

EDITORIAL

On no! Where did my “big O” go? Or could severe menopausal symptoms have stolen my orgasm?

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The often-elusive female orgasm, broadly defined as “a climax of peak sexual excitement, characterized by powerful feelings of physical pleasure and sensation centered in the genitals, which includes a discharge of accumulated erotic tension,” is the subject of a secondary analysis from a multicenter, Latin American, cross-sectional survey, by Urrunaga-Pastor et al¹ published in this edition of *Menopause*. The authors conclude that severe climacteric symptoms, a composite of somatic, psychological, and urogenital symptoms on the menopause rating scale, are associated with orgasmic dysfunction based upon comparing menopause rating scale scores with associated results on the Female Sexual Function Index. These are both validated scales in Spanish. These findings were assessed to be independent of demographic, clinical, and partner variables. Their findings parallel the results of other similar surveys from Europe, Asia, and other geographies.²⁻⁴ The strongest association in Urrunaga-Pastor et al findings was the occurrence of orgasmic dysfunction with severe urogenital symptoms (ie, sexual pain). These authors acknowledge and discuss the potential shortcomings of their study, many of which are inherent in its design. But, their findings, while not surprising to any sexual medicine or menopausal medicine practitioner, and easy to dismiss as “just menopausal vulvovaginal atrophy/genitourinary syndrome of menopause (VVA/GSM),” require further commentary.

While less prevalent than either low sexual desire or reduced sexual arousal with associated distress, orgasmic dysfunction, and associated distress was still highly prevalent in 5.2% to 6.1% of PRESIDE Survey US women.⁵ Importantly, the peak prevalence of orgasmic dysfunction occurred at about age 60 and remained elevated with increasing age (65 y and older), suggesting a variety of potential associated factors both consistent with and contrary to those described by Urrunaga-Pastor et al.¹ Quite consistent with their findings is the strong correlation with GSM which remains mild in most women shortly after menopause, but generally progresses in severity, thereafter. In my personal experience practicing gynecology for more than 40 years, sexual pain nearly always trumps orgasm, especially if the focus, as in Urrunaga-Pastor, is heterosexual, and likely to derive from penetrative intercourse.

The sixth decade of life when orgasmic dysfunction with associated distress peaks in prevalence⁵ is also more likely to involve multiple well-known correlates of sexual dysfunction as well as GSM. These include the entire spectrum of the bio/psycho/social model of sexual function/dysfunction. A short list of such correlates could include: (bio) VVA/GSM, persistent vasomotor symptoms, sleep disturbance related to both VMS and of aging per se, weight gain, sleep apnea, metabolic syndrome, type II diabetes, subclinical cardiovascular disease, etc, (psycho/social) age, length of sexual relationship, burdens of teenage children/aging parents, depression related to GSM, depressed mood related to menopause, weight gain, and body image alterations, etc. These correlates may also synergize with one another, augmenting their individual impact on sexual function while further complicating therapeutic interventions. For example, weight gain during the menopausal transition may affect metabolic syndrome, type II diabetes, and cardiovascular disease risk which, in turn, can impact vascular reactivity, an important element of both arousal and orgasm. The weight gain can also affect sleep and increase the risk of sleep apnea. Sleep apnea increases cardiovascular disease risk, augments further weight gain, and augments poor food choices, and is associated with sexual dysfunction and so on, and so on, and so on. I have not mentioned the impact of weight gain on sexual self-image, which independently of the biological correlates just mentioned, can affect sexual functioning, especially in heterosexual partnered sexual activities (the precise population reported by Urrunaga-Pastor et al).¹

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The challenges of female orgasm are not unique to aging women, however. The orgasm gap, or pleasure gap, is a social phenomenon referring to the general disparity between heterosexual men and women in terms of sexual satisfaction, specifically, the unequal frequency in achievement of orgasm during sexual encounters, and in particular heterosexual encounters like those reported here by Urrunaga-Pastor et al.¹ The orgasm gap phenomenon is not new. It was described in 1952 by Kinsey et al,⁶ further documented by Masters and Johnson⁷ noting orgasmic function was better in lesbians than in heterosexual women in 1966, and then reemphasized by Laumann⁸ in 2000. The concept was most recently popularized by University of Florida, Professor of Psychology, Mintz⁹ in her popular book: *Becoming Cliterate: Why Orgasm Equality Matters—And How to Get It*. Further, many women “fake” orgasm, so the relative differences between orgasmic prevalence in men versus women is likely to be even greater than reported.

Specific treatment of orgasmic dysfunction remains one of the major challenges in sexual medicine. Regardless of age, elimination of as many biopsychosocial impediments to orgasm remains first and foremost. As suggested by Urrunaga-Pastor et al, this should focus on urogenital symptoms, like VVA/GSM, the subject of two recent North American Menopause Society Treatment Guidelines.^{10,11} Once resolved, which might require a multipronged approach to include lubricants, moisturizers, estrogens, androgens, pelvic floor physical therapy, self-dilation, etc, a multitude of marginally helpful approaches to delayed, muted or absent orgasm still remain. These include better sexual stimulation whether by newly learned techniques including organized and tasteful teaching by one’s peers (ie, <https://www.omgyes.com>), by one’s partner, or utilizing what I call “power tools” aka sex toys/vibrators. As the world’s population ages, the rapidly growing sex toy/vibrator industry does as well, with the global market for Sex Toys estimated at US\$24.5 billion in the year 2020 and projected to reach a revised size of US\$36.1 billion by 2027. This equates to some 60 million sex toys sold every year with future growth estimates to reach a market size similar to smartphones and personal computers.¹²

Consistent with our biopsychosocial model, cognitive behavioral therapy and mindfulness-based cognitive behavioral therapy have benefit in women with orgasmic dysfunction along with medications targeting hypoactive sexual desire disorder, but with a “downstream” or “carry-over” benefit for orgasmic function.¹³ While the quality of this information is highly variable, especially for therapies used “off-label” in small trials, and some of the treatments have demonstrated efficacy only or primarily in premenopausal women, buspirone, bupropion, flibanserin, bremelanotide, tibolone,¹⁴ and testosterone¹⁵ have such “downstream” improvement in orgasmic function. While some data suggest that the phosphodiesterase-5 inhibitors in particular populations (ie, with selective serotonin reuptake inhibitor-related sexual dysfunction) likewise have “downstream” benefit for

orgasm,¹⁶ such benefit of these vasodilators has been called into question.¹⁷

To attribute altered or absent orgasmic function in menopausal women primarily or entirely to GSM and associated sexual pain would be a disservice to our patients. However, optimal treatment of secondary, acquired orgasmic dysfunction remains elusive. Significantly more research focused specifically on orgasmic function and dysfunction is desperately needed.

REFERENCES

1. Urrunaga-Pastor D, Mezones-Holguin E, Blümel JE, et al. Female orgasmic dysfunction and severe climacteric symptomatology in women aged 40-59 years: an independent association from an analysis of multicenter Latin American study. *Menopause* 2022;29:654-663. doi: 10.1097/GME.0000000000001973
2. Gozuyesil E, Gokyildiz Surucu S, Alan S. Sexual function and quality-of-life-related problems during the menopausal period. *J Health Psychol* 2018;23:1769-1780. doi: 10.1177/1359105317742194
3. Lou W-J, Chen B, Zhu L, et al. Prevalence and factors associated with female sexual dysfunction in Beijing, China. *Chinese Med J* 2017;130:1389-1394. doi: 10.4103/0366-6999.207466
4. Singh J, Tharyan P, Kekre N, et al. Prevalence and risk factors for female sexual dysfunction in women attending a medical clinic in south India. *J Postgrad Med* 2009;55:113-120. doi: 10.4103/0022-3859.52842
5. Shifren JL, Monz BU, Russo PA, et al. Sexual problems and distress in United States women: prevalence and correlates. *Obstet Gynecol* 2008;112:970-978. doi: 10.1097/AOG.0b013e3181898cdb
6. Kinsey AC, Pomeroy WB, Martin CE, Gebhard PH (May 22, 1998). *Sexual Behavior in the Human Female* (1998 reprint). Bloomington, IN: Indiana University Press; 1998: 519-520.
7. Masters WH, Johnson VE. *Human Sexual Response*. Boston, MA: Little, Brown; 1966.
8. Laumann EO (December 15, 2000). *The Social Organization of Sexuality: Sexual Practices in the United States*. Chicago, IN: University of Chicago Press; 2000: 130.
9. Mintz LB (May 15, 2018). *Becoming Cliterate: Why Orgasm Equality Matters – And How to Get It*. New York: HarperCollins; 2018.
10. The 2020 genitourinary syndrome of menopause position statement of The North American Menopause Society. *Menopause* 2020;27:976-992. doi: 10.1097/GME.0000000000001609
11. Faubion SS, Larkin LC, Stuenkel CA, et al. Management of genitourinary syndrome of menopause in women with or at high risk for breast cancer: consensus recommendations from The North American Menopause Society and The International Society for the Study of Women’s Sexual Health. *Menopause* 2018;25:596-608. doi: 10.1097/GME.00000000000001121
12. Available at: <https://www.globenewswire.com/news-release/2021/02/10/2172894/0/en/Global-Sex-Toys-Market-with-Impact-of-COVID-19-2020-2027-US-Accounted-for-Over-28-9-of-the-Market-Size-in-2020.html>. Accessed: April 4, 2022.
13. Kingsberg SA, Simon JA. Female hypoactive sexual desire disorder: a practical guide to causes, clinical diagnosis, and treatment. *J Womens Health (Larchmt)* 2020;29:1101-1112. doi: 10.1089/jwh.2019.7865
14. Laan E, van Lunsen RH, Everaerd W. The effects of tibolone on vaginal blood flow, sexual desire and arousability in postmenopausal women. *Climacteric* 2001;4:28-41.
15. Davis SR, Baber R, Panay N, et al. Global consensus position statement on the use of testosterone therapy for women. *J Clin Endocrinol Metab* 2019;104:4660-4666. doi: 10.1210/je.2019-01603
16. Nurnberg HG, Hensley PL. Selective phosphodiesterase type-5 inhibitor treatment of serotonergic reuptake inhibitor antidepressant-associated sexual dysfunction: a review of diagnosis, treatment, and relevance. *CNS Spectr* 2003;8:194-202. doi: 10.1017/s1092852900024433
17. Chivers ML, Rosen RC. Phosphodiesterase type 5 inhibitors and female sexual response: faulty protocols or paradigms? *J Sex Med* 2010;7 (2 pt 2): 858-872. doi: 10.1111/j.1743-6109.2009.01599.x