Anatomical Nervous System Influences

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Female Sexual Arousal

The Role of the Sympathetic Nervous System in Female Sexual Arousal...
sures of sexual responding: receptivity (lordosis quotient), which is the ratio of the number of spinal reflexes in response to male attempts to mate, proceptivity, which is measured as the number of ear wiggles per minute, and rejection behaviors, which are measured as the number of kicking, boxing, running away, and squealing behaviors in response to a male's attempt to mate. Application of these behaviors to human sexual behavior is obviously limited. Although there is no human equivalent of lordosis, it is considered an analog of sexual arousal in other mammals.

Yanase (1977) found that epinephrine, but not norepinephrine, stimulates lordosis behavior in estradiol-treated, ovariectomized rats. However, other findings have supported the role of norepinephrine in stimulating lordosis. Vincent and Feder (1988) found that injection of either an alpha or alpha-adrenergic agonist induced lordosis behavior in a small proportion of guinea pigs, but when used in combination, induced lordosis in 76% of the animals.

Studies examining the effects of adrenergic and antiadrenergic agents are complicated by the fact that some of the drugs used do not act exclusively on adrenergic systems. For example, yohimbine acts as both an alpha-adrenoceptor antagonist and a serotonin receptor antagonist (Broadley, 1996, p. 216). In such a case, the study must rule out the effects of different neurotransmitter systems on the phenomenon of interest. Nock and Feder (1979) observed that the dopamine beta-hydroxylase inhibitor U-14,624 abolished lordosis behavior in female guinea pigs. U-14,624 was believed to increase dopamine and serotonin availability while decreasing norepinephrine levels. After both dopamine and serotonin blockade failed to reverse the effects of U-14,624, the authors determined that only concurrent administration of the alpha-adrenergic agonist clonidine was able to restore lordosis behavior in animals treated with U-14,624. Thus, the inhibitory effects of U-14,624 on lordosis were concluded to be associated primarily with decreased availability of norepinephrine, rather than increased dopamine or serotonin levels.

Although central mechanisms have usually been implicated in the adrenergic control of lordosis, a peripheral mechanism cannot be ruled out. The facilitatory effect of norepinephrine on lordosis responses may indicate the involvement of the SNS. If so, one would expect drugs that decrease SNS activity might also decrease sexual arousal. To examine this possibility, Meston, Moe, and Gorzalka (1996) conducted a series of studies on sexual behavior in female rats treated with various drugs that inhibit SNS activity. The first study examined the influence of clonidine, an antihypertensive medication, on sexual responding. Clonidine acts centrally and peripherally as an alpha-adrenergic agonist, presumably with the effect of decreasing norepinephrine release. In the second and third studies, the effects of drugs guanethidine and naphazoline on sexual responding were examined. Naphazoline also acts as an alpha-adrenergic agonist, and guanethidine has a distinct mechanism to directly block the release of norepinephrine from sympathetic neurons. These two drugs were chosen because they are believed to exert effects similar to those of clonidine, but they do not cross the blood-brain barrier. Each study included 15 ovariectomized females treated with estrogen and progesterone (clonidine; norepinephrine) with different drugs to select rejections in which the animals received either saline or moderate or high doses of the drug.

Clonidine, guanethidine, and naphazoline all suppressed lordosis responses at both moderate and high doses. Clonidine and guanethidine significantly decreased proceptivity behavior at both moderate and high doses, suggesting a decrease in activity of the SNS. However, a decreased increase in active rejection behaviors. Because guanethidine and naphazoline act to selectively inhibit peripheral sympathetic outflow without influencing adrenergic mechanisms at a central level, the results of this study suggest that inhibition of the SNS may inhibit sexual behavior in the female rat.

Effect of Direct Stimulation of Nerves and Tissues

In vivo studies of direct nerve stimulation can differentiate genital responses to parasympathetic and sympathetic outflow. Studies of this type have used electrical stimulation of dissected nerves in order to determine specific effects on target tissues. In rats, electrical stimulation of both the parasympathetic (gastric) and hypogastric (sympathetic) nerves induced contractions of the uterine and cervical smooth muscle, which were further enhanced by pretreatment with estrogen (Sato, Hayaishi, & Garfield, 1989; Sato, Hotta, Nakayama, & Suzuki, 1996). Pelvic nerve stimulation increased uterine blood flow, while hypogastric nerve stimulation decreased blood flow. The decrease in uterine blood flow following hypogastric stimulation was diminished with phenoxybenzamine, an alpha-adrenergic antagonist (Sato et al., 1996). Similarly, in guinea pigs, stimulation of the hypogastric nerve induced uterine contractions and increased uterine sensitivity to methyldopamine (Marshall & Russe, 1970). Stimulation of the pelvic nerve, which comprises both pelvic and hypogastric nerves, increased uterine and vaginal blood flow in rats (Vachon, Zimmerman, Zabran, & Gurr, 2000). However, another study found that direct stimulation of the sympathetic chain countered the increase in vaginal blood flow resulting from pelvic nerve stimulation (Giuliano et al., 2001).

A different strategy used to examine adrenergic influences on genital
Human Models of SNS Activity and Social Absence

The neuroscientific research (Kahn et al., 2002) on the neural circuitry of social interaction, used in the context of social presence, may provide new insights into the human experience of social absence. If this is true, then the engagement of social presence in the performance of social tasks can lead to the development of specific brain regions that are involved in the experience of social absence. These regions may activate in response to social interactions with others, and the engagement of these regions may be indicative of the subjective experience of social absence.

Social presence involves the subjective experience of being present with others, and the engagement of specific brain regions may be indicative of the subjective experience of social absence. If this is true, then the engagement of these regions may be indicative of the subjective experience of social absence. These regions may activate in response to social interactions with others, and the engagement of these regions may be indicative of the subjective experience of social absence.

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Effects of Physiological Induced SNS Activation on Sexual Arousal

I. Introduction

II. Method

III. Results

IV. Discussion

V. Conclusion

References

Appendices
The distinct human ability to control the potential controlled negative state of emotion that is used to identify, label, and modulate the emotional states of others involves the interaction of the frontal lobes and the amygdala. The frontal lobes are involved in the regulation of the amygdala by processing information about the emotional significance of stimuli. The amygdala, on the other hand, is involved in the processing of emotional information and the generation of emotional responses. The interaction between these two regions allows for the modulation of emotional responses in a socially appropriate manner. This process is supported by the limbic system, which plays a crucial role in emotional regulation. The amygdala, in particular, is involved in the processing of emotional information and the generation of emotional responses.
sexual arousal and orgasm.

Neuroendocrine processes during sexual arousal and orgasm.

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Physiology and Neuroendocrinology of Sexual Response.