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Management of Menopause-Related Vasomotor, Sleep, and Mood Symptoms

Committee on Clinical Practice Guidelines–Gynecology. This Clinical Practice Guideline was developed by the American College of Obstetricians & Gynecologists' Committee on Clinical Practice Guidelines–Gynecology in collaboration with Bruce Pier, MD, Robert Kauffman, MD, MSCP, and Catherine Cansino, MD, MPH.

PURPOSE: To provide evidence-based recommendations for the management of vasomotor, sleep, and mood symptoms related to spontaneous menopause and induced menopause (at or beyond age 40 years).

TARGET POPULATION: Perimenopausal and postmenopausal individuals assigned female sex at birth (ie, women, transgender men, and gender-diverse individuals) with vasomotor, sleep, and mood symptoms related to spontaneous or induced menopause.

METHODS: This guideline was developed using an a priori protocol in conjunction with a writing team consisting of a subspecialist in reproductive endocrinology and infertility, a Menopause Society Certified Practitioner and subspecialist in reproductive endocrinology and infertility, and a specialist in obstetrics and gynecology. Studies that moved forward to the full-text screening stage were assessed by two authors from the writing team based on standardized inclusion and exclusion criteria. Included studies underwent quality assessment, and a modified GRADE (Grading of Recommendations Assessment, Development and Evaluation) evidence-to-decision framework was applied to interpret and translate the evidence into recommendation statements.

RECOMMENDATIONS: This Clinical Practice Guideline includes recommendations on pharmacologic and nonpharmacologic interventions for the management of menopause-related vasomotor, sleep, and mood symptoms. Recommendations are classified by strength and evidence quality. Ungraded Good Practice Points are included to provide clinical guidance when a formal recommendation could not be made because of inadequate or nonexistent evidence.

INTRODUCTION

Spontaneous (or natural) menopause is defined as the permanent cessation of menstruation (ie, absence of menses for 12 consecutive months) caused by the gradual depletion of functional oocytes and the progressive loss of ovarian reproductive activity (1, 2). In contrast, *induced menopause* is the abrupt cessation of menstruation and ovarian function that occurs due to medical treatment (eg, surgery, chemotherapy, radiation therapy) (2). The mean age of spontaneous menopause is

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49.9 years in the United States (3). However, there is wide variation globally, with the mean age of menopause ranging from 46 to 52 years (4). Spontaneous menopause that occurs between age 40 and 45 years is known as *early menopause* (2). *Premature ovarian insufficiency* (formerly *primary ovarian insufficiency*) is the loss of ovarian function before age 40 years (5).

Most women begin to undergo the physiologic changes associated with menopause in the years preceding the final menstrual period (6). This interval often is referred to as *perimenopause* and includes early postmenopause (7). Perimenopause is characterized by hormonal fluctuations that may contribute to vasomotor symptoms (VMS), mood changes, and sleep disturbance (6) that can adversely affect quality of life and have deleterious effects on work, life, and career (8–10). High symptom burden also is associated with significant direct and indirect economic costs due to increased use of health care resources, work absenteeism, and loss of work productivity (8, 9). This document provides evidence-based guidelines on the management of vasomotor, mood, and sleep symptoms related to spontaneous menopause and induced menopause (at or beyond age 40 years) in perimenopausal and postmenopausal women, transgender men, and gender-diverse individuals who were assigned female sex at birth (see "Use of Language" later in this document). Discussion of the management of other menopause-related symptoms is beyond the scope of this guideline. The following topics are addressed in other American College of Obstetricians & Gynecologists (ACOG) documents: premature ovarian insufficiency (5), management of urogenital symptoms in individuals with a history of estrogen-dependent breast cancer (11), female sexual dysfunction (12), osteoporosis (13, 14), and compounded bioidentical menopausal hormone therapy (MHT, including testosterone pellet therapy) (15).

SUMMARY OF RECOMMENDATIONS

Hormone Therapy

ACOG recommends the use of systemic MHT with estrogen and a progestogen (in individuals with a uterus) or estrogen alone (if endometrial protection is not needed) for the management of VMS in individuals without contraindications to hormone use. **(STRONG RECOMMENDATION, MODERATE-QUALITY EVIDENCE)**

ACOG suggests the combination of conjugated estrogen and bazedoxifene as an alternative to estrogen–progestogen therapy for the management of VMS in individuals without contraindications to hormone use. **(CONDITIONAL RECOMMENDATION, LOW-TO-MODERATE–QUALITY EVIDENCE)**

ACOG recommends the use of systemic MHT with estrogen and a progestogen (in individuals with a uterus) or estrogen alone (if endometrial protection is not needed) for the management of vasomotor-related sleep disturbance in individuals without contraindications to hormone use. **(STRONG RECOMMENDATION, MODERATE-QUALITY EVIDENCE)**

ACOG suggests the combination of conjugated estrogen and bazedoxifene as an alternative to estrogen–progestogen therapy for the management of vasomotor-related sleep disturbance in individuals without contraindications to hormone use. **(CONDITIONAL RECOMMENDATION, LOW-TO-MODERATE–QUALITY EVIDENCE)**

The available evidence is insufficient to recommend for or against the use of estrogen–progestogen or estrogen-only MHT for the treatment of menopause-related depressive symptoms. Perimenopausal and postmenopausal patients who meet diagnostic criteria for major depressive disorder (MDD) or for whom depression is the predominant symptom should be treated according to depression-management guidelines. **(GOOD PRACTICE POINT)**

The decision to initiate MHT for the management of VMS or vasomotor-associated sleep disturbance should be based on a shared decision-making discussion about the patient's symptoms and the benefits and risks of therapy, with consideration of each patient's age; medical history; baseline risks of cardiovascular disease, breast cancer, and venous thromboembolism (VTE); and individualized treatment goals and priorities. **(GOOD PRACTICE POINT)**

When MHT is selected for the management of VMS and vasomotor-related sleep disturbance, ACOG recommends initiation of therapy before age 60 years or within 10 years of the start of natural menopause in individuals without contraindications to hormone use. **(STRONG RECOMMENDATION, LOW-TO-MODERATE–QUALITY EVIDENCE)**

Persistent or unscheduled bleeding after initiation of MHT warrants evaluation for endometrial intraepithelial neoplasia/atypical endometrial hyperplasia and endometrial cancer. **(GOOD PRACTICE POINT)**

The decision about when and how to discontinue MHT should be individualized based on ongoing risk factor assessment and a shared decision-making discussion of the benefits and risks. **(GOOD PRACTICE POINT)**

Nonhormonal Pharmacologic Management

ACOG recommends selective serotonin reuptake inhibitors (SSRIs) or serotonin and norepinephrine reuptake inhibitors (SNRIs) for the management of menopausal symptoms (vasomotor and depression) in patients who do not desire or have a contraindication to hormone therapy. **(STRONG RECOMMENDATION, MODERATE-TO-HIGH–QUALITY EVIDENCE)**

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ACOG recommends neurokinin receptor antagonists for the management of VMS and vasomotor-related sleep disturbance in patients who do not desire or have a contraindication to hormone therapy. **(STRONG RECOMMENDATION, MODERATE-TO-HIGH–QUALITY EVIDENCE)**

ACOG suggests gabapentin for the management of VMS when other recommended nonhormonal pharmacologic interventions are ineffective or poorly tolerated by patients who do not desire or have a contraindication to hormone therapy. **(CONDITIONAL RECOMMENDATION, MODERATE-QUALITY EVIDENCE)**

Nonpharmacologic Interventions

ACOG suggests cognitive behavioral therapy (CBT) for the management of VMS and menopause-related sleep disturbance and mood symptoms. **(CONDITIONAL RECOMMENDATION, LOW-TO-MODERATE–QUALITY EVIDENCE)**

ACOG suggests mindfulness-based interventions (MBIs) to improve menopause-related sleep disturbance and mood symptoms. **(CONDITIONAL RECOMMENDATION, LOW-TO-MODERATE–QUALITY EVIDENCE)**

ACOG suggests physical exercise to improve menopause-related sleep disturbance and mood symptoms. **(CONDITIONAL RECOMMENDATION, LOW-TO-MODERATE–QUALITY EVIDENCE)**

ACOG suggests clinical hypnosis to reduce VMS and improve menopause-related sleep disturbance and anxiety symptoms. **(CONDITIONAL RECOMMENDATION, LOW-QUALITY EVIDENCE)**

ACOG suggests acupuncture to reduce VMS and improve menopause-related sleep disturbance and mood symptoms. **(CONDITIONAL RECOMMENDATION, LOW-QUALITY EVIDENCE)**

METHODS

ACOG Clinical Practice Guidelines provide clinical management recommendations for a condition or procedure by assessing the benefits and harms of care options through a systematic review of the evidence. This guideline was developed using an a priori protocol in conjunction with a writing team consisting of a subspecialist in reproductive endocrinology and infertility, a Menopause Society Certified Practitioner and subspecialist in reproductive endocrinology and infertility, and a specialist in obstetrics and gynecology. A full description of the Clinical Practice Guideline methodology has been published separately (16). The following description is specific to this Clinical Practice Guideline.

STRENGTH OF RECOMMENDATION

STRONG

ACOG recommends:
Benefits clearly outweigh harms and burdens. Most patients should receive the intervention.

ACOG recommends against:
Harms and burdens clearly outweigh the benefits. Most patients should not receive the intervention.

CONDITIONAL

ACOG suggests:
The balance of benefits and risks will vary depending on patient characteristics and their values and preferences. Individualized, shared decision making is recommended to help patients decide on the best course of action for them.

QUALITY OF EVIDENCE

HIGH

Randomized controlled trials, systematic reviews, and meta-analyses without serious methodologic flaws or limitations (eg, inconsistency, imprecision, confounding variables)

Very strong evidence from observational studies without serious methodologic flaws or limitations

There is high confidence in the accuracy of the findings and further research is unlikely to change this.

MODERATE

Randomized controlled trials with some limitations

Strong evidence from observational studies without serious methodologic flaws or limitation

LOW

Randomized controlled trials with serious flaws

Some evidence from observational studies

VERY LOW

Unsystematic clinical observations

Very indirect evidence from observational studies

GOOD PRACTICE POINTS

Ungraded Good Practice Points are incorporated when clinical guidance is deemed necessary in the case of extremely limited or nonexistent evidence. They are based on expert opinion as well as review of the available evidence.

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Literature Search

ACOG medical librarians completed a comprehensive literature search for primary literature within MEDLINE through Ovid and PubMed, the Cochrane Database of Systematic Reviews, and guidelines from ACOG and other organizations. Parameters for the search included human-only studies published in English. The search was restricted to studies published from 2013 to March 2024. The MeSH terms and keywords used to guide the literature search can be found in Appendix A (available online at https://links.lww.com/AOG/E979). Final supplemental literature searches were performed in July 2025 and March 2026 to ensure that any newly published, high-level sources were addressed in the final manuscript.

Study Selection

A title and abstract screen of all studies was completed by ACOG research staff. Studies that moved forward to the full-text screening stage were assessed by two authors from the writing team based on standardized inclusion and exclusion criteria. The population of interest included individuals assigned female sex at birth (ie, women, transgender men, and gender-diverse individuals) with vasomotor, sleep, and mood symptoms related to spontaneous or induced menopause. To be considered for inclusion, studies had to be conducted in countries ranked very high on the United Nations Human Development Index (17) and published in English. However, the authors considered acupuncture studies conducted in China, which is ranked high on the United Nations Human Development Index, because this was the setting for most of the relevant literature on this intervention.

Systematic reviews, meta-analyses, and randomized controlled trials (RCTs) were considered for inclusion, and observational studies were excluded. Guidelines from other medical societies also were included and used to inform ACOG recommendations. A PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) flow diagram of the included and excluded studies can be found in Appendix B (available online at https://links.lww.com/AOG/E980). Included studies underwent quality assessment and had key details extracted (study design, sample size, details of interventions, outcomes) and organized into summary evidence tables (Appendix C, available online at https://links.lww.com/AOG/E981).

Recommendation and Manuscript Development

A modified GRADE (Grading of Recommendations Assessment, Development and Evaluation) evidence-to-decision framework was applied to interpret and translate the evidence into draft recommendation statements, which were classified by strength and evidence quality (18, 19). Ungraded Good Practice Points were incorporated to provide clinical guidance in the case of extremely limited or nonexistent evidence. They are based on expert opinion as well as review of the available evidence (20).

All the recommendations and supporting evidence tables were reviewed, revised as appropriate, and affirmed by the Committee on Clinical Practice Guidelines–Gynecology at a meeting. The guideline manuscript then was written and subsequently reviewed and approved by the Committee on Clinical Practice Guidelines and other internal review bodies before continuing to publication.

CLINICAL OVERVIEW

Stages of Menopause

The 2011 Stages of Reproductive Aging Workshop +10 criteria divide spontaneous (natural) menopause into various stages based on characteristic changes in the menstrual cycle and reproductive hormone levels (7, 21). Perimenopause spans several stages and includes the *early menopausal transition* (characterized by persistent variability in menstrual cycle length), the *late menopausal transition* (defined as amenorrhea lasting 60 days or longer), the final menstrual period, and the first year of *early postmenopause* (marked by the initial rapid hormonal changes during the 12 months after the final menstrual period) (7).

Menopausal Symptoms

The menopausal transition is marked by fluctuations in hormone levels as ovarian function begins to slow down (1, 21). Serum levels of estradiol and progesterone decrease and follicle-stimulating hormone levels increase, resulting in physiologic changes and clinical symptoms (1). Although women report experiencing a variety of menopausal symptoms, VMS, sleep disturbance, and mood changes are among the most reported concerns and often co-occur (22–25). Findings from SWAN (Study of Women's Health Across the Nation), a prospective longitudinal study, show a strong bidirectional association between VMS and sleep disturbance and between VMS and frequent mood changes (eg, irritability, tearfulness, sudden anger) (22). Moreover, the presence of high levels of depression or anxiety was a strong predictor of the simultaneous occurrence of VMS, sleep disturbance, and frequent mood changes (22). However, some individuals experienced only one symptom or varying symptom clusters over time, suggesting that the interplay of these symptoms is complex and individualized (22).

Vasomotor Symptoms

Vasomotor symptoms include hot flushes and night sweats (26). Although the sudden sensation of extreme

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heat in the upper body, particularly the face, neck, and chest, is sometimes referred to as a "hot flash," the term "hot flush" is preferred because it conveys that the sensation is not necessarily momentary and typically lasts 1–5 minutes (26). Hot flushes can be accompanied by perspiration, chills, clamminess, anxiety, and, on occasion, heart palpitations (26, 27). Between 50% and 92% of women in menopause report experiencing VMS, with varying frequency and severity (28–30). Vasomotor symptoms have a peak incidence in late perimenopause and early postmenopause (31) but can begin more than 10 years before the final menstrual period and continue for 10 years or more after menopause (28).

Vasomotor symptoms are believed to result from a narrowing of the thermoregulatory (or thermoneutral) zone in the hypothalamus, such that small increases in core body temperature activate a rapid, exaggerated heat-dissipation response (vasodilation, sweating, and decreased skin resistance) (28, 32–34). Although the underlying pathophysiologic pathways that give rise to VMS are not completely understood, fluctuating and declining estrogen levels during the menopausal transition are believed to play a central role (34). For example, diminishing estrogen levels appear to alter the activity of the neurotransmitters serotonin and norepinephrine, which are involved in thermoregulation (32). The decline in circulating estrogen also triggers hyperactivation of kisspeptin/neurokinin B/dynorphin (KNDy) neurons, which innervate the thermoregulatory center of the hypothalamus (35, 36). This overstimulation causes KNDy neurons to increase secretion of the neuropeptide neurokinin B, which disrupts thermoregulatory activity and results in VMS (35, 36).

Sleep Disturbance

Approximately 16–66% of women in the United States and 8.9–73% of women worldwide report experiencing menopause-related sleep disruption (37). The most common sleep symptoms associated with menopause include frequent nighttime awakening and increased wakefulness after sleep onset (38). Menopausal sleep disturbance is believed to be the result of a complex interplay of a variety of factors, including hormonal changes that affect hypothalamic regulation of body temperature and the sleep–wake cycle, VMS, and mood symptoms such as anxiety and depression (38, 39). Sleep disturbance during midlife also can be due to a primary sleep disorder (eg, insomnia, sleep-disordered breathing, restless legs syndrome), the likelihood of which increases with advancing age (38, 39). Other contributors to menopausal sleep disruption include the use of certain medications (eg, medications that affect the central nervous system), general health impairment (eg, urologic or musculoskeletal disorders), and external stressors and psychosocial issues (eg, financial strain, unemployment, and divorce) (40, 41).

Mood Symptoms

During the menopausal transition, some individuals may experience symptoms of depression, anxiety, or both (42, 43). Although the underlying pathophysiology is not completely understood, fluctuating and declining levels of circulating estradiol and progesterone are believed to affect the activity of neurotransmitters associated with mood (serotonin, norepinephrine, dopamine, and gamma-aminobutyric acid), dysregulation of the hypothalamic-pituitary-adrenal axis, and activation of neuroinflammatory mechanisms (44). In addition to hormonal changes, genetic, epigenetic, and psychosocial factors are believed to play an important role in the development of menopausal mood symptoms (44).

Approximately 36% of perimenopausal and postmenopausal women worldwide report experiencing depressive symptoms (42). The most significant risk factor for depressive symptoms during the menopausal transition is a history of MDD (39, 45, 46). Other potential risk factors for menopause-related depression include a history of psychological disorders (eg, anxiety disorder, premenstrual dysphoric disorder, and postpartum depression) (39), the presence of VMS and menopausal sleep disturbance (22), midlife stressors (eg, caring for aging parents), health issues, and limited social support (22, 39, 45).

Based on limited evidence, there does not appear to be a universal risk of anxiety or anxiety disorders associated with menopause; however, some individuals may be more likely to experience symptoms (46). For example, VMS, depression, and sleep disturbance often co-occur among individuals undergoing the menopausal transition (22, 47). Findings from the SWAN longitudinal study and a prospective Australian study support that women without a history of anxiety may have an increased likelihood of experiencing anxiety symptoms during the menopausal transition (48, 49).

Health Inequities and Barriers to Care

Racial, cultural, and gender identity; sexuality; and socioeconomic factors affect how individuals experience menopause as well as the care they receive (50). Factors that greatly affect symptomatology, management, and sequelae include variations in awareness and attitudes about menopause, its associated symptoms, and treatment options; access to care across socioeconomic, racial, and ethnic groups; and the interplay of these factors (50). In the SWAN study, Black women experienced higher menopausal symptom burden (VMS, depression, sleep disruption) than White women but were less likely to receive treatment (51). A subsequent analysis of the factors underlying the differences in health outcomes between Black and White women in the SWAN cohort

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demonstrated that many of the disparities could be directly or indirectly attributed to social determinants of health, including systemic racism (51).

Barriers to menopausal care for transgender people, gender-diverse individuals, and sexual minority women (lesbian/gay, bisexual, and queer cisgender women) include a paucity of inclusive resources and culturally competent clinicians, as well as bias and discrimination during health care encounters (52, 53). There is also a lack of expert consensus on the definition of menopause for transgender and gender-diverse individuals because factors such as the use of gender-affirming hormone therapy and sex assigned at birth affect the timing and symptoms experienced as well as the individual perception of the physiologic changes (54).

Patient Counseling and Education

An increasing body of literature highlights the need for earlier and more comprehensive patient education about menopause to prepare and empower patients to recognize symptoms and seek treatment sooner to improve their quality of life (55, 56). Menopause therapy should be guided by patient preference, severity of symptoms, and the presence of comorbidities. During counseling, clinicians may also need to address patient concerns, including the stigma of aging, misinterpretation of results from previous clinical trials on MHT—specifically the overemphasis on MHT risks, and the commercialization of supplements targeting menopausal symptoms, which can deter patients from seeking care (57–60).

In the evaluation of patients seeking treatment for menopause-related symptoms, the continued need for contraception should be assessed until age 55 years (61–63). Patient-education materials on menopause and healthy aging also can help clinicians navigate these discussions with their patients (64).

CLINICAL RECOMMENDATIONS AND EVIDENCE SUMMARY

Hormone Therapy

Options for MHT include the systemic administration of estrogen alone, estrogen combined with a progestogen, or estrogen combined with the selective estrogen modulator bazedoxifene in a tissue-selective estrogen complex (Table 1). Estrogen is the active component of MHT and is used by itself in individuals without a uterus due to hysterectomy or complete müllerian agenesis. A progestogen or bazedoxifene is combined with estrogen to provide endometrial protection in individuals with a uterus. The available evidence does not support the use of progestogen monotherapy, dehydroepiandrosterone, or testosterone for the management of menopausal vasomotor, sleep, or mood symptoms because of their general lack of efficacy and associated adverse effects (65–67).

Systemic estrogen and progestogen are available in a variety of individual and combined formulations that can be administered orally or transdermally (Table 1). The progestogen component of MHT also can be delivered through the off-label use of a 52-mg levonorgestrel-releasing intrauterine device to provide endometrial protection as well as contraception (68, 69). The 52-mg levonorgestrel-releasing intrauterine device should be replaced after 5 years when used for MHT (68, 69). Although the etonogestrel implant is safe to use for contraception along with MHT, there is a lack of evidence to support its use to provide endometrial protection as part of MHT (61). Estrogen–progestogen therapy can be administered as a sequential regimen (ie, estrogen is taken daily and then a progestogen is added for part of the month or cycle, typically for 10–14 days) or in a continuous manner (ie, daily administration of estrogen and progestogen) (62).

There is insufficient evidence on the use of systemic MHT in transgender men (assigned female at birth) with a history of gender-affirming hormone therapy (53). Given the unclear benefits and risks, individualized shared decision making is advised in consultation with a clinician who has expertise in gender-affirming care.

Vasomotor Symptoms

ACOG recommends the use of systemic MHT with estrogen and a progestogen (in individuals with a uterus) or estrogen alone (if endometrial protection is not needed) for the management of VMS in individuals without contraindications to hormone use. **(STRONG RECOMMENDATION, MODERATE-QUALITY EVIDENCE)**

A meta-analysis of RCTs that included varying formulations, dosages, and routes of administration of estrogen therapy (with or without a progestogen) demonstrated a significant reduction in VMS frequency (relative risk [RR], 0.43; 95% CI, 0.33–0.57; N=1,104; nine trials) and severity (RR, 0.29; 95% CI, 0.17–0.50, N=503; seven trials) compared with placebo or no treatment (70). In a network meta-analysis of 32 RCTs (N=4,165) of various VMS treatments (acupuncture, transdermal estrogen plus progestogen, oral estrogen plus progestogen, tibolone, raloxifene, SSRIs or SNRIs, isoflavones, Chinese herbal medicine, black cohosh, and multibotanicals), transdermal estradiol combined with a progestogen was the most effective treatment for reducing the frequency of VMS (mean ratio, 0.23; 95% credible interval [CrI], 0.09–0.57) (71). Although oral estrogen–progestogen regimens demonstrated

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Table 1. Systemic Menopausal Hormone Therapy Options	
Treatment	**Dosage**
Estrogen (alone or combined with a progestogen)	
Oral estrogens	
17 β-estradiol	0.5–2.0 mg/d
Conjugated equine estradiol	0.3–1.25 mg/d
Transdermal estrogens	
17 β-estradiol patch	0.025–0.1 mg/d (1–2 patches/wk)
17 β-estradiol percutaneous gel	0.25–1.5 mg/d
17 β-estradiol transdermal spray	1.5 mg/d
17 β-estradiol vaginal ring	0.05–0.10 mg/d (90 d)
Progestogen (used with an estrogen)	
Oral progestogens	
Micronized progesterone	100–200 mg/d
Medroxyprogesterone acetate	2.5–10 mg/d
Norethindrone	0.35 mg/d
Norethindrone acetate	5 mg/d
Megestrol acetate	20–40 mg/d
Intrauterine progestogen delivery	
Levonorgestrel-releasing intrauterine device	52 mg*
Combination Hormone Therapies	
Oral	
Conjugated equine estradiol and medroxyprogesterone acetate	0.3–0.625 mg/1.5–5 mg/d
17β-estradiol and norethindrone acetate	0.5–1.0 mg/0.1–0.5 mg/d
17β-estradiol and micronized progesterone	0.5–1.0 mg/100 mg/d
17β-estradiol and drospirenone	0.5–1.0 mg/0.25–1.0 mg/d
17β-estradiol and norgestimate	1.0 mg/0.09 mg/d
Transdermal	
17β-estradiol and norethindrone acetate	50.0 micrograms/0.14–0.25 mg/patch
17β-estradiol and levonorgestrel	45.0 micrograms/0.015 mg/patch
Tissue-Selective Estrogen Complex	
Estrogen combined with a selective estrogen receptor modulator	Conjugated estrogen, 0.45 mg/d, and bazedoxifene, 20 mg/d

Table data from National Library of Medicine. DailyMed. Accessed July 30, 2026. https://dailymed.nlm.nih.gov/dailymed/.

*Data support the off-label use of a 52-mg levonorgestrel-releasing intrauterine device combined with estrogen to provide endometrial protection for up to 5 years (Voedisch AJ. Use of progestin-containing intrauterine systems in hormone therapy regimens: what are the data? Menopause 2026;33:86–7. doi: 10.1097/GME.0000000000002672).

benefit, they ranked lower than the transdermal estrogen–progestogen interventions (71).

ACOG suggests the combination of conjugated estrogen and bazedoxifene as an alternative to estrogen–progestogen therapy for the management of VMS in individuals without contraindications to hormone use. (CONDITIONAL RECOMMENDATION, LOW-TO-MODERATE–QUALITY EVIDENCE)

A tissue-selective estrogen complex of a conjugated estrogen and bazedoxifene is a U.S. Food and Drug Administration (FDA)–approved alternative to estrogen–progestogen therapy for the management of VMS in

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patients with a uterus. The estrogen component treats menopausal VMS, and bazedoxifene takes the place of progestogen and reduces estrogen's stimulation of the endometrium and breast tissue (72). Treatment with conjugated estrogen plus bazedoxifene is a good option for patients who prefer to avoid the potential vaginal bleeding and breast tenderness associated with progestogen use (73).

The efficacy of conjugated estrogen plus bazedoxifene for the treatment of VMS is supported by evidence from the SMART (Selective Estrogens, Menopause and Response to Therapy) trials (74, 75). In a pooled analysis of findings from SMART-1 and SMART-2, 12 weeks of conjugated estrogen plus bazedoxifene (0.45 mg/20 mg daily) was associated with a significant decrease in hot flush frequency and severity compared with placebo, regardless of time since menopause (less than 5 years or 5 years or more) (72). A subsequent post hoc analysis of SMART-2 findings showed that participants achieved a 50% reduction in hot flush frequency within 8–10 days of initiating conjugated estrogen plus bazedoxifene treatment (0.45 mg/20 mg daily) (76). Symptoms continued to improve with treatment, with a median time of 83 days to experience a consistent 90% decrease in hot flush frequency (76).

Sleep Disturbance

ACOG recommends the use of systemic MHT with estrogen and a progestogen (in individuals with a uterus) or estrogen alone (if endometrial protection is not needed) for the management of vasomotor-related sleep disturbance in individuals without contraindications to hormone use. **(STRONG RECOMMENDATION, MODERATE-QUALITY EVIDENCE)**

Menopausal hormone therapy is an option for the treatment of sleep symptoms in patients with concurrent VMS, but it is unclear whether MHT improves sleep outcomes in individuals without VMS. Most studies on systemic MHT for sleep symptoms have included women with VMS, which makes it difficult to determine whether MHT is independently associated with improvement in sleep outcomes. Evidence from RCTs demonstrates that systemic MHT with estrogen–progestogen therapy is associated with improvement in sleep disturbance (77–80). Although studies of estrogen-only therapy for sleep disturbance report mixed findings (77), estrogen-only therapy effectively treats VMS (70), which are a significant contributor to menopausal sleep disturbance (38, 39).

A systematic review and meta-analysis of 15 RCTs (N=27,715) that compared various systemic MHT regimens with placebo reported that MHT was associated with a statistically significant improvement in self-reported sleep quality assessed by questionnaire (standardized mean difference [SMD], −0.13; 95% CI, −0.18 to −0.08) but not in objective sleep parameters measured by polysomnography (ie, total sleep time, sleep latency, sleep efficiency, and number of arousals) (77). Subgroup analyses demonstrated that the magnitude of effect varied by route of administration and type of estrogen, with transdermal administration (SMD, −0.35; 95% CI, −0.64 to −0.06) and regimens with 17β-estradiol (SMD, −0.34; 95% CI, −0.51 to −0.17) associated with the greatest improvements in sleep quality (77). Treatment effect also varied based on the type of progestogen; only regimens with micronized progesterone or medroxyprogesterone acetate were associated with improvement (77). The pooled results for estrogen monotherapy did not reach statistical significance, although estrogen-only therapy was associated with benefit in the largest RCT included in the estrogen-only subgroup analysis (77). A possible explanation for the inconsistent findings for estrogen monotherapy is the heterogeneity of estrogen formulations and route of administration across trials (77). The progestogen component of combined MHT also may exert an important independent effect on sleep (77).

Two additional RCTs that were not included in the systematic review and meta-analysis reported that combined estrogen–progestogen formulations were associated with statistically significant improvements in sleep outcomes and VMS compared with placebo (78, 79). Another RCT (N=172) that controlled for VMS and depressive symptoms reported that participants who were randomized to receive 12 months of transdermal estradiol (0.1 mg/d) combined with progesterone (200 mg/d for 12 days every 3 months) experienced statistically significant reductions in minutes to fall asleep (mean difference [MD], −0.28; *P*=.02) and number of awakenings (MD, −0.11; *P*=.02) compared with those who received placebo (80).

ACOG suggests the combination of conjugated estrogen and bazedoxifene as an alternative to estrogen–progestogen therapy for the management of vasomotor-related sleep disturbance in individuals without contraindications to hormone use. **(CONDITIONAL RECOMMENDATION, LOW-TO-MODERATE–QUALITY EVIDENCE)**

Sleep disturbance was evaluated as a secondary outcome in the SMART-2 RCT (81). Among 318 postmenopausal women with seven or more moderate-to-severe hot flushes per day, 12 weeks of treatment with conjugated estrogen plus bazedoxifene (0.45 mg/20 mg daily) was associated with significant improvement in several

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domains of the Medical Outcomes Study sleep scale, including time to fall asleep, sleep disturbance, sleep adequacy, and overall sleep problems (indexes I and II), compared with placebo ($P<.001$) (81). Regression analysis showed that the observed improvements in sleep disturbance, sleep adequacy, and overall sleep problems were strongly correlated with a significant reduction in hot flush frequency ($P<.05$) (81).

In a substudy of the SMART-5 trial that included 445 postmenopausal women with bothersome moderate-to-severe VMS and sleep disturbance, conjugated estrogen plus bazedoxifene (0.45 mg/20 mg daily) was associated with reduction in time to fall asleep at 3 months and 12 months compared with placebo ($P<.05$), and statistically significant improvement in sleep disturbance was observed at 12 months ($P<.001$) (82).

Depressive Symptoms

The available evidence is insufficient to recommend for or against the use of estrogen–progestogen or estrogen-only MHT for the treatment of menopause-related depressive symptoms. Perimenopausal and postmenopausal patients who meet diagnostic criteria for MDD or for whom depression is the predominant symptom should be treated according to depression-management guidelines.
(GOOD PRACTICE POINT)

The available evidence suggests that, in perimenopausal individuals, systemic MHT with estrogen–progestogen or estrogen-only therapy may improve symptoms of depression and that MHT may enhance the effects of antidepressant treatment (83). However, studies demonstrate that MHT is not more effective than placebo for the treatment of depression in postmenopausal individuals (83). The high heterogeneity of study designs, assessment measures, and participant baseline depression status (asymptomatic, depressive symptoms, MDD diagnosis) limit confidence in the available evidence (83). Antidepressants (ie, SSRIs or SNRIs) and CBT remain first-line treatment options for perimenopausal and postmenopausal patients who meet diagnostic criteria for MDD or for whom depression is the predominant symptom (45, 84).

Risks

In general, the benefits of systemic MHT outweigh the risks for most patients with bothersome VMS and vasomotor-related sleep disturbance who are aged 50–59 years or within 10 years of menopause onset and have no contraindications to hormone therapy use (69, 84, 85). Decisions regarding MHT should be individualized, with consideration of factors that may modify risk, including age and timing of initiation; underlying risk factors and comorbidities; and hormone therapy formulation, route of administration, and duration of use (69, 84) (see "Treatment Eligibility and Initiation" later in this document for more information on contraindications and other treatment considerations).

Estrogen–Progestogen and Estrogen-Only Therapy. Most of the available data on the safety of estrogen–progestogen and estrogen-only MHT come from the Women's Health Initiative (WHI) trials, which were designed to investigate the effects of hormone therapy on the prevention of chronic disease among 27,347 postmenopausal women aged 50–79 years (86). A Cochrane meta-analysis of the results reported that, compared with placebo, the use of estrogen–progestogen therapy for a median of 5.6 years was associated with a small increased risk of stroke (RR, 1.39; 95% CI, 1.09–1.77; 5 additional cases/1,000 women), VTE (RR, 2.03; 95% CI, 1.55–2.64; 10 additional cases/1,000 women), and invasive breast cancer (RR, 1.27; 95% CI, 1.03–1.56; 5 additional cases/1,000 women) (87). Among participants who used estrogen-only therapy for a median of 7.2 years, there was not an increased risk of invasive breast cancer, but the risks of stroke (RR, 1.33; 95% CI, 1.06–1.67; 8 additional cases/1,000 women) and VTE (RR, 1.32; 95% CI, 1.00–1.74; 5 additional cases/1,000 women) were slightly elevated compared with placebo (87).

However, several important caveats need to be considered when interpreting the WHI data. Foremost among these, nearly 67% of the WHI participants were aged 60 years or older, which makes the findings difficult to generalize to younger, healthy perimenopausal or recently postmenopausal women, who have a lower absolute risk of chronic diseases such as stroke, coronary heart disease (CHD), and VTE and are the most likely to use and benefit from hormone therapy (69, 88). Subsequent subgroup analyses of WHI data and findings from other studies show that the cardiovascular risks associated with MHT vary by age and timing of initiation with respect to menopause, with a very low absolute risk for women aged 50–59 years (85, 86, 89). The WHI trials also evaluated the use of only one type of hormone therapy regimen, oral conjugated equine estrogen with or without medroxyprogesterone acetate (86), which observational data suggest may be associated with an increased risk of VTE compared with alternative regimens that include transdermal estradiol with or without micronized progesterone (90–92).

In 2025, the FDA recommended several significant updates to the labeling of MHT products to better reflect the available safety data and the nuanced considerations for prescribing and initiating MHT (93). Key updates included removing the external boxed warnings for

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cardiovascular disease, breast cancer, and probable dementia and adding guidance advising initiation of MHT in women younger than age 60 years or within 10 years of menopause onset to achieve the most favorable balance of benefits and risks (93).

Conjugated Estrogen and Bazedoxifene. Several RCTs with up to 2 years of follow-up have reported on the safety of conjugated estrogen plus bazedoxifene for the treatment of VMS (94, 95). Pooled analyses of the available data show that conjugated estrogen plus bazedoxifene (0.45 mg/20 mg daily) is not associated with an increased risk of breast cancer, endometrial cancer, VTE, stroke, or CHD compared with placebo (94, 95). However, overall confidence in the findings is limited by the low incidence of adverse events in the studies and the lack of long-term follow-up data beyond 2 years (94, 95).

Treatment Eligibility and Initiation

The decision to initiate MHT for the management of VMS or vasomotor-associated sleep disturbance should be based on a shared decision-making discussion about the patient's symptoms and the benefits and risks of therapy, with consideration of each patient's age; medical history; baseline risks of cardiovascular disease, breast cancer, and VTE; and individualized treatment goals and priorities.
(GOOD PRACTICE POINT)

Contraindications to MHT include undiagnosed abnormal genital bleeding; active or a history of breast cancer; active or a history of high-grade or advanced-stage endometrial cancer or uterine sarcoma; active or a history of low-grade serous ovarian cancer, endometrioid ovarian cancer, or granulosa cell tumors; CHD; active or a history of VTE; active or a history of arterial thromboembolic disease (eg, stroke, myocardial infarction); known thrombophilic disorders; hepatic impairment or disease; and known or suspected pregnancy (69, 96–100). With shared decision making and in consultation with a gynecologic oncologist, the use of MHT can be considered in patients with a history of early-stage (stage I–II) endometrial cancer or high-grade serous ovarian cancer (69, 99, 100).

Cardiovascular disease risk can be estimated with a validated risk calculator such as the one developed by the American College of Cardiology (101). Clinicians should carefully assess the patient's personal and family history of VTE before prescribing MHT and consider the use of transdermal forms of estrogen therapy (102). Breast cancer risk assessment can be conducted with one of the various validated assessment tools available online, including the National Cancer Institute's Breast Cancer Risk Assessment Tool and the International Breast Cancer Intervention Studies (or Tyrer–Cuzick) model (103).

When MHT is selected for the management of VMS and vasomotor-related sleep disturbance, ACOG recommends initiation of therapy before age 60 years or within 10 years of the start of natural menopause in individuals without contraindications to hormone use.
(STRONG RECOMMENDATION, LOW-TO-MODERATE–QUALITY EVIDENCE)

The available evidence (85, 86, 89) and expert guidelines (69, 73) support initiation of MHT before age 60 years or within 10 years of menopause onset to achieve the most favorable balance of benefits (ie, improvement of VMS and vasomotor-related sleep disruption) and risks. After age 60 years or more than 10 years after menopause onset, there is an age-related elevated baseline risk of VTE, stroke, and CHD; initiation of MHT at this time may further increase the risk of these adverse cardiovascular events, particularly VTE and stroke (69, 86, 89). The decision to initiate systemic MHT after age 60 years or more than 10 years after menopause onset should be individualized based on a shared decision-making discussion that considers the patient's symptoms; treatment goals; and the risk of VTE, stroke, and CHD (69, 73).

A Cochrane review that included 19 trials with 40,410 postmenopausal participants reported that those who initiated MHT before age 60 years or within 10 years of menopause onset had a lower risk of CHD (composite of death from cardiovascular causes and nonfatal myocardial infarction) (RR, 0.52; 95% CI, 0.29–0.96; N=8,311; four trials), a similar risk of stroke, but an increased risk of VTE (RR, 1.74; 95% CI, 1.11–2.73; N=9,838; three trials) compared with those who received placebo or no treatment (89). In contrast, those who initiated MHT more than 10 years after menopause onset had a greater increased risk of VTE (RR, 1.96; 95% CI, 1.37–2.80; N=27,475; nine trials), as well as an elevated risk of stroke (RR, 1.21; 95% CI, 1.06–1.38; N=22,722; eight trials), although there was no difference in the risk of CHD (89).

These findings are consistent with a more recent secondary analysis of WHI trial data that reported on the effects of MHT on cardiovascular risk among participants with VMS (85). For women aged 50–69 years with VMS, the use of MHT (estrogen–progestogen or estrogen-only therapy) was not associated with a statistically significant increased risk of atherosclerotic cardiovascular disease (a composite outcome of nonfatal myocardial infarction, hospitalization for angina, coronary revascularization, ischemic stroke, peripheral arterial disease, carotid artery disease, or cardiovascular disease death) compared with placebo (85). However, there was a significantly increased

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risk of atherosclerotic cardiovascular disease among women aged 70 years or older with VMS who used conjugated equine estrogen alone (hazard ratio, 1.95; 95% CI, 1.06–3.59; 217 excess events/10,000 person-years) or conjugated equine estrogen plus medroxyprogesterone acetate (hazard ratio, 3.22; 95% CI, 1.36–7.63; 382 excess events/10,000 person-years) (85).

Treatment Monitoring

The efficacy and tolerability of MHT should be assessed 3 months after initiation and subsequently reviewed every 12 months for the duration of treatment, unless earlier evaluation is clinically indicated (84).

Persistent or unscheduled bleeding after initiation of MHT warrants evaluation for endometrial intraepithelial neoplasia/atypical endometrial hyperplasia and endometrial cancer. (GOOD PRACTICE POINT)

Unscheduled vaginal bleeding is common within the first 3 months of MHT use. However, evaluation is warranted for endometrial intraepithelial neoplasia/atypical endometrial hyperplasia and endometrial cancer if bleeding occurs or continues after the first 6 months of MHT use (69). For more information, see ACOG's guidance on the evaluation of postmenopausal bleeding (104).

Discontinuation

The decision about when and how to discontinue MHT should be individualized based on ongoing risk factor assessment and a shared decision-making discussion of the benefits and risks. (GOOD PRACTICE POINT)

Continuation of MHT past age 60 years may be necessary to manage persistent symptoms that affect patients' quality of life (69). The decision to continue treatment should be revisited routinely and based on an ongoing assessment of benefits and risks to determine continued eligibility (see "Risks" and "Treatment Eligibility and Initiation" earlier in this document). Menopausal hormone therapy can be discontinued abruptly or gradually with tapering (73). Before cessation of MHT, patients should be counseled about the potential for recurrence of symptoms (73, 84, 105).

Nonhormonal Pharmacologic Management

Despite evidence to support the efficacy and overall favorable safety profile of MHT in healthy, newly menopausal individuals with bothersome symptoms (ie, VMS, vasomotor-related sleep disturbance), some patients may prefer not to take MHT and others may have contraindications to its use (see "Treatment Eligibility and Initiation" earlier in this document) (106). In such cases, a variety of nonhormonal pharmacotherapies are available (Table 2).

Given the limited evidence of benefit and potential for adverse effects, ACOG and other expert guidelines do not recommend clonidine as a primary treatment option for the management of VMS (62, 84, 106). Oxybutynin is an anticholinergic that has demonstrated effectiveness against moderate-to-severe VMS in two RCTs (107, 108). Although oxybutynin is included as a treatment option in some expert guidelines (62, 106), notable adverse effects such as cognitive decline in older women (older than age 65 years), dry mouth, and urinary retention (107–109) limit its use in the management of VMS.

Selective Serotonin Reuptake Inhibitors and Serotonin and Norepinephrine Reuptake Inhibitors

ACOG recommends SSRIs or SNRIs for the management of menopausal symptoms (vasomotor and depression) in patients who do not desire or have a contraindication to hormone therapy. (STRONG RECOMMENDATION, MODERATE-TO-HIGH –QUALITY EVIDENCE)

Two classes of antidepressants, SSRIs and SNRIs, have been studied for the management of VMS (Table 2) (110–113). Although the SSRI paroxetine (7.5 mg/d) is the only antidepressant currently FDA-approved for the treatment of VMS (114), studies of other SSRIs and SNRIs (ie, citalopram, escitalopram, fluoxetine, venlafaxine, and desvenlafaxine) have demonstrated a reduction in both VMS frequency and severity compared with placebo (111–113). Additionally, SSRIs and SNRIs are associated with small-to-moderate improvement in menopause-associated sleep disturbance and depression (115, 116). Commonly reported adverse effects of SSRIs and SNRIs are generally mild and include nausea, headache, dizziness, fatigue, dry mouth, and somnolence (110, 111, 116). Although adverse effects have been the principal cause of treatment discontinuation in some clinical trials (113, 116), reported dropout rates are not significantly higher for antidepressants compared with placebo (115, 116).

National and international menopause treatment guidelines recommend serotonergic antidepressants as a nonhormonal option for the management of VMS (62, 73, 106) and menopause-associated depression given their potential benefits and favorable safety profile (45, 117). However, the use of fluoxetine or paroxetine generally should be avoided in patients who are receiving tamoxifen therapy, because these agents are potent

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Table 2. Evidence-Based Nonhormonal Pharmacologic Treatment Options for Menopausal Symptoms

Treatment Class	Medication (Dosage)	Evidence of Benefit	Comments
SSRI	Paroxetine (7.5 mg/d)	VMS, depression	FDA-approved for the management of VMS; concomitant use with tamoxifen is not recommended.*
	Paroxetine (10–40 mg/d)[†] Escitalopram (10–20 mg/d)[†]	VMS, depression	Off-label option for the management of VMS; FDA-approved for the treatment of depression and anxiety; Concomitant use of paroxetine and tamoxifen is not recommended.*
	Citalopram (10–40 mg/d)[†] Fluoxetine (10–80 mg/d)[†]	VMS, depression	Off-label option for the management of VMS; FDA-approved for the treatment of depression.
SNRI	Venlafaxine (37.5–150 mg/d)[†] Desvenlafaxine (50–100 mg/d)[†]	VMS, depression	Off-label option for the management of VMS; FDA-approved for the treatment of depression and anxiety.
Neurokinin receptor antagonist	Fezolinetant (45 mg/d)	VMS	FDA-approved for the treatment of VMS; drug label includes a boxed warning about required liver function testing before and during the first 9 mo of use.[‡]
	Elinzanetant (120 mg/d)	VMS, sleep	FDA-approved for the treatment of VMS; drug label recommends liver function testing before treatment and at 3 mo after initiation.[§]
Gabapentinoid	Gabapentin (300–900 mg/d in divided doses)[‖]	VMS	Off-label option for the management of VMS.

Abbreviations: SSRI, selective serotonin reuptake inhibitor; VMS, vasomotor symptoms; FDA, U.S. Food and Drug Administration; SNRI, serotonin and norepinephrine reuptake inhibitor.

Table data from National Library of Medicine. DailyMed. Accessed July 30, 2026. https://dailymed.nlm.nih.gov/dailymed/.

*Paroxetine is a strong inhibitor of cytochrome P450 2D6 and may interfere with the conversion of tamoxifen to its active metabolite, endoxifen (Padagis US LLC. Paroxetine capsule. In: DailyMed. National Library of Medicine; 2023. Accessed July 30, 2026. https://dailymed.nlm.nih.gov/dailymed/drugInfo.cfm?setid=1383d713-79b9-45bd-bd06-65707f28bc99).

†Dosage range listed is for the treatment of depression. Although a recommended dosage range has not been established for the off-label use of SSRIs and SNRIs to treat vasomotor symptoms, studies generally have used dosages toward the lower end of the recommended treatment range for depression. See the Clinical Practice Guideline discussion for more information.

‡Before starting fezolinetant, baseline liver function testing should be performed and repeated monthly for the first 3 months and then again at months 6 and 9. See drug label for more information (Astellas Pharma US, Inc. Veozah- fezolinetant tablet, film coated. In: DailyMed. Accessed July 30, 2026. https://dailymed.nlm.nih.gov/dailymed/drugInfo.cfm?setid=cae9f798-24f9-4580-a4fc-e6c710cbda3c).

§See drug label for more information. Bayer HealthCare Pharmaceuticals Inc. Lynkuet- elinzanetant capsule. In: DailyMed. Accessed July 30, 2026. https://dailymed.nlm.nih.gov/dailymed/drugInfo.cfm?setid=f42884ff-7dff-419c-8a0c-affe2ed73818.

‖Starting dosage of 100–300 mg/d of gabapentin is recommended, with incremental dosage increase, as needed, to a maximum of 900 mg/d. Consider advising patients to take gabapentin at nighttime to avoid daytime drowsiness or to divide doses between daytime and nighttime (eg, 300 mg during the day and 600 mg at night) (Panay N, Fenton A, Hamoda H, Hillard T, Islam R, Pedder H, et al. International Menopause Society (IMS) recommendations and key messages on women's midlife health and menopause. IMS Recommendations Writing Group [published erratum appears in Climacteric 2026;29:318–20]. Climacteric 2025;28:634–56. doi: 10.1080/13697137.2025.2585487).

inhibitors of cytochrome P450 2D6 and may interfere with the conversion of tamoxifen to its active metabolite, endoxifen (106, 114, 117). Evidence does not support the superiority of any particular serotonergic antidepressant (Table 2). Thus, the choice of medication should be guided by patient preference and factors such as prior

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response to antidepressant treatment (if applicable), presence of concurrent symptoms, adverse effects, and medication interactions (eg, tamoxifen) (45, 117).

Vasomotor Symptoms. In a meta-analysis of three RCTs (N=1,198) that compared paroxetine (7.5–20 mg/d) with placebo, participants in the treatment group reported significant reductions in VMS frequency (MD, −7.36 episodes/week; 95% CI, −10.46 to −4.25) and severity (MD, −2.40; 95% CI, −4.20 to −0.60) at 12 weeks (110). Another meta-analysis of 11 RCTs (N=2,069) compared various SSRIs (paroxetine, escitalopram, citalopram, sertraline, and fluoxetine) with placebo and found that 4–8 weeks of treatment with an SSRI was associated with a small but statistically significant reduction in VMS frequency (MD, −0.93; 95% CI, −1.46 to −0.37; N=1,746; nine RCTs) and intensity (SMD, −0.34; 95% CI, −0.59 to −0.10; N=538; five RCTs) (111). The meta-analysis authors also performed a mixed-treatment comparative analysis, which showed that escitalopram was the most effective SSRI for the management of VMS; however, direct comparative trials are needed to confirm these findings (111).

The SNRIs venlafaxine and desvenlafaxine also are associated with statistically significant improvement in VMS compared with placebo (112). In a systematic review of seven RCTs, five of the trials demonstrated that treatment with venlafaxine or desvenlafaxine was associated with a 48–67% decreased frequency of VMS by weeks 8–12 compared with a 25–51% reduction observed with placebo (112). At 12 weeks, SNRI treatment also was associated with a statistically significant greater reduction in VMS severity compared with placebo (24–31% vs 12–18%, respectively) in five of the seven RCTs (112). Although there have been few head-to-head trials of SSRI and SNRI antidepressants and hormonal interventions for the treatment of VMS, the results of an indirect comparative analysis of pooled findings from three RCTs (N=899) showed that escitalopram (10–20 mg/d), venlafaxine (75 mg/d), and unopposed 17β-estradiol (0.5 mg/d) were associated with similar modest improvements in VMS frequency at 8 weeks compared with placebo (113). Low-dose estradiol had the largest effect, with 2.4 fewer VMS per day (95% CI, −3.4 to −1.3), followed by venlafaxine (−1.8 VMS/d; 95% CI, −2.8 to −0.8) and escitalopram (−1.4 VMS/d; 95% CI, −2.7 to −0.2) (113).

Depression. A meta-analysis of seven RCTs (N=1,676) reported a moderate improvement in depression severity among perimenopausal and postmenopausal women with or without diagnosed MDD who were treated with an SSRI or SNRI compared with those who received placebo (Hedges *g*, 0.44; 95% CI, 0.32–0.57) (116). Treatment duration ranged from 6 to 36 weeks, and a variety of antidepressants were investigated: desvenlafaxine, 50–200 mg/d (three trials); citalopram, 20–30 mg/d (two trials); fluoxetine, 20 mg/d (two trials); paroxetine, 25 mg/d (one trial); and venlafaxine, 75 mg/d (one trial) (116). Subgroup analyses demonstrated a significant improvement in depression regardless of whether an SSRI (Hedges *g*, 0.46; 95% CI, 0.20–0.73) or SNRI (Hedges *g*, 0.43; 95% CI, 0.28–0.58) was administered. However, only SNRIs were associated with a significantly greater likelihood of discontinuation due to adverse effects compared with placebo (odds ratio 0.53; 95% CI, 0.31–0.89; three RCTs) (116).

Sleep Disturbance. Serotonergic antidepressants may be associated with little to no benefit for menopause-related sleep disruption (115). In a meta-analysis of seven RCTs (N=1,949) that compared various SSRI and SNRI antidepressants (paroxetine, citalopram, escitalopram, sertraline, fluoxetine, and venlafaxine) with placebo, participants who received the intervention showed small but statistically significant improvement in sleep quality (Hedges *g*, 0.24; 95% CI, 0.11–0.38) (115). However, the authors noted that only three of the seven included trials reported statistically significant effects (115).

Neurokinin Receptor Antagonists

ACOG recommends neurokinin receptor antagonists for the management of VMS and vasomotor-related sleep disturbance in patients who do not desire or have a contraindication to hormone therapy. **(STRONG RECOMMENDATION, MODERATE-TO-HIGH–QUALITY EVIDENCE)**

The use of neurokinin receptor antagonists represents a new therapeutic approach to the treatment of menopausal VMS that was prompted by advances in our understanding of the pathophysiology of VMS (35). Neurokinin receptor antagonists inhibit neurokinin B from binding to KNDy neurons, which disrupts activation of the hypothalamic thermoregulatory center (35, 36). Two neurokinin receptor antagonists are FDA-approved for the management of VMS: fezolinetant, which targets neurokinin-3 receptors (118), and elinzanetant, which inhibits neurokinin-1 and neurokinin-3 receptor activity (119).

Fezolinetant. In a 2025 systematic review and meta-analysis of six RCTs of women with moderate-to-severe VMS, 12 weeks of treatment with fezolinetant (30–45 mg once daily or 30–90 mg twice daily) was associated with small-to-moderate reductions in VMS frequency (Cohen's *d*, −0.54; 95% CI, −0.74 to −0.33; N=1,278; three trials) and severity (Cohen's *d*, −0.34; 95% CI, −0.48 to −0.21; N=1,241; four trials) compared with placebo (120). Fezolinetant demonstrated similar improvements in VMS frequency and severity in a 2024 meta-analysis, which also

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reported a modest reduction in sleep disturbance (SMD, −0.18; 95% CI, −0.29 to −0.06; N=865; two RCTs) and in symptom burden ratings on the Menopause-Specific Quality of Life questionnaire (SMD, −0.37; 95% CI, −0.48 to −0.25; N=992; three RCTs) at 12 weeks (121). In a pooled analysis of findings from the SKYLIGHT 1 and 2 trials, the benefits of fezolinetant were reported as early as day 1 of treatment, and reductions in VMS frequency and severity were maintained for 52 weeks (122).

Fezolinetant also has demonstrated efficacy in indirect comparisons with hormonal therapy and other nonhormonal agents (123). In a network meta-analysis that included data from 25 RCTs, fezolinetant (45 mg) was associated with similar reductions in VMS frequency at 12 weeks compared with hormone therapy (123). Additionally, fezolinetant decreased the frequency of moderate-to-severe VMS significantly more than nonhormonal treatments: paroxetine, 7.5 mg (MD, 1.66; 95% CrI, 0.63–2.71); desvenlafaxine, 50–200 mg (MDs ranged from 1.12 [95% CrI, 0.10–2.13] to 2.16 [95% CrI, 0.90–3.40]); and gabapentin, 1,800 mg (MD, 1.63; 95% CrI, 0.48–2.81). Fezolinetant also was associated with a greater reduction in VMS severity compared with desvenlafaxine, 50 mg (MD, 0.28; 95% CrI, 0.07–0.48) but demonstrated comparable efficacy to higher-dose desvenlafaxine (100–200 mg) and gabapentin (1,800 mg) (123).

Premarketing and postmarketing surveillance have identified transient hepatic transaminase elevation in a small number of women taking fezolinetant. In the SKYLIGHT 4 trial, liver enzyme elevations more than three times the upper limit of normal occurred in 12 of 589 participants in the fezolinetant (45 mg) group, compared with 6 of 583 participants who received placebo (124). These findings and additional postmarketing surveillance data prompted the addition of an FDA boxed warning and revision of the package insert in 2024 to include guidance on liver function testing before and during treatment (118, 125).

Elinzanetant. The dual neurokinin-1 and neurokinin-3 receptor antagonist elinzanetant is another FDA-approved option for the management of VMS and vasomotor-related sleep disturbance (NLM elinzanetant product label). The efficacy of elinzanetant is supported by the results of several RCTs (126, 127). In OASIS 1 (Overall Assessment of Efficacy and Safety of Elinzanetant in Patients With Vasomotor Symptoms), a placebo-controlled, randomized trial (N=396), 12 weeks of treatment with elinzanetant was associated with significant reductions in VMS frequency (least squares mean difference [LSMD], −3.2; 95% CI, −4.8 to −1.6) and severity (LSMD, −0.4; 95% CI, −0.5 to −0.3) (126). In addition, participants in the treatment group reported significantly decreased sleep disturbance (LSMD, −5.6; 95% CI, −7.2 to −4.0) and a decrease in symptom bother ratings (LSMD, −0.4; 95% CI, −0.6 to −0.2) compared with those in the placebo arm (126). These findings are consistent with the results of a second RCT, OASIS 2 (N=400), which included a similar population and protocol (126). Although both OASIS 1 and OASIS 2 reported sustained benefit for up to 26 weeks, the trials were placebo-controlled only for the first 12 weeks (126).

Elinzanetant also has demonstrated comparable or greater efficacy in indirect comparisons with other nonhormonal agents (128). In a network meta-analysis of 17 RCTs, elinzanetant (120 mg) reduced VMS frequency more effectively at 12 weeks compared with paroxetine, 7.5 mg (MD, −2.11; 95% CI, −3.31 to −0.92); desvenlafaxine, 50–200 mg (MD ranged from −2.77 to −1.72); and gabapentin, 1,200–1,800 mg (MD ranged from −2.22 to −2.31) (128). Elinzanetant was comparable with desvenlafaxine (100–200 mg) and gabapentin for reducing VMS severity (128). In comparisons of elinzanetant and fezolinetant, both medications demonstrated similar efficacy in reducing the frequency and severity of VMS, but elinzanetant was more effective at decreasing Patient-Reported Outcomes Measurement Information System sleep disturbance scores (MD, −2.67; 95% CI, −3.92 to −1.42) (128).

Based on findings from the OASIS trials, the most common treatment-associated adverse effects reported in the elinzanetant group compared with the placebo arm, respectively, included headache (7.0–9.0% vs 2.5–2.6%) and fatigue (5.5–7.0% vs 1.5%) (126). A third RCT (OASIS 3, N=628) that investigated the safety of elinzanetant for up to 52 weeks reported that the most common adverse effects were headache, fatigue, and somnolence (127). Although there were no cases of drug-related hepatoxicity reported in phase III clinical trials of elinzanetant (126, 127), the drug label recommends liver function testing before treatment and at 3 months after initiation because 0.6% of participants treated with elinzanetant experienced liver enzyme level elevation compared with 0.4% who received placebo (119).

Gabapentin

ACOG suggests gabapentin for the management of VMS when other recommended nonhormonal pharmacologic interventions are ineffective or poorly tolerated by patients who do not desire or have a contraindication to hormone therapy. **(CONDITIONAL RECOMMENDATION, MODERATE-QUALITY EVIDENCE)**

Gabapentin, a gamma-aminobutyric acid analogue, is indicated for the treatment of postherpetic neuralgia and the adjunctive treatment of partial-onset seizures but is also used off-label to treat VMS (Table 2) (129). It is postulated that gabapentin helps improve VMS by decreasing adrenergic neurotransmitter activity and widening the thermoregulatory zone in the hypothalamus

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(130). Administration at bedtime is advised because of gabapentin's sedative effects, which may provide a secondary benefit to patients who are experiencing sleep disturbance in addition to VMS (106).

In a meta-analysis of low-to-moderate–quality RCTs that compared 12 weeks of gabapentin (900–1,800 mg/d) with placebo (or vitamin E in one study), participants who received gabapentin experienced decreases in the frequency of VMS (MD, −2.77; 95% CI, −4.29 to −1.24; N=1,913; seven RCTs) (130). The pooled results of trials that reported on severity as a separate outcome did not demonstrate a statistically significant difference between gabapentin and placebo (SMD, −1.43; 95% CI, −2.88 to 0.02; N=1,749; four RCTs) (130). However, gabapentin demonstrated a small benefit for the combined outcome of severity and frequency (SMD, 0.77; 95%, CI, −1.15 to −0.40; N=119; two RCTs) (130). Another meta-analysis that included many of the same RCTs reported a similar decrease in VMS frequency (131). The most common adverse events were dizziness, somnolence, and headache (130, 131).

Nonpharmacologic Interventions

Evidence-based nonpharmacologic interventions for menopausal vasomotor, sleep, and mood symptoms include CBT, MBIs, exercise, clinical hypnosis, and acupuncture. These approaches can be used alone but generally are recommended as adjunctive treatments to be used in combination with hormonal or nonhormonal pharmacotherapy.

Cognitive Behavioral Therapy

ACOG suggests CBT for the management of VMS and menopause-related sleep disturbance and mood symptoms. (CONDITIONAL RECOMMENDATION, LOW-TO-MODERATE–QUALITY EVIDENCE)

Cognitive behavioral therapy is a structured, time-limited approach that helps individuals change negative thought patterns that can adversely influence emotions and behaviors (132). The principal goals of CBT for menopause are to provide patients with cognitive reframing techniques and self-help coping strategies to decrease the distress and bother associated with VMS and improve related mood and sleep symptoms (45, 132, 133). Meta-analyses of data from low-to-moderate–quality RCTs demonstrate that CBT is associated with small-to-moderate improvements in vasomotor, sleep, and mood symptoms related to menopause, although the magnitude of benefit may wane over time (133–137). Cognitive behavioral therapy also is recommended as a treatment option in national and international guidelines on the management of menopausal symptoms (45, 62, 73, 84, 106, 117).

Access to CBT may be limited by several factors, including the cost of treatment, the commitment required to participate in therapy, and the limited number of mental health professionals with the required specialized training (138). Online-based and telephone-based CBT counseling interventions may help overcome some of these treatment barriers (138, 139). Patient–clinician shared decision making should include discussion of these considerations. Clinicians who plan to recommend CBT interventions to patients should consider having a current list of qualified mental health professionals for referral.

Vasomotor Symptoms. In a systematic review and meta-analysis of 14 RCTs (N=1,618), 6–12 weeks of CBT was associated with small-to-moderate improvement in hot flush frequency (Hedges *g*, 0.36; 95% CI, 0.21–0.51) and in symptom bother rating (Hedges *g*, 0.49; 95% CI, 0.34–0.64) (134). Included studies used a wait-list control group or compared CBT with usual care or menopause education (134).

Sleep Disturbance. Findings from two meta-analyses and one network meta-analysis support the benefits of CBT for menopause-related sleep disturbance (133, 136, 140). Cognitive behavioral therapy was associated with a moderate reduction in sleep disruption in a meta-analysis of four RCTs (N=581) (SMD, −0.40; 95% CI, −0.70 to −0.11) (136). The CBT interventions were delivered in person, on the telephone, or online over the course of 12–24 weeks, and sleep improvement was measured by validated questionnaires or a combination of questionnaire and polysomnography (one study) (136). These findings are consistent with the results of a more recent meta-analysis (SMD, −0.78; 95% CI, −1.32 to −0.25; N=204; two RCTs) (133). Similarly, in a network meta-analysis that included a total of 44 RCTs (N=3,512) of nonpharmacologic interventions for menopause-related insomnia, CBT ranked second only to mindfulness for sleep-quality improvement as measured by a reduction in Pittsburgh Sleep Quality Index (PSQI) scores (MD, −4.2; 95% CrI, −5.95 to −2.47; N=261; six RCTs) (140).

Mood Symptoms. Cognitive behavioral therapy also may improve menopause-related depression and anxiety symptoms. A meta-analysis of six RCTs (N=771) reported that participants who received 4–12 weeks of CBT reported a small-to-moderate reduction in menopause-related depressive symptoms compared with those in the control group (wait list or no intervention) (SMD, −0.33; 95% CI, −0.45 to −0.21) (137). These results are consistent with another, smaller meta-analysis of two RCTs (N=124), which demonstrated that 4–6 weeks of CBT was associated with a large reduction

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in depressive symptoms (SMD, −3.55; 95% CI, −5.48 to −1.61) (133). A meta-analysis of five RCTs (N=691) reported that CBT was associated with a small but statistically significant reduction in anxiety symptoms (SMD, −0.22; 95% CI, −0.35 to −0.10) (137).

Mindfulness-Based Interventions

ACOG suggests MBIs to improve menopause-related sleep disturbance and mood symptoms. (CONDITIONAL RECOMMENDATION, LOW-TO-MODERATE–QUALITY EVIDENCE)

Mindfulness-based interventions, such as meditation, body scanning, breathing exercises, and mind–body exercises (eg, yoga), train individuals to focus their thoughts and attention on the present moment with nonjudgmental awareness, which may help decrease the emotional distress and bother associated with menopausal symptoms (141–143). The benefits of MBIs to manage menopausal-related sleep disruption and mood symptoms (depression, stress, and anxiety) are supported by evidence from low-to-moderate–quality RCTs, and MBIs are included as a treatment option in guidelines from the International Menopause Society (62). Limitations of the evidence base include high heterogeneity across studies (eg, in trial design, intervention delivery, and outcome measures), issues with participant blinding, and a lack of long-term outcome data (141, 144). However, given the potential benefits and negligible risks, the use of MBIs can be considered, particularly as an adjunct to pharmacologic treatment as part of a holistic approach to the management of menopausal symptoms (for additional discussion on the use of yoga to manage menopausal symptoms, please see the "Exercise" section later in this guideline).

Sleep Disturbance. In a meta-analysis of 13 RCTs (N=1,146), perimenopausal and postmenopausal women who participated in MBIs (mindfulness-based stress reduction, yoga, Pilates, music therapy, and Qigong) experienced statistically significant reductions in sleep disturbance compared with those who received usual care, no intervention, or sleep hygiene education (SMD, −0.91; 95% CI, −1.28 to −0.54) (144). Interventions were delivered in two to eight weekly sessions for 4–24 weeks, and sleep outcomes were measured by validated questionnaires such as the PSQI. Subgroup analyses showed that yoga, mindfulness-based stress reduction, and music therapy were associated with the largest reductions in sleep disturbance (144). Similarly, another systematic review and meta-analysis reported that MBIs were associated with statistically significant beneficial effects on sleep quality (SMD, −0.92; 95% CI, −1.65 to −0.20; N=405; five trials) compared with the control condition (usual care, waiting list, or no intervention) (141). The MBI duration was 8 weeks in most studies, and several types of MBIs were administered (mindfulness stress reduction, mindfulness-based cognitive therapy, and acceptance and commitment therapy) (141).

Mood Symptoms. Several meta-analyses of RCT findings support that MBIs can help improve menopause-related symptoms of depression, anxiety, and stress (137, 141, 143, 144). A systematic review and meta-analysis of 19 RCTs (N=1,670) reported that MBIs were associated with statistically significant reductions in depressive symptoms (SMD, −0.91; 95% CI, −1.30 to −0.53; N=845; 10 RCTs), anxiety (SMD, −1.03; 95% CI, −1.42 to −0.65; N=1,230; 13 RCTs), and stress (SMD, −0.85; 95% CI, −1.55 to −0.15; N=434; four trials) compared with the control condition (usual care, waiting list, or no intervention) (141). Several other meta-analyses of studies on MBIs have reported similar benefits for menopause-related depression, anxiety (137, 144), and stress (143).

Exercise

ACOG suggests physical exercise to improve menopause-related sleep disturbance and mood symptoms. (CONDITIONAL RECOMMENDATION, LOW-TO-MODERATE–QUALITY EVIDENCE)

The results of several systematic reviews and meta-analyses support that physical exercise (aerobic exercise, mind–body techniques such as yoga, resistance training, or a combination of approaches) is associated with improvement in menopause-related sleep disturbance, depression, and anxiety (145–150). Confidence in the findings is limited by the high heterogeneity in types of interventions, regimens, and outcome measures across studies. However, given the overall health benefits associated with exercise, it can be recommended as an adjunct to pharmacologic or nonpharmacologic treatment as part of a holistic approach to the management of menopause-related sleep and mood symptoms (for more information on the use of yoga to manage menopausal symptoms, please see the "Mindfulness-Based Interventions" section earlier in this guideline).

Sleep Disturbance. Findings from several meta-analyses of low-to-moderate–quality RCTs support the benefits of various types of exercise (ie, aerobic exercise, walking, yoga, Pilates, resistance training, flexibility) for menopause-related sleep disruption (145–148). The largest analysis (25 RCTs, N=2,757) showed that exercise was associated with a significant reduction in sleep

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disruption scores on the PSQI or Insomnia Severity Index compared with no intervention or usual activity (SMD, −1.60; 95% CI, −2.03 to −1.18) (145). Another meta-analysis showed a dose–response relationship between exercise session duration and improvement in sleep disruption, with the largest effects reported among those who exercised for 70–90 minutes per session (SMD, −0.64; 95% CI, −1.08 to −0.21), followed by 60 minutes per session (SMD, −0.52; 95% CI, −0.73 to −0.32) and 30–50 minutes per session (SMD, −0.27; 95% CI, −0.41 to −0.14) (146). Although one meta-analysis of nine RCTs (N=1,579) reported that exercise was not associated with a statistically significant improvement in sleep quality or insomnia, the included studies specifically enrolled women with VMS, which suggests that exercise may be less effective for VMS-related sleep disturbance (151).

Mood Symptoms. Two meta-analyses of low-to-moderate–quality RCTs support the efficacy of various types of physical activity, including aerobic exercise and yoga, to relieve menopause-associated depression and anxiety (149, 150). The first meta-analysis, which included 21 RCTs that enrolled perimenopausal and postmenopausal women (N=2,020), reported that low-to-moderate–intensity exercise (aerobic, walking, yoga, Pilates, tai-chi) was associated with significant improvement in validated symptom questionnaire scores measuring depression (SMD, −0.66; 95% CI, −0.99 to −0.33; N=1,990; 20 RCTs) and anxiety (SMD, −0.55; 95% CI, −0.82 to −0.27; N=1,411; 12 RCTs) compared with no intervention or a wait-list control condition (149). A second meta-analysis of RCTs that compared yoga interventions (varied formats, 60–90-minute sessions 1–3 times/wk for 8–24 weeks) with a usual-care or wait-list control condition showed that the mind–body discipline was associated with a significant reduction in validated symptom scores of depression (SMD, −1.55; 95% CI, −2.36 to −0.74; N=824; 10 RCTs) and anxiety (SMD, −0.96; 95% CI, −1.82 to −0.09; N=648; eight RCTs) among perimenopausal and postmenopausal women (150).

Clinical Hypnosis

ACOG suggests clinical hypnosis to reduce VMS and improve menopause-related sleep disturbance and anxiety symptoms. **(CONDITIONAL RECOMMENDATION, LOW-QUALITY EVIDENCE)**

Clinical hypnosis is a mind–body therapy that combines focused attention with mental imagery and therapeutic suggestion during a relaxed or altered state of consciousness, ultimately leading to modified thoughts, feelings, and behaviors aimed at achieving a particular health goal (152). Findings from several RCTs suggest that clinical hypnosis is associated with improvement in VMS (153–155), and The Menopause Society and the International Menopause Society recommend clinical hypnosis as a treatment option for VMS (62, 106). Limited evidence suggests that clinical hypnosis also may help improve menopause-related sleep disturbance (152, 153) and anxiety symptoms (156).

Although these findings are favorable, confidence in the evidence is limited by the small number of studies and methodologic concerns, such as unclear blinding of participants, missing outcome data, and a lack of long-term efficacy data. Another consideration is that access to clinical hypnosis may be limited by the cost of treatment, lack of insurance coverage, limited availability of hypnotherapists, and the need for in-person appointments (155, 157), although the use of smartphone applications and self-hypnosis are under investigation and have the potential to increase access to this form of treatment (155, 157). Despite these limitations, given the potential benefits and negligible risks, it is reasonable to consider the use of clinical hypnosis in combination with pharmacologic or other nonpharmacologic treatments as part of a multimodal holistic approach to VMS and menopausal sleep disturbance and anxiety symptoms.

Vasomotor Symptoms. Findings from several RCTs support that clinical hypnosis is associated with improvements in the frequency and severity of VMS (153–155). One RCT (N=187) reported that, compared with a therapist-led sham intervention, clinical hypnosis (five weekly 45-minute sessions) was associated with greater, statistically significant mean reductions in hot flush frequency (74.16% vs 17.13%), severity (80.32% vs 15.38%), and physiologically monitored hot flushes (56.86% vs 9.94%) at 12-week follow-up (153). In an 8-week randomized pilot study (N=71), therapist-delivered clinical hypnosis and venlafaxine were associated with similar reductions in hot flush scores (composite measurement of frequency and severity) compared with placebo (154). A more recent RCT (N=250) that compared a self-administered hypnosis program (ie, hypnosis educational material and daily 20-minute audio-recorded hypnosis sessions) with sham hypnosis (ie, hypnosis educational material and daily 20-minute recordings of white noise) reported that self-hypnosis was associated with statistically significant greater reductions in mean hot flush scores of severity and frequency (53.4% vs 40.9% decrease) and mean ratings of hot flush daily interference (49.3% vs 37.4% decrease) at 6 weeks, although the differences in hot flush scores were within the margin of error at 12-week follow-up (155).

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Sleep Disturbance. Two studies have investigated the effects of clinical hypnosis on sleep disruption in individuals with and without VMS (152, 153). In the first RCT (N=187), participants with VMS who received five weekly 45-minute sessions of clinical hypnosis reported a statistically significant greater reduction in sleep disruption, as measured by the validated PSQI, compared with a therapist-led sham intervention (53.63% vs 10.34%) (153). The second trial was a pilot study that randomized 90 postmenopausal women with sleep disturbance to receive an at-home hypnosis audio-recording program paired with either three to five therapist-delivered hypnosis sessions or three to five therapist check-in calls focused on encouragement of home practice (152). At 8 weeks, all groups reported similar improvement in subjective measures of sleep (quality, duration, and bother) and objective sleep duration measured by wrist actigraphy (152).

Anxiety Symptoms. Based on very limited evidence, clinical hypnosis also may improve anxiety symptoms in women experiencing concurrent VMS (156). An RCT randomized 187 postmenopausal women with VMS to receive clinical hypnosis delivered in five 45-minute weekly in-person sessions or a control intervention consisting of five 45-minute weekly interactive sessions with a therapist who discussed symptoms and provided encouragement (156). At 5-week and 12-week follow-up, clinical hypnosis was associated with greater mean reductions in anxiety scores (as measured by validated anxiety symptom questionnaires) compared with the control intervention (156).

Acupuncture

ACOG suggests acupuncture to reduce VMS and improve menopause-related sleep disturbance and mood symptoms. **(CONDITIONAL RECOMMENDATION, LOW-QUALITY EVIDENCE)**

Acupuncture is a complementary and alternative medicine intervention that involves the use of thin, solid metallic needles that are inserted into the skin at specific anatomic points and then activated through manual manipulation (manual acupuncture) or electric stimulation (electroacupuncture) to achieve therapeutic effects (158). It is theorized that acupuncture may reduce menopausal vasomotor, sleep, and mood symptoms through inhibition of hypothalamic-pituitary-adrenal and hypothalamic-pituitary-ovarian activity, modulation of reproductive hormone and hormone receptor activity, and regulation of neurotransmitters and signaling pathways (158).

Studies on the use of acupuncture for menopausal symptom management report mixed findings, with many demonstrating little-to-no difference compared with sham acupuncture, which is commonly used as the control condition (158–160). In addition, there is high heterogeneity across trials in the type, frequency, and duration of treatment regimens (159, 160), and most studies have been conducted in China, which may limit the generalizability of the findings. There also is a lack of expert consensus on the use of acupuncture for menopausal symptoms, with some guidelines recognizing electroacupuncture as an adjunctive treatment option for VMS (62) and other medical societies advising that further study is needed (106).

However, given acupuncture's potential benefits and low risk of harm, it is reasonable to consider the use of acupuncture in combination with pharmacotherapy or other nonpharmacologic treatments as part of a multimodal holistic approach to menopausal symptom management. Acupuncture may not be covered by insurance (161), which should be taken into consideration when counseling patients who are interested in acupuncture as a treatment option for menopausal symptoms.

Vasomotor Symptoms. In a meta-analysis of 12 RCTs (N= 869), acupuncture was associated with small to moderate statistically significant reductions in VMS frequency (Hedges *g*, −0.35; 95% CI, −0.5 to −0.21; 10 studies) and severity (Hedges *g*, −0.44; 95% CI, −0.65 to −0.23; eight studies) compared with sham acupuncture and usual care, with improvements still present at 3 months after treatment (159). Similarly, a Cochrane review of studies that compared active and sham acupuncture reported a small but statistically significant reduction in VMS severity favoring active acupuncture (SMD, −0.45; 95% CI, −0.84 to −0.05; N=297; six RCTs), but there was not a significant difference in VMS frequency (MD, −1.13; 95% CI, −2.55 to 0.29; N=414; eight RCTs) (160).

Sleep Disturbance. Acupuncture also may be associated with improvement in menopause-related sleep disruption (162). A meta-analysis of 26 RCTs (N=2,326) reported that the use of acupuncture as an adjunct to conventional pharmacotherapy or nonpharmacologic treatment for perimenopausal insomnia was associated with a significant reduction in sleep disruption (measured by PSQI scores) compared with standard therapy alone (SMD, −1.00; 95% CI, −1.21 to −0.79) (162).

Mood Symptoms. A 2020 meta-analysis of RCTs that compared acupuncture with pharmacotherapy (fluoxetine, fluoxetine plus hormone therapy, escitalopram, or an antipsychotic plus tricyclic antidepressant) for the treatment of perimenopausal depression reported that manual acupuncture (MD, −2.35; 95% CI, −2.93 to −1.77; N=758; 10 RCTs) and electroacupuncture (MD, −1.20; 95% CI, −1.92 to −0.48, N=413, four RCTs) were associated with a greater reduction in symptom

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scores on the Hamilton Depression Rating Scale (163). A more recent systematic review and network meta-analysis that included a total of 54 RCTs (N= 4,406) found that the use of acupuncture alone or in combination with Western medicine (ie, antidepressants [SSRI, tricyclic, or tetracyclic], MHT, tibolone) was more effective than Western therapies alone in reducing scores on the Hamiton Depression Rating Scale (164).

Natural Products and Supplements

The available evidence does not support the efficacy or safety of phytoestrogens (eg, soy isoflavones) or herbal supplements for the treatment of vasomotor or other menopausal symptoms (165–167). Although multiple studies have been conducted to assess the efficacy of these substances for the management of VMS, confidence in the findings is limited by the small sample sizes as well as the heterogeneity of formulations, doses, and treatment durations across trials (165–167). In addition, none of these complementary therapies are regulated by the FDA, and they have not been tested for safety, efficacy, or purity because they are considered nutritional supplements. For these reasons, phytoestrogens and herbal supplements are not included as recommended treatment options in expert guidelines for the management of menopausal symptoms (73, 84, 106).

USE OF LANGUAGE

ACOG recognizes and supports the gender diversity of all patients who seek obstetric and gynecologic care. In original portions of this document, the authors seek to use gender-inclusive language or gender-neutral language. When describing research findings, this document uses gender terminology reported by the investigators. ACOG's policy on inclusive language can be reviewed at https://www.acog.org/clinical-information/policy-and-position-statements/statements-of-policy/2025/inclusive-language.

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APPENDICES

Supplemental Digital Content

A. Literature search strategy: available online at https://links.lww.com/AOG/E979

B. PRISMA diagram: available online at https://links.lww.com/AOG/E980

C. Evidence tables: available online at https://links.lww.com/AOG/E981

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