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A neurologic excitation/inhibition “faucet model” for orgasm and pain

Sexual Medicine Reviews (SMR) is privileged to bring you, our readers, not only outstanding review articles but also cutting-edge theories and concepts for your edification and contemplation. Please enjoy this new concept that may change the way that you think about orgasm and pain.

A prevalent concept of neuronal inhibition in sexual response is that it opposes the effects of neuronal excitation. One model of this concept is the classical arm balance in which excitation and inhibition are on opposite arms of the balance such that when one is up, the other is down.¹ Another such either-or model is that of Bloemers et al: “low sexual desire in women (with HSDD [hypoactive sexual desire disorder]) could be due to either a relative insensitive brain system for sexual cues or to enhanced activity of sexual inhibitory mechanisms.”² A unitary role of inhibition attributed to specific neurotransmitters was emphasized by Georgiadis et al: “the activation of inhibitory pathways for sexual arousal and desire generates a state of reduced sexual wanting.”³ However, as emphasized in the recent article in SMR, the nervous system is actually organized such that the intensity of neuronal inhibition usually increases concurrently with an increase in excitation, thereby attenuating opposing activity and actually *enabling* the excitation intensity to increase.⁴ This principle is exemplified in the case of even the most basic functions of the nervous system (ie, the spinal reflexes) in which excitation of the simplest reflex, the leg extensor reflex, simultaneously activates inhibition of the antagonistic flexor muscles. Alternatively, excitation of the flexor reflex simultaneously activates inhibition of the antagonistic extensor muscles. Thus, the inhibition enables the excitation to function unopposed by the antagonistic muscles; it also serves to protect the opposing muscles from being torn from their tendon.

In other motor contexts, the intensity of inhibition increases to match the intensity of excitation, as exemplified in graceful and nonspastic movement of precision, such as moving a chess piece, as well as in intense movement, as in running up a flight of stairs (cf Figure 1 in Komisaruk and Rodriguez del Cerro).⁴ Inhibition also plays an essential role in the sensory system: pain activates the endogenous pain gate (ie, pain-inhibiting) system as a protective mechanism. Furthermore, in the case of sexual response, as the intensity of response to vaginal self-stimulation increases from pressure to pleasure to orgasm, so does the intensity of analgesia (ie, the intensity of pain inhibition), as measured by a >100% increase in pain

threshold at orgasm over baseline, in simple pressure, or in pleasurable threshold prior to orgasm.⁴

As reviewed in the SMR article, the neuronal system that transmits genital sensation to the brain (ie, the spinothalamic tract) also transmits pain. In the brain, some of the major components that are activated by pain are also activated by orgasm (eg, insula, anterior cingulate cortex, periaqueductal gray, raphé system).⁴ These are likely sites at which there are inhibitory interactions between genital afferent activity and pain perception. In a more general sense, as proposed in the review article, when the intensity of excitation surpasses that of concurrent inhibition, the excitation is perceived as aversive or painful (cf Figure 2 from Komisaruk and Rodriguez del Cerro).⁴ Thus, neuronal inhibition enables excitation to increase in intensity without becoming aversive or painful, up to the peak of orgasm.⁴ As such, activation of the pain system, albeit at a “subpain” intensity maintained by the inhibitory action of the genital afference, is essential to orgasm, in conjunction with genital sensory excitatory activity.

We suggest a metaphor of this view of concurrent excitation and inhibition—with each increasing in intensity as hot water (excitatory) and cold water (inhibitory) jointly flowing from a faucet: a *faucet model*. This metaphor is different from that of classical balance¹ in that it incorporates variations in intensity of excitation and inhibition independently as well as concurrently and jointly, rather than just reciprocally, which we believe more appropriately represents the function of the nervous system. Thus, at extremes, a low-vs-high flow rate of exclusively hot water would represent mild or intense pain, whereas a high flow rate of hot and cold water would represent intense but nonpainful/comfortable excitation, as in orgasm, with perhaps the hot water intensity just exceeding the cold at orgasm.

In the case of pleasure dissociative orgasmic disorder or a weak anhedonic orgasm—as described by a woman in the SMR review as “I expected a mountain and got an anthill”—each would be represented by the faucet model as hot and cold water at a very low flow rate,⁴ with the intensities of the hot just exceeding the cold at orgasm. Premature ejaculation could be represented by the same combination of hot and cold at a low flow rate, with the hot just exceeding the cold at the point of ejaculation. The faucet model could represent cases of anorgasmia and/or hypoactive sexual desire disorder as cold consistently surpassing hot at any

flow rate, from low to high, depending on the dynamic qualities of the anorgasmia—in other words, not “getting going” or perhaps “getting close but not there” with regard to orgasm.

It is an empirical question whether this faucet model could be effectively applied therapeutically depending on the symptomatology, such as by 1 or a combination of pharmaceuticals that are primarily stimulatory or *hot* (eg, Adderall or pseudoephedrine) or inhibitory or *cold* (eg, diazepam or baclofen). For example, in the case of orgasmic anhedonia, would a titrated combination of Adderall and diazepam enable a more delayed and intense orgasm?

Read this and other editorials and reviews in your *SMR* to learn of the latest theories in sexual medicine. As a new year starts, we are excited to bring new concepts for your consideration.

Conflicts of interest: None declared.

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