NAMS
THE NORTH AMERICAN MENOPAUSE SOCIETY

MENOPAUSE CARE UPDATES

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Menopause Care Updates presents reviews of important, recently published scientific articles selected by The North American Menopause Society (NAMS), the leading nonprofit scientific organization dedicated to improving women's health and quality of life through an understanding of menopause and healthy aging. Each review has commentary from a recognized expert that addresses its clinical relevance. Oversight for this e-newsletter was by Nicole Jaff, NCMP, Chair-elect of the 2016 NAMS Education Committee. Opinions expressed in the commentaries are those of the authors and are not necessarily endorsed by NAMS or by Dr. Jaff.

New American Cancer Society breast cancer screening guidelines continue confusion, controversy for women and their providers

Oeffinger KC, Fontham ET, Etzioni R, et al. Breast cancer screening for women at average risk: 2015 guideline update from the American Cancer Society. *JAMA*. 2015;314(15): 1599-1614.

Summary. The American Cancer Society (ACS) has updated its 2003 guidelines for screening mammography. The new evidence-based guidelines focus on women at average risk for breast cancer.

Average risk for breast cancer means

- No personal history of breast cancer
- No confirmed or suspected genetic mutation known to increase risk of breast cancer (eg, *BRCA*)
- No history of radiotherapy to the chest at a young age
- No significant family history of breast cancer
- No prior diagnosis of benign proliferative breast disease
- No significant mammographic breast density

The new guidelines are graded according to the strength of the recommendation as being either "strong" or "qualified." The ACS defines a strong recommendation as one that most women should follow. A qualified recommendation indicates that clinicians should acknowledge that there may be different choices for different women and that they must help each woman arrive at a management decision based on her values and preferences.

The new recommendations are

- Regular screening mammography starting at age 45 years (strong recommendation)
- Annual screening in women aged 45 to 54 years (qualified recommendation)
- Biennial screening beginning at age 55, unless the woman prefers to continue annual screening (qualified recommendation)
- Women who desire to initiate annual screening between the ages of 40 and 44 years should be accommodated (qualified recommendation)
- Screening mammography should continue as long as a woman is in good health and has a life expectancy of at least 10 years (qualified recommendation)

- Clinical breast examination is not recommended at any age (qualified recommendation)

Shortly after the ACS revised guidelines were released, the American Congress of Obstetricians and Gynecologists (ACOG) issued a statement in response, reaffirming their current clinical guidelines, saying, "ACOG strongly supports shared decision making between doctor and patient, and in the case of screening for breast cancer, it is essential. We recognize that guidelines and recommendations evolve as new evidence emerges, but currently ACOG continues to support routine mammograms beginning at 40 years as well as continued use of clinical breast examinations."

ACOG's current recommendations are

- Screening mammography every year for women aged 40 to 49 years
- Screening mammography every year for women aged 50 years or older
- Breast self-awareness has the potential to detect palpable breast cancer and can be recommended
- Clinical breast examination every year for women aged 19 years or older

Commentary by

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What are the controversies surrounding screening for breast cancer?

The primary controversies are the recommendations *against* routine screening in women aged 40 to 44 years, of *biennial* (rather than annual) screening beginning at 55 years, and *against* women performing breast self-examination.

Those in favor of the new ACS screening guidelines are concerned about lifetime radiation exposure and the costs, in terms of money, time, and mental well-being, of callbacks or false-positive biopsies.

Reasons against continuing annual mammography include psychological harms (anxiety) to the woman, excess or unnecessary imaging tests, unnecessary biopsies in women who don't turn out to have cancer, and inconvenience and fear when false-positive screening results occur.

Concern is raised regarding overdiagnosis of a breast cancer that might never become clinically apparent during a woman's lifetime and unnecessary treatments of early, nonaggressive breast cancer that may become clinically apparent but would not actually shorten a woman's life.

The differences are based on interpretations of the benefits and harms of screening. Regular mammogram screening finds breast cancer earlier when it is easier to treat, has less effect on women's quality of life, and reduces breast cancer deaths.

On the other side, an abnormal finding that requires further testing to investigate but has no effect on prolonging life or decreasing mortality leads to unnecessary risks of further tests or treatments.

In order to facilitate discussions about the new mammographic recommendations with patients, we turned to one of our nationally recognized breast-imaging specialists, Dr. Jennifer Harvey.

Dr. Harvey discusses her recommendations for women at average and high risk for breast cancer, including why she continues to recommend screening beginning at age 40 and continuing annually and her suggestions of 3D-tomosynthesis or other imaging modalities for those with dense breasts or at higher risk.

Commentary by

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Screening mammography saves lives, and women should continue to have this important test. Breast cancer mortality reductions of up to 48% have been demonstrated for women aged 40 to 79 years who undergo regular screening exams.[1] The debate is really about the age at which to initiate screening and the screening interval. The greatest benefit from mammography will be obtained from annual screening beginning at age 40 years.

Initiation of screening. Breast cancer diagnosed in young women represents a disproportionate percentage of breast cancer deaths,[2] likely because of the greater incidence of high-grade cancers. Faster tumor doubling times observed in younger women require more frequent screening intervals to affect mortality, and therefore, women aged younger than 55 years should obtain a mammogram every year.

Early initiation of screening increases the likelihood of a false-positive mammogram, which may lead to anxiety. For the vast majority of women, this represents a minimal and temporary state of anxiety until additional images are obtained that resolve the questioned screening finding. Certainly, some women will experience a much higher level of anxiety related to false-positive imaging, but these likely represent a low percentage of patients. Women who are anxious about a false positive should have 3D-tomosynthesis, because this technology reduces recalls by 15% to 30% while increasing detection of invasive cancers by 30% to 40%.[3-6]

Screening intervals. Annual screening results in the largest reduction in breast cancer mortality. The tradeoff is a greater risk of false-positive mammography and false-positive biopsy. Again, the risk of anxiety related to a false-positive mammogram is low for the vast majority of women compared with the benefit of reduction in breast cancer mortality. The risk of false-positive biopsy over a decade is 7% for annual and 4.8% for biannual screening.[7] The absolute increase in false-positive biopsies by using annual compared to biannual mammography translates to two false-positive biopsies being performed each year per 1,000 women screened. At the per-woman level, this risk is minimal. In addition, more than 95% of diagnostic breast biopsies are minimally invasive image-guided needle biopsies.

Can women at average risk safely reduce screening intervals at age 55? The big problem with moving to a risk-based approach to screening is that risk identification strategies are terribly inaccurate. Current breast cancer risk models have high calibration (an indicator of function at the population level) but low discrimination (an indicator of performance at the individual level), even for models including extensive risk factors such as the Tyrer-Cuzick model.[8]

Current models do not account for dense breast tissue, which increases the risk of breast cancer by 2 to 4 times and increases the risk of a false-negative mammogram.[9] Women with dense breasts should continue to undergo yearly mammography and consider additional screening with ultrasound.

Women wanting to maximize the benefits of screening should start at age 40 and obtain annual mammograms for as long as they remain in good health and have a life expectancy of 10 years or longer. Women with dense breast tissue should continue annual screening and at least consider ancillary screening. Women who are anxious about false-positive results should strongly consider having 3D-tomosynthesis mammography. Regular screening not only

reduces breast cancer mortality but also reduces the treatment of disease when detected.

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Review of menopause symptom treatment stresses staying current on data and individualizing care

Kaunitz AM, Manson JE. Management of menopausal symptoms. *Obstet Gynecol*. 2015;126(4):859-876.

Summary. A comprehensive review article reports on updates in the treatment of menopause symptoms, including vasomotor symptoms (VMS) and the genitourinary syndrome of menopause. The latest information on hormone therapy (HT), including the different HT options (oral vs transdermal) and varying doses, is presented and explained.

Nonhormonal treatment options, including behavioral treatments for hot flashes, selective serotonin reuptake inhibitors, serotonin-norepinephrine reuptake inhibitors, paroxetine, escitalopram, citalopram, venlafaxine, desvenlafaxine, gabapentin, and pregabalin, are explained, along with their benefits and risks.

Other information included in this review article is an overview of menopause, including symptoms, demographics, natural history, and the risk factors for different symptoms.

Special patient populations that may be particularly challenging are also addressed. These include women with early menopause, women with a history of or increased risk for breast cancer or who carry the *BRCA* gene, women with a history of venous thromboembolism or endometriosis, and women who have persistent menopause symptoms of a very long duration or who may request an extended duration of HT treatment.

Rather than stopping systemic HT at age 65, the authors suggest that length of treatment should be individualized on the basis of a woman's risk profile and preferences. They also suggest the use of benefit-risk profile tools for hormone and nonhormone options to help women make sound decisions on treatment.

Commentary by

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This excellent, updated review article is worthy of attention by any practitioner

caring for perimenopausal, postmenopausal, and menopausal women. It capsulizes an enormous literature and fairly presents a major evolution of changes in our approach to therapy for menopause symptoms since the publication of the Women's Health Initiative (WHI) study.

That stated, there are some additional points that should be included, understanding that this is a review article and not a book. For one, although we all agree that the pathophysiology of VMS remains poorly characterized, Freedman has not only shown that the set point of the thermoregulatory system is lowered as stated, but he has helped us to rule out the decreased endorphins and pulsatile luteinizing hormone secretion as causative factors.[1]

In addition, when only moderate and severe flashes are considered worthy of treatment, an attempt should be made to define them, even though it is certainly subjective. The US Food and Drug Administration gave these definitions as guidelines to industry that are widely used in studies: Mild feeling of heat with no sweating; moderate feeling of heat with sweating but able to continue activity; severe feeling of heat with sweating causing cessation of activity.[2]

It is important to consider the associated morbidity that accompanies symptoms.[3] Whiteley and associates observed that in postmenopausal women, a greater severity of VMS is significantly associated with lower levels of health status and work productivity and greater healthcare resource use.[4]

Thurston and colleagues' extensive Study of Women's Health Across the Nation showed that hot flashes were associated with a higher incidence of insulin resistance, and to a lesser extent, higher glucose.[5]

And in a subgroup analysis from the Women's Health Initiative trials of HT, higher risks of cardiovascular disease were shown in women with a higher burden of menopausal symptoms.[6] Those experiencing symptomatic menopause had significantly increased risk for coronary heart disease (hazard ratio [HR], 5.08; 95% confidence interval [CI], 2.08-12.40) compared with their counterparts with a lower burden of menopausal symptoms. Similarly, the risk of stroke was significantly elevated (HR, 3.94; 95% CI, 1.09-1.14). This analysis has supported the hypothesis that menopause symptoms convey cardiovascular risk.

It is also apparent from Greene's positron emission tomography studies in 2007 that there is significant decrease in cerebral blood flow during a hot flash.[7] This explains a woman's inability to continue her tasks during a hot flash.

The issue of estrogen alone versus estrogen-progestin in the benefit-risk ratio is certainly covered but should be stressed. The off-label use of progestin-releasing intrauterine devices is noted, but the availability of a lower-dose, smaller device should be mentioned as more appropriate for menopausal women.[8]

The conjugated estrogen (CEE)-bazedoxifene (BZA) combination may indeed be one answer as we receive more information. Clarkson's monkey study is hopeful in showing that CEE inhibits the progression and complications of both coronary and iliac artery atherosclerosis, and BZA has no adverse effects on atherosclerosis in postmenopausal monkeys.[9]

Because decrease in sexual desire is often a menopausal symptom, testosterone issues need to be addressed, although no testosterone medication has been approved by FDA. In a 6-month placebo-controlled study, followed by a 4-year follow-up of treated patients, a 300-µg patch daily (considered a low physiologic dose) showed statistically significant improvement in all aspects of sexual function, including successful and satisfying sexual desire.[10]

Kaunitz and Manson should be applauded for their exhaustive review and for including many controversies with their opinions. Of particular note is the reference to the package insert for low-dose vaginal estrogens, which becomes problematic in clinical use, and the outdated

recommendation of the American Geriatric Society to not treat women aged older than 65 years with estrogen. This has led to denial of medication by insurance companies.

The authors have given deserved credit to The North American Menopause Society and to the American Congress of Obstetricians and Gynecologists for fighting for individualizing hormone medications and treating symptoms regardless of age.

The authors should be commended as well for helping to bring clinicians up-to-date on the enormous significant data that address the benefit-risk profile of HT and point out that although there are contraindications, absolute risks are small. Their conclusion that remaining abreast of new information is essential in aiding women regarding management of menopausal symptoms should be a guiding principle.

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Merits, demerits of proposed redefined diagnostic criteria for osteoporosis

Kanis JA, McCloskey EV, Harvey NC, Johansson H, Leslie WD. Intervention thresholds and the diagnosis of osteoporosis. *J Bone Miner Res*. 2015;30(10):1747-1753.

Summary. A recent position paper of the National Bone Health Alliance (NBHA) has recommended that diagnostic criteria for osteoporosis be redefined to more closely reflect National Osteoporosis Foundation (NOF) treatment thresholds. Current World Health Organization (WHO) diagnostic criterion for osteoporosis is based on the measurement of bone mineral density (BMD). Osteoporosis is described as a BMD at the femoral neck of 2.5 standard deviations (SD) or more below the young female adult mean (T-score ≤–2.5 SD).

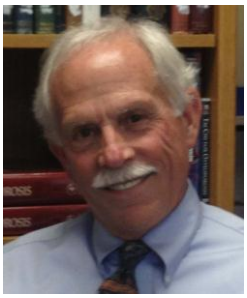
The NBHA proposes to broaden the definition of osteoporosis that adds additional criteria to include patients with fragility fractures and at "high risk for fracture." Using clinical, global, and economic reasoning, Kanis and colleagues provide arguments for and against this definition of osteoporosis.

Ultimately, Kanis and colleagues find the redefined diagnostic criteria "cumbersome" and likely to deter the management of osteoporosis in routine clinical practice.

They suggest an eventual balance for the diagnostic criteria for osteoporosis between sensitivity and clinical justification, as done recently for cardiovascular disease. They argue that the diagnostic criteria should still be based on BMD and question the need for diagnostic

criteria when the field, as other fields, is moving toward risk-based assessment and intervention.

Commentary by



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Confusion exists between the definition of and the diagnostic criteria for multifactorial clinical disorders such as osteoporosis, hypertension, and hyperlipidemia.

Each of these disorders has several pathogenetic mechanisms, and each becomes clinically apparent only when complications such as fracture, stroke, or heart attack occur.

At the beginning of my career, osteoporosis was the diagnosis when a patient, typically an older postmenopausal woman, presented with a fracture of her spine, hip, or shoulder. Osteoporosis was defined as a disorder of low bone mass, specifically, "a medical condition in which the bones become brittle and fragile from loss of tissue, typically as a result of hormonal changes, or deficiency of calcium or vitamin D."[1]

Osteoporosis was one of the *osteopenias* (the original meaning of that word), but unlike osteomalacia and other forms of osteopenia, the quality of bone in osteoporosis appeared normal by light microscopy.

However, based on histomorphometric studies by Parfitt and others, we came to appreciate that disrupted microarchitecture, in addition to low bone mass, was an important component of osteoporosis.[2]

The beautiful scanning electron microscopic images of trabecular bone by David Dempster[3] and of cortical porosity by Dr. Zabeze[4] augmented this understanding.

As the complex pathogenesis of skeletal fragility unfolded and our insight into the multiple determinants of bone strength improved, the definition of osteoporosis evolved, leading to the inclusion of "microarchitectural deterioration" as a requisite component.[5]

Although this is an accurate description of the disease, the influence of structure is difficult to incorporate into a diagnostic criterion because, until recently, it could not be assessed in individual patients, and even today, we can only do that with expensive research techniques.

The confusion between the definition and diagnosis of osteoporosis is amplified for two historical reasons: First, the term osteoporosis was originally (as is still) used as the diagnosis in patients who presented with a fragility fracture of the hip or spine.

However, in 1994, on the basis of epidemiologic but not clinical considerations, the World Health Organization (WHO) defined osteoporosis in postmenopausal women as a bone mineral density (BMD) T-score value of −2.5 or less.[6] By this definition, osteoporosis was redefined; no longer a clinical event, but as an important (but not the only) risk factor for fracture.

Unlike the relationship between high blood pressure (risk factor) and stroke (a clinical event), the same word was used to define both the risk factor and the clinical consequence. We are then left with the confusing situation of making the diagnosis of osteoporosis on both clinical criteria (patients who present with spine or hip fracture), regardless of T-score, and in those who meet the BMD diagnostic criteria.

Second, the term low bone mass (or unfortunately, *osteopenia*) was defined by the WHO as a T-score value between −1 and −2.5, overlapping the normal range of BMD in healthy young adults, which is −2 up to +2. Thus, the true definition of low bone mass is a T-score value of less than −2.

The article by Kanis and colleagues comes in response to a paper by the National Bone Health Alliance (NBHA), an American organization, that proposed broadening the definition of osteoporosis to include patients with certain fractures and patients at high risk for fracture.[7] That suggestion was based on the reluctance of American insurance companies to cover osteoporosis medications in patients who are at high risk for fracture but who do not meet the WHO BMD diagnostic criteria. Wedding the diagnostic and treatment criteria would perhaps solve this uniquely American problem.

Kanis reviews the history of and rationale for the diagnostic criteria for osteoporosis on the basis of BMD and clearly makes the distinction between how we define and diagnose the condition and how we decide who should receive osteoporosis treatment. The necessary disparity between diagnostic and treatment criteria is shared with other clinical disorders.

Updated treatment guidelines for using statin therapy to reduce cardiovascular risk have moved away from specific low-density lipoprotein values as a treatment threshold toward identifying and treating patients at high risk, just as we have done in the osteoporosis field.[8] Not surprisingly, those new guidelines engendered a spirited debate.[9,10]

Kanis also makes the important point that the WHO operational definition of osteoporosis has been well received by and functions very well in the rest of the world, in which the awkward relationships between payers and patients or physicians found in our country do not exist.

The Kanis paper is an important reference outlining the relationships between definitions, diagnostic criteria, and treatment thresholds. It should be read (and re-read) by all of us who make decisions about when to treat osteoporosis, hypertension, or hyperlipidemia and especially for those who take part in the challenging task of developing diagnostic criteria and treatment guidelines for complex medical problems with multiple distinct and independent determinants.

Were we to have a fresh start in the osteoporosis field, we would do a much better job of being specific and precise in our terminology. We would define low bone mass correctly as a T-score of less than −2, emphasizing the importance of this risk factor for fracture, just as *elevated blood pressure* and *high lipid levels* are used in other fields.

The utility of combining low bone mass with other risk factors such as advanced age and history of prior fracture to make assessments of fracture risk in individual patients would be recognized, as we have done with the FRAX tool. We would clearly define our therapeutic goal—to prevent fractures, or as we do in Portland, to make it even clearer to patients, to prevent "bone attacks."

The term osteoporosis would either revert to its early meaning as a specific pathologic condition and would not be used to define a risk factor for fracture or a patient with a fracture or become a more general term, analogous to cerebrovascular disease.

More practically, the problem that the NBHA attempted to address could be more easily solved by a simple change in the labeling of osteoporosis mediations by FDA. By approving drugs "for the treatment of patients at high risk of fracture" rather than for treating osteoporosis, a major component of the reimbursement difficulties would evaporate.

There is precedent in the lipid field in which statins are indicated to "reduce the risk of MI, stroke . . . in patients with CHD or without CHD, but with multiple risk factors."[11] Such a proposal was made at a recent FDA workshop on osteoporosis therapy. We should advocate for such a strategy, rather than confusing ourselves with yet another definition of osteoporosis.

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Disclosure: Dr. McClung reports no relevant conflicts of interest.

In Other News

NAMS presents summaries of other recently published articles for your review

Menopause status affects onset of asthma and other respiratory symptoms

Triebner K, Johannessen A, Puggini L, et al. Menopause as a predictor of new-onset asthma: a longitudinal Northern European population study [published online ahead of print October 1, 2015]. *J Allergy Clin Immunol*.

Because of limited and conflicting evidence on the effect of menopause on asthma, researchers in this longitudinal population-based study sought to learn whether the incidence of asthma and respiratory symptoms differ by menopause status.

The Respiratory Health in Northern Europe study provided questionnaire data pertaining to respiratory and reproductive health at baseline and follow-up.

The study cohort included women aged 45 to 65 years at follow-up, without asthma at baseline, and not using hormone therapy (n=2,322).

Menopause status was defined as nonmenopausal, transitional, early post-menopausal, and late postmenopausal.

Associations with asthma (defined by the use of asthma medication, having asthma attacks, or both) and respiratory symptoms scores were analyzed by using logistic (asthma) and negative binomial (respiratory symptoms) regressions, adjusting for age, body mass index, physical activity, smoking, education, and study center.

The odds of new-onset asthma were increased in women who were transitional (odds ratio [OR], 2.40; 95% confidence interval [CI], 1.09-5.30), early postmenopausal (OR, 2.11; 95% CI, 1.06-4.20), and late postmenopausal (OR, 3.44; 95% CI, 1.31-9.05) at follow-up compared with nonmenopausal women. The risk of respiratory symptoms increased in early postmenopausal (coefficient, 0.40; 95% CI, 0.06-0.75) and late

postmenopausal (coefficient, 0.69; 95% CI, 0.15-1.23) women.

These findings were consistent regardless of smoking status and across all study centers.

Menopause Editor's picks for November 2015

NAMS spotlights selections from the most recent issue of the Society's official journal, *Menopause*, chosen by its editor in chief, Isaac Schiff, MD.

Associations between body mass index and sexual functioning in midlife women: the Study of Women's Health Across the Nation
Lisa M. Nackers, PhD, MPH; Bradley M. Appelhans, PhD; Eisuke Segawa, PhD; Imke Janssen, PhD; Shelia A. Dugan, MD; and Howard M. Kravitz, DO, MPH

♦

Cluster analysis of midlife women's sleep-related symptoms: racial/ethnic differences
Eun-Ok Im, PhD, MPH, RN, CNS, FAAN; Ko Young, PhD; Eunice Chee, BS(c); and Wonshik Chee, PhD

♦

Confirmatory factor analysis of the Pittsburgh Sleep Quality Index in women with hot flashes
Julie L. Otte, PhD, RN; Kevin L. Rand, PhD; Carol A. Landis, PhD, RN, FAAN; Misti L. Paudel, PhD; Katherine M. Newton, PhD; Nancy Woods, PhD, RN, FAAN; and Janet S. Carpenter, PhD, RN, FAAN

♦

One-year treatment persistence with local estrogen therapy in postmenopausal women diagnosed as having vaginal atrophy
David Portman, MD; Lee Shulman, MD; Jason Yeaw, MPH; Sha Zeng, MSc; Chioma Uzoigwe, MPH; Ricardo Maamari, MD, NCMP; and Neeraj N. Iyer, PhD

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