screening and questionnaire session; a 45-min No-exercise experimental session; and a 1-hr Exercise experimental session. Order of the two experimental sessions, Exercise and No-exercise, was counterbalanced across subjects within each experimental condition (Immediate, Delayed, Residual). During each experimental session subjects viewed one of two 7-min videotaped sequences, referred to here as Sequence A and Sequence B. Each sequence consisted of a 1-
Table I.
Psychometric Characteristics of Subjects in the Immediate, Delayed, and Residual Conditions of Experiment 1.

<table>
<thead>
<tr>
<th>Measure</th>
<th>Immediate Condition</th>
<th>Delayed Condition</th>
<th>Residual Condition</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean/SEM</td>
<td>Range</td>
<td>Mean/SEM</td>
</tr>
<tr>
<td>DSFI Subtests</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Information</td>
<td>52.6+/-2.2</td>
<td>38-62</td>
<td>53.2+/-3.0</td>
</tr>
<tr>
<td>Experience</td>
<td>51.3+/-2.5</td>
<td>35-64</td>
<td>51.3+/-1.8</td>
</tr>
<tr>
<td>Drive</td>
<td>58.3+/-2.0</td>
<td>47-67</td>
<td>60.9+/-2.4</td>
</tr>
<tr>
<td>Attitude</td>
<td>29.9+/-0.8</td>
<td>25-34</td>
<td>29.4+/-1.6</td>
</tr>
<tr>
<td>Symptoms (BSI)</td>
<td>40.9+/-2.6</td>
<td>30-53</td>
<td>43.8+/-2.5</td>
</tr>
<tr>
<td>Affect</td>
<td>42.1+/-2.7</td>
<td>30-58</td>
<td>46.4+/-3.1</td>
</tr>
<tr>
<td>Gender Role</td>
<td>42.5+/-4.6</td>
<td>20-60</td>
<td>54.2+/-2.3</td>
</tr>
<tr>
<td>Fantasy</td>
<td>60.8+/-2.9</td>
<td>48-79</td>
<td>60.8+/-2.6</td>
</tr>
<tr>
<td>Body Image</td>
<td>35.9+/-2.5</td>
<td>29-53</td>
<td>44.4+/-4.4</td>
</tr>
<tr>
<td>Satisfaction</td>
<td>48.1+/-3.2</td>
<td>30-63</td>
<td>52.3+/-2.1</td>
</tr>
<tr>
<td>DSFI Global Scores</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SFI</td>
<td>41.8+/-2.2</td>
<td>31-53</td>
<td>49.8+/-3.0</td>
</tr>
<tr>
<td>GSSI</td>
<td>55.7+/-2.3</td>
<td>47-70</td>
<td>54.5+/-3.1</td>
</tr>
<tr>
<td>OFQ</td>
<td>94+/-2.6</td>
<td>70-100</td>
<td>96+/-2.0</td>
</tr>
</tbody>
</table>

Note: DSFI = Derogatis Sexual Functioning Inventory, OFQ = Orgasmic Functioning Questionnaire, BSI = Brief Symptom Inventory, SFI = Sexual Functioning Index, GSSI = Global Sexual Satisfaction Index. Means for Derogatis Sexual Functioning Inventory items are based on raw scores that were converted to established percentile rankings (T scores). Means for the OFQ are based on the highest estimated percentage of trials on which a subject achieved orgasm by any method.
min display of the word "relax" followed by a 3-min neutral travelogue film and then a 3-min erotic film.

Sequence A and B differed only in the content of the neutral and erotic films. The neutral film in Sequence A depicted geographic scenes from the Antarctic; Sequence B depicted wildlife scenes from the Antarctic. In both Sequence A and B, the erotic films depicted a nude, heterosexual couple engaging in foreplay and intercourse. The erotic films were accompanied by fast-paced music and included explicit sexual communication by the couple. The two erotic films used in Sequences A and B were matched on the number, order, type, and duration of sexual acts, and included the same actors and settings. The erotic films were adapted by E.M. Palace from those used by John P. Wincze. Both the neutral and erotic films were identical to those used by Meston and Gorzalka (1995).

The two experimental sessions were scheduled at approximately three day intervals and excluded times during which the subjects were menstruating. Phase of the menstrual cycle was not controlled given that sexual arousability to erotic stimuli in laboratory situations, measured either subjectively or physiologically, is only minimally, if at all, influenced by the menstrual cycle (Hoon, Bruce, & Kinchloe, 1982; Meuwissen & Over, 1992). All subjects were asked to abstain from psychoactive drugs (including caffeine and alcohol) and to refrain from engaging in any strenuous physical activity for 24 hr prior to each experimental session.

Session 1 (orientation/screening). Following an initial telephone screening, subjects were scheduled for a first session with the female experimenter. During this session, subjects were shown the laboratory facilities and equipment, were given verbal instructions on the use of the photoplethysmograph, were informed of the plethysmograph sterilization procedures, and were encouraged to ask any questions related to the experiment. Subjects were told that they would be participating in an experiment which involved the effects of exercise on female sexual arousal. They were told that they would view brief visual stimuli, some of which may
include erotic content. To minimize a possible sense of coercion, subjects were given the option of either participating in the first session on that day, or telephoning within a week, regarding their decision to participate. All subjects chose to begin the study that day.

After signing the standard consent form, subjects completed the DSFI, the OFQ, three additional sexuality items, and the Physical Readiness Exam for Fitness Test in a private room. The three additional sexuality items were (1) "Given the appropriate situation, what is the highest level of sexual arousal you think that you could experience?", (2) "What is the highest level of sexual arousal you have ever experienced?", and (3) "What is the highest level of sexual arousal you think that you could experience viewing an erotic film alone?". Subjects answered on a 7 point Likert scale from 1 (low) to 7 (intense). These questions were used to assess (1) the level of sexual arousal that subjects were willing to admit to having experienced or being able to experience, and (2) how reported levels of sexual arousal to the experimental films compared with subjects' expected ability to become aroused by an erotic film. The Physical Readiness Exam for Fitness Test, assesses subjects' current medical functioning, and is designed to screen for subjects who would be put at risk when exercising (see Appendix).

To minimize potential experimental demand, subjects were instructed as follows:

When you fill out these questionnaires, please be as honest as possible. Every person is unique and there are absolutely no right or wrong answers. I realize that these questionnaires ask very personal information and so I would like to assure you again that all information is kept strictly confidential. All of the forms that you will be filling out are numerically coded; there will be no identifying information on any of the forms. Only myself and the female research assistant will have access to this coded information.

After completing the questionnaires, subjects took a 10 min break, and then began Session 2.
Sessi on 2 and 3 (experimental). The second and third sessions were the two experimental sessions: Exercise and No-exercise. The order of these two sessions was counterbalanced across subjects, within each experimental condition (Immediate, Delayed, Residual). Both experimental sessions were conducted inside the Sexual Psychophysiology Laboratory at the University of British Columbia. This laboratory has an adjoining, private, internally-locked subject room. Communication with subjects is made possible via an intercom system between subject and experimenter rooms. The room is kept at a constant 21.7°C. A 41 cm color television monitor is positioned 205 cm from the subject, a distance which allows subjects to sit comfortably in a recliner with a full view of the screen. A bicycle ergometer is positioned to the rear of the room.

During the No-exercise session, subjects entered the private, internally locked room together with the female experimenter. They were told that, when the experimenter notified them, via the intercom system, they were to sit in the chair and insert the photoplethysmograph so as to allow approximately a 2.5 cm distance between the end of the probe and the vaginal opening. They were also asked to remain as still as possible throughout the session in order to minimize potential movement artifacts. Subjects were instructed as follows:

I will be giving you step by step instructions, via the intercom system as we go along, but I'll also explain to you now what will take place during this session. When I leave the room, I'd like you to remove any clothing necessary to insert the photoplethysmograph and then have a seat in this chair and relax. You can recline the chair a little and cover yourself with this blanket if it makes you feel more comfortable. After 1 minute (or 11 minutes, or 26 minutes, depending on the experimental condition) I will inform you via the intercom system to insert the photoplethysmograph. Once you have inserted the plethysmograph, please say "ready". I will be able to hear you through the intercom. As soon as
you say "ready", a series of short films will begin on the television. They will last about 10 minutes. It is very important that you remain as still as possible during the film presentations. The photoplethysmograph is very sensitive and any movement makes it very hard to read the information. Immediately after the films have ended, I'd like you to fill out this short questionnaire. Please be as honest as possible about how you feel about the films. Remember, every person is unique in their responses and there are no right or wrong answers.

Also remember that all information is kept strictly confidential.

As soon as the experimenter left the subject room, she began the stop watch. Subjects in the Immediate condition were notified to insert the plethysmograph after 1 min had passed, subjects in the Delayed condition were notified after 11 min had passed, and subjects in the Residual condition were notified to insert the plethysmograph after 26 min had passed. This procedure was used to ensure that the plethysmograph adaptation period was equivalent for all experimental conditions. When subjects notified the experimenter, via the intercom system, that they had finished inserting the plethysmograph, the films began. Subjects viewed either videotaped sequence A or B. Each sequence consisted of the word "relax" (1 min), a neutral travelogue (3 min), followed by an erotic film (3 min). Immediately following the erotic film, subjects were asked to fill out the subjective rating scale.

During the Exercise session subjects entered the private, internally locked room with the female experimenter and were informed of the experimental procedure as in the No-exercise session. Subjects were then asked to cycle for 20 min on a Get Fit 200-II stationary bicycle. The subjects' heart rates were monitored continuously using a Hear: Speedometer model 8719 (Computer Instruments Corp., Westbury, NY). Subjects were asked to cycle at a constant 70% of their maximum heart rate. Maximum heart rate was determined using the standardized formula

$$HR_{\text{max}} = 220 - \text{age in years}$$

(Golding, Meyers, & Sinning, 1982). Subjects were given continual visual feedback on their heart rate levels, and were asked to
cycle faster or slower if their heart rate indicated they were below or above the required exertion level. By ensuring that all subjects worked at equivalent levels of their maximum heart rate, differences in physiological responses resulting from variations in fitness levels are minimized (Grossman & Moretti, 1986). Fitness levels were not assessed, given that Meston and Gorzalka (1995) reported no correlation between fitness levels and physiological measures of sexual arousal when subjects exercised at equivalent levels of their maximum heart rate.

When one min of cycling time remained, the experimenter left the room. Subjects had been instructed to continue cycling until the timer signaled 20 min, then remove any clothing necessary to insert the plethysmograph, cover themselves with a blanket, and then sit in the chair until notified by the experimenter (via the intercom system) to insert the plethysmograph. As in the No-exercise session, subjects in the Immediate condition were notified to insert the plethysmograph after 1 min had passed, subjects in the Delayed condition were notified after 11 min had passed, and subjects in the Residual condition were notified to insert the plethysmograph after 26 min had passed. When the subject had finished inserting the plethysmograph, the films began. Subjects viewed one of the two videotaped sequences (A or B). The total time from the cessation of exercise to the onset of the erotic stimulus was approximately 5 min for the Immediate condition (1 min rest period, 1 min display of the word "relax", 3-min neutral film), 15 min for the Delayed condition (11 min rest period, 1 min display of the word "relax", 3-min neutral film), and 30 min for the Residual condition (26 min rest period, 1 min display of the word "relax", 3-min neutral film). Immediately following the erotic film, subjects were asked to fill out the subjective rating scale. With the exception of 20 min of cycling, all experimental procedures were identical to those of the No-exercise session.
Upon completion of Session 3, subjects were thoroughly debriefed, informed about the additional purposes and goals of the study, and given an opportunity to view the records of their vaginal responses. All subjects were paid $15.00 for their participation.

**Data Sampling and Reduction**

**Physiological Measurements**

Physiological measures were obtained using a vaginal photoplethysmograph (Sintchak & Geer, 1975). The photoplethysmograph was washed with Hibitane and sterilized by soaking in Cidex, 2% glutaraldehyde, 98% inert ingredients (long-life activated dialdehyde solution: Surgikose Canada, Peterborough, Ontario) for 10 hr between uses. Changes in VBV, VPA, and heart rate were monitored simultaneously during all experimental sessions. Light and heating effects were minimized by allowing the photoplethysmograph a 45-min warm-up period prior to insertion.

The signal from the Geer gauge and module (Farrall Instruments, Grand Island, NE) was channeled through an optical isolator-power supply. Vaginal blood volume was transduced using a Beckman Type 9806AB coupler and amplified to yield 0.1 V/mm with the high frequency response filter set at 22 Hz. and the time constant set to dc. Pulse amplitude was transduced using a Sensormedics Type 9853A coupler and amplified to yield 10 mV/mm with the low frequency response filter set at 5.3 Hz. The VBV signal was recorded at a sampling rate of 5 times/s with a Data Translation (Marlborough, MA) analog-digital converter and Labtech Acquire Program (Laboratory Technologies Corp., 1986) installed on a Samtron SC-386 microcomputer. The VPA and VBV signals were channeled and recorded on a Beckman model R612 dynagraph (Scheller Park, IL) with a rectilinear pen system, and chart speed set at 2.5 mm/s. Heart rate was extracted from the VPA recordings. The software program timed the administration of the stimuli and used an audio trigger signal to mark all stimulus changeovers.
**Vaginal pulse amplitude.** Vaginal pulse amplitude was recorded throughout the entire 180 s of neutral film and 180 s of erotic film. The data were hand scored from the polygraph recordings by a research assistant who was kept blind to the experimental manipulations. For each experimental condition, an average peak to peak amplitude was computed for both the neutral and erotic films by summing the amplitudes of each peak during the middle 20 s of the neutral or erotic film stimulus and dividing by the number of peaks per interval. Difference scores were computed for each experimental condition by subtracting the average VPA score during the neutral film from the average VPA score during the erotic film.

**Vaginal blood volume.** Vaginal blood volume was sampled during the last 80 s of neutral film, and during the entire 180 s of erotic stimuli. Because there is no absolute method of calibrating VBV and, hence, no zero point, the data were scored as 0.0001 mV units of blood volume deviation from a baseline reference level defined as the mean of the last 80 s of the neutral stimulus.

**Heart rate.** Heart rate was scored from the VPA polygraph records by counting the number of beats across the entire 180 s of neutral and 180 s of erotic film. The scores were averaged across time to yield 2 measures (bpm) for each subject per experimental session (one measure during each of the neutral and erotic films).

The data sampling and reduction procedures for VBV, VPA, and heart rate were identical to those used by Meston and Gorzalka (1995).

**Subjective Measurements**

A self-report rating scale, adapted from Heiman and Rowland (1983), was used as a subjective measure of sexual arousal. This scale has been shown to be a sensitive indicator of emotional reactions to erotic stimuli (Heiman 1980; Heiman & Hatch 1980; Heiman & Rowland 1983; Morokoff & Heiman 1980). The scale consists of 33 items: sexual arousal (1 item), perceptions of physical sexual change (4 items), autonomic arousal (5 items), anxiety (1 item), positive affect (11 items) and negative affect (11 items) (see Appendix for complete
item list). Subjects rated each of these items, depending on the degree to which they experienced the sensations, on a 7-point Likert Scale, from not at all (1) to intensely (7). Subjective sexual arousal was defined by the following five items on the scale: Sexually aroused, warmth in genitals, genital wetness or lubrication, genital pulsing or throbbing, and any genital feelings. Research indicates that there are no significant differences in subjective reports of sexual arousal obtained by methods of discrete versus continuous subjective measurement (Steinman et al., 1981).

**Results**

**Analyses of Physiological Measures**

**Vaginal pulse amplitude.** A Session (Exercise vs. No-exercise) x Condition (Immediate vs. Delayed vs. Residual) analysis of variance was conducted on VPA difference scores. Results revealed a significant main effect of exercise on VPA difference scores, $F(1, 33) = 5.85$, $p = .021$, and a marginally significant main effect of condition on VPA difference scores, $F(2, 33) = 3.10$, $p = .058$. There was a significant interaction between session and condition, $F(2,33) = 6.63$, $p = .004$. A follow-up one-way analysis of variance between VPA difference scores during the No-exercise sessions revealed no significant difference in VPA difference scores between the No-exercise Immediate, Delayed, or Residual conditions ($F<1$). A follow-up one-way analysis of variance between VPA difference scores during the Exercise sessions revealed significant differences in VPA difference scores between the Exercise Immediate, Delayed, and Residual conditions, $F(2, 33) = 7.93$, $p = .002$. Newman Keuls tests with a significance level set at $p<.05$ indicated a significant difference in VPA difference scores between the Immediate and Delayed conditions, and between the Immediate and Residual conditions during the Exercise session. Planned follow-up t-tests were conducted on VPA difference scores between the No-exercise and Exercise conditions in each of the Immediate, Delayed, and Residual conditions. Due to accumulating Type I error on mean comparisons across the variables, only mean difference of $p<.02$ ($p<.05/3$) should be
considered statistically reliable. Results revealed a marginally significant decrease in VPA scores with exposure to exercise in the Immediate condition, \( t(11) = -2.57, p = .026 \), a significant increase with exposure to exercise in the Delayed condition, \( t(11) = 3.65, p = .004 \), and a trend toward increasing VPA responses with exposure to exercise in the Residual condition, \( t(11) = 2.31, p = .041 \). Mean VPA difference scores for the No-exercise and Exercise sessions are presented in Figure 1.

In order to verify that the erotic films facilitated VPA responses, one-tailed, paired samples t-tests were conducted on VPA raw scores between neutral and erotic films within each experimental condition and session. Due to accumulating Type I error on mean comparisons across the variables, only mean difference of \( p < .008 \) (\( p < .05/6 \)) should be considered statistically reliable. Results revealed a significant increase in pulse amplitude responses with exposure to an erotic film in the No-exercise Immediate condition, \( t(11) = 6.82, p < .001 \); Exercise Immediate condition, \( t(11) = 5.83, p < .001 \); No-exercise Delayed condition, \( t(11) = 9.99, p < .001 \); Exercise Delayed condition, \( t(11) = 8.33, p < .001 \); No-exercise Residual condition, \( t(11) = 4.46, p < .001 \); and Exercise Residual condition, \( t(11) = 7.08, p < .001 \).

To determine whether order of experimental sessions influenced VPA responses, Session (Exercise vs. No-exercise) x Order (Exercise, No-exercise vs. No-exercise, Exercise) analyses of variance were conducted on VPA difference scores within each experimental condition. There was no significant effect of order on VPA responses in either the Immediate, Delayed, or Residual condition, and no significant interaction between session and order in any of the experimental conditions.

**Vaginal blood volume.** Deviation scores in blood volume were compared using a Session (Exercise vs. No-exercise) x Condition (Immediate vs. Delayed vs. Residual) analysis of variance. Mean VBV deviation scores during the Exercise and No-exercise sessions are presented in Figure 2. Results revealed a significant main effect of exercise on VBV deviation.
The No-exercise and Exercise conditions of Experiment 1, measured at 5 min, 15 min, and 30 min post-exercise.

Figure 1. Mean vaginal pulse amplitude (millimeters of pen deflection + SEM) between neutral and erotic stimulus presentations during the No-exercise and Exercise conditions of Experiment 1.
Figure 2. Mean vaginal blood volume (millivolts deviation from baseline) ± SEM between neutral and erotic stimulus presentations during the No-exercise and Exercise conditions of Experiment 1, measured at 5 min, 15 min, and 30 min post-exercise.
scores, $F(1, 33) = 5.50$, $p = .025$, a significant main effect of condition on VBV deviation scores, $F(2, 33) = 4.45$, $p = .020$, and a significant interaction between session and condition, $F(2, 33) = 5.57$, $p = .008$. A follow-up one-way analysis of variance between VBV deviation scores during the No-exercise sessions revealed no significant difference in VBV deviation scores between the No-exercise Immediate, Delayed, or Residual conditions ($F<1$). A follow-up one-way analysis of variance between VBV deviation scores during the Exercise sessions revealed significant differences in VBV deviation scores across conditions, $F(2, 33) = 5.60$, $p = .008$. Newman Keuls tests with a significance level set at $p<.05$ indicated a significant difference in VBV deviation scores between the Immediate and Residual conditions. Planned follow-up t-tests were conducted on VBV deviation scores between the No-exercise and Exercise conditions in each of the Immediate, Delayed, and Residual conditions. Due to accumulating Type I error on mean comparisons across the variables, only mean differences of $p<.02$ ($p<.05/3$) should be considered statistically reliable. Exercise produced a marginally significant increase in VBV deviation scores during the Residual condition, $t(11) = -2.55$, $p = .027$, and showed a trend toward increasing VBV responses during the Delayed condition, $t(11) = -2.07$, $p = .063$. Exercise had no significant effect on VBV scores during the Immediate condition. Mean VBV deviation scores are presented in Figure 2.

In order to verify that the erotic films facilitated VBV responses, one-tailed, paired samples t-tests were conducted on VBV raw scores between neutral and erotic films within each experimental condition and session. Due to accumulating Type I error on mean comparisons across the variables, only mean difference of $p<.008$ ($p<.05/6$) should be considered statistically reliable. Results revealed a marginally significant increase in blood volume responses with exposure to an erotic film in the No-exercise Residual, $t(11) = -2.63$, $p<.01$, Exercise Residual, $t(11) = -3.02$, $p<.01$, and Exercise Delayed, $t(11) = -2.66$, $p<.01$, conditions, and a trend toward increasing VBV responses to erotic stimuli in the No-exercise...
Immediate, \( t(11) = -2.18, p < .05 \), and No-exercise Delayed, \( t(11) = -1.95, p < .05 \), conditions. The erotic films failed to increase VBV responses during the Exercise Immediate condition.

To determine whether order of experimental sessions influenced VBV responses, Session (Exercise vs. No-exercise) x Order (Exercise, No-exercise vs. No-exercise, Exercise) analyses of variance were conducted on VBV deviation scores within each experimental condition. There was no significant effect of order on vaginal blood responses in either the Immediate, Delayed, or Residual condition, and no significant interaction between session and order in any of the experimental conditions.

**Heart rate.** A repeated-measures Session (Exercise vs. No-exercise) x Film (neutral vs. erotic) analysis of variance of heart rate was conducted within each experimental condition to examine whether heart rate was altered with exposure to exercise and/or an erotic film. Mean heart rates for each of the experimental conditions, sessions, and films are presented in Table II. Results revealed a significant increase in heart rate with exposure to exercise during each of the Immediate, \( F(1, 11) = 40.13, p < .001 \); Delayed, \( F(1, 11) = 51.91, p < .001 \); and Residual conditions, \( F(1, 11) = 21.05, p < .001 \). No difference in heart rate was found between neutral and erotic films in either the Immediate, Delayed, or Residual conditions (all \( F \)s < 1), and there was no significant interaction between session and film for any of the experimental conditions.

Results from a one-way analysis of variance indicated that there were no significant differences in mean heart rate across films, between conditions during the No-Exercise sessions, (\( F < 1 \)), but significant differences in heart rate between conditions during the Exercise sessions, \( F(2, 33) = 7.08, p = .003 \). Post-hoc analyses using Newman Keuls tests with a significance level of \( p < .05 \) revealed differences in heart rate between the Exercise Immediate and Exercise Delayed conditions, and between the Exercise Immediate and Exercise Residual conditions. There was no significant difference in heart rate between the Exercise Delayed and Exercise Residual conditions.
Table II.
Mean (±SEM) Heart Rate Responses to Neutral and Erotic Films During the No-exercise and Exercise Sessions of Subjects in the Immediate, Delayed, and Residual Conditions of Experiment 1.

<table>
<thead>
<tr>
<th>Condition</th>
<th>No-exercise neutral film</th>
<th>No-exercise erotic film</th>
<th>Exercise neutral film</th>
<th>Exercise erotic film</th>
</tr>
</thead>
<tbody>
<tr>
<td>Immediate</td>
<td>70.1±/-2.8</td>
<td>70.2±/-2.7</td>
<td>96.0±/-3.4</td>
<td>97.2±/-2.7</td>
</tr>
<tr>
<td>Delayed</td>
<td>67.9±/-2.2</td>
<td>68.5±/-2.0</td>
<td>87.4±/-2.5</td>
<td>86.9±/-2.5</td>
</tr>
<tr>
<td>Residual</td>
<td>70.1±/-3.2</td>
<td>70.2±/-3.1</td>
<td>80.9±/-3.6</td>
<td>80.5±/-3.6</td>
</tr>
</tbody>
</table>

*Note: Heart rate means are based on raw scores averaged across the entire 180s of neutral and 180s of erotic film stimuli, during each experimental condition and session.*
Analyses of the Relationship Between Vaginal Blood Volume and Vaginal Pulse Amplitude Responses

In order to determine the relationship between blood volume and pulse amplitude responses, Pearson product-moment correlation coefficients were calculated between VPA difference scores and VBV deviation scores of the middle 20s of erotic film, during both the No-exercise and Exercise sessions (due to the small sample size, correlations were conducted across experimental conditions). There was a significant correlation between VBV and VPA responses during the Exercise, \( r(36) = .57, \ p < .001 \), but not the No-exercise, \( r(36) = .26, \ p = .12 \) condition.

Analyses of Subjective Measures

Subjective ratings of sexual arousal, autonomic arousal, anxiety, positive affect, and negative affect in response to erotic stimuli were analyzed using 2 x 3 (Session x Condition) analyses of variance. Mean subjective ratings are presented in Table III. There were no significant effects of session or condition, nor were there significant interactions between session and condition on any of the subjective measures.

Separate paired samples t-tests were conducted between subjective ratings of sexual arousal during the Exercise condition and responses to each of the following three questions: "Given the appropriate situation, what is the highest level of sexual arousal that you think you could experience?", "What is the highest level of sexual arousal that you have ever experienced?", and "What is the highest level of sexual arousal that you think that you could experience viewing an erotic film alone?". The purpose was to examine whether the levels of reported subjective arousal might represent the highest levels that could be expected given the experimental stimuli used, or whether they might represent the highest levels of sexual arousal that subjects were willing to admit to. Mean subjective ratings of predicted/past sexual arousal are presented in Table IV. Due to accumulating Type I error on mean comparisons across the variables, only mean differences of \( p < .006 \ (p < .05/9) \) should be
### Table III.
Mean (+/- SEM) Subjective Ratings of Sexual Arousal, Autonomic Arousal, Positive Affect, Negative Affect, and Anxiety During the No-Exercise and Exercise Sessions of Subjects in the Immediate, Delayed, and Residual Conditions of Experiment 1.

<table>
<thead>
<tr>
<th>Condition</th>
<th>Session</th>
<th>Sexual</th>
<th>Autonomic</th>
<th>Pos. Affect</th>
<th>Neg. Affect</th>
<th>Anxiety</th>
</tr>
</thead>
<tbody>
<tr>
<td>Immediate</td>
<td>No-exercise</td>
<td>4.1 +/- 0.4</td>
<td>3.0 +/- 0.3</td>
<td>3.2 +/- 0.5</td>
<td>1.4 +/- 0.1</td>
<td>1.7 +/- 0.4</td>
</tr>
<tr>
<td>Immediate</td>
<td>Exercise</td>
<td>3.8 +/- 0.5</td>
<td>2.9 +/- 0.3</td>
<td>3.1 +/- 0.5</td>
<td>1.4 +/- 0.1</td>
<td>1.8 +/- 0.4</td>
</tr>
<tr>
<td>Delayed</td>
<td>No-exercise</td>
<td>4.2 +/- 0.4</td>
<td>2.9 +/- 0.3</td>
<td>3.5 +/- 0.4</td>
<td>1.4 +/- 0.1</td>
<td>1.3 +/- 0.4</td>
</tr>
<tr>
<td>Delayed</td>
<td>Exercise</td>
<td>4.0 +/- 0.5</td>
<td>3.1 +/- 0.3</td>
<td>3.4 +/- 0.4</td>
<td>1.3 +/- 0.1</td>
<td>1.3 +/- 0.4</td>
</tr>
<tr>
<td>Residual</td>
<td>No-exercise</td>
<td>3.7 +/- 0.5</td>
<td>2.8 +/- 0.4</td>
<td>2.5 +/- 0.3</td>
<td>1.6 +/- 0.5</td>
<td>1.5 +/- 0.4</td>
</tr>
<tr>
<td>Residual</td>
<td>Exercise</td>
<td>3.9 +/- 0.5</td>
<td>3.0 +/- 0.4</td>
<td>2.9 +/- 0.3</td>
<td>1.4 +/- 0.4</td>
<td>1.3 +/- 0.4</td>
</tr>
</tbody>
</table>

*Note: Means are based on an item response format of low (1) to intense (7).*
Table IV.
Mean (+/-SEM) Subjective Ratings of Predicted and Past Sexual Arousal of Subjects in the Immediate, Delayed, and Residual Conditions of Experiment 1.

<table>
<thead>
<tr>
<th>Item</th>
<th>Immediate</th>
<th>Delayed</th>
<th>Residual</th>
</tr>
</thead>
<tbody>
<tr>
<td>Given the appropriate situation, what is the highest level of sexual arousal that you think that you could experience?</td>
<td>6.4+/-0.3</td>
<td>6.9+/-0.1</td>
<td>6.9+/-0.1</td>
</tr>
<tr>
<td>What is the highest level of sexual arousal that you have ever experienced?</td>
<td>6.2+/-0.3</td>
<td>6.8+/-0.1</td>
<td>6.6+/-0.3</td>
</tr>
<tr>
<td>What is the highest level of sexual arousal that you think that you could experience viewing an erotic film alone?</td>
<td>4.4+/-0.3</td>
<td>4.6+/-0.3</td>
<td>4.2+/-0.4</td>
</tr>
</tbody>
</table>

Note: Means are based on an item response format of low (1) to intense (7).
considered statistically reliable. There were significant differences between subjective ratings of sexual arousal and ratings of the highest level of sexual arousal believed possible in each of the Immediate, \(t(11) = 4.99, p<.001\), Delayed, \(t(11) = 5.74, p<.001\), and Residual, \(t(11) = 6.47, p<.001\), conditions. There were also significant differences between subjective ratings of sexual arousal and ratings of the highest level of sexual arousal previously attained in each of the Immediate, \(t(11) = 4.06, p<.002\), Delayed, \(t(11) = 5.45, p<.001\), and Residual, \(t(11) = 5.17, p<.001\), conditions. No significant differences were noted between subjective ratings of sexual arousal and ratings of the highest expected level of arousal to an erotic film. A Pearson correlation revealed a significant relationship between subjective ratings of sexual arousal and ratings of expected levels of arousal to an erotic film, \(r(36) = .41, p=.01\).

**Analyses of the Relationship Between Physiological and Subjective Sexual Responses**

Pearson product-moment correlation coefficients were calculated separately for the No-exercise and Exercise experimental sessions in order to investigate the degree of association between physiological and subjective ratings of sexual arousal (due to the small sample size, correlations were conducted across experimental conditions). There was a significant correlation between pulse amplitude responses and subjective ratings during the No-exercise sessions, \(r(36) = .55, p < .001\), but no significant correlation between pulse amplitude responses and subjective ratings during the Exercise conditions, \(r(36) = .23, p = .18\). There were no significant correlations between blood volume responses and subjective ratings during either the No-exercise, \(r(36) = .03, p = .87\), or Exercise, \(r(36) = .22, p = .21\), condition.

**Discussion**

The present investigation examined the effects of SNS activation, via acute exercise, on subjective and physiological sexual arousal in women. Exercise was used to elicit SNS activity based on evidence which suggests that exercise, at the intensity, duration, and time frame used in the present study, elicits significant SNS arousal (e.g., Cleroux et al., 1985;
Kraemer et al., 1991; Strobel et al., 1993). Indirect support for the effectiveness of the experimental procedures and controls in eliciting SNS activity was provided by the finding that heart rate remained significantly elevated during the Exercise versus No-exercise sessions in each of the Immediate, Delayed, and Residual conditions. The finding that heart rate was significantly higher during the Immediate Exercise than the Residual Exercise condition suggests that, as intended, levels of SNS activity had declined to markedly lower levels post-exercise. The finding that the erotic films elicited significant increases in VPA during each of the experimental conditions indicates that the experimental stimuli were successful in altering physiological sexual arousal. Analyses of order effects revealed that order of presentation of experimental conditions did not influence physiological measures of sexual arousal. In other words, familiarity with the experimental procedures and/or with viewing an erotic film, did not influence subjects' responses. Together, these results suggest that the experimental manipulations and controls were effective.

In the presence of an erotic stimulus, acute exercise inhibited physiological sexual arousal when measured immediately following exercise, and facilitated physiological sexual arousal when measured 15 or 30 min post-exercise. These effects included a marginally significant decrease in VPA at 5 min post-exercise, a significant increase in VPA and a trend toward increasing VBV at 15 min post-exercise, and a marginally significant increase in VBV and a trend toward increasing VPA responses at 30 min post-exercise. These findings suggest that there is an optimal level of SNS activation for facilitation of physiological sexual arousal in women.

In contrast to the reported changes in physiological sexual arousal with exposure to exercise, exercise had no significant effect on subjective ratings of sexual arousal in either of the Immediate, Delayed, or Residual conditions. These findings replicate those of Meston and Gorzalka (1995) who found significant increases in physiological but not subjective ratings of sexual arousal at 15 min post-exercise. Exercise also had no significant effect on subjective
ratings of either positive affect, negative affect, or anxiety in any of the experimental conditions. This suggests that the exercise-induced changes in physiological sexual arousal cannot be explained exclusively in terms of cognitive factors. That is, the reported increases or decreases in physiological sexual arousal cannot be attributed to differences in subjects' mood with exposure to exercise, or to a positive feedback system between cognitive and physiological components of the sexual response.

EXPERIMENT 2

The Effects of Sympathetic Inhibition During Heightened Nervous System Arousal on Subjective and Physiological Sexual Arousal in Women

The results of Experiment 1 replicated the finding that acute exercise significantly increases physiological sexual responses to an erotic stimulus when measured at 15 min post-exercise (Meston and Gorzalka, 1995). Experiment 2 was designed to examine the effects of SNS inhibition on sexual arousal in women, and to determine whether the sexually facilitatory influence of exercise can be attributed primarily to SNS activation or to the most likely alternative, the hormonal consequences of exercise. By having subjects exercise in each of two experimental conditions, and blocking SNS activity in only one condition, Experiment 2 allowed for comparison of the hormonal and SNS influences of exercise with the hormonal influences of exercise alone.

In Experiment 2, SNS activity was blocked using the commonly prescribed antihypertensive medication clonidine. Clonidine was chosen as a means of inhibiting SNS activity based on numerous studies which indicate that the drug's hypotensive effects are attributable to central stimulation of alpha-2-adrenoreceptors which, in turn, causes a significant decrease in peripheral sympathetic outflow (e.g., Engelman et al., 1989; Flacke et al., 1987; Kooner, Birch, Frankel, Peart, & Mathias, 1991; Maze & Tranguilli, 1991; Quintin et al., 1991). The reduction in sympathetic activity following clonidine administration has been documented using both indirect measures, such as plasma catecholamine levels (e.g.,
Engelman et al., 1989; Flacke et al., 1987; Kooner et al., 1991; Maze & Tranguilli, 1991; Quintin et al., 1991), and direct measures, such as sympathetic nerve recordings (e.g., Muzzi, Goff, Kampine, Roerig, & Ebert, 1992). Several studies also indicate that acute, moderate doses of clonidine suppress peripheral sympathetic responses to exercise (e.g., Joffe et al., 1986; Maurer, Hausen, Kramer, & Kubler, 1983; Virtanen, Janne, & Frick, 1982), and that they do so without altering central alpha-adrenergic-mediated hormonal responses to exercise (Joffe et al., 1986). In addition, clonidine has been reported to have no effect on hormonal levels of either prolactin (e.g., Lal, Tolis Martin, Brown, & Guyda, 1975; Schindler, Muller, Keller, Goser, & Runkel, 1979), follicle stimulating hormone (e.g., Lal et al., 1975; Lanes, Herrera, Palacios, & Moncada, 1983), luteinizing hormone (e.g., Lal et al., 1975; Lanes et al., 1983), thyroid stimulating hormone (e.g., Lal et al., 1975), estrogen (e.g., Schindler et al., 1979), or testosterone (e.g., Boyar et al., 1980).

**Method**

**Subjects**

Fifteen sexually functional women (M age = 23.40 years, range = 19-35) participated in two experimental conditions, Clonidine and Placebo. The subjects were recruited via advertisements, in the university and local Vancouver newspapers, which requested volunteers for the current experiment. All subjects were employed in professions outside the university. Racial background of the subjects was: Caucasian (14), and African (1). All subjects were currently involved in sexual relationships; one subject was married. Subjects were paid $40.00 for their participation. Initial telephone screening criteria were: between the ages of 18-45 years, no use of medications known to affect vascular or sexual functioning, no history of treatment for sexual dysfunction, no medical condition that may put the subject at risk when exercising, and current involvement in a heterosexual relationship. Further inclusion criteria, based on subject information from the DSFI, the OFQ, a brief medical history questionnaire, the Physical Readiness Exam for Fitness Test, and a general
cardiovascular examination included: absence of general psychopathology, absence of sexual
dysfunction, absence of anorgasmia, within the normative range of sexual experience, no
history of heart disease or cardiovascular dysfunction, no history of high or low blood
pressure, no history of dizzy spells or "light-headedness", and no bone or joint problem that
might be aggravated by 20 min of cycling.

Profile descriptions of all subjects were obtained via the DSFI and the OFQ. Mean
scores by experimental condition for the DSFI subscales and the OFQ are presented in Table
V. All subjects employed in the study scored greater than or equal to the 30th percentile
(i.e., within two standard deviations of the normative mean) on the SFI, GSSI, BSI, and the
Drive and Experience subscales of the DSFI. Data from the OFQ was used to screen for
absence of orgasmic dysfunction. All subjects employed in the study were able to achieve
orgasm by some means (e.g., intercourse, oral sex, masturbation) on at least 50 percent of the
attempted trials.

Two subjects were eliminated from the study because they scored below the cutoff
criterion for general psychopathology, and one subject was eliminated because she did not
pass the general cardiovascular examination. Fifteen subjects met all inclusion criteria and
served as subjects in the study.

Design and Procedure

The procedure consisted of three sessions: a 2-hr orientation screening and questionnaire
session, a 2-hr clonidine-exercise experimental condition, and a 2-hr placebo-
exercise experimental condition. Order of the two experimental conditions, Clonidine and
Placebo, was counterbalanced across subjects (7 subjects received the Placebo condition
prior to the Clonidine condition). During each experimental session, subjects viewed one of
two 7-min videotaped sequences. Each sequence consisted of a 1-min display of the word
"relax" followed by a 3-min neutral travelogue film and then a 3-min erotic film. The films
Table V.
Psychometric Characteristics of Subjects in Experiment 2.

<table>
<thead>
<tr>
<th>Measure</th>
<th>Mean/SD</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>DSFI Subtests</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Information</td>
<td>51.1 +/-1.8</td>
<td>38-68</td>
</tr>
<tr>
<td>Experience</td>
<td>48.5 +/-2.7</td>
<td>30-63</td>
</tr>
<tr>
<td>Drive</td>
<td>55.0 +/-3.2</td>
<td>36-71</td>
</tr>
<tr>
<td>Attitude</td>
<td>31.5 +/-1.0</td>
<td>24-37</td>
</tr>
<tr>
<td>Symptoms (BSI)</td>
<td>43.6 +/-2.3</td>
<td>30-61</td>
</tr>
<tr>
<td>Affect</td>
<td>46.7 +/-2.3</td>
<td>36-61</td>
</tr>
<tr>
<td>Gender Role</td>
<td>55.3 +/-1.7</td>
<td>40-60</td>
</tr>
<tr>
<td>Fantasy</td>
<td>58.1 +/-3.7</td>
<td>35-80</td>
</tr>
<tr>
<td>Body Image</td>
<td>38.5 +/-2.8</td>
<td>28-66</td>
</tr>
<tr>
<td>Satisfaction</td>
<td>48.0 +/-2.2</td>
<td>37-63</td>
</tr>
<tr>
<td><strong>DSFI Global Scores</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SFI</td>
<td>54.3 +/-3.3</td>
<td>33-74</td>
</tr>
<tr>
<td>GSSI</td>
<td>53.0 +/-2.7</td>
<td>35-70</td>
</tr>
<tr>
<td>OFQ</td>
<td>87 +/-5.2</td>
<td>50-100</td>
</tr>
</tbody>
</table>

*Note: DSFI = Derogatis Sexual Functioning Inventory, OFQ = Orgasmic Functioning Questionnaire, BSI = Brief Symptom Inventory, SFI = Sexual Functioning Index, GSSI = Global Sexual Satisfaction Index. Means for Derogatis Sexual Functioning Inventory items are based on raw scores that were converted to established percentile rankings (T scores). Means for the OFQ are based on the highest estimated percentage of trials on which a subject achieved orgasm by any method.*
were identical to those used by Meston and Gorzalka (1995), and to those used in Experiment 1.

The two experimental sessions were scheduled at approximately one week intervals and excluded times during which the subjects were menstruating. To control for daily circadian fluctuations which may influence drug sensitivity, subjects were scheduled at approximately the same time during both sessions (i.e., either morning, afternoon, or evening). All subjects were asked to abstain from psychoactive drugs (including caffeine and alcohol) and to refrain from engaging in any strenuous physical activity for 24 hr prior to each experimental session. Because drug metabolism may be influenced by food in the stomach, subjects were also asked to refrain from eating for four hours prior to the experimental sessions.

Session 1 (orientation/screening). Following an initial telephone screening, subjects were scheduled for a first session with the female experimenter. During this session, subjects were shown the laboratory facilities and equipment, were given verbal instructions on the use of the photoplethysmograph, were informed of the plethysmograph sterilization procedures, and were encouraged to ask any questions related to the experiment. Subjects were told that they would be participating in an experiment which examines the effects of clonidine, a drug commonly used in the treatment of hypertension, on female sexual arousal. They were informed of the risks and reported side effects of clonidine, and told that, in order to measure sexual arousal, they would view brief visual stimuli, some of which may include erotic content. To minimize a possible sense of coercion, subjects were given the option of either participating in the first session on that day, or telephoning within a week, regarding their decision to participate. All subjects chose to begin the study that day.

After signing the standard consent form, subjects were asked to complete a brief medical history questionnaire (designed to screen for subjects with cardiovascular dysfunction) and the Physical Readiness Exam for Fitness Test. Subjects whose medical
histories indicated that they would not be put at risk when exercising and/or taking clonidine were then given a cardiovascular examination by a fourth year medical student from the Faculty of Medicine, University of British Columbia. The examination was conducted in both a supine and standing position, and covered the following: peripheral pulse, ankle swelling, heart sounds, apex beat, jugular venous pressure, breathing/chest sounds. Subjects who were not considered at risk when exercising and/or taking clonidine were then asked to complete the DSFI and OFQ in a private room. After completing the questionnaires, subjects took a 10 min break and then began Session 2.

Sessions 2 and 3 (experimental). The second and third sessions were the two experimental sessions: Clonidine and Placebo. Both experimental sessions were conducted inside the Sexual Psychophysiology Laboratory at the University of British Columbia. During the experimental sessions, subjects were given either a placebo (icing sugar) or clonidine (0.2 mg, mixed with icing sugar) capsule. Both capsules were taken orally with 250 ml of water. Neither the subject nor the experimenter knew in which session the subject received the placebo and in which session she received clonidine. Subjects' heart rates and blood pressure were monitored using an oscillometric electronic digital blood pressure and pulse monitor (Omron Healthcare Inc., Vernon Hill, IL) 10 min prior to drug administration and every 15 min for 60 min after the drug had been ingested. Both systolic and diastolic blood pressure measures were taken. These measures were taken to assess whether clonidine influenced subjects' resting levels of heart rate and blood pressure. Reports on the effects of clonidine on resting levels of blood pressure and heart rate in normotensive persons have been mixed. Acute doses of clonidine, higher than those employed in the present study (i.e., 1.5 mg - 3.0 mg), have been shown to significantly decrease resting levels of systolic blood pressure (e.g., Joffe et al., 1986; Maurer et al., 1983; Wing, Reid, Hamilton, Sever, Davies, & Dollery, 1977; Yeragani, Pohl, Balon, & Berchou, 1992), decrease resting levels of diastolic blood pressure (e.g., Joffe et al., 1986; Wing et al., 1977; Yeragani et al, 1992; ), and decrease
resting heart rate (e.g., Maurer et al., 1983), although some studies fail to reveal an effect of clonidine (e.g., Ceremuzynski, Lada, Maruchin, Dluzniewski, & Herbaczynska-Cedro, 1979; Yeragani et al., 1992). The 1-hr waiting period following drug administration was used to ensure that the pharmacological effects of clonidine had taken place. During all heart rate and blood pressure recordings, the subject was seated comfortably inside the Sexual Psychophysiology Laboratory.

During both experimental sessions, one hour following clonidine/placebo administration, subjects engaged in 20 min of stationary cycling prior to viewing the films. During the first session, subjects were asked to cycle at a constant 70% of their maximum heart rate. Subjects were given continual feedback on their heart rate levels, and were asked to cycle faster or slower if their heart rate indicated they were below or above the required exertion level. Because clonidine has in some cases (e.g., Maurer et al., 1983) been shown to decrease heart rate responses to acute exercise, subjects' workload and cycle speed (rpm) were recorded during the first session, and subjects were asked to cycle at the same speed and intensity during the second exercise session. This procedure was used to ensure that subjects were exercising at equivalent intensities during the Clonidine and Placebo conditions.

When one min of cycling time remained, the experimenter left the room. Subjects were instructed to continue cycling until the timer signaled 20 min, then remove any clothing necessary to insert the plethysmograph, and then sit in the chair, cover themselves with a blanket, and insert the plethysmograph so as to allow approximately a 2.5 cm distance between the end of the probe and the vaginal opening. They were also asked to remain as still as possible throughout the session in order to minimize potential movement artifacts. They were asked to notify the experimenter, via the intercom system, when they were ready, at which time a 10-min adaptation recording was taken in order to allow the plethysmograph time to adapt to subjects' body temperatures. This procedure is identical to that used by Meston and Gorzalka (1995), and was conducted in order to further minimize potential light
and heating effects. Following the adaptation period, subjects viewed one of the two videotaped sequences. Each sequence consists of the word "relax" (1 min), a neutral travelogue (3 min), followed by an erotic film (3 min). Immediately following the erotic film, subjects were asked to fill out the subjective rating scale. With the exception of the capsule contents, experimental procedures for the Clonidine and Placebo conditions were identical. At the end of the second experimental session, subjects were thoroughly debriefed, informed about the additional purposes and goals of the study, and given an opportunity to view the records of their vaginal responses. All subjects were paid $40.00 for their participation.

**Data Sampling and Reduction**

**Physiological Measurements**

- **Vaginal pulse amplitude and vaginal blood volume.** All data sampling and reduction procedures for both VBV and VPA were identical to those used in Experiment 1.

- **Heart rate.** Heart rate prior to the film presentations was monitored using an oscillometric electronic digital blood pressure and pulse monitor 10 min prior to drug administration and every 15 min for 60 min following clonidine/placebo ingestion. These scores yielded five measures (bpm) for each subject per experimental condition. Heart rate during film presentations was scored from the VPA polygraph records as in Experiment 1.

- **Blood pressure.** Systolic and diastolic blood pressure were monitored using an oscillometric electronic digital blood pressure and pulse monitor 10 min prior to drug administration and every 15 min for 60 min following clonidine/placebo ingestion. These scores yielded five systolic and five diastolic blood pressure measures for each subject per experimental condition.

**Subjective Measurements**

Data sampling and reduction procedures for all subjective measures were identical to those used in Experiment 1.
Results

Analyses of Physiological Measures

Vaginal pulse amplitude. To examine whether clonidine influenced VPA scores during either the neutral or erotic films, and to determine whether the erotic films facilitated VPA responses, a Condition (Clonidine vs. Placebo) x Film (neutral vs. erotic) analysis of variance was conducted on VPA raw scores. Results revealed a significant effect of the erotic films on VPA scores, $F(1, 14) = 39.38, p < .001$, a marginally significant effect of clonidine on VPA scores, $F(1, 14) = 4.15, p = .061$, and a significant interaction between condition and film, $F(1, 14) = 10.99, p = .005$. Planned, paired-samples t-tests were conducted between the Clonidine and Placebo conditions during each of the neutral and erotic films. Due to accumulating Type I error on mean comparisons across the variables, only mean differences of $p < .025$ ($p < .05/2$) should be considered statistically reliable. There was no significant difference in VPA scores during the neutral films, but a significant decrease in VPA scores with clonidine administration during the erotic films, $t(14) = 2.57, p = .022$. Eleven of 15 subjects showed a decrease in VPA responses to erotic stimuli with clonidine administration. Mean VPA difference scores during the Clonidine and Placebo conditions are presented in Figure 3.

To determine whether order of experimental conditions influenced VPA responses, a Condition (Clonidine vs. Placebo) x Order (Clonidine, Placebo vs. Placebo, Clonidine) analysis of variance was conducted on VPA difference scores. There was no significant effect of order on VPA responses and no significant interaction between condition and order.

Vaginal blood volume. A paired samples t-test conducted on VBV deviation scores revealed a significant decrease in VBV with clonidine administration, $t(14) = 5.40, p < .001$. All of 15 subjects showed a decrease in VBV responses to erotic stimuli with clonidine administration. In order to verify that the erotic films facilitated VBV responses, one-tailed, paired-samples t-tests were conducted on VBV raw scores between neutral and erotic films.
Following exercise, during the Clonidine and Placebo conditions of Experiment 2.

Figure 3. Mean subjective ratings of sexual arousal, autonomic arousal, positive affect, negative affect, and anxiety in response to erotic stimuli during placebo and Clonidine conditions.
within each experimental condition. Due to accumulating Type I error on mean comparisons across the variables, only mean differences of $p<.025$ ($p<.05/2$) should be considered statistically reliable. Results revealed a significant increase in blood volume responses with exposure to an erotic film in both the Clonidine, $t(14) = 6.45$, $p < .001$, and Placebo, $t(14) = 7.35$, $p < .001$ conditions. Mean VBV deviation scores during the Clonidine and Placebo conditions are presented in Figure 4.

To examine whether order of experimental conditions influenced VBV responses, a Condition (Clonidine vs. Placebo) x Order (Clonidine, Placebo vs. Placebo, Clonidine) analysis of variance was conducted on VBV deviation scores. There was no significant effect of order on VBV responses and no significant interaction between condition and order.

**Heart rate.** To examine whether heart rate was influenced with clonidine administration during the 1-hr waiting period prior to exercise and film presentations, a Condition (Clonidine vs. Placebo) x Time (4 time blocks following clonidine/placebo administration) analysis of variance of heart rate was conducted. There were no significant effects of either clonidine or time on heart rate levels, and no significant interaction between condition and time. To ensure there were no differences between conditions in resting levels of heart rate prior to clonidine/placebo administration, a paired-samples t-test was conducted on heart rate levels measured 10 min prior to drug administration. There was no significant difference in resting heart rate levels between conditions. Mean heart rates, prior to and following clonidine/placebo administration, for each of the experimental conditions are presented in Table VI.

A Condition (Clonidine vs. Placebo) x Film (neutral vs. erotic) analysis of variance of heart rate was conducted to examine whether heart rate (following exercise and during film presentation) was altered with clonidine administration and/or an erotic film. Results revealed a significant decrease in heart rate with clonidine administration, $F(1, 14) = 4.85$, $p = .045$. There was no significant difference in heart rate between neutral and erotic films, and
Figure 4. Mean vaginal pulse amplitude (millimeters of pen deflection) + SEM between neutral and erotic stimulus presentations during the Clonidine and Placebo conditions of Experiments 2 and 3.
Table VI.
Mean (+/- SEM) Heart Rate Responses Prior to and Following Drug Administration During the Clonidine and Placebo Conditions of Experiments 2 and 3.

<table>
<thead>
<tr>
<th>Condition</th>
<th>Pre Drug</th>
<th>15 min</th>
<th>30 min</th>
<th>45 min</th>
<th>60 min</th>
</tr>
</thead>
<tbody>
<tr>
<td>Experiment 2</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Placebo</td>
<td>61.9 +/- 3.4</td>
<td>61.7 +/- 3.3</td>
<td>64.9 +/- 2.1</td>
<td>65.5 +/- 2.8</td>
<td>65.4 +/- 2.9</td>
</tr>
<tr>
<td>Clonidine</td>
<td>64.8 +/- 2.1</td>
<td>68.2 +/- 2.0</td>
<td>68.2 +/- 2.0</td>
<td>68.2 +/- 2.2</td>
<td>66.2 +/- 2.8</td>
</tr>
<tr>
<td>Experiment 3</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Placebo</td>
<td>66.8 +/- 3.2</td>
<td>64.1 +/- 2.4</td>
<td>62.3 +/- 2.5</td>
<td>65.4 +/- 2.9</td>
<td>67.7 +/- 2.5</td>
</tr>
<tr>
<td>Clonidine</td>
<td>65.2 +/- 3.2</td>
<td>66.4 +/- 4.2</td>
<td>63.8 +/- 2.7</td>
<td>62.1 +/- 3.2</td>
<td>62.4 +/- 4.0</td>
</tr>
</tbody>
</table>

Note: Heart rate recordings were taken 10 min prior to clonidine/placebo administration and 15, 30, 45, and 60 min following drug ingestion.
no significant interaction between condition and film. Mean heart rates during film presentations for each of the experimental conditions and films are presented in Table VII.

**Blood pressure.** Condition (Clonidine vs. Placebo) x Time (4 time blocks following clonidine/placebo administration) analyses of variance of blood pressure were conducted separately for systolic and diastolic measures. These analyses were conducted in order to examine whether clonidine influenced subjects' resting levels of blood pressure. Results revealed no significant effect of either clonidine or time on resting systolic or diastolic blood pressure levels, and no significant interaction between condition and time for either of the measures. To ensure there were no differences between conditions in resting levels of blood pressure prior to clonidine/placebo administration, paired-samples t-tests were conducted on systolic and diastolic blood pressure levels measured 10 min prior to drug administration. There were no significant differences between conditions in resting levels of either systolic or diastolic blood pressure. Mean systolic and diastolic blood pressure levels, prior to and following clonidine/placebo administration, for each of the experimental conditions are presented in Table VIII.

**Analyses of the Relationship Between Vaginal Blood Volume and Vaginal Pulse Amplitude Responses**

In order to determine the relationship between physiological sexual responses, Pearson product-moment correlation coefficients were calculated between VPA difference scores and VBV deviation scores of the middle 20 s of erotic film, during both the Clonidine and Placebo condition. There was a significant correlation between VBV and VPA responses during the Placebo, \( r(15) = .74, p = .001 \), but not Clonidine, \( r(15) = .19, p = .49 \), condition.

**Analyses of Subjective Measures**

A paired-samples t-tests between Clonidine and Placebo conditions was conducted on subjective ratings of sexual arousal. Results revealed a significant decrease in subjective sexual arousal with clonidine administration, \( t(14) = 2.27, p = .04 \). A Condition (Clonidine
Table VII.
Mean (+/- SEM) Heart Rate Responses to Neutral and Erotic Films During the Clonidine and Placebo Conditions of Experiments 2 and 3.

<table>
<thead>
<tr>
<th>Condition</th>
<th>Experiment 2</th>
<th></th>
<th>Experiment 3</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>neutral film</td>
<td>erotic film</td>
<td>neutral film</td>
</tr>
<tr>
<td>Placebo</td>
<td>82.6 +/- 3.2</td>
<td>81.5 +/- 3.4</td>
<td>64.2 +/- 2.5</td>
</tr>
<tr>
<td>Clonidine</td>
<td>73.8 +/- 3.7</td>
<td>73.1 +/- 3.8</td>
<td>62.3 +/- 3.1</td>
</tr>
</tbody>
</table>

*Note: Heart rate means are based on raw scores averaged across the entire 180s of neutral and 180s of erotic film stimuli, during each experimental condition and session.*
Table VIII.
Mean (+/- SEM) Systolic and Diastolic Blood Pressure Responses Prior to and Following Drug Administration During the Clonidine and Placebo Conditions of Experiments 2 and 3.

<table>
<thead>
<tr>
<th>Condition</th>
<th>Pre Drug</th>
<th>15 min</th>
<th>30 min</th>
<th>45 min</th>
<th>60 min</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Experiment 2</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SBP (Placebo)</td>
<td>109.5 +/- 3.3</td>
<td>109.6 +/- 3.4</td>
<td>104.8 +/- 2.0</td>
<td>106.9 +/- 3.1</td>
<td>101.8 +/- 2.3</td>
</tr>
<tr>
<td>DBP (Placebo)</td>
<td>72.1 +/- 3.4</td>
<td>70.0 +/- 4.0</td>
<td>64.9 +/- 2.3</td>
<td>69.9 +/- 3.8</td>
<td>59.8 +/- 2.6</td>
</tr>
<tr>
<td>SBP (Clonidine)</td>
<td>109.1 +/- 7.0</td>
<td>101.5 +/- 3.0</td>
<td>103.9 +/- 3.2</td>
<td>105.1 +/- 5.3</td>
<td>104.9 +/- 5.8</td>
</tr>
<tr>
<td>DBP (Clonidine)</td>
<td>72.3 +/- 7.1</td>
<td>64.4 +/- 2.0</td>
<td>66.5 +/- 4.7</td>
<td>70.9 +/- 6.1</td>
<td>69.1 +/- 7.0</td>
</tr>
<tr>
<td><strong>Experiment 3</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SBP (Placebo)</td>
<td>104.1 +/- 3.8</td>
<td>103.1 +/- 4.8</td>
<td>102.2 +/- 3.0</td>
<td>104.3 +/- 3.5</td>
<td>103.1 +/- 3.0</td>
</tr>
<tr>
<td>DBP (Placebo)</td>
<td>66.9 +/- 3.2</td>
<td>66.5 +/- 4.6</td>
<td>65.1 +/- 2.7</td>
<td>63.6 +/- 2.4</td>
<td>62.2 +/- 2.3</td>
</tr>
<tr>
<td>SBP (Clonidine)</td>
<td>110.3 +/- 6.9</td>
<td>102.0 +/- 4.7</td>
<td>104.7 +/- 3.6</td>
<td>98.2 +/- 4.1</td>
<td>102.5 +/- 5.1</td>
</tr>
<tr>
<td>DBP (Clonidine)</td>
<td>78.4 +/- 7.6</td>
<td>62.0 +/- 2.7</td>
<td>69.2 +/- 3.6</td>
<td>60.3 +/- 1.8</td>
<td>68.5 +/- 5.7</td>
</tr>
</tbody>
</table>

*Note:* Blood pressure recordings were taken 10 min prior to clonidine/placebo administration and 15, 30, 45, and 60 min following drug ingestion. SBP = systolic blood pressure, DBP = diastolic blood pressure.
vs. Placebo) x Measure (positive affect vs. negative affect vs. autonomic arousal vs. anxiety) analysis of variance was conducted to examine whether clonidine influenced subjects' mood and perceptions of autonomic changes to erotic stimuli. There was a significant effect of measure on subjective ratings, $F(3, 42) = 10.25, p < .001$, but no significant effect of clonidine on subjective measures of autonomic arousal, anxiety, positive affect, or negative affect, and no significant interaction between condition and measure. Mean subjective ratings are presented in Figure 5.

In order to examine whether the levels of reported subjective arousal might represent the highest levels that could be expected given the experimental stimuli used, or whether they might represent the highest levels of sexual arousal that subjects were willing to admit to, separate paired samples t-tests were conducted between subjective ratings of sexual arousal during the Placebo condition and responses to each of the three predicted arousal questions. Due to accumulating Type I error on mean comparisons across the variables, only mean differences of $p < .02 (p < .05/3)$ should be considered statistically reliable. There were significant differences between subjective ratings of sexual arousal and ratings of the highest level of sexual arousal believed possible, $t(14) = 5.27, p < .001$, and between subjective ratings of sexual arousal and ratings of the highest level of sexual arousal previously attained, $t(14) = 4.00, p = .001$. There was no significant difference between subjective ratings of sexual arousal and ratings of the highest expected level of arousal to an erotic film. A Pearson correlation revealed a significant relationship between subjective ratings of sexual arousal and ratings of expected levels of arousal to an erotic film, $r(15) = .60, p = .017$. Mean ratings of the highest personal level of sexual arousal believed possible, the highest level of sexual arousal ever experienced, and the highest level of sexual arousal believed possible when viewing an erotic film alone were, 6.6 (+/-0.2), 5.7 (+/-0.3), 3.7 (+/-0.4), respectively.
Figure 5: Mean vaginal blood volume (millivolts deviation from baseline) +/- SEM between neutral and erotic stimulus presentations during the Clonidine and Placebo conditions of Experiments 2 and 3.
Analyses of the Relationship Between Physiological and Subjective Sexual Responses

Pearson product-moment correlation coefficients were calculated separately for the Clonidine and Placebo conditions in order to investigate the degree of association between physiological and subjective ratings of sexual arousal. There was a significant correlation between VPA difference scores and subjective sexual arousal during the Placebo, $r(15) = .56$, $p = .032$, but not Clonidine, $r(15) = .17$, $p = .54$, condition. There were no significant correlations between VBV deviation scores and subjective sexual arousal during either the Clonidine, $r(15) = -.21$, $p = .462$, or Placebo, $r(15) = .31$, $p = .254$, condition.

Discussion

The present investigation examined the effects of SNS inhibition, via clonidine administration, on subjective and physiological sexual arousal in women. Clonidine was used to inhibit SNS activity based on evidence which indicates that clonidine acts centrally as an alpha$_2$ adrenergic agonist, and peripherally as a sympathetic outflow blocker (e.g., Engelman et al., 1989; Flacke et al., 1987; Kooner et al., 1991; Maze & Tranguilli, 1991; Quintin et al., 1991). Indirect support for an inhibitory influence of clonidine on SNS activity was provided by the finding that clonidine significantly decreased heart rate responses to exercise. The finding that clonidine influenced VPA responses to erotic but not neutral stimuli verifies that the plethysmograph measured only the sexual, and not other potential "nonsexual", autonomic effects of clonidine. Support for the effectiveness of the experimental films in eliciting sexual arousal was provided by the finding that VPA and VBV responses were significantly increased during both the Clonidine and Placebo conditions. Analyses of order effects indicated that order of presentation of experimental conditions did not influence sexual responding. The above findings suggest that the experimental manipulations and controls used in the present investigation were effective.

When exercising subjects received clonidine, as opposed to placebo, the facilitatory effects of exercise on physiological sexual arousal were suppressed. Because clonidine has
been reported to significantly inhibit SNS responses to exercise (e.g., Engelman et al., 1989; Flacke et al., 1987) without altering most hormonal consequences of exercise (e.g., Joffe et al., 1986). Experiment 2 allowed for comparison of the hormonal and SNS influences of exercise on sexual arousal (i.e., Placebo condition), with the hormonal influences alone (i.e., Clonidine condition). The finding that the facilitatory effects of exercise on VPA and VBV responses were blocked when the SNS, but not hormonal, responses to exercise were blocked, provides support for the assertion that exercise facilitates sexual responding via SNS mechanisms.

In addition to significantly decreasing physiological sexual arousal, clonidine significantly decreased subjective ratings of sexual arousal. The subjective decreases were noted in the absence of significant changes in reports of either positive affect, negative affect, or anxiety. This suggests that the reported decreases in sexual arousal are not likely attributable to cognitive factors such as mood, which may potentially have been altered with clonidine administration. The finding that clonidine significantly decreased subjective and physiological responses without altering mood state, provides the first empirical support for an inhibitory influence of SNS inhibition on sexual arousal in women.

EXPERIMENT 3

The Effects of Sympathetic Inhibition during Baseline Nervous System Arousal on Physiological and Subjective Sexual Arousal in Women

Experiment 2 demonstrated that clonidine inhibited both subjective and physiological sexual arousal when subjects were in a state of heightened nervous system arousal. Experiment 3 was designed to examine the effects of clonidine on female sexual arousal when subjects were at baseline levels of autonomic arousal. Subjects participated in two experimental conditions in which sexual arousal to an erotic film was measured following either clonidine or placebo administration. The purpose is to further examine the effects of
clonidine on sexual responding, and to determine whether the sexually inhibitory effects, reported in Experiment 2, can be attributed primarily to SNS inhibition or, to other potential centrally-mediated, nonspecific effects of clonidine such as sedation. If clonidine fails to significantly influence VBV and VPA responses in Experiment 3, it is unlikely that the inhibitory influence of clonidine reported in Experiment 2 could be explained in terms other than inhibition of the SNS. On the other hand, if clonidine has a similar inhibitory influence on physiological sexual arousal during baseline levels of autonomic arousal as it does during heightened levels of arousal, this would suggest that clonidine may be acting to suppress sexual responding via either central or peripheral mechanisms.

Method

Subjects

Subjects were 15 sexually functional women (M age = 26.67, range = 18–42). Mean scores for the DSFI subscales and the OFQ are presented in Table IX. Two subjects were students at the University of British Columbia, and the remaining 13 subjects were employed in professions outside the university. All subjects were currently involved in heterosexual relationships; none of the subjects were married. Racial background of subjects was: Caucasian (13), Asian (2). The manner in which the subjects were recruited, the measures used to screen the subjects, the subject selection criteria, and the subject payment and debriefing were identical to that used in Experiment 2

One subject was eliminated because she scored below the cutoff criterion for sexual experience, and one subject was eliminated because she did not pass the general cardiovascular examination. Data from two subjects were eliminated because of technical difficulties which may have influenced the results. Fifteen subjects met all inclusion criteria and served as subjects in the study.
Table IX.
Psychometric Characteristics of Subjects in Experiment 3.

<table>
<thead>
<tr>
<th>Measure</th>
<th>Mean/SD</th>
<th>Range</th>
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<tbody>
<tr>
<td><strong>DSFI Subtests</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Information</td>
<td>53.9+/-2.4</td>
<td>38-75</td>
</tr>
<tr>
<td>Experience</td>
<td>52.3+/-2.43</td>
<td>32-63</td>
</tr>
<tr>
<td>Drive</td>
<td>55.5+/-3.1</td>
<td>37-72</td>
</tr>
<tr>
<td>Attitude</td>
<td>31.1+/-0.6</td>
<td>27-35</td>
</tr>
<tr>
<td>Symptoms (BSI)</td>
<td>45.8+/-3.7</td>
<td>30-75</td>
</tr>
<tr>
<td>Affect</td>
<td>43.0+/-3.3</td>
<td>30-68</td>
</tr>
<tr>
<td>Gender Role</td>
<td>50.0+/-3.5</td>
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<tr>
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<td>Body Image</td>
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<tr>
<td>Satisfaction</td>
<td>52.2+/-2.8</td>
<td>30-63</td>
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<td><strong>DSFI Global Scores</strong></td>
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</tr>
<tr>
<td>GSSI</td>
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</tr>
<tr>
<td>OFQ</td>
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<td>80-100</td>
</tr>
</tbody>
</table>

*Note: DSFI = Derogatis Sexual Functioning Inventory, OFQ = Orgasmic Functioning Questionnaire, BSI = Brief Symptom Inventory, SFI = Sexual Functioning Index, GSSI = Global Sexual Satisfaction Index. Means for Derogatis Sexual Functioning Inventory items are based on raw scores that were converted to established percentile rankings (T scores). Means for the OFQ are based on the highest estimated percentage of trials on which a subject achieved orgasm by any method.*
Design and Procedure

The procedure was identical to that used in Experiment 2 with the exception of the following: Subjects were not required to exercise in either experimental condition. Sexual arousal, in response to the films, was measured after a 1-hr waiting period following drug administration, and a 10-min recorded adaptation period.

Data Sampling and Reduction

All data sampling and reduction procedures were identical to those used in Experiment 2.

Results

Analyses of Physiological Measures

Vaginal pulse amplitude. A Condition (Clonidine vs. Placebo) x Film (neutral vs. erotic) analysis of variance was conducted on VPA raw scores to determine whether clonidine influenced VPA scores during either the neutral or erotic films, and to examine whether the erotic films facilitated VPA responses. Results revealed a significant effect of the erotic films on VPA responses, \( F(1, 14) = 26.48, p < .001 \). Despite the fact that clonidine decreased VPA responses to erotic stimuli among nine of 15 subjects, the influence of clonidine on VPA scores did not reach statistical significance. There was no significant interaction between condition and film. Planned, paired-sample t-tests were conducted on VPA responses between neutral and erotic during the Placebo and Clonidine conditions. Due to accumulating Type I error on mean comparisons across the variables, only mean differences of \( p < .025 \) (\( p < .05/2 \)) should be considered statistically reliable. Results revealed significant increases in VPA responses to the erotic films in both the Placebo, \( t(14) = -4.50, p < .001 \), and Clonidine, \( t(14) = -3.78, p = .002 \), conditions.

To determine whether exercise increased VPA responses to erotic stimuli, an independent samples t-test was conducted on VPA difference scores between the Placebo condition in Experiment 2 and the Placebo condition in Experiment 3. Results indicated a
significant increase in VPA responses with exposure to exercise in Experiment 2, $t(28) = 2.01, p = .054$. Mean VPA difference scores during the Clonidine and Placebo conditions are presented in Figure 3.

To examine whether order of experimental conditions influenced VPA responses, a Condition (Clonidine vs. Placebo) x Order (Clonidine, Placebo vs. Placebo, Clonidine) analysis of variance was conducted on VPA difference scores. There was no significant effect of order on VPA responses and no significant interaction between condition and order.

Vaginal blood volume. Results from a paired-samples t-test revealed no significant effect of clonidine administration on VBV deviation scores. Only seven of 15 subjects showed decreases in VBV responses to erotic stimuli with clonidine administration. One-tailed, paired samples t-tests were conducted on VBV raw scores between neutral and erotic films within each experimental condition in order to verify that the erotic films facilitated VBV responding. Due to accumulating Type I error on mean comparisons across the variables, only mean differences of $p < .025 (p < .05/2)$ should be considered statistically reliable. Results revealed a significant increase in blood volume responses with exposure to an erotic film in both the Clonidine, $t(14) = 5.16, p < .001$, and Placebo, $t(14) = 4.76, p < .001$ conditions. Mean VBV deviation scores during the Clonidine and Placebo conditions are presented in Figure 4.

To examine whether exercise facilitated VBV responses to erotic stimuli, an independent samples t-test was conducted on VBV deviation scores between the Placebo condition in Experiment 2 and the Placebo condition in Experiment 3. Results indicated a marginally significant increase in VBV responses with exposure to exercise in Experiment 2, $t(28) = 1.97, p = .059$. To determine whether order of experimental conditions influenced VBV responses, a Condition (Clonidine vs. Placebo) x Order (Clonidine, Placebo vs. Placebo, Clonidine) analysis of variance was conducted on VBV deviation scores. There was
no significant effect of order on VBV responses and no significant interaction between condition and order.

**Heart rate.** To examine whether heart rate was influenced with clonidine administration during the 1-hr waiting period prior to film presentation, a Condition (Clonidine vs. Placebo) x Time (4 time blocks following clonidine/placebo administration) analysis of variance of heart rate was conducted. There were no significant effects of either clonidine or time on heart rate levels, and no significant interaction between condition and time. A paired-samples t-test conducted on heart rate levels measured 10 min prior to drug administration, revealed there were no significant differences between conditions in resting levels of heart rate prior to clonidine/placebo administration. Mean heart rates, prior to and following clonidine/placebo administration, for each of the experimental conditions are presented in Table VI.

A Condition (Clonidine vs. Placebo) x Film (neutral vs. erotic) analysis of variance of heart rate was conducted to examine whether heart rate (during film presentations) was altered with clonidine administration and/or an erotic film. There was no significant difference in heart rate between neutral and erotic films, or between Clonidine and Placebo conditions, and no significant interaction between condition and film. Mean heart rates, during film presentations, for each of the experimental conditions and films are presented in Table VII.

To determine whether heart rate, in Experiment 2, was influenced with exposure to exercise, independent samples t-tests were conducted on heart rate between the Placebo condition in Experiment 2 and the Placebo condition in Experiment 3 for each of the neutral and erotic films. Heart rate was significantly increased following exercise during both the neutral, $t(28) = 4.54, p < .001$, and erotic, $t(28) = 3.30, p = .003$, films.

**Blood pressure.** To examine whether blood pressure was influenced with clonidine administration during the 1-hr waiting period prior to film presentation, Condition (Clonidine
vs. Placebo) x Time (4 time blocks following clonidine/placebo administration) analyses of variance of blood pressure were conducted separately for systolic and diastolic measures. Results revealed no significant effect of either clonidine or time, and no significant interaction between condition and time for either systolic or diastolic blood pressure levels. To ensure there were no differences between conditions in resting levels of blood pressure prior to clonidine/placebo administration, paired-samples t-tests were conducted on systolic and diastolic blood pressure levels measured 10 min prior to drug administration. There were no significant differences between conditions in resting levels of either systolic or diastolic blood pressure. Mean systolic and diastolic blood pressure levels, prior to and following clonidine/placebo administration, by experimental condition are presented in Table VIII.

Analyses of the Relationship Between Vaginal Blood Volume and Vaginal Pulse Amplitude Responses

In order to assess the relationship between physiological responses, Pearson product-moment correlation coefficients were calculated between VPA difference scores and VBV deviation scores of the middle 20 s of erotic stimulus, during both the Clonidine and Placebo conditions. Results revealed a marginally significant correlation between VBV and VPA responses during the Placebo, $r(15) = .47$, $p = .075$, but not Clonidine, $r(15) = .10$, $p = .71$, condition.

Analyses of Subjective Measures

A paired-samples t-tests was conducted on subjective ratings of sexual arousal between the Clonidine and Placebo conditions. Results revealed a marginally significant decrease in subjective ratings of sexual arousal, $t(14) = 1.97$, $p = .06$, with clonidine administration. A Condition (Clonidine vs. Placebo) x Measure (positive affect vs. negative affect vs. autonomic arousal vs. anxiety) analysis of variance was conducted to examine whether clonidine influenced subjects' mood and perceptions of autonomic changes to erotic stimuli. There was a significant effect of measure on subjective ratings, $F(3, 42) = 9.05$, $p <
.001, but no significant effect of clonidine on subjective measures of autonomic arousal, anxiety, positive affect, or negative affect, and no significant interaction between condition and measure. Mean subjective ratings are presented in Figure 6.

To examine whether exercise influenced subjective sexual arousal, an independent samples t-test was conducted on subjective ratings of sexual arousal between the Placebo condition in Experiment 2 and the Placebo condition in Experiment 3. Exercise had no significant influence on subjective ratings of sexual arousal.

Separate paired samples t-tests were conducted between subjective ratings of sexual arousal during the Placebo condition and responses to each of the three predicted arousal questions. The purpose was to examine whether the levels of reported subjective arousal might represent the highest levels that could be expected given the experimental stimuli used, or whether they might represent the highest levels of sexual arousal that subjects were willing to admit to. Due to accumulating Type I error on mean comparisons across the variables, only mean differences of $p<.02$ ($p<.05/3$) should be considered statistically reliable. There was a significant difference between subjective ratings of sexual arousal and ratings of the highest level of sexual arousal believed possible, $t(14) = 4.28, p<.001$, and a marginally significant difference between subjective ratings of sexual arousal and ratings of the highest level of sexual arousal previously attained, $t(14) = 2.58, p=.02$. There was no significant difference between subjective ratings of sexual arousal and ratings of the highest expected level of arousal to an erotic film. A Pearson correlation revealed a significant relationship between subjective ratings of sexual arousal and ratings of expected levels of arousal to an erotic film, $r(15) = .60, p=.017$. Mean ratings of the highest personal level of sexual arousal believed possible, the highest level of sexual arousal ever experienced, and the highest level of sexual arousal believed possible when viewing an erotic film alone were, 6.7 (+/-0.2), 6.0 (+/-0.3), 4.3 (+/-0.3), respectively.
Figure 6. Mean subjective ratings of sexual arousal, autonomic arousal, positive affect, negative affect, and anxiety in response to erotic stimuli during the Clonidine and Placebo conditions of Experiment 3.
Analyses of the Relationship Between Physiological and Subjective Sexual Responses

Separate Pearson product-moment correlation coefficients were calculated for each of the Clonidine and Placebo conditions in order to investigate the degree of association between physiological and subjective ratings of sexual arousal. There were no significant correlations between VPA difference scores and subjective ratings during either the Placebo, r(15) = .19, p=.49, or Clonidine, r(15) = -.10, p=.73, condition, and no significant correlations between VBV deviation scores and subjective sexual arousal during either Placebo, r(15) = .25, p=.38, or Clonidine, r(15) = .32, p=.25, condition.

Discussion

Support for the effectiveness of the experimental films in eliciting sexual arousal was provided by the finding that VPA and VBV responses were significantly increased during both the Clonidine and Placebo conditions. Comparison of heart rate between the Placebo conditions of Experiments 2 and 3 revealed a significant increase in heart rate with exposure to exercise. This finding provides indirect support for the use of exercise in Experiment 2 as a means of eliciting SNS activity. Analyses of order effects indicated that, as in Experiment 2, order of presentation of experimental conditions did not influence sexual responding.

Clonidine marginally decreased subjective ratings of sexual arousal and showed a trend toward decreasing physiological sexual arousal. These findings are consistent with those of Experiment 2, and with the results of Hodge et al. (1991) who found an inhibitory effect of clonidine on subjective ratings of sexual desire in hypertensive women. The fact that clonidine significantly inhibited physiological sexual arousal in Experiment 2, during heightened nervous system arousal, and only marginally inhibited physiological sexual arousal in Experiment 3, during baseline arousal, can be explained in terms of differences in levels of SNS inhibition between studies. In Experiment 2, clonidine significantly decreased SNS activity, as suggested by the significant decrease in heart rate following clonidine administration. In Experiment 3, clonidine only moderately inhibited SNS activity, as
suggested by the slight but insignificant decrease in heart rate responses following drug administration.

The fact that clonidine had a differential ability to inhibit physiological sexual responses, dependent upon the level of SNS activation attained, suggests that the inhibitory influence of clonidine on VPA and VBV responses in Experiment 2 are not likely attributable to centrally-mediated influences of clonidine. While, to the author's knowledge, there have been no reports of central adrenergic activity having a direct influence on sexual responding, it is possible that indirect central effects, such as sedation, may have influenced sexual responding. If clonidine were acting centrally to inhibit sexual responding, however, one would expect it to do so equally effectively during both heightened and baseline nervous system arousal. The fact that clonidine had a greater inhibitory effect on sexual arousal during increased autonomic arousal suggests that the drug acted to suppress sexual responding via peripheral (i.e., SNS inhibition), as opposed to central, mechanisms. As in Experiment 2, clonidine had no significant effect on subjective ratings of positive affect, negative affect, or anxiety. This suggests that the reported decreases in subjective sexual arousal with clonidine administration cannot be explained in terms of potential changes in subjects' mood with drug administration.

Comparison of sexual responses during the Placebo conditions of Experiments 2 and 3 allowed for an additional examination of the effects of SNS activation on sexual responding. The finding that exercise significantly increased VPA, marginally increased VBV, and had no effect on subjective sexual responses replicates the findings of Experiment 1 at 15 min post-exercise, and the findings of Meston and Gorzalka (1995). The exercise-induced increases in VPA and VBV measures during the Placebo condition of Experiment 2 constitute the third empirical demonstration of a facilitatory influence of moderate levels of SNS activation on physiological sexual arousal in women.
EXPERIMENT 4

The Differential Effects of Sympathetic Activation on Sexual Arousal in Sexually Dysfunctional and Functional Women

The results of Experiment 1 demonstrated that acute exercise enhances physiological sexual arousal in women at 15 min post-exercise, and the results of Experiment 2 suggested that this effect is attributable to increased SNS activity. Experiment 4 was designed to examine whether the sexually facilitatory effects of SNS activation could be extended to a group of sexually dysfunctional women. Subjects participated in two experimental conditions in which they viewed a neutral film followed by an erotic film. In one of these sessions subjects engaged in 20 min of intense exercise prior to viewing the films. Profile descriptions, obtained using the DSFI and OFQ, were used to classify subjects as either sexually functional, low sexual drive, or anorgasmic. This is the first empirical study to consider potential differences in sexual responding between subgroups of women with sexual difficulties. Based on the results of outcome studies which show a differential effectiveness in using anxiety-reduction techniques to treat sexual arousal and orgasm difficulties (for review, see Andersen, 1983), this study entertained the notion that orgasmic and anorgasmic women may differ intrinsically at some neurophysiological level.

Method

Subjects

Thirty-six women participated in this investigation. The subjects were recruited through a psychology department undergraduate research participant pool and through local newspaper advertisements requesting volunteers for the present experiment. Initial telephone screening criteria were: between the ages of 18-45 years, no use of medications known to affect vascular or sexual functioning, no medical condition that may put the subject at risk when exercising, and current involvement in a heterosexual relationship. Further inclusion criteria, based on subject information from the Physical Readiness Exam for Fitness Test
included: no history of heart disease or cardiovascular dysfunction, no history of dizzy spells or "light-headedness", and no bone or joint problem that might be aggravated by 20 min of cycling.

The Functional group (mean age = 24 years, range, 19-38) consisted of five undergraduate students and seven individuals employed in professions outside the university. The Low Sexual Desire group (mean age = 25 years, range, 18-45) and the Anorgasmia group (mean age = 29 years, range, 19-36) were each comprised of four undergraduate students and eight individuals employed in professions outside the university. Racial background of the subjects was Caucasian with the exception of one East Asian in the Functional group, three East Asians in the Low Sexual Drive group, and three East Asians in the Anorgasmia group. One subject in the Functional group was married and two subjects in each of the Low Sexual Drive and Anorgasmia groups were married.

Profile descriptions of all subjects were obtained via the DSFI and the OFQ. Mean scores by group for the DSFI subtests and the OFQ are presented in Table X. Data from the OFQ and the Drive subscale of the DSFI were used to validate differences between groups. Subjects in the Functional group scored higher than 50% on the OFQ (i.e., able to achieve orgasm by some means on more than 50% of the attempted trials), and higher than the 50th percentile on the Drive subscale. Subjects in the Low Sexual Drive group scored higher than 50% on the OFQ, and less than or equal to the 50th percentile on the Drive subscale. Subjects in the Anorgasmia group scored less than or equal to 50% on the OFQ. Because of the high comorbidity of anorgasmia with low sexual drive, the Drive subscale was not used as a criterion for the Anorgasmia group. Of the 12 women with anorgasmia, 10 met the criterion for primary anorgasmia (never achieved orgasm by any means), and two met the criterion for secondary anorgasmia (had difficulty achieving orgasm). In addition, all subjects employed in the study scored greater than or equal to the 30th percentile (i.e., within two standard deviations of the normative mean) on the BSI. Data from the Experience subtest of the DSFI
<table>
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<tr>
<th>Measure</th>
<th>Functional Low Sexual Drive Anorgasmia</th>
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</thead>
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<td>Gender Role</td>
<td>DSFI Subtests</td>
</tr>
<tr>
<td>Affect</td>
<td>Symptom (BSI)</td>
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<td>Attitude</td>
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<tr>
<td>Drive</td>
<td>Experience</td>
</tr>
<tr>
<td>Information</td>
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</tr>
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</table>
were used to ensure that all subjects were within the normative range of sexual experience (i.e., within two standard deviations of the normative mean), and to verify that the women in the Functional, Low Sexual Drive, and Anorgasmiagroups had experienced a similar repertoire of sexual behaviors. All subjects scored above the 30th percentile on the Experience subtest.

Two subjects were eliminated from the study because they scored below the cutoff criterion for general psychopathology, and one subject was eliminated because she scored below the cutoff criterion for sexual experience. Data from five subjects were eliminated because of technical difficulties during physiological recording. Thirty-six subjects met all inclusion criteria and served as subjects in the study. Subjects were paid $25.00 for their participation.

**Design and Procedure**

The design and procedure of Experiment 4 was identical to that used in the Delayed (i.e., 15 min) condition of Experiment 1, with the exception of the following: After subjects inserted the plethysmograph, a 10-min recorded adaptation was taken prior to the onset of the films to allow the plethysmograph time to adapt to subjects' body temperatures. This procedure is identical to that used by Meston and Gorzalka (1995) and to that used in Experiments 2 and 3. The total time from the cessation of exercise to the onset of the erotic stimulus was approximately 15 min (1 min to insert the plethysmograph, 10-min adaptation period, 1 min display of the word "relax", 3-min neutral film). This time interval was based on the results of Experiment 1 which indicated that moderate (i.e., 15 min post-exercise) levels of SNS activation may be optimal in facilitating physiological sexual arousal in women. The methodology of Experiment 4 replicates that of Meston and Gorzalka (1995) in a sexually dysfunctional sample of women.
Data Sampling and Reduction

All data sampling and reduction procedures were identical to those used in Experiment 1.

Results

Analyses of Group Differences in Sexual Functioning

In order to validate differences between sexually functional, low sexual drive, and anorgasmic groups, one-way analyses of variance were conducted on DSFI subscale scores and OFQ scores. Due to accumulating Type I error on mean comparisons across the variables, only mean differences of $p < .004 (p < .05/13)$ should be considered statistically reliable. As can be seen in Table X, the groups differed significantly on measures of sexual drive and orgasmic ability. Newman Keuls tests with a significance level set at $p < .05$ indicated significantly higher sexual drive scores among sexually functional than low sexual drive or anorgasmic subjects. Newman Keuls tests also indicated significantly higher sexual drive scores among anorgasmic subjects than low sexual drive subjects. With respect to orgasmic ability, subjects in the Functional or Low Sexual Drive groups were significantly more likely to achieve orgasm than subjects in the anorgasmia group. Reports of sexual satisfaction differed significantly between groups. Newman Keuls tests with a significance level set at $p < .05$ indicated significantly greater sexual satisfaction among sexually functional subjects than either low sexual drive or anorgasmic subjects. There was no significant difference in sexual satisfaction between low sexual drive and anorgasmic subjects.

Analyses of Physiological Measures

Vaginal pulse amplitude. A Condition (Exercise vs. No-exercise) x Group (Functional vs. Low Sexual Drive vs. Anorgasmia) analysis of variance was conducted on VPA difference scores. Mean VPA difference scores for the No-exercise and Exercise sessions are presented in Figure 7. Results revealed a significant main effect of exercise, $F(1, 33) = 5.76, p = .022$, and a significant interaction between session and group, $F(2,33) = 7.19,$
Figure 7. Mean vaginal pulse amplitude (millimeters of pen deflection) 
+/- SEM for sexually functional, low sexual drive, and anorgasmic women during the No-exercise and Exercise conditions of Experiment 4.
p = .003. A follow-up one-way analysis of variance between VPA difference scores during the No-exercise sessions indicated no significant difference between the Functional, Low Sexual Drive, or Anorgasmia groups (F<1). A follow-up one-way analysis of variance between VPA difference scores during the Exercise sessions, however, revealed a significant difference between the Functional, Low Sexual Drive, and Anorgasmia groups, F(2, 33) = 5.02, p = .01. Newman Keuls tests with a significance level set at p< .05 indicated a significant difference in VPA difference scores between the Functional and Anorgasmia groups, and between the Low Sexual Drive and Anorgasmia groups. There was no significant difference in VPA difference scores between the Functional and Low Sexual Drive groups. Planned follow-up t-tests were conducted between the No-exercise and Exercise conditions within each of the Functional, Low Sexual Drive, and Anorgasmia groups. Due to accumulating Type I error on mean comparisons across the variables, only mean differences of p<.02 (p<.05/3) should be considered statistically reliable. Results revealed a significant increase in VPA scores with exposure to exercise among functional subjects, t(11) = -2.58, p=.02, a significant increase with exposure to exercise among low sexual drive subjects, t(11) = -2.67, p=.02, and a marginally significant decrease with exposure to exercise among anorgasmic subjects, t(11) = 2.30, p=.042.

In order to verify that the erotic films facilitated VPA responses, Film (neutral vs. erotic) x Group (Functional, Low Sexual Drive, Anorgasmia) analyses of variance were conducted on VPA raw scores within each experimental condition. Results revealed a significant increase in pulse amplitude responses with exposure to an erotic film in both the No-exercise, F(1,33) = 63.30, p<.0001, and Exercise, F(1,33) = 130.93, p<.0001 conditions. There was no significant main effect of group in either the No-exercise or Exercise conditions.

To determine whether order of experimental sessions influenced VPA responses, Condition (Exercise vs. No-exercise) x Order (Exercise, No-exercise vs. No-exercise,
analyses of variance were conducted on VPA difference scores within each subject group. There were no significant effects of order on VPA responses among either sexually functional, low sexual drive, or anorgasmic subjects, and no significant interaction between condition and order for any of the subject groups.

**Vaginal blood volume.** Deviation scores in VBV were compared using a Condition (Exercise vs. No-exercise) x Group (Functional vs. Low Sexual Drive vs. Anorgasmia) analysis of variance. Mean VBV deviation scores during the Exercise and No-exercise sessions are presented in Figure 8. Results revealed a significant main effect of exercise, $F(1, 33) = 4.50, p = .042$, and a significant interaction between condition and group, $F(2,33) = 5.70, p = .008$. Follow-up one-way analyses of variance revealed no significant difference in VBV deviation scores between the Functional, Low Sexual Drive, and Anorgasmia groups during either the No-exercise, ($F<1$), or Exercise, $F(2, 33) = 2.50, p = .097$, conditions. Planned follow-up t-tests were conducted between the No-exercise and Exercise conditions within each of the Functional, Low Sexual Drive, and Anorgasmia groups. Due to accumulating Type I error on mean comparisons across the variables, only mean differences of $p < .02$ ($p < .05/3$) should be considered statistically reliable. Results revealed a significant increase in VBV deviation scores with exposure to exercise among functional subjects, $t(11) = -3.38, p = .006$, a significant increase among low sexual drive subjects, $t(11) = -2.92, p = .014$, and no effect among anorgasmic subjects.

In order to verify that the erotic films facilitated VBV responses, one-tailed, paired samples t-tests were conducted on vaginal blood volume raw scores between neutral and erotic films within each experimental condition and group. Due to accumulating Type I error on mean comparisons across the variables, only mean differences of $p < .008$ ($p < .05/6$) should be considered statistically reliable. Results revealed a significant increase in blood volume responses to an erotic film among functional subjects in both the No-exercise, $t(11) = -7.61, p < .001$ and Exercise, $t(11) = -5.82, p < .001$ conditions, among low sexual drive subjects in
Figure 8. Mean regional blood volume (millivolts deviation from baseline) +/- SEM for sexually functional, low sexual drive, and anorgasmic women during the No-exercise and Exercise condition of Experiment 4.
the No-exercise, $t(11) = -3.60, p = .004$ and Exercise, $t(11) = -4.33, p = .001$ conditions, and among anorgasmic subjects in both the No-exercise, $t(11) = -4.41, p < .001$ and Exercise, $t(11) = -4.15, p = .002$ conditions.

To examine whether order of experimental sessions influenced VBV responses, Condition (Exercise vs. No-exercise) $\times$ Order (Exercise, No-exercise vs. No-exercise, Exercise) analyses of variance were conducted on VBV deviation scores within each subject group. There were no significant effects of order on VBV responses among either sexually functional, low sexual drive, or anorgasmic subjects, and no significant interaction between condition and order for any of the subject groups.

**Heart rate.** Mean heart rates for each of the experimental conditions, groups, and films are presented in Table XI. A Condition (Exercise vs. No-exercise) $\times$ Film (neutral vs. erotic) $\times$ Group (Functional vs. Low Sexual Drive vs. Anorgasmia) MANOVA was computed to examine whether heart rate was altered with exposure to exercise and/or an erotic arousal during the film. Results revealed a significant main effect of condition, $F(1, 33) = 98.51, p < .0001$, indicating that exercise significantly increased heart rate among sexually functional, low sexual drive, and anorgasmic subjects. There was no significant difference in heart rate between exposure to neutral and erotic films, or between subject groups, and there were no significant interactions between condition, group or film (all $F$s $< 1$).

**Analyses of the Relationship Between Vaginal Blood Volume and Vaginal Pulse Amplitude Responses**

In order to determine the relationship between blood volume and pulse amplitude responses, Pearson product-moment correlation coefficients were calculated between VPA difference scores and VBV deviation scores of the middle 20s of erotic film, during both the No-exercise and Exercise conditions (due to the small sample size, correlations were conducted across subject groups). There were no significant correlations between VBV and
Table XI. Mean (+/-SEM) Heart Rate Responses to Neutral and Erotic Films During the No-exercise and Exercise Conditions of Sexually Functional, Low Sexual Drive, and Anorgasmic Subjects in Experiment 4.

<table>
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<tr>
<th>Group</th>
<th>No-exercise neutral film</th>
<th>No-exercise erotic film</th>
<th>Exercise neutral film</th>
<th>Exercise erotic film</th>
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<tr>
<td>Functional</td>
<td>68.3 +/- 2.5</td>
<td>74.7 +/- 6.2</td>
<td>92.1 +/- 2.4</td>
<td>90.4 +/- 2.5</td>
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<tr>
<td>Low Sexual Drive</td>
<td>72.4 +/- 3.1</td>
<td>74.0 +/- 2.8</td>
<td>91.3 +/- 4.3</td>
<td>92.1 +/- 4.8</td>
</tr>
<tr>
<td>Anorgasmia</td>
<td>67.4 +/- 2.4</td>
<td>66.9 +/- 2.4</td>
<td>88.3 +/- 3.0</td>
<td>86.6 +/- 3.2</td>
</tr>
</tbody>
</table>

*Note:* Heart rate means are based on raw scores averaged across the entire 180s of neutral and 180s of erotic film stimuli, within each experimental group and condition.
VPA responses during either the Exercise, $r(36) = .21$, $p = .21$, or No-exercise, $r(36) = .05$, $p = .80$, condition.

**Analyses of Subjective Measures**

Subjective ratings of sexual arousal, autonomic arousal positive affect, negative affect, and anxiety in response to erotic stimuli, were analyzed separately using 2 x 3 (Condition x Group) analyses of variance. Mean subjective ratings are presented in Table XII. There were no significant effects of condition (No-exercise vs. Exercise) on measures of subjective sexual arousal, autonomic arousal, positive affect, negative affect, or anxiety (all $F$s $< 1$). There were no significant effects of group (Functional vs. Low Sexual Drive vs. Anorgasmia), and there were no significant interactions between condition and group for any of the subjective ratings (all $F$s $< 1$).

In order to examine whether the levels of reported subjective arousal might represent the highest levels that could be expected given the experimental stimuli used, or whether they might represent the highest levels of sexual arousal that subjects were willing to admit to, separate paired samples t-tests were conducted between subjective ratings of sexual arousal during the Exercise condition and responses to each of the three predicted arousal questions. Mean subjective ratings of predicted/past sexual arousal are presented in Table XIII. Due to accumulating Type I error on mean comparisons across the variables, only mean differences of $p < .006$ ($p < .05/9$) should be considered statistically reliable. There were significant differences between subjective ratings of sexual arousal and ratings of the highest level of sexual arousal believed possible among sexually functional women, $t(11) = 4.98$, $p < .001$, women with low sexual drive, $t(11) = 5.00$, $p < .001$, and anorgasmic women, $t(11) = 4.18$, $p = .002$. There were also significant differences between subjective ratings of sexual arousal and ratings of the highest level of sexual arousal previously attained among sexually functional women, $t(11) = 4.10$, $p = .002$, and women with low sexual drive, $t(11) = 3.36$, $p < .006$. There was no significant difference between subjective ratings of sexual arousal and
Table XII.

Mean (+/−SEM) Subjective Ratings of Sexual Arousal, Autonomic Arousal, Positive Affect, Negative Affect, and Anxiety During the No-Exercise and Exercise Sessions of Sexually Functional, Low Sexual Drive, and Anorgasmic Subjects in Experiment 4.

<table>
<thead>
<tr>
<th>Condition</th>
<th>Session</th>
<th>Sexual Arousal</th>
<th>Autonomic Arousal</th>
<th>Positive Affect</th>
<th>Negative Affect</th>
<th>Anxiety</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anorgasmia</td>
<td>Exercise</td>
<td>1.4+/−0.3</td>
<td>4.4+/−0.4</td>
<td>2.8+/−0.5</td>
<td>1.2+/−0.1</td>
<td>1.2+/−0.1</td>
</tr>
<tr>
<td></td>
<td>No-exercise</td>
<td>1.4+/−0.3</td>
<td>4.4+/−0.4</td>
<td>2.0+/−0.3</td>
<td>1.2+/−0.1</td>
<td>1.2+/−0.1</td>
</tr>
<tr>
<td>Low Drive</td>
<td>Exercise</td>
<td>1.4+/−0.3</td>
<td>3.4+/−0.5</td>
<td>2.2+/−0.6</td>
<td>1.2+/−0.1</td>
<td>1.4+/−0.3</td>
</tr>
<tr>
<td></td>
<td>No-exercise</td>
<td>1.4+/−0.3</td>
<td>3.4+/−0.5</td>
<td>2.2+/−0.6</td>
<td>1.2+/−0.1</td>
<td>1.4+/−0.3</td>
</tr>
<tr>
<td>Functional</td>
<td>Exercise</td>
<td>1.4+/−0.3</td>
<td>3.4+/−0.3</td>
<td>3.1+/−0.5</td>
<td>1.4+/−0.3</td>
<td>1.4+/−0.3</td>
</tr>
<tr>
<td></td>
<td>No-exercise</td>
<td>1.4+/−0.3</td>
<td>3.4+/−0.3</td>
<td>3.1+/−0.5</td>
<td>1.4+/−0.3</td>
<td>1.4+/−0.3</td>
</tr>
<tr>
<td>Anorgasmia</td>
<td>Exercise</td>
<td>1.5+/−0.3</td>
<td>4.5+/−0.4</td>
<td>2.7+/−0.6</td>
<td>1.4+/−0.3</td>
<td>1.4+/−0.3</td>
</tr>
<tr>
<td></td>
<td>No-exercise</td>
<td>1.5+/−0.3</td>
<td>4.5+/−0.4</td>
<td>2.7+/−0.6</td>
<td>1.4+/−0.3</td>
<td>1.4+/−0.3</td>
</tr>
</tbody>
</table>

Note: Means are based on an item response format of low (1) to intense (7).
### Table XIII.

**Mean (+/-SEM) Subjective Ratings of Predicted and Past Sexual Arousal of Sexually Functional, Low Sexual Drive, and Anorgasmic Subjects in Experiment 4.**

<table>
<thead>
<tr>
<th>Item</th>
<th>Sexually Functional</th>
<th>Low Sexual Drive</th>
<th>Anorgasmic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Given the appropriate situation, what is the highest level of sexual arousal that you think you could experience?</td>
<td>6.7 +/- 0.3</td>
<td>6.8 +/- 0.1</td>
<td>6.1 +/- 0.4</td>
</tr>
<tr>
<td>What is the highest level of sexual arousal that you have ever experienced?</td>
<td>6.6 +/- 0.3</td>
<td>6.2 +/- 0.3</td>
<td>5.5 +/- 0.3</td>
</tr>
<tr>
<td>What is the highest level of sexual arousal that you think you could experience viewing an erotic film alone?</td>
<td>4.5 +/- 0.4</td>
<td>4.4 +/- 0.5</td>
<td>4.6 +/- 0.5</td>
</tr>
</tbody>
</table>

*Note: Means are based on an item response format of low (1) to intense (7).*
ratings of the highest level of sexual arousal previously attained among anorgasmic women. No significant differences were noted between subjective ratings of sexual arousal and ratings of the highest expected level of arousal to an erotic film among sexually functional, low sexual drive, or anorgasmic women. Results from a Pearson correlation revealed a significant relationship between subjective ratings of sexual arousal and ratings of expected levels of arousal to an erotic film, $r(34) = .50, p = .003$.

**Analyses of the Relationship Between Physiological and Subjective Sexual Responses**

Pearson product-moment correlation coefficients were calculated separately for the No-exercise and Exercise experimental sessions in order to investigate the degree of association between physiological and subjective ratings of sexual arousal (due to the small sample size, correlations were conducted across experimental groups). There were no significant correlations between VPA responses and subjective ratings during either the No-exercise, $r(36) = .05, p = .78$, or Exercise, $r(36) = .15, p = .39$, condition, or between VBV responses and subjective ratings during either the No-exercise, $r(36) = -.08, p = .66$, or Exercise, $r(36) = -.20, p = .25$, condition.

**Discussion**

As in Experiment 1, exercise was used as a means of increasing SNS activity. The finding that heart rate was significantly increased with exposure to exercise indirectly suggests that the intensity and duration of exercise used in the present study were sufficient to elicit significant SNS activity. The fact that exercise did not differentially influence heart rate among sexually functional, low sexual drive, or anorgasmic subjects suggest that, as intended, levels of attained SNS activation were comparable across subject groups. Support for the effectiveness of the experimental films in eliciting sexual arousal was provided by the finding that both VPA and VBV responses were significantly increased with exposure to the erotic films among all subject groups. As in Experiments 1 through 4, analyses of order effects revealed familiarity with the experimental procedures or with viewing an erotic film
had no significant influence on subjects' sexual responses. The findings that subjects in the sexually functional and low sexual drive groups were significantly more likely to achieve orgasm than were subjects in the anorgasmia group, and sexually functional subjects reported significantly higher levels of sexual drive than did low sexual drive or anorgasmic subjects, validate the designation of women in the present study as sexually functional, low sexual drive, and anorgasmic. Together, the above results suggest that the experimental manipulations and control procedures used in this study were effective.

In the presence of an erotic stimulus, acute exercise facilitated physiological sexual arousal among sexually functional women and women with low sexual drive. This effect included a significant increase in both VPA and VBV responses. These results replicate those of Experiment 1 at 15 min post-exercise and those of Meston and Gorzalka (1995), and extend the findings to a group of women with inhibited sexual drive. In contrast to these facilitatory effects, exercise marginally decreased VPA responses and had no effect on VBV responses among anorgasmic women. These findings reveal potential differences in physiological sexual response mechanisms among women with and without orgasmic dysfunction. Subjective ratings of sexual arousal, positive affect, negative affect, and anxiety, were unaffected by exercise among either sexually functional, low sexual drive, or anorgasmic women. It is, consequently, unlikely that these cognitive factors could explain the reported exercise-induced changes in physiological sexual arousal.

As can be seen in Table X, subjects in the sexually functional, low sexual drive, and anorgasmic groups did not differ significantly on measures of sexual experience, information, psychopathology, affect, gender role, fantasy, or body image. This suggests that the reported group differences in physiological sexual arousal are not likely attributable to group differences in any of these variables. There were also no significant group differences in subjective ratings of positive affect, negative affect, anxiety, or sexual arousal. In other words, the erotic films had similar cognitive effects on sexually functional and dysfunctional
women. Cognitive factors cannot, therefore, solely explain the reported differences in physiological sexual arousal between orgasmic and anorgasmic women.

**GENERAL DISCUSSION**

The findings from this investigation provide new insight into the autonomic pathways and processes by which sexual arousal is facilitated in women. Specifically, the results reveal:

1. Moderate levels of SNS activation facilitate physiological sexual arousal in sexually functional women and in women who suffer from low sexual drive,
2. Moderate levels of SNS activation do not facilitate physiological sexual arousal in anorgasmic women,
3. There appears to be an optimal level of SNS activity for facilitation of physiological sexual arousal in sexually functional women,

These results have potential clinical, theoretical, and research implications.

**The Role of the Sympathetic Nervous System in Female Sexual Arousal**

The results of Experiment 1 revealed that exercise significantly increased physiological sexual responses to an erotic stimulus when measured at 15 min post-exercise. Comparison of findings from the Placebo condition of Experiment 2 with that of Experiment 3 also revealed significant increases in physiological sexual arousal with exposure to exercise. The results of Experiment 4 provided further demonstration of increased physiological sexual arousal with exposure to acute exercise in both sexually functional women and women with low sexual drive. These findings replicate and extend the findings of Meston and Gorzalka (1995) which indicated significant increases in VPA and marginally significant increases in VBV responses to an erotic film at 15 min post-exercise. The fact that, in Experiment 2, clonidine suppressed the exercise-induced increase in SNS activity and also suppressed
physiological sexual arousal is consistent with the idea that exercise facilitates physiological sexual responding through activation of the SNS. Together, these results provide strong support for a facilitatory influence of moderate levels of SNS activation on sexual arousal in women.

Because the effects of exercise were measured at 5, 15, and 30 min post-exercise, Experiment 1 allowed for examination of the effects of high, moderate, and low levels of SNS activation on sexual responding. The finding that exercise marginally decreased VPA responses at 5 min post-exercise, significantly increased VPA and showed a trend toward increasing VBV responses at 15 min, and marginally facilitated VBV and showed a trend toward increasing VPA responses at 30 min post-exercise suggests that there is a relation between the level of SNS activation and the level of physiological sexual arousal in women. That is, it seems that an optimal level of SNS activation exists beyond and below which physiological sexual arousal is suppressed or unaffected. Exactly where this optimal point may be cannot be concluded from the results of this study but seems to be indirectly related to levels of heart rate roughly between 80-90 bpm. The finding that moderate levels of SNS activation may be particularly beneficial in enhancing sexual arousal is consistent with previous research which has shown a curvilinear relation between levels of general arousability and sexual function in women (Jupp & McCabe, 1989).

The fact that exercise marginally decreased VPA responses at 5 min post-exercise leads one to question whether too much SNS activity may inhibit sexual arousal in women. Possibly, a ceiling exists beyond which increasing levels of SNS activity no longer play a role in facilitating sexual responding. However, a number of additional physiological changes, secondary to intense, acute exercise make this hypothesis highly speculative. Research indicates that, during and immediately following exercise, a decrease in vascular resistance of working muscles causes a significant increase in blood flow to the exercising muscles, and arterial vasoconstriction decreases blood flow to the nonexercising muscles, skin, kidney,
spleen, liver, and intestines (Christensen & Galbo, 1983). Consequently, immediately following exercise, blood flow may have been shifted away from the genital region to help restore working muscles. The lack of available blood in the genital region may have impaired sexual responding. This assertion is supported by the finding that, at 5 minutes post-exercise, VBV responses to erotic stimuli were not only suppressed but extinguished. Further research is needed to examine the effects of intense levels of SNS activation elicited by means that do not cause significant increases in peripheral muscle activity (e.g., epinephrine injections) on sexual responding in women. Research of this nature would provide insight into whether the inhibitory effects noted at 5 min post exercise are in fact attributable to intense SNS activity or, alternatively, to residual, nonsexual effects of acute exercise.

While definitive conclusions regarding the effects of intense SNS activation on physiological sexual arousal cannot be made from the present findings, the possibility that intense SNS activation inhibits physiological sexual responding remains tenable. Evidence for an inhibitory influence of intense SNS activation on sexual responding would support the long-held notion that anxiety inhibits sexual arousal not only through cognitive (i.e., distraction), but through physiological (i.e., increased SNS activity) mechanisms. It would not, however, support the use of relaxation training to treat these anxiety-related sexual disorders. While the present findings suggest that intense SNS activity may inhibit sexual arousal, the findings also strongly suggest that moderate levels of SNS activation may be important in facilitating the sexual response in women.

The notion that low to moderate levels of SNS activation facilitate physiological sexual responding contrasts with previous assumptions regarding the nervous system regulation of sexual arousal in women. Early researchers such as Kinsey et al. (1953), Wolpe (1958), and Wenger et al. (1956) proposed that female sexual arousal was largely dependent upon PNS rather than SNS activation. More recently, Kaplan (1974) proposed a biphasic model in which the initial stage of sexual arousal in women, characterized by engorgement of
tissue, is mediated by the PNS, and the later stage of arousal, characterized by orgasm, is mediated by the SNS. The present results suggest that SNS influences may be important in facilitating sexual responding not only during the later stages of sexual arousal/orgasm, but during the initial stages of arousal as well.

Evidence that activation of the SNS can facilitate physiological sexual arousal provides a possible explanation for the recently reported enhancing effects of anxiety on sexual responding (e.g., Hoon et al., 1977; Palace & Gorzalka, 1990). In these studies, anxiety was induced by exposing subjects to films depicting events such as threatened amputation and tragic automobile accidents. Because, in addition to activating the nervous system, these stimuli undoubtedly altered cognitions, researchers have often explained the findings in terms of cognitive processes such as an anxiety relief phenomenon, or a misattribution. Palace and Gorzalka (1990) were the first to propose that anxiety may be working through neural processes to enhance sexual responding. In the present study, the physiological component of anxiety was simulated (i.e., increased nervous system activity) without altering cognitions. That is, exercise significantly increased heart rate responses, but had no significant effect on subjective ratings of sexual arousal, positive affect, negative affect, or anxiety. The fact that the physiological component alone facilitated physiological sexual responding adds credence to Palace and Gorzalka's (1990) speculation that anxiety may have facilitated sexual arousal through activation of the SNS. The fact that increased SNS activity alone enhanced sexual responding also leads one to question whether an experimental paradigm which focused on enhancing both physiological and cognitive components might have an even greater facilitatory influence on sexual arousal. One such possibility would be the induction of laughter.

In Experiment 2, during heightened nervous system activity, clonidine significantly decreased subjective and physiological responses to an erotic film. In Experiment 3, during baseline levels of nervous system activity, clonidine marginally decreased subjective sexual
arousal and showed a trend toward decreasing physiological sexual responses. Together, these findings provide the first empirical evidence of an inhibitory influence of SNS inhibition on sexual arousal in women. The possibility that inhibition of the SNS inhibits female sexual arousal may help to explain the high reported incidence of inhibited sexual arousal/orgasm secondary to psychotherapeutic drug use (for review, see Meston and Gorzalka, 1992). Numerous antipsychotic and anxiolytic medications have been reported to inhibit sexual arousal and orgasm in women (Meston & Gorzalka, 1992; Shen & Sata, 1990). Many of these same drugs have also been shown to inhibit peripheral SNS activity (Physicians' Desk Reference, 1993).

The finding that clonidine decreased sexual responses to erotic stimuli also provides evidence of an inhibitory influence of antihypertensive medication on sexual function in women. While drug-induced sexual dysfunction is well known to occur with antihypertensive drugs in men, research on the effects of these drugs in women has been largely ignored (Bateman, 1980). The paucity of research in this area is surprising given that approximately 43% of patients with hypertension are female (Moss & Procci, 1982) and that reports of the incidence of sexual dysfunction among women with hypertension include estimates of up to 23% (Poloniecki & Hamilton, 1985). The results from Experiments 2 and 3 provide empirical support for a detrimental influence of antihypertensive treatment on sexual function in women, and provide an explanation for the mechanism of action by which some antihypertensive drugs may influence sexual responding. Although clonidine is known to act both centrally and peripherally, reports of sexual dysfunction secondary to antihypertensive drug use have more often been attributed to clonidine's central effects (e.g., Duncan & Bateman, 1993). The finding that clonidine had a greater inhibitory effect on sexual arousal during increased than baseline autonomic arousal, suggests that the sexual side effects of clonidine are more likely attributable to peripheral (i.e., SNS inhibition), than central, mechanisms.
Implications For Deriving an Etiological Theory of Inhibited Female Orgasm

In Experiment 4, exercise enhanced physiological sexual responding among women with low sexual drive. This finding is consistent with reports of increased VBV responses to anxiety-evoking stimuli among women with heterogeneous sexual difficulties (Palace & Gorzalka, 1990). Evidence that SNS activation has no effect on physiological sexual arousal in women with orgasmic dysfunction, however, has not previously been reported. The only study to compare the effects of nervous system activation among sexually functional and dysfunctional women (Palace & Gorzalka, 1990) did not differentiate response patterns between women with low sexual desire, anorgasmia, and dyspareunia. The present investigation is the first to consider potential differences in sexual arousal between subgroups of women with sexual difficulties.

The finding that anorgasmic women differ from orgasmic women in their physiological responses to SNS activation is consistent with the idea that there may be fundamental differences at a neurophysiological level. In the absence of anatomical problems, orgasmic difficulties have generally been regarded as psychogenic (Andersen, 1983). This notion dates at least back to Freud (1938) who claimed that the inability to have vaginal orgasms was the hallmark of female neuroticism and "infantile sexuality". More recently, Kaplan (1974) described a host of psychological factors which may contribute to orgasmic dysfunction. These include: ambivalence about relationship commitment, fear of abandonment, fear of asserting independence, guilt about sexuality, hostility toward the sexual partner, fear of "letting go", fear of men, and a variety of unconscious conflicts about sexuality which stem from restrictive upbringings. Despite numerous psychological theories of inhibited female orgasm, research has generally failed to find consistent psychological differences between women who can and cannot experience orgasm. Heiman, Gladue, Roberts, and LoPiccolo (1986) found overall quality of home life, parental attitudes and openness about sex, early religious attitudes, parental strictness, or family abuse did not
distinguish sexually functional from dysfunctional women. In an extensive 10-year study of 
female sexual responsiveness, Fisher (1973) found no consistent relation between degree of 
femininity, aggressiveness, passivity, guilt, impulsiveness, or narcissism, and orgasmic ability. 
Similarly, neurotic illness (Winokur, Guze, & Pfeiffer, 1959), or neuroticism (Cooper, 1969b) 
have been found unrelated to sexual responsiveness in women. Possibly, primary anorgasmia 
in women who are not experiencing anatomical or psychological problems, and who are 
sufficiently sexually experienced, may be explained in terms of neurophysiological differences. 
The present investigation provides the first neurophysiological explanation for inhibited 
female orgasm.

One way in which orgasmic and anorgasmic women may differ at a neurological level 
is that anorgasmic women require either higher or lower levels of SNS activation for sexual 
arousal/orgasm than do orgasmic women. The results of Experiment 1 suggest that an 
optimal level of SNS activation may exist, below and above which SNS activity may inhibit 
or play less of a facilitatory role on physiological sexual arousal. Possibly, anorgasmic women 
have different "optimal levels" of SNS activation than do orgasmic women. If this is the case, 
the moderate levels of SNS activation used in Experiment 4 may have either been too high or 
too low to facilitate the sexual response.

The notion that anorgasmic women may require low levels of SNS activation to 
facilitate sexual responding is supported by occasional case reports of women who are 
anorgasmic with men that they find sexually exciting, but orgasmic with men who do not 
strongly appeal to them (e.g., Kaplan, 1974). On the other hand, if low levels of SNS 
activation facilitate sexual arousal/orgasm among anorgasmic women, one would expect 
higher success rates in treating anorgasmia using techniques such as systematic 
desensitization and relaxation training, which decrease nervous system arousal. Control group 
or factorial design studies (as opposed to case reports) which have examined the 
effectiveness of systematic desensitization in treating primary anorgasmia reveal either no
change in orgasmic status (Sotile & Kilmann, 1978) or only insignificant to moderate gains (Nemetz, Craig, & Reith, 1978; Husted, 1972; Wincze & Caird, 1976). Future research which examines the effects of varying levels of SNS activation on sexual arousal in anorgasmic women may provide insight into whether SNS activation at an "optimal level" facilitates sexual arousal in anorgasmic women, or whether SNS activation plays a paradoxical, inhibitory role among these women.

The fact that differences in VPA and VBV responses between orgasmic and anorgasmic women were apparent only with exposure to exercise suggests that differences in physiological sexual responding between functional and dysfunctional women may be apparent only during heightened nervous system arousal. If one assumes that laboratory studies induce lower levels of sexual/nervous system arousal than those which occur during the sexual act, this may explain why research has failed to reveal consistent differences in physiological sexual arousal between sexually functional and dysfunctional women. Differences between orgasmic and anorgasmic women that would become apparent and problematic during sexual activity may be disguised in laboratory settings which induce lower levels of nervous system arousal. Future studies which examine potential differences in sexual responding between functional and dysfunctional women may do better to measure sexual responses to erotic stimuli after inducing a state of heightened nervous system activity.

Despite group differences in physiological sexual responses with exposure to exercise, sexually functional, low sexual drive, and anorgasmic subjects did not differ in their subjective sexual responses. This finding is consistent with previous research which has found no difference in subjective sexual responses to an erotic film between sexually functional and dysfunctional women (e.g., Wincze et al., 1976; Palace & Gorzalka, 1990). The fact that physiological, but not subjective, measures differentiated low sexual drive from anorgasmic women suggests that subjective measurement of sexual arousal may not be a valid diagnostic means of assessing sexual dysfunction among anorgasmic women.
The unexpected finding that SNS activation facilitates physiological sexual arousal in sexually functional women, and women with low sexual drive, but not among anorgasmic women, raises a number of novel questions: 1. Does SNS activation facilitate physiological sexual arousal among orgasmic women but inhibit sexual arousal in anorgasmic women? 2. Do anorgasmic women require lower or higher levels of SNS activation for sexual arousal/orgasm than do orgasmic women? 3. Can neurophysiological differences between orgasmic and anorgasmic women help explain why, among female sexual disorders, anorgasmia has a comparably lower treatment success rate? The present study is the first examination and discovery of differences in physiological sexual responding between orgasmic and anorgasmic women. Future research which examines the influences of various levels of SNS activation, as well as SNS inhibition, on the sexual responses of anorgasmic women may provide important insight into the etiology and treatment of inhibited female orgasm.

Implications for the Treatment of Sexual Dysfunction in Women

Since Wolpe's introduction of systematic desensitization (1958), anxiety-reduction techniques have been widely adopted in the treatment of sexual dysfunction. These techniques are thought to facilitate sexual responding by decreasing negative cognitions which disrupt the processing of erotic cues and by inducing a state of relaxation which increases PNS and decreases the presumably inhibitory SNS influences. With respect to the cognitive aspect of these treatments, numerous outcome studies have shown anxiety-reduction techniques to be highly successful in altering negative performance cognitions (for review, see Andersen, 1983). With regard to the physiological component of these treatments, however, the effects of decreasing SNS and increasing PNS activity on sexual arousal had not previously been examined. The findings from Experiment 2, which revealed clonidine significantly decreased physiological sexual arousal, strongly suggest that decreasing SNS activity during the early stages of sexual arousal may be detrimental to the
sexual response. This suggests that treatments for sexual dysfunction such as systematic desensitization and sensate focus may be desynchronous in their effectiveness for treating components (i.e., cognitive vs. physiological) of the female sexual response. This would not be surprising given it has long been established that, although linked, the three response systems (i.e., cognitive, behavioral, physiological) which conceptualize human behaviors do not necessarily change at the same time, in the same manner, or even in the same direction (Rachman & Hodgson, 1974).

The finding that SNS activation facilitates VPA and VBV responses in women with low sexual drive suggests that, rather than decreasing SNS activity, increasing SNS activity may prove beneficial in treating these women. This is consistent with Palace and Gorzalka's (1990) notion that SNS activation may provide sexually dysfunctional women with a "jump start" for sexual arousal. One method of treating sexual difficulties while maintaining or increasing SNS activity would be to train women, via imagery techniques, to associate increases in nervous system activity (e.g., increased heart rate, blood pressure, perspiration) with sexually pleasurable thoughts. For example, women could fantasize about being in a sexual scenario in which they were highly aroused, and were experiencing "sexual sensations" such as increased perspiration, heart rate, etc. This technique might serve both to break the association between changes in physiological processes and negative performance-anxiety cognitions, and to enhance SNS activation. Psychophysiological research has lent broad support to the notion that imagination of emotionally arousing scenes can elicit a number of psychophysiological changes including heart rate, respiration rate, muscular tension, and, less consistently, skin conductance (e.g., Folkins, Lawson, Option, & Lazarus, 1968; Grossberg & Wilson, 1968; Lang, Melamed, & Hart, 1970; Marzillier, Carroll, & Newland, 1979; May, 1977; Weerts & Lang, 1978). Recent studies have also shown that imagination of exercise can elicit physiological changes (e.g., oxygen consumption, ventilation, heart rate, blood pressure) similar to those which occur during the actual event (Morgan, 1985; Morgan,
While the effects of imagined exercise on sexual responses have not yet been examined, there is no apparent reason why imagination of exercise or other similarly arousing events could not also facilitate VBV and VPA responses. The proposal that imagery be used to enhance physiological sexual arousal is not intended to imply that it become the exclusive treatment for sexual dysfunction. Rather, it is suggested that imagery techniques, aimed at enhancing SNS activity, become an addition to already existing multimodal treatment techniques. The finding that heightened nervous system activity may enhance sexual arousal in women also suggests that when treating sexual disorders which include an anxiety/fear element, clinicians may do better to adopt a habituation, as opposed to a reciprocal inhibition, paradigm. Repeated exposure to the fear-evoking sexual stimulus would extinguish the anxiety response (as does pairing the stimulus with a relaxed state), but would be less likely than systematic desensitization to indirectly teach the patient that heightened nervous system arousal is an undesirable state.

In addition to imagery techniques, more active means of eliciting SNS activity may also prove beneficial in treating disorders of inhibited sexual arousal. Engaging in acute, intense exercise prior to the sexual act stands out as the obvious possibility, although other means such as using nasal epinephrine sprays or drinking a shot of strong espresso might also prove surprisingly helpful in providing a "jump start" for sexual arousal. The finding that there may be an ideal level of SNS activation for heightened sexual responding to occur suggests that proper integration of new techniques which aim at increasing SNS activity may require a certain degree of trial and error before optimal levels are discovered.

Research Implications

The finding that SNS activation can potentially either facilitate or inhibit sexual arousal, depending on the level of activation attained, suggests that future studies of the influence of nervous system arousal on sexual responding would benefit from measuring
nervous system activity using some objective, physiological means. Research on the effects of anxiety on sexual arousal, for example, has operationalized anxiety in a variety of different ways including receiving performance demand instructions, viewing films of threatened amputation, receiving epinephrine injections, crossing a fear-arousing suspension bridge, and receiving shock threats. While it is highly likely that these diverse stimuli elicit differential levels of nervous system activity, studies of this nature have generally failed to provide physiological indices of the level of nervous system arousal attained. Consequently, it is difficult, if not impossible to make comparisons of the effects of anxiety/nervous system arousal on sexual arousal across studies. Moreover, it is possible that current discrepancies in the literature on the effects of anxiety on physiological sexual arousal in men may, at least in part, be due to differences in attained levels of SNS activation.

The finding that exercise had no significant effect on subjective ratings of sexual arousal among either sexually functional, low sexual drive, or anorgasmic women contrasts with evidence that anxiety-evoking stimuli decrease subjective ratings of sexual arousal in sexually functional and dysfunctional women (Palace & Gorzalka, 1990). The fact that exercise also had no significant effect on subjective ratings of positive or negative affect similarly contrasts with reports that anxiety-evoking films significantly alter subjects' ratings of feeling "worried" (Palace & Gorzalka, 1990). The differential influence of exercise and anxiety-evoking films on subjective ratings indicates that exercise may have less of an effect on cognitive processes than does a film of threatened amputation. Exercise, rather than anxiety-evoking films, may thereby provide a better means for examining the sexual effects of nervous system arousal in women, given that interpretation of findings is not confounded by the possible contributory role of negative cognitions.

The findings of a low correspondence between physiological measures in the present investigation has implications for future research which employs vaginal photoplethysmography in the measurement of sexual arousal. With the exception of the
Meston and Gorzalka (1995) study, the present investigation was the first to use both VBV and VPA measures to assess the effects of heightened nervous system arousal on sexual responding in women. Across most areas of research on female sexuality, investigators have generally employed only one or the other of these two measures to assess sexual arousal. Which of these two measures better serve as an indicator of sexual responding has been the topic of much debate. Some investigators have found that VPA to be a more sensitive index (e.g., Geer, Morokoff, & Greenwood, 1974; Heiman, 1977; Osborn & Pollack, 1977), and to show a more rapid return to baseline after stimulus presentation has ended (Henson, Rubin, & Henson, 1979). Other researchers have chosen to use VPA because VBV appears to be complicated by variability in the resting dc signal both between and within subjects (e.g., Beck, Sakheim, & Barlow, 1983; Henson et al., 1979). On the other side, Hoon, Wincze, and Hoon (1976) and Palace and Gorzalka (1990) have argued that VBV is a more relevant measure, given changes in VPA account for only a small percentage of total blood volume during engorgement.

The use of both VBV and VPA measures in the present investigation allowed for comparison of these measures across situations of both heightened and lowered nervous system activity. In Experiment 1, as in the Meston and Gorzalka (1995) study, VPA responses were significantly increased at 15 min post-exercise but VBV responses showed only a trend toward increasing. At 5 min post-exercise VPA responses were marginally suppressed while VBV responses were unaffected. Similarly, among anorgasmic women, VPA responses were marginally decreased while VBV responses remained unaltered. These findings support the notion that VPA, compared with VBV, may be a more sensitive detector of physiological sexual arousal in women. The finding that, with clonidine administration, decreases in VPA were paralleled by decreases in VBV, while exercise led to a general desynchrony between physiological responses, may indicate that changes in body temperature resulting from exercise had more of an influence on VBV than VPA indices. This assertion is
consistent with research which indicates that the dc signal used in the measurement of VBV is more likely affected by temperature confounds than is the ac signal used in the measurement of VPA (Beck et al., 1983).

The issue of which of the two physiological signals may be a more appropriate indicator of sexual responding in women is also related to validity concerns. As noted by Rosen and Beck (1988), one approach to the establishment of validity is to examine correlations between physiological and subjective measures. Past research of this nature has revealed inconsistent findings. Heiman (1977) reported higher correlations between VPA and subjective arousal than between VBV and self-ratings, while Cerny (1978) found the reverse. There is no obvious explanation for this inconsistency. In the present study, significant correlations were noted between VPA and subjective sexual responses during the No-exercise condition of Experiment 1, and during the Placebo condition of Experiment 2. Vaginal blood volume, by contrast, revealed no significant correlation with subjective sexual responses in any of the experimental conditions of Experiments 1 through 4. The finding that VPA showed higher correlations with subjective responses than did VBV provides support for VPA as the more valid indicator of sexual arousal in women.

While the findings from the present investigation suggest that VPA may be a more sensitive and valid indicator of sexual arousal than VBV and, possibly, less influenced by temperature confounds, the low correlation between these two measures in the present investigation leads one to question whether VPA and VBV responses can be meaningfully compared. Significant correlations were noted between VPA and VBV measures only during the Exercise condition of Experiment 1, and during the Placebo conditions of Experiments 2 and 3. That is, there were no significant correlations between physiological measures during the Exercise condition of Experiment 1, the Clonidine conditions of Experiments 2 or 3, or either the Exercise or No-exercise conditions of Experiment 4. This low correspondence between physiological responses is consistent with previous research which has generally
shown a lack of correlation between these two measures (e.g., Meston & Gorzalka, 1995; Zingheim & Sandman, 1978). Heiman (1976) suggested that the magnitude of correspondence between the two signals may be dependent on the level of arousal achieved. That is, the higher level of physiological arousal, the higher the correspondence between measures. In support of Heiman's (1976) explanation, the present results indicated a higher correlation between measures during the Exercise versus No-exercise, and Placebo versus Clonidine conditions of Experiments 1 to 3. The higher correspondence between measures in these conditions was accompanied by higher levels of both VBV and VPA responses.

An alternative explanation for the low correlation between physiological measures is that the VBV and VPA signals assess separate features of the vasocongestive response. The ac signal, used in the measurement of VPA, represents changes in pulse rate and other cardiac fluctuations. As noted by Rosen and Beck (1988), the significance of VPA in the complex process of vaginal engorgement is not well understood, although it is believed that increases in VPA lead to increases in peripheral vascular dilation (Palti & Bercovici, 1967). Vaginal blood volume, on the other hand, appears to reflect the difference between rates of blood inflow and outflow to a specified site. Given these measures reflect different aspects of the vasocongestive response and are sampled and analyzed differently, it is not surprising that they do not necessarily work together in perfect synchrony. Together with previous research which has shown a low correlation between VBV and VPA measures, the findings from the present investigation suggest that the two signals may need to be used in combination in order to provide complete data on the engorgement process.

**Theoretical Implications**

**SNS Activation and Sexual Arousal are not Mutually Inhibitory.** The results of the present investigation which indicate that SNS activation may enhance physiological sexual arousal challenges Wolpe's (1958) theory that SNS activation and sexual arousal are mutually inhibitory. As noted earlier, only one study, to date, has been aimed at directly assessing
Wolpe's reciprocal inhibition theory with regard to sexual behavior (i.e., Hoon et al., 1977). This study (Hoon et al., 1977), which examined the effects of an erotic film and an anxiety film on sexual arousal, revealed support both for and against Wolpe's assertion. In support of Wolpe's theory, exposure to the anxiety film following the erotic film suppressed sexual responding. In contrast to Wolpe's theory, exposure to the anxiety film prior to the erotic film, enhanced sexual responding. Wolpe criticized the study on the grounds that there was no indication of increased heart rate following exposure to the anxiety film and, consequently, no evidence that the effects of the anxiety and sexual stimuli were present simultaneously. The results of the present investigation revealed that heart rate was significantly increased during the Exercise conditions of Experiments 1 and 4, and that this increase remained unchanged throughout the entire 180 seconds of erotic exposure. These data verify that the effects of SNS activation and erotic stimuli were present simultaneously. Consequently, the present study provided an indirect, but adequate, test of the reciprocal inhibition hypothesis.

If SNS activity and sexual arousal are mutually inhibitory, one would expect either a decrease in sexual arousal during the exercise condition or, alternatively, a decrease in heart rate during the erotic exposure. Neither of these outcomes were noted. Increased SNS activity facilitated physiological sexual arousal, and increased physiological sexual arousal did not alter SNS activity. Perhaps, then, SNS activation only inhibits sexual arousal when accompanied by negative cognitions. Perhaps SNS activation can be considered an "engine" which drives cognitions to a behavioral response. If negative cognitions are in play, then SNS activation provides the force to decrease sexual arousal. In this regard SNS activation and sexual arousal are mutually inhibitory. On the other hand, if sexual cognitions are in play, SNS activation may provide the force behind increasing sexual arousal. In this sense SNS activation and sexual arousal are mutually facilitatory.
Cognitive and Physiological Components of the Female Sexual Response are not Desynchronous. Examination of the relation between subjective and physiological indices of the female sexual response has become one of the more pervasive issues in laboratory studies of female sexuality. In contrast to research in men which has found relatively high correlations between subjective and physiological indices of sexual responding (e.g., Heiman & Rowland, 1983; Steinman et al., 1981), research in women has generally revealed a low concordance between these measures (e.g., Heiman et al., 1991; Palace & Gorzalka, 1990, 1992; Meston & Gorzalka, 1995; Morokoff & Heiman, 1980; Steinman et al., 1981; Wincze et al., 1976). The results of the present investigation also reveal a low concordance between subjective and physiological sexual responses. Significant correlations between VPA and subjective sexual responses were found only during the No-exercise condition of Experiment 1, and during the Placebo conditions of Experiments 2 and 3. That is, there were no significant correlations between VPA and subjective sexual responses during either the No-exercise condition of Experiment 1, the Clonidine conditions of Experiments 2 and 3, or during either the No-exercise or Exercise conditions of Experiment 4. There were no significant correlations between VBV and subjective ratings during any of the experimental conditions.

Authors have explained the low correspondence between genital blood flow measures and subjective arousal in women in terms of a potential desynchronous relationship. Heiman (1977) suggested that this desynchrony may be attributable to a less direct feedback system (erection vs. vasocongestion) in the female, which would allow bodily cues to be more easily ignored. Heiman (1976) also suggest that the desynchrony may be due to an inability to detect the more subtle, experimentally induced physiological sexual changes. As noted by Rosen and Beck (1988), studies which have produced higher levels of physiological sexual arousal have occasionally resulted in improved response concordance. Consistent with this explanation, in the present investigation, higher levels of physiological sexual arousal during
the Placebo conditions of Experiments 2 and 3, were paralleled by higher correlations between subjective and physiological measures. Inconsistent with this explanation, however, is the finding from Experiment 1 which indicated that subjects attained significantly lower levels of VPA responses during the No-exercise versus Exercise conditions, but displayed a higher concordance between subjective and physiological measures. In addition, subjects in the present study attained extremely high levels of physiological sexual arousal. For example, during the Delayed condition of Experiment 1, subjects attained a 135% increase in VPA responses. By contrast, previous research has noted increases in VPA responses of approximately 60% in response to erotic stimuli (e.g., Morokoff & Heiman, 1980). Consequently, it is unlikely that the low concordance between sexual measures in women can be explained solely in terms of an inability to detect physiological changes.

An alternative explanation offered for the low correlation between subjective and physiological measures is that women may estimate their degree of subjective arousal according to standards other than physiological sexual changes (Korff & Geer, 1983). That is, changes in affect or various somatic changes may be used as a criterion for estimating the degree of sexual arousal. This explanation is supported by the finding that there were no significant differences in ratings of positive affect, negative affect, or anxiety between the Exercise and No-exercise conditions of Experiments 1 and 4, and also there were no significant differences in ratings of sexual arousal. Possibly, subjects had intrinsically employed mood state as a criterion for assessing their level of sexual arousal and did not detect any differences between conditions. The results from Experiments 2 and 3 shed doubt on this explanation, however. As in Experiments 1 and 4, the results of Experiments 2 and 3 indicated no significant difference between the Clonidine and Placebo conditions in subjective ratings of affect, but did reveal significant differences in subjective ratings of sexual arousal.

The possibility that subjects employed somatic cues as indicators of sexual arousal is consistent with the findings from Experiment 2 which revealed that significant decreases in
heart rate with clonidine administration were accompanied by significant decreases in subjective sexual arousal. It is not, however, consistent with the fact that exercise significantly increased heart rate responses in Experiments 1 and 4 but did not significantly alter ratings of sexual arousal. If subjects had focused on nonsexual somatic cues to estimate their level of sexual arousal, one would expect the substantial changes in heart rate, breathing, and sweating with exposure to exercise to be accompanied by changes in subjective ratings.

With respect to the lack of concordance between subjective and physiological sexual responses in Experiments 1 and 4, it is possible that subjects misinterpreted the increases in physiological sexual arousal as residual, nonsexual consequences of exercise. This explanation is consistent with findings by Giroda (1973) who reported subjects underestimated their level of arousal to an erotic film when cues from an earlier aroused state were still readily apparent. According to the Excitation Transfer Theory (Zillmann, 1972), emotional states may be intensified by highly arousing, potentially unrelated experiences, but only when the individual has lost track of the physiological cues (Cantor et al., 1975). Possibly, in the present study, the cues from exercise (e.g., heart pounding, sweating) were more apparent than the physiological cues of sexual arousal (e.g., changes in VBV, VPA). If this were the case, however, one would expect exercise, in Experiment 1, to decrease or have no effect on subjective sexual arousal at 5 or 15 min post-exercise when the cues from exercise were readily available, but to facilitate subjective ratings of sexual arousal at 30 min post-exercise when autonomic arousal was still present but the cues less apparent. Exercise in the present study had no effect on subjective ratings of sexual arousal at either 5, 15, or 30 min post-exercise, providing only partial support for this explanation. It should be noted, however, that subjective ratings of residual excitation were not taken in the present study and, consequently, it is possible that cues from exercise were readily available during all experimental conditions.
Results of the relation between predicted and actual sexual arousal in the present investigation lend support for a previously unconsidered explanation for the often reported low concordance between subjective and physiological components of the female sexual response. In Experiments 1 through 4, there were no significant differences between subjects' responses to the question "What is the highest level of sexual arousal you think that you could obtain viewing an erotic film alone?" and their actual ratings of sexual arousal to the experimental films. For example, subjects in the Placebo condition of Experiment 2 predicted a mean level of sexual arousal of 3.73, and attained a mean level of sexual arousal of 3.60. Subjects in the Immediate condition of Experiment 1 predicted a mean level of sexual arousal of 4.42, and attained a mean level of sexual arousal of 4.00. These findings indicate that subjects in the present investigation attained close to the highest level of sexual arousal which they believed possible when viewing an erotic film alone. In other words, on a scale of 1 to 7, 4 may be the highest approximate level of cognitive arousal that subjects can be expected to attain under these, and similar, experimental conditions. This assertion is consistent with the finding that, on the same scale, subjects in the Palace and Gorzalka (1990) study attained levels of subjective sexual arousal to an erotic film of 4.5 and 3.8 with preexposure to neutral and anxiety-evoking films, respectively. If different "ceilings" existed on cognitive and physiological aspects of sexual arousal, this might explain why there has generally been a lack of concordance between these two measures. The possibility of different "ceilings" between measures seems feasible when one compares the ability of the photoplethysmograph to detect even minute changes in physiological sexual arousal (i.e., millivolts deviation), with the ability of 4 points on a Likert scale to detect changes in subjective sexual arousal. Differences in range restriction between subjective and physiological measures could statistically have the effect of lowering correlations between indices of sexual arousal (Pagano, 1990).

While there were no significant differences between ratings of subjective sexual arousal and ratings of expected arousal to an erotic film in Experiments 1 through 4, there
were significant differences between ratings of subjective sexual arousal and ratings of both the highest expected level of sexual arousal and the highest level of previously attained sexual arousal, in each of the four experiments. For example, subjects in the Delayed condition of Experiment 1 attained a mean level of sexual arousal to the experimental films of 3.91, reported previously experiencing a mean level of sexual arousal of 6.75, and predicted being able to experience a mean level of sexual arousal of 6.91 if given the appropriate situation. These findings argue against Palace and Gorzalka's (1990) assertion that the lack of correspondence between subjective and physiological measures in women can be explained in terms of social dictates which prevent women from acknowledging that they are sexually aroused. If the reason subjects reported their level of arousal to be only a 4.0, on a scale of 1 to 7, was because they believed it to be socially desirable or appropriate, it is not clear why they would admit to having experienced levels of close to 7 in the past. This finding adds further support to the notion that 4 may be approximately the highest level of subjective sexual one can expect subjects to attain when viewing an erotic film alone in a laboratory setting.

Future Directions

The body of research which comprises this dissertation provides only a small start to understanding the neural pathways by which female sexual arousal is initiated. The studies are limited by the exclusive reliance upon exercise to increase SNS activity, clonidine to decrease SNS activity, heart rate to indirectly assess SNS activity and, most importantly, the use of a contrived laboratory setting to study a very private emotional and physical experience. Given the restrictive nature of these findings, the value of this research may lie not so much in the questions that it has answered, but in the many questions that it has raised.

The unexpected discovery of a difference between orgasmic and anorgasmic women in their response to SNS activation opens an entirely new area of investigation. Studies which examine the effects of various neuropharmacological agonists and antagonists on sexual
arousal and orgasm among these women are required before in-depth understanding and therapeutic integration of this finding is possible. The finding that SNS activation had a differential influence on physiological sexual arousal, depending on the intensity used, calls for further investigation of the effects of varying intensities of SNS activation on sexual responding in both sexually functional and dysfunctional women. To this regard, the use of relatively specific SNS agonists would allow for more selective examination of the effects of SNS activation on sexual arousal. While exercise proved to be an effective means of activating the SNS because of its minimal influence on cognitive factors and its relatively unobtrusive nature, the findings it helped to reveal were confounded by a number of post-exercise physiological changes. The shifting of blood flow to restore working muscles, a rapid decline in SNS activity immediately following the cessation of exercise, and the onset of restorative PNS activity are some of the primary complicating variables. Administration of a drug which acted more selectively than exercise to increase SNS activity would have the benefit of ruling out some of these alternate explanations. The finding that SNS activation facilitated sexual responding among women with low sexual drive calls for exploration of new treatment techniques for inhibited sexual desire which aim at enhancing not only the cognitive component of sexual arousal, but the physiological component as well. To this regard, combining existing treatment techniques, such as directed masturbation or sensate focus, with imagery to increase heart rate, blood pressure, and other SNS functions, may warrant examination. Lastly, the finding that clonidine inhibited sexual arousal highlights the need for empirical exploration of the effects of other commonly prescribed psychotherapeutic drugs on sexual function in women. Research of this nature would provide insight into the pathophysiological processes of sexual dysfunctions and may aid in the development of pharmacological agents which enhance, rather than inhibit, sexual function.
BIBLIOGRAPHY


Duncan, L., & Bateman, D. N. (1993). Sexual function in women: Do antihypertensive drugs have an impact? Drug Safety, 8, 225-234.


ORGASMIC FUNCTIONING QUESTIONNAIRE (OFQ)

1. Below is a list of various ways of attaining orgasm. Please specify whether you have ever experienced an orgasm by any of these means and, if so, whether you have during the past 6 months. If you have attained an orgasm by any of these means, please indicate approximately how often you attain orgasm out of every ten times you attempt to reach orgasm.

   a) Masturbating by oneself by stimulating your clitoral/vulva area with your hand
      yes ___ past 6 months ___ number out of 10 times ___
      no ___ past 6 months ___ have never tried ___

   b) Masturbating by oneself using a vibrator
      yes ___ past 6 months ___ number out of 10 times ___
      no ___ have never tried ___

   c) Manual clitoral stimulation by a partner
      yes ___ past 6 months ___ number out of 10 times ___
      no ___ have never tried ___

   d) Intercourse plus manual clitoral stimulation
      yes ___ past 6 months ___ number out of 10 times ___
      no ___ have never tried ___

   e) Intercourse alone (without manual stimulation)
      yes ___ past 6 months ___ number out of 10 times ___
      no ___ have never tried ___

   f) Oral stimulation by a partner
      yes ___ past 6 months ___ number out of 10 times ___
      no ___ have never tried ___

   g) Other (specify)

2. If you have attained orgasm by one or more of the preceding means (in the past 6 months), how long does it generally take to achieve orgasm? (specify means)

3. Do you ever experience pain with intercourse?
   yes ___ If yes, number out of 10 times ___
   no ___

4. Do you feel that your partner often is unable to maintain an erection for a sufficient period of time?
   yes ___
   no ___

5. Do you have any of the following concerns associated with the sexual act? (please check which ones)
   _____ fear of becoming pregnant
   _____ fear of contracting a disease
   _____ concern with cleanliness
   _____ religious concerns
   _____ fear of not having an orgasm
   _____ fear of being judged negatively by one's partner
   _____ other (specify)

Meston, Jung, Hansen, and Gorzalka, 1993
PHYSICAL READINESS EXAM FOR FITNESS TEST

For most of us, physical activity poses no problem or hazard. This test has been designed to identify the small number of individuals who should seek medical advice prior to engaging in strenuous exercise.

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<th>YES</th>
<th>NO</th>
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<td>Has your doctor ever said you have heart trouble?</td>
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<td>Do you often have pains in your heart or chest?</td>
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<td>Do you often feel faint or have spells of severe dizziness?</td>
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<td>Has a doctor ever said your blood pressure was too high or too low?</td>
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<td>Has your doctor ever told you that you have a bone or joint problem that has been aggravated by exercise, or might be made worse with exercise?</td>
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<td>Are you currently using any prescription medications?</td>
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<td>Are you using any recreational drugs?</td>
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<td>Have you ever smoked?</td>
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<td>If you currently smoke, how many cigarettes per day?</td>
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<td>If you have quit smoking, when was it?</td>
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<td>Do you drink coffee?</td>
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<td>If yes, how many cups per day?</td>
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<td>Are you currently involved in a regular exercise program?</td>
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<td>Do you regularly walk or run one or more miles continuously?</td>
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<td>If yes, average number of miles per day</td>
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<td>Do you practice weight lifting or home calisthenics?</td>
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<td>Are you involved in an aerobics program?</td>
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<td>If yes, how frequently?</td>
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<td>Do you frequently participate in competitive sports?</td>
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<td>If yes, how frequently?</td>
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<td>Have you suffered any serious medical problems that you consider important for us to know?</td>
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<td>Is there any physical reason not mentioned above why you should not engage in exercise?</td>
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Signature
FILM SCALE

Instructions: Please use the following scale to evaluate how you felt during the last film. Please answer honestly and carefully. On the scale, circle any of the numbers from 1 (not at all) to 7 (intensely).

During the film, I felt:
1. Faster breathing___________________ 1 2 3 4 5 6 7
2. Faster heart beat___________________ 1 2 3 4 5 6 7
3. Perspiration________________________ 1 2 3 4 5 6 7
4. Feelings of warmth__________________ 1 2 3 4 5 6 7
5. Any physical reaction at all_________ 1 2 3 4 5 6 7

Continue on to the next page.
Instructions: Please use the following scale to evaluate how you felt during the last film. Please answer honestly and carefully. On the scale, circle any of the numbers from 1 (not at all) to 7 (intensely).

During the film, I felt:

6. Breast sensations
7. Warmth in genitals
8. Genital wetness or lubrication
9. Genital pulsing or throbbing
10. Any genital feelings
11. Sexually aroused
12. Worried
13. Anxious
14. Angry
15. Disgusted
16. Embarrassed
17. Guilty
18. Sensuous
19. A desire to be close to someone
20. Pleasure
21. Interested
22. Attracted
23. Excited
24. Sexy
25. Dirty
26. Loving
27. Sexually attractive
28. Inhibited
29. Easy to arouse
30. Incompetent
31. Sexually turned off
32. Offended
33. Bored
34. Feminine

Stop and wait for further instructions.