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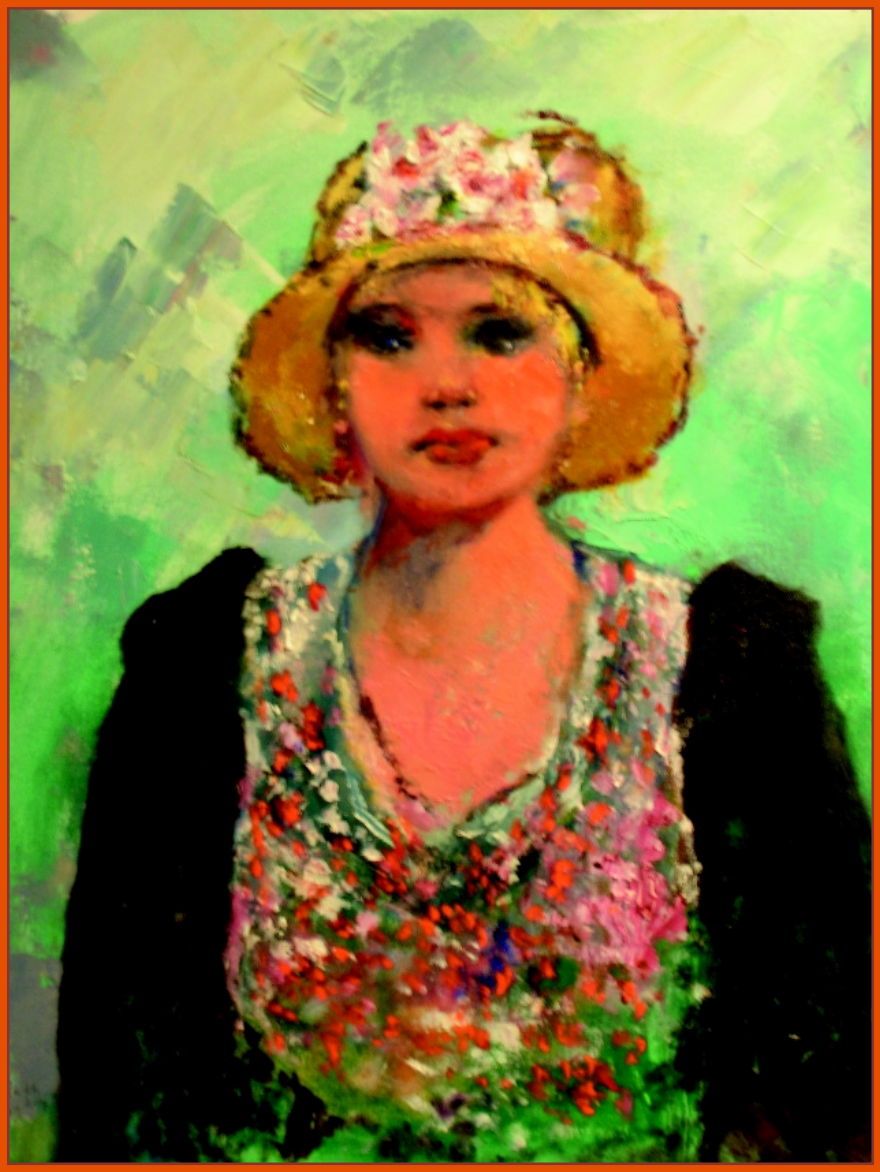

Menopause

What's in a Name???

GOOD - authentic, honest, just, kind, pleasant, skillful, valid

NEIGHBOR - friend, near

ALLIANCE - affiliation, association, marriage, relationship

CORPORATION - company, business establishment

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COMMENTARIES

CONTRIBUTIONS

SPECIAL ISSUE: Menopause
Guest Editor: **Kelly A. McGarry, MD**

COLUMNS

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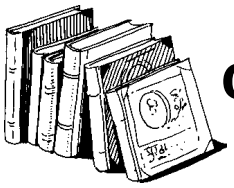

Commentaries

My Ties To Big Pharma

Recently I was reviewing a submission to the journal and wondered why the author hadn't discussed a certain medication. While this particular use of the medicine was "off label," it was a common use. Some of the other drugs mentioned in the article were also commonly used, and also were being recommended for "off label" use. As soon as I wrote my comment I realized that I had a conflict of interest. I was a speaker for the company that sold the drug I recommended. Not just that either, I was a "KOL!" (I learned about KOLs when I was told that my speaking engagements would be limited so that the company didn't run into trouble with the FDA because I was a KOL. Since I didn't know what a KOL was, I asked, and learned that I was a "Key Opinion Leader.") So, here I am, a national spokesman, sort of, for a company that makes a drug I'm touting in a review article by someone else for an off label indication.

This raised an immediate alarm within my head. How was I to edit a journal when I had a variety of potential conflicts of interest? Of course, some readers will note that no matter how objective I may seem, or fool myself and others into seeming, the very fact that I take money from drug companies indicates a certain political slant that many readers do not share. So at that point I immediately instituted our current policy of reporting all conflicts of interest. Not only do the authors list their conflicts, as is standard in national journals, but the editor (i.e, me) now lists them as well, something I've not seen in national journals.

I have many ties to the pharmaceutical industry. I participate in clinical trials. I occasionally consult, and I give lectures. I have no stock interests. It is mainly the lectures that cause problems for me. I give these for a few reasons. Income is certainly near the top of the list, but I also like to educate doctors and health professionals about the areas I have expertise in. I only talk about things I'm knowledgeable about and I never recommend things I don't do myself. My "promotional" talks are no less balanced than my grand rounds lectures. But there is a downside to this. The biggest for me was having to turn down an invitation to write an editorial for *Neurology* because the topic involved discussion of a drug I spoke for. The solicitation was a reward for 15 years of work, and I had to decline. A blow to my vanity, but the journal and I vouchsafed our integrity.

I also occasionally solicit funds for neurology grand rounds to subsidize speakers from out of town. Our grand rounds have no budget, so I am limited to local speakers, which luckily includes Boston and Worcester.

Although journals do not require that their reviewers reveal any conflicts, at least none of the many that I've reviewed for, I have begun to list them anyway for the benefit of the editors. It is important that an editor realize that my panning or praising may be related to some tangible interest of mine that might cloud my judgment. I have been thinking of suggesting that editors of journals and all reviewers reveal their conflicts. I suspect that most of us have been doing this when we think there is a "real" danger of a conflict or a seeming conflict, and we may do this in advance of accepting a reviewing assignment. "No, I can't review this article by my best friend." "No, I can't review this because my retirement depends on this bad result not causing the drug company to tank."

The *New England Journal* tried to exclude doctors with any conflicts from writing their review articles and concluded that it was impossible. Virtually all the physicians with sufficient expertise and fame in almost any field had financial ties to industries that might be affected by, or affect, what they wrote. They ended up with a policy like every other journal's, namely, stating the conflict of interest.

The *New England Journal* has continued to be interested in the topic and recently published an article detailing the prevalence and types of ties doctors have with pharmaceutical companies (the biotech and bio-device industry is no different than the drug companies). Over 25% of doctors surveyed received fees for consulting, lecturing or participating in clinical trials. Over three quarters accepted drug samples. Over a third accepted sponsorship for continuing medical education and 83% accepted food in the work setting. Articles in the news media revealed that most local speakers for drug companies were chosen entirely based on their being high volume prescribers, not being expert in their field. Since the FDA regulates what is allowed to be discussed at promotional, rather than talks given for CME credit, that is, the usual talks given at dinners, run by drug reps, in which post-its and pens are handed out, this makes perfect sense because the speakers are now forbidden from saying anything not on a slide, and having to say everything that is on all of the slides. It is forbidden, for example, for me to illustrate a movement disorder I'm discussing by showing an example. So, if expertise is going to be untapped, why use an expert? Better to reward a big user of the drug with the honorarium and a chance to shine in front of local colleagues.

It is quite clear to me that there has to be a close relationship between doctors and drug companies, and that this relationship needs to be public and above board. But how to achieve this is uncertain. While I agree that it is a good idea to keep drug reps from trainees, it is more politically correct than efficacious to reject all drug company relationships. Expert doctors should be running drug company trials. Who can do them better and more reliably? Expert doctors should be talking about the drugs. Expert doctors should be providing counsel to companies, telling them in what way their drugs are

better, worse or the same as others in their field. Some companies use the gifts of fame and fortune to mask their real intentions of getting an endorsement under the guise of flattery and close personal ties.

The question really is how do we keep from being corrupted when the aim of the company is to corrupt us? Government edicts won't work. Ethics can't be mandated. Our journal has begun, somewhat belatedly, with publication of the conflicts of interests of the authors.

– JOSEPH H. FRIEDMAN, MD

Disclosure of Financial Interests

Joseph Friedman, MD, Consultant: Acarta Pharmacy, Ovation, Transoral; Grant Research Support: Cephalon, Teva, Novartis, Boehringer-Ingelheim, Sepracor, Glaxo; Speakers' Bureau: Astra Zeneca, Teva, Novartis, Boehringer-Ingelheim, GlaxoAcadia, Sepracor, Glaxo Smith Kline

A Slothful Afternoon On an Alabama Farm

Sloth is one of those miserable human failings with a reputation that loiters somewhere between heresy and high treason. In a world that forgives child labor as family enrichment and caning as progressive encouragement, sloth remains resolutely, steadfastly an unforgivable shortcoming; not a momentary lapse nor even a youthful indiscretion but a cardinal sin, indeed one of the seven deadly ones.

Sloth, from an old English word meaning slow, is more than a brief contemplative reflection; more than a Scripturally-sanctioned sabbatical rest; rather, it now defines a form of moral or physical laziness. [It is surprising, incidentally, how many English words, beginning with the letters, **sl-**, signify demeaning sloth-like human qualities; words such as slovenly, sloppy, sleazy, slacker, slow-witted, slap-dash, slattern, slanderer, sluggish and slipshod.]

Sloth is mentioned repeatedly in the Book of Proverbs, appearing as a warning against excessive idleness. In Proverb 12, the unhappy fate of the slothful is made abundantly clear. And later, we learn that slothfulness will lead eventually to hunger and banishment. The sins that were considered deadly became codified by the 3rd Century. Pope Gregory I then declared seven sins as cardinal [extravagance, gluttony, greed, sadness, wrath, envy and pride.]; but not until the 17th Century was sloth distinguished as a major vice. Previously, it had been subsumed under the sinful category of sadness.

But with so many nasty human failings, transgressions, villainies, trespasses and lapses, how did sloth ever manage to beat out so many others to reach the Final Seven ? It emerged, first, under the name of *akedia*, a Greek word describing carelessness, indifference or lethargy, an essentially secular failing. But akedia, or sloth, also came to define an indifference to moral responsibilities, a sluggish torpor which avoids religious obligations during that arduous journey toward adulthood; sloth was, hence, contrary to the obligations required of those abiding by the first of the Mosaic Commandments. In medieval texts, sloth was viewed as a spiritual fatigue, as an arrogant affront to the Divine Authority, punishable, in Hell, by being thrown into deep snake pits.

The Seven Deadly Sins were not misdeeds of equal severity. Pride was generally identified as the worst of the seven sins. Sloth, on the other hand, was considered a sin of omission, an absence of diligent piety, in contrast to the sins of excess such as lust, gluttony and greed. But as the Industrial Revolution overtook Europe, however, the moral lapse called sloth gradually evolved into a major transgression. Sloth, sometimes called loafing or laziness amongst the poor and leisure amongst the landed, became an affront to industrial progress, with but few remaining safe pockets of sloth. It was said, for example, that vacation spas were enclaves of rest designed specifically for those seeking leisure to meditate upon the sin of sloth.

And why seven sins? Why not nine or 49? Seven was given special meaning in the Bible. It was a number sacred to the Hebrews marking the cycle of creation in days. The Hebrew candlestick had seven branches; and " seven" is mentioned over 700 times in the Bible. The Book of Revelation mentions seven repeatedly [seven churches, seven spirits, seven stars, seven seals.]

Sin, of course, is sin; but being free of sin may merely indicate that circumstances had not yet been contrived to make the keys to sin available; or, alternatively, that the person currently free of sin has not as yet learned how.

And, on the other hand, can sloth have arisen in some people, not by a consciously sinful act or a moral poverty, but by banal circumstances beyond their control ? Consider rural Alabama, for example, in the year 1905. An average farm might house a handful of children, most of them going barefoot. This typical farmhouse has neither electricity nor indoor privies. A closer scrutiny of the children, particularly the pre-adolescent males, shows what many adults might call laziness, shiftlessness—or just plain sloth.

And what are the characteristics of these slothful children? They tend to be black, visibly undernourished, slow in movement and sluggish in vocal response. Their speech is filled with hesitations and its cadence is maddeningly halting. An alert pediatrician will recognize this slothful behavior, not as a moral shortcoming but rather as a manifestation of a parasitic infestation called hookworm, a disease widely endemic in the rural south until the 1930s.

Hookworms are small parasites which attach themselves to the inner lining of the young victim's small intestine constantly extracting small amounts of blood; and if there are sufficient numbers of these worms, the victim becomes severely anemic with secondary weakness, slowness of thinking and speech, chronic diarrhea and retarded growth. The eggs of these worms are shed in the victim's feces; and if defecation takes place in some secluded spot on the farm rather than in a privy, the parasitic eggs mature into larval worms which then invade the naked feet of children walking over the farmland. The development of interior plumbing and insistence that the children wear shoes has now reduced hookworm disease to a rare disorder. And with the disappearance of hookworm in the American South, the nation has also witnessed the disappearance of that demeaning racial stereotype of the shuffling, sleepy-headed, slow-speaking "darkie."

James Thurber, who relished his slothful afternoons, declared: "It is better to have loafed and lost than never to have loafed at all."

– STANLEY M. ARONSON, MD

Disclosure of Financial Interests

Stanley M. Aronson, MD, has no financial interests to disclose.

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Menopause: Introduction

Kelly A. McGarry, MD

Prior to the findings of the Women's Health Initiative, health care providers often turned to estrogen replacement as the sole management for women with significant menopausal symptoms. It was believed that estrogen could help all menopausal symptoms and, to a certain extent, it was true. However, armed with the knowledge that estrogen replacement may be more harmful than beneficial, providers must learn about alternative ways to help those women who are significantly affected by menopausal symptoms. Importantly, most women will go through the menopause transition without experiencing any negative or burdensome symptoms. Our goal in these articles is to address several common symptoms and provide up-to-date information about the options for women who request your help. The first article by Dr. Tong reviews the treatment options for hot flashes. In it she provides an excellent evidence-based review of the medications, including complementary therapies, for the treatment of hot flashes. The second article by Dr. Duffy is designed to help providers assess and treat women who experience sexual dysfunction around menopause. She discusses the importance of the psychosocial and medical history, illuminating the ways in which both can impact on a woman's sexual functioning. Treatment options including the role of psychotherapy and testosterone are discussed. Finally, Dr. Landau and I discuss how menopause can affect mood. We outline the possible psychosocial and hormonal connections between menopause and mood. We also highlight the potential treatments. We hope that these succinct reviews provide useful information to aid providers in the care of women significantly affected by menopausal symptoms.

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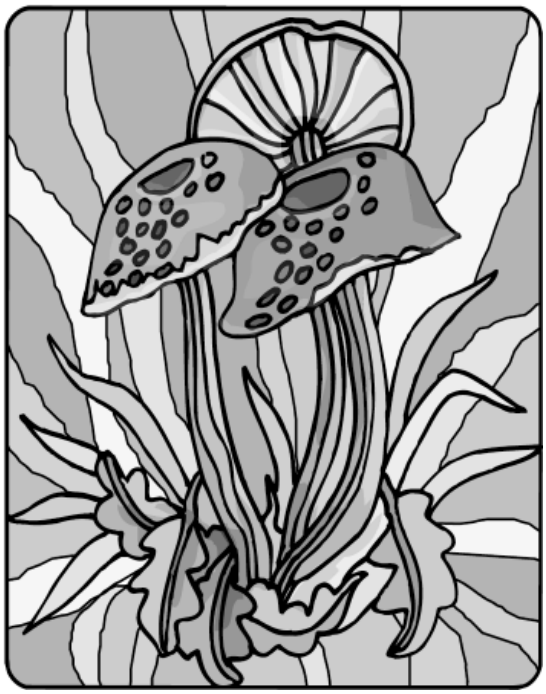

Treatment of Menopausal Hot Flashes

Iris L. Tong, MD

The onset of vasomotor symptoms occurs in the perimenopausal state. Approximately two-thirds of postmenopausal women will experience hot flashes; 10-20% will experience severe symptoms. For most women, symptoms spontaneously resolve within a few years.[1] However, one third of postmenopausal women experience symptoms for up to 5 years, and 20% will have symptoms for up to 15 years.[2]

In the United States, an estimated 25 million women are symptomatic, and 4 million women have severe symptoms.[3]

While the exact cause of hot flashes is unknown, vasomotor symptoms result from thermoregulatory dysfunction at the level of the hypothalamus. Inappropriate peripheral vasodilatation occurs, followed by rapid heat loss and a decrease in core body temperature. Shivering ensues to restore the core body temperature to normal. Norepinephrine and serotonin act on the thermoregulatory nucleus to affect heat loss mechanisms, and estrogen has been demonstrated to affect norepinephrine and serotonin levels.[4]

Examining the Role of Hormone Therapy (HT)

HT has been shown to improve symptoms in 80% - 90% of women. However, the **Women's Health Initiative (WHI)** demonstrated that the risks of HT outweigh its benefits in postmenopausal women. Over 16,000 healthy, postmenopausal women were randomized to receive either conjugated equine estrogen (CEE) 0.625 mg with progesterone 2.5 mg daily or placebo.[5] The study was stopped early after 5.2 years, secondary to a statistically significant higher number of adverse events in the treatment arm. Compared to placebo, there were 7 more cases of nonfatal myocardial infarction or death related to coronary artery disease, 8 more cases of stroke, 8 more cases of pulmonary embolism, and 8 more cases of invasive breast cancer per 10,000 person-years in the treatment group. As part of the WHI, 10,739 postmenopausal women who had undergone hysterectomy were randomized to receive either estrogen 0.625mg daily or placebo. This trial was also stopped early after 6.8 years secondary to the higher number of cases of stroke in the treatment arm (12 additional cases of stroke per 10,000 person-years).[6]

In a secondary analysis of the WHI data, study subjects were divided into three age-categories (50-59 years, 60-69 years, 70-79 years of age) and by years since menopause (<10 years, 10-20 years, >20 years since menopause).[7] The authors analyzed the timing of HT and its effect on cardiovascular events. When the women were analyzed according to age and years since menopause, only women between 60 and 69 years old who received estrogen alone had a statistically significant higher risk of stroke (hazards ratio = 1.62, confidence interval = 1.15-2.27). With respect to cardiac events, there was a statistically significant increased risk of cardiac events among women taking estrogen plus progesterone who were between 70 and 79 years old (hazards ratio 1.48, confidence interval 1.04-2.11) or who were greater than 20 years since menopause (hazards ratio = 1.66, confidence interval 1.14-2.41). Among the women who were distant from menopause, the cardiac events clustered in those with moderate or severe hot flashes. From this data, the authors suggest that hot flashes may be a marker for endothelial dysfunction. There was a trend for reduced risk of cardiac events among women 50-59 years of age taking estrogen plus progesterone (hazards ratio = 0.63, confidence interval 0.36-1.09) and among women who were less than 10 yrs since the onset of menopause taking estrogen alone (hazards ratio = 0.48, confidence interval = 0.20-1.17). The authors suggest that HT can be safely used for the treatment of hot flashes among postmenopausal women who are younger or who have recent onset of menopause given the trend for lower risk of coronary events in these two subsets of women. While vasomotor symptoms remain an indication for HT use, the **Federal Drug Administration (FDA)** advises prescribing the lowest dose for the shortest duration possible.

When discontinuing HT, many women experience a recurrence of symptoms. In a cross-sectional survey of 8,405 women who had participated in the WHI estrogen plus progesterone trial, women in both the treatment and placebo arms reported their hot flash symptoms after they were instructed to discontinue the medication.[8] Women in the treatment arm were more likely to experience moderate to severe symptoms than women in the placebo arm, whether they reported hot flashes at baseline (56% vs. 22%, respectively) or not (21% vs. 5%, respectively). All women were instructed to discontinue HT abruptly in this trial.

Some women are more successful in discontinuing HT by tapering off the medication rather than stopping the medication abruptly. A standard tapering regimen is to decrease HT by one pill per week (i.e., six pills per week, then five pills per week, etc.) until the taper is completed over six weeks. For women who experience symptoms while tapering, a slower taper may be attempted. In a trial of 91 postmenopausal women on HT for greater than 3 years who were randomly assigned either to discontinue HT abruptly or to discontinue HT gradually over 6 months, hot flash symptoms were more severe in the abrupt discontinuation group in the first 3 months after HT withdrawal.[9] However, at 6 months after HT withdrawal, symptoms were worse in the gradual discontinuation group, and by 9-12 months after HT withdrawal, there was no difference between groups. Overall, a lower number of women in the gradual discontinuation arm resumed HT secondary to vasomotor symptoms as compared to the abrupt discontinuation arm (36.6% vs 42%, respectively). Therefore, tapering HT may offer a slightly greater rate of successful discontinuation of HT when compared to abrupt discontinuation. The length of taper in this study occurred over 6 months; it is not known if tapering over 6 weeks would have a comparable success rate of discontinuing HT.

Nonhormonal Therapies

Given the potential adverse events associated with HT, other options for the treatment of vasomotor symptoms have emerged recently, including behavior modification, complementary medicines, and pharmacologic agents.

Behavior Modification

For women with mild symptoms, keeping room temperatures cool and dressing in layered clothing may provide adequate relief. Small studies have demonstrated that paced respiration, a relaxation-based method which consists of slow, rhythmic breathing, has a modest effect on hot flashes.[10] Exercise, while beneficial in other respects, has not been consistently shown to improve hot flash symptoms.[11] Cigarette smoking has been associated with a higher risk of experiencing vasomotor symptoms, and the risk appears to be directly proportional to the number of cigarettes smoked.[12] Given the additional health benefits of tobacco cessation, smoking cessation should be encouraged.

Complementary Medicine

Approximately 50-75% of postmenopausal women will use alternative medicines to treat their hot flashes.[13] Many complementary medicines have been studied in the treatment of hot flashes. A randomized trial of 120 subjects receiving 800 international units of Vitamin E daily demonstrated a modest benefit over placebo, with an average reduction of one hot flash per day.[14] No evidence supports the use of red clover, dong quai, evening primrose oil, wild yam, or black cohosh in the treatment of vasomotor symptoms.[15,16] Of the complementary therapies, soy and black cohosh have been the most investigated.

Soy is the most common plant containing phytoestrogens, plant-based estrogens which bind to estrogen receptors. A review of 16 trials investigated the effect of soy on hot flash symptoms.[16] Eight trials demonstrated that soy was superior to placebo, and 8 studies demonstrated that soy was comparable to placebo in the treatment of hot flashes. From the trials demonstrating benefit, the average reduction in symptoms ranged from 25-55% compared to an average reduction of 20-30% with placebo. Importantly, most of the trials on soy (average length of 12 weeks, with a range of 4-52 weeks) are longer in duration than trials on pharmacological therapies (average length of 4-6 weeks, with a range of 4-36 weeks). Given the natural history of menopausal symptoms, longer trials are less likely to demonstrate a treatment effect.

No evidence supports the use of red clover, dong quai, evening primrose oil, wild yam, or black cohosh in the treatment of vasomotor symptoms.

There have been no adverse effects associated with soy. However, phytoestrogens bind to estrogen receptors and can exert estrogenic and antiestrogenic effects. Therefore, they may cause potential harm in women with estrogen-dependent tumors. Phytoestrogens may antagonize the antitumor effect of tamoxifen. Because of these concerns, soy is typically avoided in women with a personal history of breast cancer, at high risk for breast cancer, or who are undergoing treatment with tamoxifen.

Black Cohosh originates from the plant, *Cimicifuga racemosa*, native to the eastern United States and Canada. It has been used in Europe to treat hot flashes for over 50 years. Preparations of black cohosh may contain phytoestrogens and therefore should be avoided in breast cancer survivors. Several small randomized trials comparing black cohosh to placebo in the treatment of hot flashes have demonstrated conflicting results, but in a recent, large trial,[15] 350 women between 45 and 55 years old were randomized to one of five arms: 1) Black cohosh, 160 mg daily, 2) a multibotanical containing 200mg black cohosh daily, 3) a multibotanical plus dietary soy counseling, 4) CEE 0.625 with or without 2.5 mg progesterone daily, or 5) placebo. Fifty-two percent of the women were perimenopausal. After one year of treatment, all three black cohosh treatment arms were comparable to placebo. Subjects in the hormone therapy arm had a statistically significant reduction in symptoms compared to the other treatment arms and placebo (59.7% vs. 0% reduction in symptoms beyond placebo, respectively). The authors concluded that black cohosh was not superior to placebo in the treatment of hot flashes.

Pharmacologic Agents

Several pharmacologic agents have been used as therapy for vasomotor symptoms. The most common agents used are clonidine, antidepressants, and gabapentin. Clonidine is a centrally acting α_2-adrenergic agonist which decreases norepinephrine release. Three small randomized trials have demonstrated a significant reduction in symptoms when compared to placebo (40-80% vs. 25-30%).[17,18,19] Most of the subjects were women with a prior history of cancer who were taking tamoxifen. The doses of clonidine used in the trials ranged from 0.1 mg to 0.4 mg daily, and the length of the trials ranged from 8-12 weeks. Significant side effects included dry mouth, insomnia, and dizziness.

The most common antidepressant medications used to treat hot flashes are **serotonin-reuptake inhibitors (SSRI)** and a **serotonin/norepinephrine reuptake inhibitor (SNRI)**, venlafaxine. SSRIs that have been investigated include paroxetine, fluoxetine, and citalopram. These agents alter norepinephrine and/or serotonin levels, and both neurotransmitters are thought to affect temperature control at the level of the hypothalamus.[4]

Two randomized trials evaluated paroxetine in the treatment of hot flashes.[20,21] In the first trial, 165 postmenopausal women were randomized to controlled-release paroxetine 12.5 mg daily, 25 mg daily, or placebo for six weeks.[20] Subjects in the 12.5 mg and 25mg arms had significantly greater reductions in symptoms than subjects in the placebo arm (62.2 %, 64.6%, and 37.8% reduction, respectively, $p<.05$), and the 12.5 mg dose was comparable to the 25mg dose. In the second study, 151 women were randomized to paroxetine 10mg daily, 20mg daily, or placebo in an 8-week crossover trial.[21]

Over 80% of the women had a history of breast cancer. Women in the 10mg and the 20mg arm had statistically significant reductions in hot flash frequency compared to women receiving placebo (40.6%, 51.7%, and 26.6% reduction, respectively, p<0.002). Common side effects included headache, nausea, and insomnia, but nausea was the only symptom significantly associated with the treatment arm in both studies. Weight gain was not a significant side effect in either study. Paroxetine reduces the formation of a major active metabolite of tamoxifen and therefore should not be used in women taking tamoxifen.

Two other SSRIs that have been studied are citalopram and fluoxetine. In one of the longest trials conducted on pharmacological agents, 150 postmenopausal women were randomized to receive Citalopram 10mg daily, Fluoxetine 10mg daily, or placebo for nine months.[22] Doses were increased to 20mg daily at 1 month and to 30 mg daily at 6 months. Over the 9-month study period, subjects in all three arms improved, and there was no statistically significant difference between the treatment and placebo arms. There was a 62%, 64%, and 58% reduction in hot flash frequency with fluoxetine, citalopram, and placebo, respectively.

Venlafaxine, an SNRI, has been studied in two randomized trials. In the first trial, 191 postmenopausal women with or without a history of breast cancer were randomized to extended-release venlafaxine 37.5 mg daily, 75mg daily, 150mg daily, or placebo and followed for 4 weeks.[23] Sixty-nine percent of subjects were taking tamoxifen. All three treatment arms were superior to placebo (37%, 61%, 61%, and 27% reduction in symptoms, respectively, p<0.0001), and the 75mg dose was equally as effective as the 150mg dose. Efficacy was similar between patients with and without tamoxifen use. In a second trial, 80 postmenopausal women without a history of breast cancer were randomized to extended-release venlafaxine 75mg daily or placebo for 12 weeks.[24] Although there was no difference in symptom reduction between the treatment and placebo arms, subjects in the treatment arm perceived a reduction in symptoms. Significant side effects from both studies included dry mouth, decreased appetite, insomnia, nausea, and constipation. Increase in blood pressure was not a significant side effect in either study.

Gabapentin is a α-aminobutyric acid (GABA) analogue and may regulate calcium channels in the ventromedial hypothalamus, which is involved in thermoregulation. There have been 3 randomized, placebo-controlled trials evaluating gabapentin in the treatment of vasomotor symptoms, one of which enrolled women with a history of breast cancer.[25,26,27] All three trials demonstrated that gabapentin was superior to placebo. Subjects in these studies were followed for 8-12 weeks, and effective doses ranged from 900mg to 2400mg daily in divided doses. In a recent study, 118 postmenopausal women with inadequate hot flash control with antidepressants were randomized either to switch to gabapentin 300mg tid or to add gabapentin 300mg tid to their regimen for 5 weeks.[28] Most patients were taking venlafaxine or paroxetine. There was a significant reduction in hot flash frequency in both groups, 49% reduction with gabapentin alone and 54% reduction with combined treatment. The authors concluded that switching to gabapentin is an option for women who continue to have symptoms despite antidepressant therapy. However, because there was no placebo arm in this study, it is difficult to conclude definitively that the improvement in symptoms was secondary to gabapentin. Three studies reported no significant side effects with gabapentin. In one study, the most commonly reported side effects were somnolence and dizziness. Given its side effect of somnolence, gabapentin may be a good option for women with vasomotor symptoms as well as sleep disturbances.

Interpreting the Data

In reviewing the current studies on complementary medicines and pharmacologic agents, there may be a bias in favor of pharmacologic agents because most of these trials are sponsored by the manufacturers. In addition, there have been a greater number of randomized controlled trials conducted on complementary medicines, soy in particular, than pharmacological therapies. Moreover, the length of complementary medicine trials is significantly longer than that of pharmacologic agent trials. Trials investigating complementary medicines typically range from 12 to 24 weeks, with the longest being 52 weeks, whereas trials investigating pharmacologic therapies range from 4 to 12 weeks, with the longest being 36 weeks. Given the natural history of menopausal symptoms, most women will have spontaneous resolution within one to a few years. Hence, the longer the trial, the more likely there will be less of a difference between the treatment and placebo arms. Perhaps if more and longer studies were conducted on pharmacologic agents, it might be demonstrated that these agents are also comparable to placebo. Finally, in most trials, there is consistently a 20-30% reduction of hot flash symptoms in the placebo arm. This significant placebo effect highlights the importance of placebo-controlled trials in the investigation of treatment options for vasomotor symptoms.

Conclusions

Hormone replacement therapy remains the most effective therapy for postmenopausal vasomotor symptoms. However, as demonstrated by the WHI, HT is associated with an increased risk of cardiovascular events, pulmonary embolism, and breast cancer. While menopausal symptoms remain an indication for HT, the FDA advises using the lowest dose for the shortest duration possible. A recent secondary analysis of the WHI suggests that HT may be safe in women with recent onset of menopause with respect to risk of cardiac events.

Alternatives to HT include behavior modification, complementary medicines, and pharmacologic agents. Behavior modification has a modest effect on hot flash symptoms. Complementary medicines such as Vitamin E, dong quai, evening primrose oil, red clover, and black cohosh have been shown to be comparable to placebo. Soy has been the most heavily studied and seems the most effective alternative therapy. The most effective pharmacologic agents include clonidine, paroxetine or paroxetine controlled-release, venlafaxine extended-release, and gabapentin. Therapeutic and side effect profiles of these treatment options can aid in the choice of therapy for women with vasomotor symptoms. Women with concomittent hypertension

may benefit from treatment with clonidine; women with concomittent depression may benefit from treatment with paroxetine or venlafaxine; and women with insomnia may benefit from treatment with gabapentin.

With patients who are experiencing hot flash symptoms, I review the options of behavioral modification, dietary soy, pharmacological agents (paroxetine, venlafaxine, neurontin), and hormone therapy. Many patients have a preference with respect to which therapy they would like to initiate, have additional symptoms that help guide our choice of therapy (i.e., sleep disturbance which may improve with gabapentin or underlying anxiety which may improve with paroxetine), or have a contraindication to a given therapy (i.e., patients with hypertension may have an increase in blood pressure with venlafaxine or patients at high risk for breast cancer should avoid soy). I typically do not prescribe clonidine because of the potential side effects in women without underlying hypertension and the potential occurrence of rebound hypertension with its abrupt discontinuation in women with hypertension. In addition, I typically do not prescribe HT initially given the potential risks but reserve it for patients with severe symptoms who have failed other therapeutic options. I would then only recommend HT for women with recent onset of menopause, using the lowest dose for the shortest period of time.

References

1. Kronenberg F. *Ann N Y Acad Sci* 1990; 592:52.
2. Kronenberg F. *Exp Gerontol* 1994; 29: 319-36.
3. Stearns V, Ullmer L, et al. *Lancet* 2002; 360: 1851-61.
4. Gonzales GF, Cariilo C. *Maturitas* 1993; 17: 23-9.
5. Rossouw JE, Anderson GL, et al. *JAMA* 2002;288:321-33.
6. The Women's Health Initiative Steering Committee. *JAMA* 2004; 291:1701-12.
7. Rossouw JE, Prentice RL, et al. *JAMA* 2007; 297:1465-77.
8. Ockene JK, Barad DH, et al. JAMA 2005; 294:183-93.
9. Haimov-Kochman R, Barak-Glantz E, et al. *Menopause* 2006; 13: 370-6.
10. Freedman RR, Woodward S. *Am J Obstet Gynecol* 1992;167:436-9.
11. Aiello EJ, Yasui Y, et al. *Menopause* 2004; 11:382–8.
12. Whiteman MK, Staropoli CA, et al. *Obstet Gynecol* 2003; 101:264–72.
13. Newton, KM, Buist, DS, et al. *Obstet Gynecol* 2002; 100:18.
14. Barton DL, Loprinzi CL, et al. *J Clin Oncol* 1998;16:495-500.
15. Nelson HD, Vesco KK, et al. *JAMA* 2006; 295: 2057 - 71.
16. Newton KM, Reed SD, et al. *Ann Intern Med* 2006; 145: 869-79.
17. Laufer LR, Erlik Y, et al. *Obstet Gynecol* 1982; 60:583-6.
18. Nagamani M, Kelver ME, Smith ER. *Am J Obstet Gynecol* 1987;156:561-5.
19. Pandya KJ, Raubertas RF, et al. *Ann Intern Med* 2000;132:788-93.
20. Stearns V, Beebe KL, et al. *JAMA* 2003; 289:2827-34.
21. Stearns V, Slack R, et al. *J Clin Oncol* 2005; 23: 6919-30.
22. Suvanto-Luukkonen E, Koivunen R, et al. *Menopause* 2005;12:18-26.
23. Loprinzi CL, Kugler JW, et al. *Lancet* 2000;356:2059-63.
24. Evans ML, Pritts E, et al. *Obstet Gynecol* 2005; 105:161-6.
25. Guttuso T Jr, Kurlan R, et al. *Obstet Gynecol* 2003;101:337-45.
26. Pandya KJ, Morrow GR, et al. *Lancet* 2005; 366:818-24.
27. Reddy SY, Warner H, et al. *Obstet Gynecol.* 2006;108:41-8.
28. Loprinzi CL, Kugler JW, et al. *J Clin Oncol* 2007; 25:308-12.

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Sexual Dysfunction After Menopause: Assessment and Treatment

Christine Duffy, MD, MPH, and Kelly A. McGarry, MD, FACP

Sexual activity generally declines with age[1,2] and between 25-63% of peri and postmenopausal women report some sexual dysfunction.[3,4,5,6,7] This wide variability has been attributed to methodology: differences in the questionnaires, the populations studied, and the time period studied.[8] However, even low estimates indicate that this is a common complaint in the menopausal woman.

Many physicians do not bring up issues related to sexual satisfaction, fearing that they will have nothing to offer patients. The following will provide an overview of the most common sexual complaints and dysfunction in postmenopausal women as well as treatments and interventions to improve sexual functioning.

Sexual Function in Menopause

Physiology

Early in menopause, women begin to experience menstrual irregularity due to changing hormonal patterns. A declining follicle number leads to a rise in the serum FSH levels with initial preservation of estradiol secretion, but with low luteal phase progesterone concentrations. Later in menopause, fluctuations in serum FSH and estradiol concentrations can be striking; hormone levels may be in the premenopausal range confusing the clinical picture. Ultimately, when ovarian follicles are depleted, the ovary no longer secretes estradiol, but does continue to secrete androgens due to continued stimulation of LH.

These changes in hormone levels may lead to a variety of manifestations, including irregular bleeding, hot flashes, sleep disturbance, and genitourinary symptoms. Pathophysiologically, there may be thinning of the vaginal mucosal epithelium and atrophy of the vaginal wall smooth muscle, vaginal dryness and increased vaginal pH, reduction in intraurethral pressure, changes in thickness of mucous membranes, reduction in pelvic muscle tone, and an increased sensitivity to pain. While many of these physiologic changes and menopausal symptoms can have a direct effect on sexual functioning, the physiology of menopause is but one small part influencing the complex experience of sexual function.

Defining Sexual Dysfunction

Sexual *behavior* declines with age. Reasons include lack or loss of a partner, partners' sexual problems, health and physiological changes.[9] Research among populations of women does not show a clear association between menopause and a decline in sexual *functioning*. While some studies have found lower sexual functioning among peri- and postmenopausal women as compared to premenopausal women, others have found no such association.[8,10,11] Data from the population-based Massachusetts Women's Health Study concluded that menopause status, but not estrogen levels, is related to some aspects of sexual functioning (desire, interest and changes in arousal), but not others (satisfaction, frequency or difficulty reaching orgasm).[8]

Neither menopause nor age has been correlated with satisfaction of one's sexual relationship, and psychological and health factors are often reported as more important determinants of sexual functioning than ovarian function, including availability of a partner, psychological functioning, partner limitations, quality of the relationship, and previous sexual behavior and enjoyment.[3,9,11] In a secondary analysis of the **Reproductive Risk Factors for Incontinence Study at Kaiser** (**BRISK**) which included women aged 40-69, among women who were sexually active (75%), two-thirds reported being at least somewhat satisfied.[3] While changes in sexual behavior and desire may occur in postmenopausal women, those who continue to be sexually active appear to maintain sexual satisfaction and enjoyment.

DSM-IV describes 4 types of female sexual dysfunction: hypoactive sexual desire disorder, sexual aversion disorder, female sexual arousal disorder and female orgasmic disorder. In addition, two sexual pain disorders are also described: dyspareunia and vaginismus.[12] While all these disorders can occur in the menopause, hypoactive sexual desire disorder and dyspareunia are the most common complaints in the postmenopausal population and this review will focus on these disorders. It is important to remember that such classifications fail to incorporate the strong role of contextual factors in female sexuality.

Diagnosis & Evaluation/ Sexual History

Sexual History and Functioning

The first step to diagnosing sexual dysfunction is to ask about sexual functioning as part of a routine physical. Many women will not spontaneously volunteer problems, but most appreciate when their physician raises the issue. A straight-for-

Table 1. DSM-IV Sexual Disorders

- Hypoactive sexual desire disorder – persistent or recurrent deficiency or absence of sexual fantasies, thoughts and or desire for sexual activity, which causes personal distress
- Sexual arousal disorder – the persistent or recurrent inability to attain or maintain sufficient sexual excitement, which causes personal distress (subjective or somatic responses such as lubrication)
- Orgasmic Disorder – the persistent or recurrent difficulty of, delay in, or absence of attaining orgasm after sufficient sexual stimulation and arousal, which causes personal distress
- Dyspareunia – Recurrent or persistent genital pain associated with sexual intercourse
- Vaginismus – the persistent or recurrent involuntary spasm of the musculature of the outer third of the vagina that interferes with vaginal penetration and causes personal distress

Table 2. Common Classes of Drugs That Can Cause Sexual Dysfunction

- Antipsychotics
- SSRIs
- Tricyclic antidepressants
- Beta-blockers
- GnRh agonists
- Anticholinergics
- Antihistimines
- Narcotics
- Diuretic (spironilactone)
- Recreational drugs (heroin, marijuana, alcohol)

ward, non-judgmental approach is important. Sample questions (including information about same sex partnerships) include:

"Are you currently involved in a sexual relationship with men, women or both?"

"Do you have any sexual concerns that you would like to discuss?"

"Do you have difficulty with the desire to have sex, getting aroused, or having an orgasm?"

It is important to ask women whether sexual concerns or issues cause them any personal or relationship stress. DSM-IV criteria for sexual disorders include distress. Some women may not be distressed by a lack of desire, or lack of sex in their relationship.

If sexual dysfunction causes personal distress, providers should ask about onset, duration and whether it is situation-specific or global. Women should be asked about desire, arousal, orgasm, any pain related to sex, fantasy and self-stimulation. It is important to determine whether the dysfunction is partner-specific. Previous sexual satisfaction and frequency are important predictors of current and future sexual function. If a woman has never achieved orgasm, then referral to a therapist experienced in treating primary orgasmic disorders is paramount. If a woman fantasizes and engages in self-stimulation, but has little desire with her current partner, dysfunction may be primarily psychosocial and exploration of relationship factors (including sexual practices) is important. If a woman reports lack of interest because of chronic pain, then intervention would be aimed at treating her underlying pain disorder.

Medical History

The medical history should focus on identifying causative medical or gynecologic conditions, and determining the social context (relationship quality and current stressors). It is important to question women about medications that can impact desire, arousal or orgasm. (Table 2) In addition, a number of conditions result in low testosterone, including bilateral oophorectomy, hypothalamic/ pituitary/ adrenal insufficiency (including Sheehan's syndrome and Addison's disease), systemic glucocorticoid therapy, oral estrogen therapy, hyperthyroidism, and chronic illness such as diabetes, depression, and advanced cancer. Gynecologic symptoms such as dryness, discharge, or bleeding should be elicited, as well as urinary incontinence, which may be a sign of weakened pelvic floor muscles. A history of pelvic surgeries or chronic pelvic pain should be noted.

Psychosocial

Women's sexual satisfaction and functioning are intimately related to interpersonal relationships and social factors. Important psychosocial factors include availability of partner, emotional well-being, depressive symptoms, stress, expectations, partner limitations and previous sexual behavior and enjoyment. Questions regarding past sexual abuse or trauma should be prefaced by a statement indicating that all women are asked such questions. If a woman indicates such a history, referral to a therapist is warranted. Postmenopausal women are especially likely to suffer a partner loss or difficulty finding a partner. Screening for clinical depression is vital as it has a negative effect on sexual desire, beyond any dysfunction caused by SSRIs.

Physical Exam

When performing the physical exam, clinicians should note signs of underlying chronic disease that may affect sexual function, including arthritis, neuropathy, hypothyroidism, pituitary adenomas and chronic heart or lung disease. A careful gynecologic exam should be performed. For some women with a history of negative sexual experiences, the pelvic exam may cause anxiety. It is important to explain what will be done during the exam, and to indicate what is being done as the exam progresses. External genitals should be evaluated for any rashes, ulcers, or signs of atrophy and the clitoris should be examined for any signs of adhesions. Strength of the levator ani, assessment of **cervical motion tenderness** (**CMT**), and palpation of adenexa, uterus, and vaginal wall should be performed, as well as assessment for prolapse (cystocele, rectocele and uterine prolapse). Cultures and pH should be performed if women complain of any discharge, have CMT, or report unprotected sex. Typical changes associated with the menopause include a thinning of the epithelial layers within the vagina and a general decrease in elasticity. Decreased secretions and an increase in the vaginal pH may be seen. Pelvic floor strength is often decreased as well.

Labs and Diagnostic Tests

Labs and diagnostic tests targeted toward identifying reversible causes of sexual function should be obtained. These may include CBC, TSH, prolactin and pelvic US. Measuring testosterone has uncertain clinical utility since there is no clearly defined level considered deficient.[13]

Interventions for Common Sexual Complaints

Hypoactive sexual desire

Other causes of and contributors to sexual dysfunction should be sought before assuming a woman has hypoactive sexual desire. For instance, low desire may be a result of dyspareunia from vaginal atrophy, or problems within the relationship. Hypoactive sexual desire has often been attributed to low testosterone, but there is no level of testosterone that clearly relates to hypoactive sexual desire.

A recent Cochrane review of the role of testosterone for peri- and postmenopausal women concluded that there was a dearth of high quality studies, hampering the ability to provide conclusions about efficacy and safety. They did find evidence that adding testosterone to **hormone therapy** (**HT**) has a beneficial effect on sexual function (measured by a score that combined libido, sexual activity, satisfaction, pleasure, fantasy and orgasm) in postmenopausal women. The only significant adverse effect observed was a decrease in HDL. The short duration of treatment (6 months) for the studies prevents any conclusions regarding long-term use. [14]

The North American Menopause Society, in a position statement, concluded that postmenopausal women with decreased sexual desire associated with personal distress and no other identifiable cause may be candidates for testosterone therapy. However, they recommended a careful clinical evaluation before starting therapy. In addition to psychosexual and psychosocial history, a complete medical history and exam, laboratory testing such as TSH, CBC, prolactin and pelvic US should be considered to exclude other causes of low desire. [13] However, the Society of Obstetricians and Gynecology of Canada noted that testosterone therapy should be viewed as investigational and should be prescribed only by clinicians who are knowledgeable about sexual dysfunction in women.[15] In a recent review, testosterone therapy was not recommended, pending more and longer-term data.[16]

Testosterone for hypoactive sexual desire disorder is an off-label use of testosterone. There is also little data on long-term use (>6 months) and relatively few women have taken testosterone. The doses required for women are about 1/10th that of men, which makes delivering appropriate levels to women difficult. There are only two FDA approved drugs with testosterone: Estratest, a combination of estrogen and testosterone given orally, and testosterone enanthate/estradiol valerate, which is given intramuscularly. The former is FDA approved as a treatment for menopausal symptoms resistant to estrogen therapy, while the latter is FDA approved as a treatment for female metastatic breast cancer and rarely used. Estratest has the disadvantage of first-pass liver metabolism and its consequent effects on lipids (lowers HDL). Patches, commonly used in men, avoid this issue but the doses available are much too high for women and they cannot be cut or altered. Some clinicians may modify the dose of testosterone gels for off-label use in women, but it is difficult to regulate the amount of testosterone a woman receives. Compounding pharmacies can create a testosterone compound that a woman rubs into her skin daily. An appropriate dose of such a compound would be 0.5 g/d of a 1% gel cream or ointment.[13] However, there is a lack of standardization and no quality control.

If after discussing the potential risks of testosterone therapy a woman and her provider decide to pursue treatment, the best way to monitor response is to ask women about their experiences. Testosterone can be titrated up every 3 months, but women should also be monitored for signs of androgen excess, such as clitoromegaly, hirsutism, and acne. Once a woman is on a stable dose, LFTs and fasting cholesterol panel should be checked, and then rechecked every 6 months.

Dyspareunia

Dyspareunia in postmenopausal women is most often caused by vaginal changes which result from a low-estrogen state. However, other causes such as vulvodynia or vaginismus should be ruled out by history and physical. Non-hormonal options for dyspareunia include lubricants such as KY jelly or astroglide, or agents such as Replens which increase vaginal moisture. They can cause irritation in some women, but are generally well-tolerated.

A Cochrane review of the use of local estrogen therapy for vaginal atrophy found that estrogen decreased vaginal dryness. [17] All forms (cream, tablets, ring) were effective in relieving the symptoms; however, the cream was associated with more uterine bleeding, breast pain and perineal pain, and greater endometrial stimulation. Women tended to favor the estrogen ring for overall ease of use, comfort and overall satisfaction. Although endometrial stimulation from unopposed estrogen is a theoretical risk with any of these therapies, the Cochrane review concluded that only women receiving estrogen creams in doses above 0.5mg daily required progesterone protection. Clearly any woman receiving any intra-vaginal estrogenic preparations who has bleeding should have endometrial investigation.

Table 3. Use of Testosterone for Hypoactive Sexual Desire

Testosterone:

Careful clinical evaluation before starting therapy including:

- psychosexual and psychosocial history
- complete medical history including medications that may impact sexual functioning
- physical exam
- laboratory testing such as TSH, CBC, prolactin and pelvic US
- Free testosterone index (ratio of total testosterone to SHBG) or the Sodergard equation if lab can perform

At baseline and 3 months, then every 6 months:

- Serum lipids
- Liver function tests
- Free testosterone index (not dose adjustment but for supra-physiologic levels)

Therapeutic monitoring:

Subjective assessments of sexual response, desire and satisfaction, twice yearly CBE and yearly mammogram and physical exam

Testosterone products used for sexual dysfunction in women:[13]

Estratest (sometimes used for treatment of sexual dysfunction)

- Estratest HS (0.625 mg esterified estrogen/1.25 methyltestosterone), orally 1/day
- Estratest (0.625 mg esterified estrogen/2.5 methyltestosterone), orally 1/day

Approved in Canada, not US for treatment of menopausal symptoms:

- Climacteron 150mg/mL testosterone enanthate, 7.5 mg/mL estradiol dienanthate,
- 1mg/ML estradiol benzonate; 0.5 – 1.0 mL intramuscularly q4-6 weeks

Testosterone cream or ointment from compounding pharmacy (not FDA approved):

- Appropriate dose of such a compound would be 0.5 g/d of a 1% gel cream or ointment (delivers ~ 5mg/testosterone/day)

** testosterone patches should not be used to treat women

Table 4. When to consider referral to therapist

- History of sexual abuse
- Domestic violence
- Significant marital discord
- Previous sexual dysfunction or poor sexual functioning
- When patient is not responding to treatment
- If patient requests

SSRI-related sexual dysfunction

While not exclusive to postmenopausal women, the high prevalence of depression in women in general means that many patients will be on **selective serotonin reuptake inhibitors (SSRIs)**. Estimates of sexual dysfunction vary considerably, but range from 20-60%. Difficulty reaching orgasm or anorgasmia and lack of desire is most common. In a recent Cochrane review of the treatment of antidepressant-related sexual dysfunction, no clear successful treatment emerged. [18] There was some evidence that changing to non-SSRI antidepressants such as bupropion, trazodone or mirtazapine might be helpful, but no definitive conclusions could be made. Since there may be individual differences in side effects to particular SSRIs, it also may be reasonable to switch to a different SSRI.

General measures

General strategies for improving sexual function include elimination of routine, changing time, position or place, setting time aside for sexual activity, encouraging masturbation to help determine what sensations are pleasurable, kegel exercises to improve pelvic tone, sensual massage, and oral stimulation. If anxiety appears to be playing a central role, then relaxation techniques can be helpful such as a warm bath before sex, soothing music, and progressive relaxation. Referral to therapists who specialize in sexual dysfunction may be helpful as well. Sex therapists usually use a combination of cognitive behavioral approaches, including sensate focus techniques that begin with non-sexual touch, with gradual progression toward sexual touch, with the couple providing feedback about what is pleasurable. Indications for referral to a sex therapist, couples or general therapist are listed in Table 4.

Conclusions

Sexual dysfunction is a common complaint among postmenopausal women when they are asked about their sexual life. A careful history and examination are key to identifying the causes, paying particular attention to relationships and stressors. Once the problem is identified, there are psychosocial and medical interventions available to assist postmenopausal women in improving their sexual functioning. The first step is becoming comfortable asking about these issues in the primary care setting.

References

1. Dennerstein L, Burrows GD. *Clin Enocrinol Metab* 1982;11:661-79.
2. Cain VA, Johannes CB, et al*J Sex Res* 2003; 40:266-76.
3. Addis IB, Van Den Eeden SK, et al. *Obstet Gynecol* 2006;107:755-64.
4. Frank E, Anderson C, Rubenstein D. *NEJM* 1978;299:111-5.
5. Spector IP, Carey MP. *Arch Sexual Behavior* 1990; 19:389-408.
6. Rosen RC, Taylor JF, et al. *J Sex Marital Therapy* 1993;19:171-88.
7. Laumann EO, Paik A, Rosen RC. *JAMA* 1999;281:537-44.
8. Avis NE, Stellato R,, et al. *Menopause* 2000;7:297-309.
9. Avis NE. *J Gender Specific Med* 2000;3:37-41.
10. Dennerstein L, Smith AMA, et al. *J Psychosom Obstet Gynecol* 1994;15:59-66.
11. Hawton K, Gath D, Day A. *Arch Sex Behav* 1994;23:375-95.
12. American Psychiatric Association. *Diagnostic and statistical manual of mental disorders* (4th ed., text revision). Washington, DC: American Psychiatric Association, 2000.
13. North American Menopause Society *Menopause* 2005; 12:497-511.
14. Somboonporn W, Davis S, et al. Testosterone for peri- and postmenopausal women. Cochrane Database of Systematic reviews 2005, Issue 4. Art. No. CD004509. DOI:10.1002/14651858.CD004509.pub2.
15. Blake J, Belise S, et al. *J Obstet Gynecol Can* 2006;28:Suppl:1-92.
16. Basson R. *NEJM* 2006;354:1497-506.
17. Suckling J, Lethaby A, Kennedy R. Local estrogen for vaginal atrophy in postmenopausal women. Cochrane database of Systematic reviews 2006, Issue 4. Art. No: CD001500. DOI:10.1002/14651858.CD001500.pub2.
18. Rudkin L, Taylor MJ, Hawton, H. Strategies for managing sexual dysfunction induced by antidepressant medication. *Cochrane Database of Systematic Reviews* 2004, Issue 4. Art. No.: CD003382. DOI: 10.1002/14651858.CD003382.pub2.

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Menopause and Mood

Kelly A. McGarry, MD, and Carol Landau, PhD

Depressed mood is a frequent complaint of midlife women. Women are twice as likely as men to develop a depressive disorder, with higher lifetime rates of major depression (21 vs. 12.7%).[1] Women who seek healthcare have even higher rates of depression. Given that mood disorders are so prevalent throughout the lifecycle, controversy exists as to whether there is something unique about the menopausal transition, which further predisposes women to mood alterations. Regardless of etiology, it is imperative that health care professionals identify mood disorders and incorporate the biopsychosocial approach to treatment. Under- treatment exists despite excellent tools for diagnosis and effective treatments.

The Scope of Depressive Disorders

Not all women who report changes in mood meet criteria for a mood disorder. It is important to distinguish **major depressive disorder** (**MDD**) and dysthymia from mood changes unlikely to require treatment, such as reactions to an acute, but transient life stressor. Women experiencing a stressful life event will benefit from support and possibly counseling, but are unlikely to require medications, psychiatric intervention, or long-term psychotherapy

Screening and Assessment

The **US Preventive Services Task Force** (**USPSTF**) recommends screening for depression.[2] Simplified Screening tools can be easily adapted for the primary care setting, including the **Beck Depression Inventory** (**BDI**) and the **Center for Epidemiologic Study Depression Scale** (**CES-D**). However, two questions may be as effective: "Over the past two weeks have you felt down, depressed or hopeless?" and "Over the past two weeks have you felt little interest or pleasure in doing things?" Answering "yes" to either question was found to have a sensitivity of 96% and a specificity of 57% for the diagnosis of depression.[3] Patients with a positive response to either question should undergo a full diagnostic interview to assess whether they meet the DSM IV R criteria for depression.

Defining Menopause

Menopause refers to the cessation of menstrual periods due to declining estrogen and progesterone production by the ovaries. Women must be free of menses for one year to be "postmenopausal." Before the complete cessation of menses, the pattern of menstrual cycles, including duration, frequency, and amount of bleeding, becomes less predictable. According to the World Health Organization, "perimenopause" refers to this period of time prior to menopause and includes the first year after the final menstrual period. The hormonal changes and symptoms during the menopausal transition may last several years.[4] Symptoms which result from changing hormone levels include: hot flashes, vaginal symptoms and urinary incontinence. These can lead to sleep disturbances, poor concentration, and decreased libido, possibly contributing to and confounding a diagnosis of depression.

Does Menopause Affect Mood?

Patients approaching menopause should be counseled that natural menopause is just that, "natural," and does not usually lead to a mood disorder. Longitudinal studies do suggest, however, that certain groups of women may be more vulnerable to mood disturbances during this transition. The Massachusetts Women's Health Study found that women with a history of depression or Premenstrual Dysphoric Disorder appear to be at greater risk for a recurrence of depressive symptoms around menopause.[5] Additionally, women suffering from untreated vasomotor symptoms are more likely to report being depressed. Epidemiological studies consistently reveal that women with high levels of stress, especially family or medical problems, are at increased risk as well.

Recent data suggest that mood disorders around menopause are often multifactorial, and support the idea that:

1) Menopausal symptoms may be causative;
2) Declining hormones may be causative;
3) Psychosocial issues/stressors may be contributory.

Table 1. Symptoms of Major Depressive Disorder

At least five of the following symptoms during the same two-week period, and is this a change from usual functioning?

1. depressed mood most of the day, nearly every day or
2. markedly diminished interest or pleasure in all, or almost all activities most of the day, nearly every day and
3. significant weight loss or weight gain when not dieting (e.g., more than 5 percent of body weight in a month), or decrease or increase in appetite nearly every day.
4. insomnia or hypersomnia nearly every day.
5. psychomotor agitation or retardation nearly every day.
6. fatigue or loss of energy nearly every day.
7. feelings of worthlessness or excessive or inappropriate guilt nearly every day.
8. diminished ability to think or concentrate, or indecisiveness, nearly every day.
9. recurrent thoughts of death (not just fear of dying), recurrent suicidal ideation without a specific plan, or a suicide attempt or a specific plan.

*If answered YES to 1 or 2 and YES to four items from 3–9, this indicates a major depressive episode.

Adapted from the *Diagnostic and Statistical Manual of Mental Disorders*, Fourth Edition, 1994.

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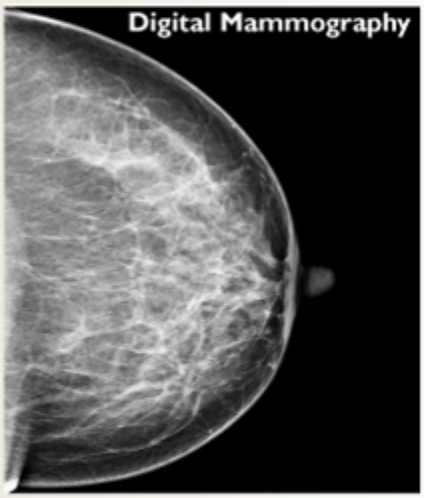

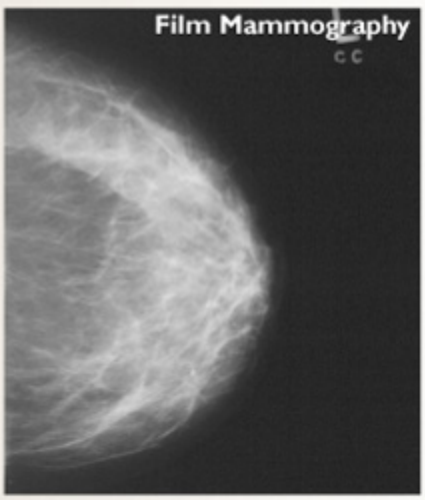

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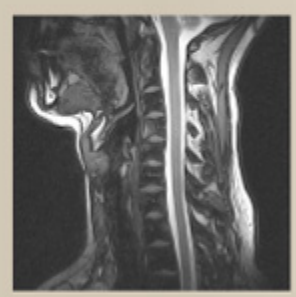

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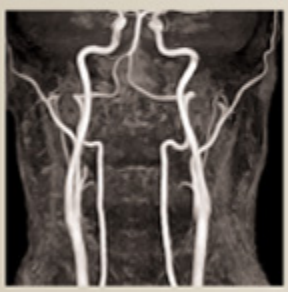
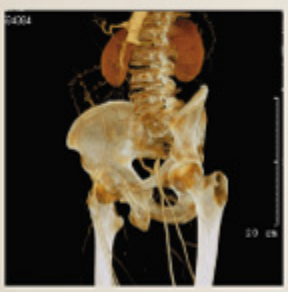
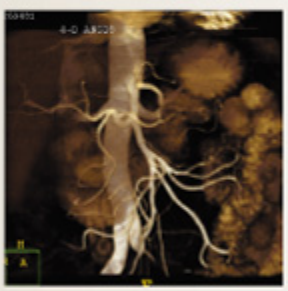
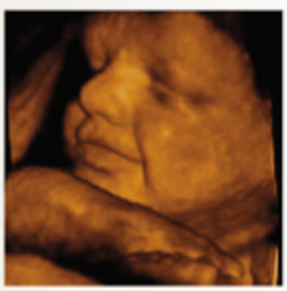




CT and 3D CT



CTA



3D Ultrasound

WARWICK
250 Toll Gate Rd.
401.921.2900

CRANSTON
1301 Reservoir Ave.
401.490.0040

CRANSTON (annex)
1500 Pontiac Ave.
401.228.7901

N. PROVIDENCE
1500 Mineral Spring
401.533.9300

E. PROVIDENCE
450 Vets. Mem. Pkwy. #8
401.431.0080

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Table 2. Symptoms of Dysthymic Disorder

1. Depressed mood for most of the day, more days than not, for at least two years?
2. At least *two* of the following?
   1) poor appetite or overeating
   2) insomnia or hypersomnia
   3) low energy or fatigue
   4) low self-esteem
   5) poor concentration or difficulty making decisions
   6) feelings of hopelessness
3. No manic episode–overly high amounts of energy, poor judgment.
4. No medications that can cause depression.

Adapted from the *Diagnostic and Statistical Manual of Mental Disorders*, Fourth Edition, 1994.

Vasomotor symptoms, such as hot flashes and night sweats, are correlated with negative mood.[6] A subset of the **Study of Women's Health Across the Nation (SWAN)** examined the association between psychologic distress and natural menopause in a community sample of ethnically diverse women. The relationship between sleep problems and distress was striking; nearly 40% of women with sleep problems compared with 15% of women without sleep problems, reported psychologic distress. Further, nearly 37% of women with vasomotor symptoms reported distress compared with 18% of women without vasomotor symptoms. Rates of psychologic distress were highest in early menopause (28.9%) and lowest in premenopause (20.9%) and postmenopause (22%) even after adjustment for potential confounders, including vasomotor and sleep symptoms.[7] These findings suggest that vasomotor and sleep problems do not fully explain the relationship between menopausal status and distress and that menopausal status is an independent contributor to psychologic distress.

While it is not fully understood how estrogen exerts its function in the brain, it is known that estrogens exert a positive effect on serotonergic activity and influence dopaminergic and norepinephrine activity. It is likely that estrogen enhances mood through these actions. Hormone changes may cause mood alterations in some women, although no studies have consistently demonstrated hormone levels that can predict which women will experience depression. Thus, there is no role for testing hormone levels in depressed menopausal women. Three double-blind, placebo-controlled trials using similar methodologies and identical estrogen preparations have evaluated the efficacy of estrogen treatment in peri- and postmenopausal women who met criteria for depression.[8,9,10] Two out of three studies examined perimenopausal women and found significant clinical responses in both major and minor depression in those treated with estradiol. One study showed a full or partial therapeutic response in 80% of subjects treated with estradiol compared with 22% of those receiving placebo.[8] The benefits were observed in women with and without hot flashes, reinforcing the idea that the effect of estrogen on depression is not mediated solely through its ability to reduce hot flashes. In contrast, a study of estradiol therapy in women 5 to 10 years postmenopausal demonstrated no improvement in depression symptoms, suggesting that the hormonal changes at perimenopause may alter mood.[10]

Unique psychosocial issues/stressors arise for women in mid-life, which may predispose them to increased rates of depression. The negative effect of stress on mood is well-documented.[11] Stressors may include an individual's or partner's health problems, marital dissatisfaction, family difficulties and social isolation. Additionally, economic concerns, including the stressors of forced retirement and financial assistance for adult children, elderly parents, or both, often arise at this time. Finally, many midlife women find access to health care problematic. Importantly, social support can buffer the effect of stress.[15]

Treatment

Most women will go through menopause without significant impairment in their mood. However, as health care professionals, we are likely under- treating many women who may benefit from psychotherapy or medical therapy for mood disturbances around menopause. Some women feel that significant mood disturbances, from transient distress to a major depressive episode, are a natural part of menopause and "something they have to get through." Therefore, they do not discuss these symptoms with providers. Additionally, in the busy clinical setting, health care providers may not be asking and, similar to patients, may not appreciate the need for intervention.

Should we limit treatment to women with major depression? Individuals with depressive and anxiety symptoms, but not meeting DSM-IV criteria for specific disorders, may experience functional impairment, including lost work and disability. Additionally, subclinical symptoms are associated with an increased risk of future clinical episodes.[13] Treatment of subclinical symptoms may prevent escalation of the symptoms into a major depressive episode. Thus, limiting treatment to women who meet criteria for major depression may result in providers missing the opportunity to help women experiencing significant psychologic distress.

Treatment options include counseling and lifestyle modification, as well as traditional and complementary medications. It is important to determine contributing factors for depressed mood in each patient. For those plagued by symptoms of menopause, therapies targeting the specific symptoms may be the first priority. Dr. Tong reviews these therapies in this issue. When life stressors are the prime reason for the mood disturbance, psychotherapy is of paramount importance. Two forms of psychotherapy have been documented as effective treatment for depression.[14] **Cognitive Behavioral Therapy (CBT)** is an active focused process that targets negative thoughts and predictions and increased assertiveness. **Interpersonal therapy (IPT)**, also short-term and active, focuses on improving a woman's social relationships with respect to conflict resolution, loss and social role change. Therapists often use a combination of modalities.

Even though declining hormone levels may play a direct role in mood alterations and evidence suggests that estrogen replacement may improve mood, **hormone replacement therapy (HRT)** is not recommended as the firs- line treatment for depressed mood in menopause, given the risks documented in the Women's Health Initiative. If, however, severe hot flashes are impacting a women's mood and **selective serotonin reuptake inhibitors (SSRIs)** have not resulted in significant improvement, then the lowest dose of estrogen used for the shortest amount of time possible to control symptoms may be used.

For many women, multiple factors may affect mood and a combination of approaches may be necessary. Additionally, severity of symptoms may dictate the intervention. Mild to moderate depression responds equally well to either medication or psychotherapy.[15] Patients with severe depression benefit more from medications or from combined medication/therapy than from psychotherapy alone. Providers can use the self-administered 9-item Patient Health Questionnaire to assess the severity of depression.[16] In addition to severity of symptoms, patients may have individual treatment preferences. Finally, medications may help target both depression and menopausal symptoms. For example, one woman may desire to try a combination of psychotherapy for coping with her life stressors and an SSRI or SNRI for her hot flashes. Another woman may require an SSRI or SNRI for hot flashes and a hypnotic for her sleep disturbance.

Studies examining depression treatment in menopausal women have demonstrated treatment efficacy similar to studies of depression in the general population.[17] The duration of antidepressant therapy has not been specifically investigated in menopausal women, however, antidepressant therapy should probably be continued for 6 to 12 months after clinical symptoms have improved to reduce the risk of relapse. Antidepressants are associated with adverse effects including sexual dysfunction, insomnia, headache and gastrointestinal symptoms. The complementary medicine, St. John's wort, may benefit women with subsyndromal depression or those who do not wish to take conventional therapy for mild depression.[18] It has not been shown to benefit women with moderate to severe depressive symptoms and should not be prescribed to these women. All women with mood changes around menopause should be prescribed exercise if they do not already engage in routine exercise. Mood has been shown to be improved with regular exercise.[19] Women should be advised to engage in an aerobic activity they enjoy for at least 30 minutes most days of the week.

Most women will go through menopause without significant impairment in their mood.

Conclusion

Many women will not experience any mood disturbance in the perimenopausal or menopausal period. This can be a reassuring fact to share with premenopausal women. However, for those who seem vulnerable to adverse changes in mood around menopause, there is much to offer. It is paramount that providers identify these women in order to institute appropriate treatment. Mood disturbances are usually multifactorial and may relate to life stressors, menopausal symptoms, and/or changes in hormone levels. As such, appropriate treatments may include a combination of psychotherapy, medication therapy and lifestyle modification. Identifying and treating mood disorders associated with menopause will enhance the quality of life of our patients.

References

1. Kessler RC, McGonagle KA, et al.. *Arch Gen Psychiatry* 1994;51:8-19.
2. Pignone MP, Gaynes BN, et al. *Ann Intern Med* 2002 136:765-76.
3. Mulrow CD, Williams JW Jr. *Ann Intern Med* 1995;122:913-21.
4. NIH State-of-the-Science Panel. *Ann Intern Med* 2005;142(12 part 1):1003-13.
5. Avis NE, et al. *Ann Epidemiol* 1994;4:214-20.
6. Juang KD, Wang SJ, et al*Maturitas* 2005;52:119-26.
7. Bromberger JT, Meyer PM, et al. *Am J Pub Health* 2001;91:1435-42.
8. Schmidt PJ, Nieman L, et al. *Am J Obstet Gynecol* 2000;183:414-20.
9. Soares CN, Almeida OP, et al *Arch Gen Psychiatry* 2001;58:529-34.
10. Morrison MF, Kallan MJ, et al. *Biol Psychiatry* 2004;55:406-12.
11. Kawachi I, Berkman LF. *J Urban Health* 2001;78:458-67.
12. Phongsavan P, Chey T, et al. *Soc Sci Med* 2006;63:2546-61.
13. Broadhead WE, et al. *JAMA* 1990;264:2524-8.
14. Fava GA, et al. *Am J Psychiatr* 1998;155:1443-5.
15. Thase ME, Greenhouse JB, et al. *Arch Gen Psych* 1997;54:989-91.
16. Lowe B, Unutzer J, et al. *Med Care* 2004;42:1194-201.
17. Joffe H, Soares CN, Cohen LS. *Psychiatr Clin North Am* 2003;26:563-80.
18. Geller SE, Studee L. *Menopause* 2007;14:541-9.
19. Brosse AL, Sheets ES, et al. *Sports Med* 2002;32:741-60.

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Diabetes Mortality In Rhode Island:
Comparing Underlying Cause of Death versus Any Listed Cause of Death

Lauren M. Wier and Annie Gjelsvik, PhD

In 2005, an estimated 20.8 million people in the United States had diabetes and nearly 30% of these cases were undiagnosed.[1] The age-adjusted mortality of individuals with diabetes is approximately twice that of non-diabetic individuals.[2] In 2005, diabetes was listed as the sixth leading cause of death in the United States.[3] National cause of death statistics have historically been based on the underlying cause of death listed on the death certificate, where the underlying cause is defined as "the disease or injury that initiated the train of events leading directly to death." This method of reporting mortality statistics does not account for other conditions mentioned on the death certificate described as "the disease or condition contributing but not leading directly to death."[4] Studies have found that only 30 to 40 % of decedents with diabetes have diabetes listed anywhere on the death certificate[5-7] and only about 10 to 15% had it listed as the underlying cause of death.[5,8]

Diabetes is a well-established risk factor for the development of serious complications, such as cardiovascular disease, which may ultimately be coded as the underlying cause of death; consequently, reporting only underlying cause of death data may substantially underestimate the death burden of diabetes. Using any listed cause of death, defined as the disease or condition mentioned as underlying or attributing cause of death, reported on death certificates may more appropriately capture mortality attributable to diabetes. This report compares the number and rates of deaths in Rhode Island with diabetes coded as the underlying cause of death versus deaths coded with diabetes as any listed cause of death.

Methods

The Office of Vital Records of the Rhode Island Department of Health provided an analytic file of death certificate data for state residents from 1995 through 2005 for this study. Diabetes was coded as the underlying cause of death if it was listed as such on the death certificate. Diabetes was coded as any listed cause if diabetes was mentioned on the death certificate as either the underlying or a contributing cause of death.

Numbers and rates of deaths with diabetes coded as the underlying cause and as any listed cause were compared based on age, gender, race, and Hispanic origin. (Race and Hispanic origin data were available in comparable format only for the years 2003-2005). Denominators were obtained from the 2000 US census data; for analyses by demographic characteristics, the Census denominators were adjusted to produce estimates of the number of diabetics through weighting with three-year age-, race-, and sex- specific average prevalence rates for diabetes from the Rhode Island **Behavioral Risk Factor Surveillance System (BRFSS)**. Death rates were age adjusted to the 2000 United States Standard Population using age groups 0-44, 45-64 and 65+. For each rate, the 95% **confidence interval (CI)** was calculated in order to test for statistically significant differences between the two types of rates and between groups.

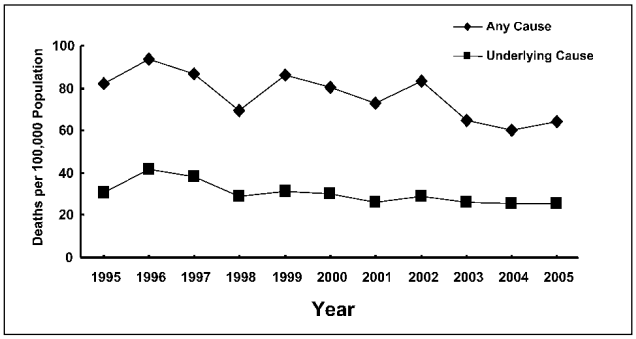

Figure 1: Age-adjusted diabetes mortality rates per 100,000 population, Rhode Island, 1995-2005.

Results

From 1995 to 2005, 2,657 diabetes deaths were identified with diabetes mentioned as underlying cause of death and 7,360 diabetes deaths were identified with diabetes mentioned as any listed cause of death. The average age-adjusted diabetes death rate per 100,000 Rhode Island residents was 30.2 (95% CI: 28.3-32.1) for diabetes mentioned as underlying cause and 76.8 (95% CI: 74.3-79.3) for diabetes mentioned as any listed cause of death. (Table 1) Between 1995 and 2005, the annual mortality rate in Rhode Island from diabetes as the underlying cause of death decreased slightly; the annual mortality rate from diabetes as any listed cause of death declined variably over time. (Figure 1)

Over the eleven-year period 1995 to 2005, older age group was positively correlated with mortality, with individuals aged 18-44 having the lowest mortality rates per 10,000 diabetics and individuals aged 65+ having the highest rates, regardless of diabetes cause of death coding. (Figure 2. Death rates for the age group 0-17 are not presented due to small numbers.) By gender, the average age-adjusted mortality rate was slightly higher for both underlying and any listed cause for

Table 1: Average Annual Age-Adjusted* Death Rate for Diabetes as Underlying Cause of Death and as Any Listed Cause of Death, Rhode Island, 1995-2005.

Diabetes Cause of Death Code	Death Rate per 100,000	95% Confidence Interval
Underlying	30.19	28.27, 32.12
Any Listed	76.80	74.31, 79.28

*Age-adjusted to 2000 US standard population

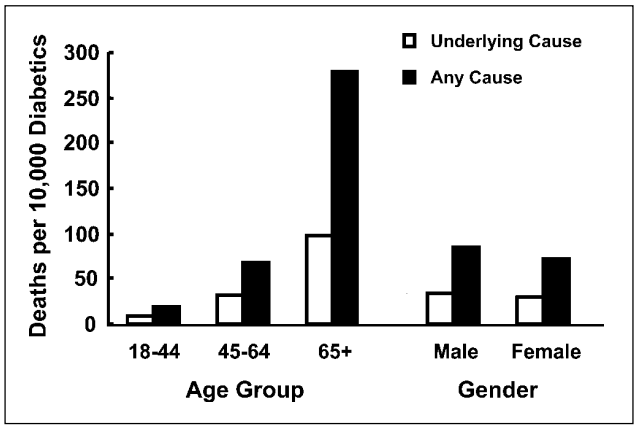

Figure 2. Average annual diabetes mortality rates per 10,000 diabetic population, by age group and gender (age-adjusted), Rhode Island, 1995-2005.

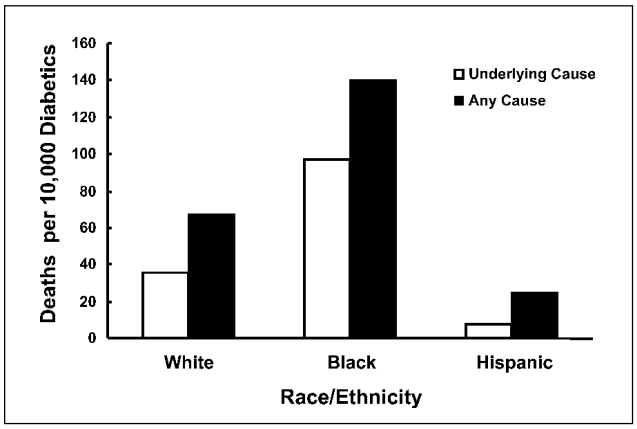

Figure 3. Average annual age-adjusted diabetes mortality rates per 10,000 diabetic population, by race and ethnicity, Rhode Island, 2003-2005.

males (33.0 and 84.4, respectively) than for females (28.9 and 71.5, respectively), with any listed cause greater for both genders. (Figure 2) With respect to race, from 2003 to 2005, Black diabetics had the highest average annual age-adjusted mortality rate from diabetes, while rates for White and Hispanic diabetics were substantially lower. Using underlying cause of death underestimated diabetes deaths in all races, most notably in Hispanics, in which about 3.5 times more diabetes deaths were captured with diabetes coded as any listed cause of death than with diabetes as the underlying cause of death. (Figure 3)

Discussion

This report indicates that use of underlying cause of death data leads to substantial underreporting of mortality in Rhode Island related to diabetes. About 2.5 times more diabetes deaths were captured with diabetes mentioned as any listed cause of death than with diabetes listed as the underlying cause of death. This finding is consistent with numbers reported elsewhere.[5-8]

As expected, since chronic disease mortality increases with age, diabetic residents aged 65+ had the highest rates of diabetes deaths compared to diabetics in other age groups. Among all age groups, classifying diabetes deaths only by underlying cause underestimated death rates, most notably in the 65+ age group. The average annual mortality rate for diabetes was slightly higher for males with diabetes than for females for both any and underlying cause. Reporting based only on underlying cause of death underestimated diabetes deaths similarly in both genders.

From 2003 to 2005, Blacks had the highest average annual mortality rate from diabetes mentioned as the underlying or any listed cause of death, whites had the second highest rate, and mortality rates from diabetes among Hispanics were lowest. Underlying cause of death underestimated diabetes deaths in all races, most notably in Hispanics, in which about 3.5 times more diabetes deaths were captured with diabetes coded as any listed cause of death than underlying cause of death. (Figure 4)

Including deaths where diabetes is identified as a contributing cause increases the number of diabetes–related deaths in Rhode Island during 1995-2005 by 4,703, or an average of 428 deaths per year, an increase of 177% relative to the number of deaths with diabetes identified as the underlying cause. These deaths occur among all demographic groups based on age, gender, race and ethnicity. These findings strongly support the conclusion that reporting diabetes mortality statistics with diabetes mentioned as any listed cause rather than based on underlying cause of death would more accurately and appropriately illustrate the extent of diabetes death burden in Rhode Island.

References

1. National Institute of Diabetes and Digestive and Kidney Diseases. National Diabetes Statistics fact sheet: general information and national estimates on diabetes in the United States, 2005. Bethesda, MD: US Department of Health and Human Services, National Institute of Health, 2005.
2. Gu K, Cowie CC, Harris MI. Mortality in adults with and without diabetes in a national cohort of the US population, 1971-1993. *Diabetes Care* 1998;21:1138-45.
3. Kung HC, Hoyert DL, et al. Deaths: Final data for 2005. *National vital statistics reports*; vol 56 no 10. Hyattsville, MD: National Center for Health Statistics. 2008.
4. National Center for Health Statistics. NCHS Definitions: cause-of-death. Available at: http://www.cdc.gov/nchs/datawh/nchsdefs/list.htm. Accessed August 1, 2007.
5. Bild DE, Stevenson JM. Frequency of recording of diabetes on US death certificates. *J Clin Epidemiol* 1992;54:275-81.
6. Will JC, Vinicor F, Stevenson J. Recording of diabetes on death certificates. *J Clin Epidemiol* 2001;54:239-44.
7. McEwen LN, Kim C, et al. Diabetes reporting as a cause of death. *Diabetes Care* 2006;29:247-53.
8. Geiss LS, Herma WH, Smith PJ. Mortality in non-insulin dependent diabetes. In: Harris MI, Cowie CC, et al, eds. Diabetes in America. 2nd ed. Washington, DC: US Government Printing Office; 1995: 233-57.

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Biomonitoring In Rhode Island

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Biomonitoring is the direct measurement of environmental toxins found in human specimens. Measurement of these toxins in human blood (including cord blood collected at the time of birth), saliva, urine, and hair may have a direct influence on public health standards and may result in improved regulation of toxic chemicals. A local application of biomonitoring exists in a pilot project involving the measurement of mercury, lead and cadmium in human cord blood. This on-going project is a cooperative effort between the Rhode Island Department of Health (the State Health Laboratories and the Office of Environmental Health Risk Assessment) and Memorial Hospital of Rhode Island's Departments of Family Medicine and Obstetrics.

Clinicians at Memorial Hospital, with the mother's consent, are collecting cord blood at the time of vaginal delivery or cesarean section and sending specimens to the laboratory at the Rhode Island Department of Health for toxin measurement. This process is extremely non-invasive, yet provides accurate information about a mother's (and indirectly, a fetus's) exposure to mercury, lead and/or cadmium. Each of these chemicals is potentially dangerous, yet has different effects on our health.

MERCURY

Mercury exists in several forms, each of which differs in bioavailability and toxicity. Inorganic mercury (I-Hg) is derived mainly from dental amalgams, where the fetus is potentially exposed to the toxin through maternal mastication. Other sources of environmental exposure to mercury include that released from broken thermometers, button cell batteries, electrical switches, and antiseptics. Until substantially elevated, I-Hg is excreted into the urine.

When inorganic mercury is discharged into lakes, rivers, oceans, and the air, microorganisms convert it to methylmercury (MeHg), the most toxic form of mercury. The primary route of exposure to methylmercury is through the consumption of contaminated fish and other seafood. In contrast to I-Hg, MeHg accumulates in erythrocytes at a wide range of exposure levels.[1,2] Ingested MeHg is almost completely absorbed and crosses the placenta and blood-brain barrier. Pregnant women who consume large amounts of fish, especially lipophilic species such as tuna, may expose the fetus to MeHg.

The concern about MeHg toxicity was initially based on several international health disasters. In the 1950s, the Japanese community of Minamata was exposed to fish contaminated with high levels of MeHg from an industrial source, and significant negative health outcomes such as growth delay and neurological compromise were noted. A similar episode followed in the 1970s in Iraq, where people consumed bread that contained a fungicide that had high levels of MeHg. Additionally, in the 1980s, miners in Tagum were exposed to high levels of mercury during their ore processing, which included an amalgam containing a mercury mixture. These three cases demonstrate that high levels of MeHg are neurotoxic, causing seizures, microcephaly, mental retardation, and cerebral palsy, especially in the prenatal period.[3,4,5,6] The susceptibility of the infant's developing brain to MeHg is due to the ability of lipophilic MeHg to cross the placenta and concentrate in the central nervous system, where it can inhibit developmental functions such as neuronal cell division and migration.[7]

These disasters provoked further research into common forms of MeHg exposure, such as fish consumption. In two studies conducted in the Faeroe Islands[8] and New Zealand,[9,10] prenatal exposure to MeHg from seafood consumption showed adverse neurological effects in children. A study in Poland suggested that cord and maternal blood mercury levels are associated with delays in psychomotor development as measured by the Bayley Scales of Infant Development in the first year of life.[11] Other studies, such as the Tagum study mentioned earlier, concluded that cord blood positive for Hg was correlated to smaller head circumference and increased risk of meconium stained amniotic fluid.[12] A second Tagum study, which included follow-up information at two years of age, suggested that prenatal Hg exposure correlated with lower neurodevelopmental screening, especially in the linguistic pathway.[13]

Not all studies reported negative outcomes associated with mercury exposure. A study in Seychelles suggested that no adverse developmental effects were found in children up to 9 years old following prenatal MeHg exposure.[14] Because of this conflicting evidence, a meta-analysis was conducted by the National Academy of Sciences in 2000. This review concluded that there is strong evidence for the fetal neurotoxicity of methyl mercury, even at low levels of exposure.[15]

LEAD

In the US, lead is found in lead-based paint and in soil contaminated with emissions from cars that burn leaded gas. Although lead was banned from paint in 1978 and phased out of gasoline in the 1970s, the lead from these products remains in the environment. Other sources of lead include drinking water (if the pipes contain lead solder,) foods stored in lead-glazed pottery, industrial emissions, various consumer products, and folk remedies. Despite public awareness and changes in federal regulations regarding removal of lead paint, dust from deteriorating lead paint is the most common source of lead poisoning.

While blood lead levels have decreased dramatically in the past decade, estimates show that 1.7 million children between the ages of 1 and 5 in the United States still have el-

evated blood lead levels.[16] Lead exposure can have a variety of effects including cognitive impairment, anemia, interstitial nephritis, headache, muscle wasting, and vast neurological deficits including seizures, delirium, coma, and even death.

Since lead crosses the placenta, the infant blood lead concentration reflects that of maternal blood.[17] Maternal exposure to lead during gestation contributes to the infant's lead burden at birth. In pregnancy, lead exposure has been associated with diminished neurologic function, low birth weight, and premature birth.[18,19,20] Higher lead levels in cord blood are also correlated with deteriorating performance on tests of infant development up to two years of life.[21]

Cadmium

Cadmium is primarily used in the manufacturing of electrical conductors, photography, and batteries. Cadmium exposure in Asia is primarily though dietary intake of rice.[22,23] In the US, the dominant source of human exposure is from cigarette smoking, since cadmium bioaccumulates in the tobacco leaf.

Cadmium primarily affects the kidney, and can lead to irreversible glomerular and tubular damage. Urinary levels of cadmium as low as 1 mcg/gram of creatinine may be associated with subtle kidney injury and an increased risk of low bone mineral density.[24]

During pregnancy, cadmium absorption from the gastrointestinal tract is increased, though there is no significant change in urine concentrations. This may suggest that during pregnancy, more cadmium is retained in the body, [25,26] potentially making pregnant women and the fetus more susceptible to cadmium toxicity. Cadmium can accumulate in the placenta and cause changes in placental morphology,[27] which is subsequently linked to preterm labor, decreased birth weight[28], and decreased birth length.[29]

Laboratory Measurements

Biomonitoring has been made possible by recent advents in laboratory instrumentation, which permit accurate measurements of many environmental chemicals in human tissues at lower (but clinically significant) levels. In particular, a technique called ICP/MS (inductively coupled plasma/mass spectrometry) can be employed to test whole blood for low levels of toxic heavy metals such as mercury, lead and cadmium.

Cord blood measurements of these heavy metals are an important step in understanding the effects of gestational exposure. Since these heavy metals have been shown to correlate with developmental delay and/or physiologic impairment, early determination of toxin exposure will lead to earlier diagnosis and treatment.

There are few guidelines for pregnant women related to mercury, lead, and cadmium. The US Environmental Protection Agency has defined the toxic limit of maternal mercury intake as 0.1 micrograms/kilogram/day; however, the toxic limit for the fetus could be lower. While there are no clear guidelines for screening pregnant women for mercury toxicity, pregnant women are advised to limit their intake of fish and seafood that are high in mercury. Pregnant women are advised to avoid lead exposure and to conduct safe home renovations; however, lead screening guidelines are currently geared towards children under the age of six,[30] and do not include recommendations for screening the mother prenatally or the infant at the time of birth. Currently, there are no set guidelines for screening for cadmium at any age.

Our study, aimed at determining levels of these toxins in cord blood, is the first of its kind in Rhode Island. By educating all providers about the effects of these chemicals, and creating simple screening measures for women and their growing

fetuses, we may prevent the toxicity from occurring in screen positive women.

Over the coming months, partners at the Rhode Island Department of Health and Memorial Hospital of Rhode Island plan to expand the study to other hospitals, so that potentially all pregnant women in Rhode Island will be screened and treated accordingly. At this point, there is not enough evidence to suggest that screening and treatment prevent negative outcomes such as preterm birth. In further studies, we will expand our parameters to include birth outcomes such as prematurity, birth weight and head circumference in order to help delineate a relationship between these negative birth outcomes and toxin exposure.

References

1. Kershaw TG, Clarkson TW, Dhahir PH. *Arch Environ Health* 1980; 35:28-36.
2. Svensson BG, Schutz A, et al. *Sci Total Environ* 1992;126:61-74.
3. Marsh DO, Myers GJ, et al. *Ann Neurol* 1980;7:348-53.
4. Cox C, Clarkson TW, et al. *Environ Res* 1989,31:640-9.
5. Harada Y. Congenital(or fetal) Minamata disease. In: Study Group of Minamata Disease eds. *Minamata Disease*. Japan; Kumamoto University, 1968:93-118.
6. Florentine MJ, Sanfilippo DJ. *Clin Pharm* 1991,10:213-21.
7. Choi BH, Lapham LW, et al. *J Neuropathol Exp Neurol* 1978;87:719-33.
8. Grandjean P, Weihe P, et al. *Neurotoxicol Teratol* 1997;19:417-28.
9. Kjellstron T, Kennedy P, et al. Physical and mental development of children with prenatal exposure to mercury from fish.. Stage 1. Solna, Sweden: National Swedish Environmental Board Report. No 3080: 1986.
10. Kjellstron T, Kennedy P, et al. Physical and mental development of children with prenatal exposure to mercury from fish. Stage 2. Solna, Sweden: National Swedish Environmental Board Report. No 3642: 1989.
11. Jedrychowski W, Jankowski J, et al. *AEP* 2006; 16:439-47.
12. Ramirez GB, Cruz CV, et al. *Pediatrics* 2000; 106:774-81.
13. Ramirez GB, Pagulayan O, et al. *Pediatrics* 2003; 111: 289-95.
14. Davidson PW, Myers GJ, et al. *JAMA* 1998; 280:701-7.
15. Trasande L, Landrigan PJ, Schechter C. *Env Health Per* 2005; 113:590-6.
16. Pirkle JL, Brody DJ, et al. *JAMA* 1994,272:284-91.
17. Goyer RA. *Environ Health Perspect* 1990;89:101-8.
18. Andrews KW, Savitz DA, Hertz-Picciotto I. *Am J Indust Med* 1994;26:13-32.
19. Recknor JC, Reigart Ret al. *J Ped* 1997;130:123-7.
20. Dietrich KN. *Appl Toxicol* 1991; 16:17-9.
21. Bellinger D, Leviton A, et al. *NEJM* 1987;316:1037-43.
22. Ikeda M, Zhang Z-W, et al. *Sci Total Env* 249:373-384.
23. Watanbe T, Zhang Z-W, et al. *Int Arch Occup Environ Health* 73:26-34.
24. Centers for Disease Control and Prevention. The third national report on human exposure to environmental chemicals. July 2005.
25. Hernandez M, Schuhmacher M, et al. *Biol Trace Elem* 1996; 53:203-12.
26. Bhatacharyya MH, Whelton BD, Peterson DP. *Toxicol App Pharm* 1982; 66:368-75.
27. Bush, PG. Mayhew TM, et al. *Placenta* 2000; 21:247-56.
28. Salpietro CD, Gangemi S, et al. *J Perinat Med* 2002; 30:395-9.
29. Zhang YL, Zhao YC, et al. *J Env Sci Health* 2004; A39:2507-15.
30. American Academy of Pediatrics. *Pediatrics* 2005;116:1036-46.

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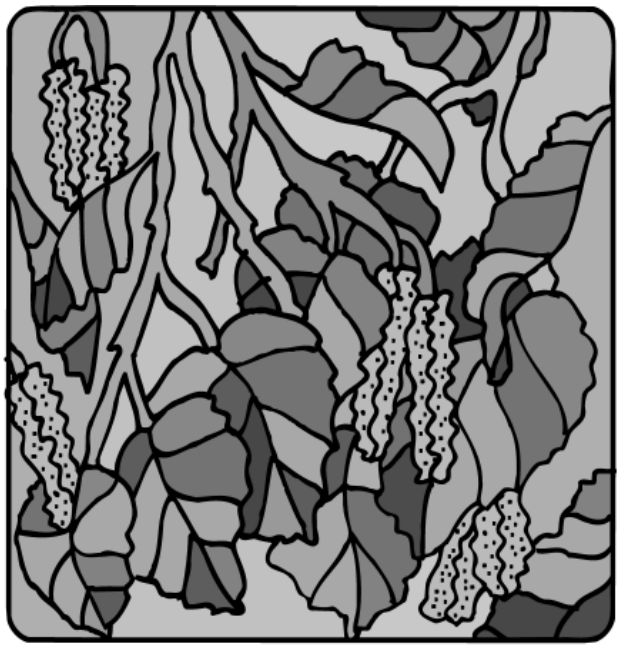

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GERIATRICS FOR THE PRACTICING PHYSICIAN

QUALITY PARTNERS OF RHODE ISLAND
Quality Partners of RI
Edited by Ana C. Tuya, MD

House Calls and Home Care

Tom J. Wachtel, MD

House calls and domiciliary visits

Introduction

A growing number of people in the United States are homebound and need in-home health care services.

"House calls" refer to the provision of physician services to patients in their homes or apartments, including independent living centers. Domiciliary visits refer to physician services provided to patients who reside in assisted living facilities, boarding houses or group homes.

Home visits may be provided as part of an interdisciplinary team or by a solo physician; they may be episodic or exist as ongoing care to patients. Furthermore, the diagnostic house call can provide information to the physician about how the patient functions within the home environment. (Table 1).

TABLE 1

Problems to Address During Geriatric Home Visit:

Safety

Access to telephone or other means of calling for help
Appliances
Smoke detector
CO monitor
Household risks (e.g. loose rugs, night lights, bathroom rails)
Yard risks (e.g. cracks in cement)
Security

Psychosocial Situation

Mental status and affect
Support (family, friends, loneliness)
Financial resources

Ethical Issues/Advance Directives including Decision to (not to) hospitalize.

Medications and Allergies

Functional Status

Activities of daily living (ADL)
Instrumental activities of daily living (IADL)
Mobility
Vision and hearing assessment
Continence (Urine and stool)

Nutrition

Ability to prepare and eat meals
Availability of food

Medical Problems

Diagnose and treat acute illness
Manage chronic conditions
Primary prevention (e.g. vaccinations, hygiene)

The actual visit[1]

The history and physical exams in the patient's home are similar to office work. In addition, permission should be requested to inspect the living quarters. Is the home clean? Is there food in the house? Can the patient get around? Is the environment safe? Are there loose rugs, nightlights, rails in the bathroom? Medications should be reviewed.

An office visit, no matter how comprehensive, cannot provide a complete understanding of the patient's daily routine.

In many situations, a family member or other caregiver should be present during the visit. When the patient's condition requires substantial nursing care, the visiting nurse should be present during some visits, enabling the team to discuss the care plan. Observing the interaction between caregivers and patients is also a valuable source of information. In the home setting, people may be more likely to display their usual patterns of interaction. In some cases (e.g. abuse or neglect), the physician may need to contact an agency that provides adult protective services.

The goals of house calls vary. A "sick" visit may simply address an acute complaint (e.g., respiratory symptoms, a fall). In the case of home-based long term care, the data described in Table 1 should be collected over time or during a comprehensive intake session; included are information on medical problems, physical function (e.g., ADL, IADL) and social and role function, such as visits by friends and relatives; and mental function, affect and advance directives. Unlike the office setting, much of this information can be collected by direct observation during a home visit.

Blood and urine tests, electrocardiograms and portable x-rays can be obtained in the home but they must be scheduled in advance and are rarely available on an emergency basis.

Logistics and Time Management

The logistics of house calls explain why many physicians, busy with their office and hospital work, find house calls inefficient. However, the physician with a substantial caseload of homebound patients can cluster visits geographically. Except for first encounters, multiple house calls can be scheduled per hour when visits are clustered.

Routine house calls can replace idle time caused by cancellations in the office, and improve efficiency. Urgent visits can be made at day's end. However, it should be made clear to homebound patients that emergencies cannot always be addressed at home, and may require hospital ED.

Payment Codes for House Calls and Domiciliary Visits (effective 1/1/2008)

The CPT codes for house calls and domiciliary visits are different. (Table 2)

Table 2

	House Call CPT	Domiciliary Visit CPT	History	Examination	Decision-making complexity
New patient-Level 1	99341	9324	Problem focused	Problem focused	Straight forward
New patient-Level 2	99342	99325	Expanded problem focused	Expanded problem focused	Low
New patient-Level 3	99343	99326	Detailed	Detailed	Moderate
New patient-Level 4	99344	99327	Comprehensive	Comprehensive	Moderate
New patient-Level 5	99345	99328	Comprehensive	Comprehensive	High
Established-Level 1	99347	99334	Problem-focused interval	Problem-focused	Straight forward
Established-Level 2	99348	99335	Expanded problem focused interval	Expanded problem focused	Low
Established-Level 3	99349	99336	Detailed	Detailed	Moderate
Established-Level 4	99350	99337	Comprehensive	Comprehensive	Moderate or high
Home care and domiciliary care oversight CPT codes applying only to patients who are **not** enrolled in a home care agency or hospice (face to face with patient is not required).					
99339: Physician supervision for 15-29 minutes per billing calendar month					
99340: Physician supervision for 30 minutes or more per billing calendar month					

Table 3. Examples of Homebound Cases According to Medicare Criteria[4]

Mobility restricted by a disease process such as unsteady gait, draining wounds, or pain.

Poor cardiac reserve, shortness of breath or activity intolerance as a result of an unstable or exacerbated disease process.

Bedridden or wheelchair bound patients who require physical assistance to move any distance.

Patients who require caregiver help with assistive devices such as walker, wheelchair or other special device to leave home.

A tracheotomy, abdominal drains, colostomy, Foley catheter or nasogastric tube that restricts ambulation.

Psychotic ideation, confusion, or impaired mental status that restricts functional abilities outside the home.

Fluctuating blood pressure or blood sugar levels that can cause syncope.

Inability to negotiate stairs or uneven surfaces without assistance of a caregiver.

Postoperative patients whose activity has been restricted by the physician.

Patients who are legally blind or cannot drive.

TABLE 4

Clinical Vignettes Related to Medicare Home Care Eligibility Criteria[4]

Vignette	Home Confined	Skilled Care needed	Meets Medicare Eligibility Criteria
Patient with unsteady gait who requires assistance for ambulation and whose blood pressure is 190/110	Yes	Yes	Yes
Patient with a dense hemiparesis who is bedridden or chair bound and who has a pressure ulcer	Yes	Yes	Yes
Patient with severe peripheral neuropathy who is blind and wheelchair-dependent for mobility and whose chronic conditions are well controlled with 12 or more oral medications	Yes	No	No
Patient with advanced Alzheimer's dementia, incontinent of urine, and living in his daughter's home; no other medical problem	Yes	No	No
Patient with a draining venous ulcer who is able to walk and drive her car independently	No	Yes	No
Patient with severe emphysema and cor pulmonale who is ambulatory and stable on home oxygen therapy and medication	No	No	No

Formal Home Care – Physician Role[2]

Introduction

Formal home care is that care provided to homebound patients by home care agencies. Most agencies are certified as Medicare providers; a few are not. Some have service contracts with health insurers or managed care organizations. They typically provide short term, skilled nursing services; rehabilitation services including physical, occupational and speech therapy; and personal care. Such care must be provided under physician approval and oversight.

Regulations

When physicians prescribe home care services for Medicare beneficiaries, they must certify that the patient is homebound; is in need of intermittent skilled nursing care or physical, speech, or occupational therapy; and under the physician's ongoing care. By signing the Medicare authorization form, the physician verifies that the patient has met the three eligibility criteria. The physician must also review the home care plan periodically, but no less often than every 2 months, and re-certify the patient if appropriate.

Homebound Criteria

In order to be eligible for homecare, patients need not be bedridden; Medicare considers patients homebound if they cannot leave their residence independently. Such patients may leave their homes with the aid of assistive devices or another person, but absences from the home must be relatively short and in most instances, be for the purpose of medical treatment. Patients are also considered homebound if leaving the home is medically contraindicated.

Table 3 lists qualifying clinical situations for Medicare criteria for home confinement.

The Skilled Service Requirement

A homebound person is not eligible for home care unless criteria for intermittent skilled care are also met. Skilled care must be provided by a registered nurse or physical, occupational, or speech therapist. However, just because a service is provided by one of these health professionals does not necessarily mean it is skilled. A service is skilled because of its complexity, its appropriateness for the patient's condition and because it meets accepted standards of medical and nursing practice. "Intermittent" means that the skilled services are required less frequently than 7 days per week, but at least once every 60 days.

Table 4 provides examples of met and unmet eligibility requirements. Documentation should describe the patient's condition and the complexity of required services. An assessment of the risk of bad outcomes should the services become unavailable is also required.

The Physician as a Gatekeeper

The eligibility criteria for home care are stringent because the intent of the Medicare program is to cover acute care rather than long-term care. However, the clinical reality is that chronic conditions exacerbate and improve over time, causing home care eligibility to change and complicating the physician's role in approving services. The (re)certification plan-of-care forms, completed by home care agency staff, should document not only patients' current needs for skilled care, but also the reasons for their homebound status. Without firsthand knowledge or other reliable information of the patient's condition, the physician should not certify the patient in a perfunctory manner by signing a form.

In many cases, there is no doubt that the patient meets the Medicare criteria for home confinement. Still, physicians should not allow long periods of time to go by without seeing the patient (e.g., 6 months if stable); house calls may be required for some patients. Given that an assessment of functional status is an integral component of geriatric care, the medical record should contain current information about function, in addition to usual medical management issues that will justify patients' eligibility in case of audit.

The gatekeeping role can be particularly frustrating for patients with chronic conditions, such as congestive heart failure (CHF) or emphysema who meet criteria for skilled services only during episodes of exacerbation. In-home nursing services for some of those conditions have been shown to reduce exacerbations and hospitalizations, yet regulations impede the provision of evidence-based proven interventions.[3]

Physician payment codes for formal home care oversight

Medicare also pays physicians for overseeing the work done by home care agencies. The CPT codes for these services are:

a. G0180 Certification for home care
b. G0179 Re-certification for home care
c. 99374 Care plan oversight for home care: requires at least 15-29 minutes per month and ability to document the time spent.
   99375 Care plan oversight for home care: 30 minutes or more per month
d. 99377 and 99378 Care plan oversight for hospice (same respective time requirements as for home care).

References

1. Unwin B, Jerant A. The home visit. *Am Fam Physician* 1999;60:1481-8.
2. Wachtel TJ, Gifford DR. Eligibility for home care certification. *J Gen Intern Med* 1998;13:705-9.
3. Stewart S, Pearson S, Horowitz JD. Effects of a home-base intervention among patients with CHF discharged from acute hospital care. *Arch Intern Med* 1998;158:1067-72.
4. Wachtel TJ. Home Care and House Calls. In*: Practical Guide to the Care of the Geriatric Patient*. Third Edition. Wachtel TJ, Fretwell MD. (eds.) St. Louis, 2007; Mosby: 497-511.

Additional Readings

American Academy of Home Care Physicians: Executive Summary, Public Policy Statement, 2005. Edgewood, MD: American Academy of Home Care Physicians, 2005. http://wwwaahcp.org/public_policy_2005.pdf

Boling P, Abbey L, Keenan J: Home care. In Ham R, Sloane P, Warshaw G (eds): *Primary Care Geriatrics, ed 4*. St. Louis, Mosby, 2002: 217-28.

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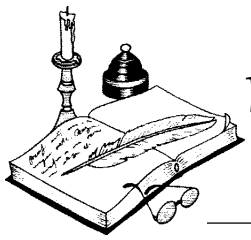

Physician's Lexicon

The Shape of Medical Terms

Paired prefixes, Latin or Greek, serve to provide parallel meanings in the realm of straightness, slantedness, curvature and flatness to a variety of medical and non-medical words.

The Greek word for flat [***platys***] appears in the technical name for flat worms [***Platyhelminthes***], the flat cervical muscle [***musculus platysma***], and skull flatness [***platycephaly***.] The prefix also defines such words as platypus [the duckbill], plate [an older term for sheets of metal] and platinum [from ***platina del Pinto***, the little silver of the River Pinto in Spain]. Plato, the celebrated Greek philosopher [427? – 347 BCE] was given the name, Aristocles, at birth but in early maturity was renamed Plato because of his broad and flat shoulders.

The Latin equivalent for flat, ***planus***, is seen in medical words such as the sole of the foot [plantar] and non-medical terms such as planar, planaria, planchette and plane. [The word, planet, however is from the Greek word, ***planetus***, meaning wanderer.]

The Greek word for straight or upright [***orthos***] defines such medical words as orthopedics [upright children], orthodontia [straight teeth] orthopnea [difficulty in breathing in any but the upright position] and orthophrenia [normal mentation.] Non-medical words include orthodoxy [upright beliefs] and orthogonal [right-angled.]

The Latin equivalent for upright or straight is ***rectus*** as in such technical words as rectangle, ***rectus abdominus*** and ***rectum*** [the straight colon.] A host of non-medical words are based on ***rectus***, including rectilinear, rectify, rectitude, rectangle, correction, rectory and director [one who keeps things straight.]

A cluster of Greek prefixes convey the meaning of curved, distorted or bent: ***skolios*** [as in scoliosis]; ***kampylos*** [as in ***Campylobacter***, the curved bacillus]; ***ankylos*** [as in ankylosis, a deformed joint]; and ***grypos*** [as in arthrogryposis, a crooked, inflexible joint.] The Latin counterpart is ***clivus***, meaning a sloping or slanted surface as in words such as proclivity, decline, inclination, declension and clinical [an adjective pertaining to the bed.] Another Latin equivalent, ***crispare***, meaning to wave or curl, is rarely used except in words such as crispation, meaning to curl, crisp [as in potato chips] and St. Crispin, 3rd Century patron saint of leather workers [see Shakespeare's Henry V.].

The Latin word for slanted, ***obliquus***, appears in words such as obliquity [a divergence, a slanting away from moral rectitude] and oblique but not obliterate [literally, ***ob – littero***, to erase the words.]

Medicine employs still other words of descriptive geometry such as triangle [Alsberg's, inguinal and others] and circle [such as the Circle of Willis] but few trapezoids [***musculus trapezius***] or ovals [ovalocyte] and no squares.

– **Stanley M. Aronson, MD**

Rhode Island Department of Health
David Gifford, MD, MPH
Director of Health

Vital Statistics

Edited by Colleen Fontana, State Registrar

Rhode Island Monthly Vital Statistics Report Provisional Occurrence Data from the Division of Vital Records

Underlying Cause of Death	Reporting Period			
	March 2007	12 Months Ending with March 2007		
	Number (a)	Number (a)	Rates (b)	YPLL (c)
Diseases of the Heart	223	2,775	259.4	3,739.0
Malignant Neoplasms	181	2,289	214.0	5,947.5
Cerebrovascular Diseases	40	385	36.0	494.5
Injuries (Accidents/Suicide/Homicde)	40	560	52.3	8,775.5
COPD	28	461	43.1	437.5

Vital Events	Reporting Period		
	September 2007	12 Months Ending with September 2007	
	Number	Number	Rates
Live Births	1,171	13,680	12.8*
Deaths	680	9,915	9.3*
Infant Deaths	(7)	(108)	7.9#
Neonatal Deaths	(4)	(76)	5.6#
Marriages	899	6,885	6.4*
Divorces	237	3,096	2.9*
Induced Terminations	430	4,696	343.3#
Spontaneous Fetal Deaths	73	1,004	73.4#
Under 20 weeks gestation	(65)	(931)	68.1#
20+ weeks gestation	(8)	(73)	5.3#

(a) Cause of death statistics were derived from the underlying cause of death reported by physicians on death certificates.

(b) Rates per 100,000 estimated population of 1,067,610

(c) Years of Potential Life Lost (YPLL)

Note: *Totals represent vital events which occurred in Rhode Island for the reporting periods listed above. Monthly provisional totals should be analyzed with caution because the numbers may be small and subject to seasonal variation.*

* Rates per 1,000 estimated population

\# Rates per 1,000 live births

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Ninety Years Ago, March 1918

Frank E. Peckham, MD, in "The Treatment of Poliomyelitis," suggested tapping the spinal cord and withdrawing fluid: "…it has been observed many times that there was immediate improvements in all symptoms, including the paralysis…To have such immediate relief proves that it was due to mechanical pressure within the membrane, because when the infiltrate returns, the symptoms, including paralysis, promptly return, to be relieved by tapping and again removing the mechanical pressure…Therefore, the earlier stages of paralysis are due to mechanical pressure and not to cellular death." The author also suggested adding orange juice and beef juice to children's diets, because often the correct diagnosis was not poliomyelitis, but scurvy.

Henry A. Cooke, MD, in "Vaccine Treatment in 15 Cases of Typhoid Fever at The Rhode Island Hospital," noted that 3 patients died, but still advanced his "personal feeling…that it merits further trial."

John G. O'Meara, MD, in "Current Public Health Legislation," spotlighted a few key bills: legislation that would force all manufacturers to provide fresh drinking water to their employees "during working hours;" legislation that would prohibit the "use of common drinking cup and a common towel…in all factories, manufacturing or business establishments;" and legislation that would increase the number of state factory inspectors from 4 to 5, with 1 a physician. (In Massachusetts, all 23 factory inspectors were physicians.)

Fifty Years Ago, March 1958

Francesco Ronchese, MD, in "The Care of the Skin," discussed the care of hands, exposure to sun, and hair and scalp problems.

William P Buffum, MD, in "Asthma in Infancy," noted: "The prognosis in asthma, occurring before 2 years of age, is probably…worse than when the asthma begins later."

Charles J. Ashworth, MD, President, Physicians Service, noted in the Report for 1957 that the insurance service gained 483 contracts in 1957. The net gain reflected key losses: 70 companies went out of business, 25 companies downsized, and 9 companies dropped Physicians Service insurance.

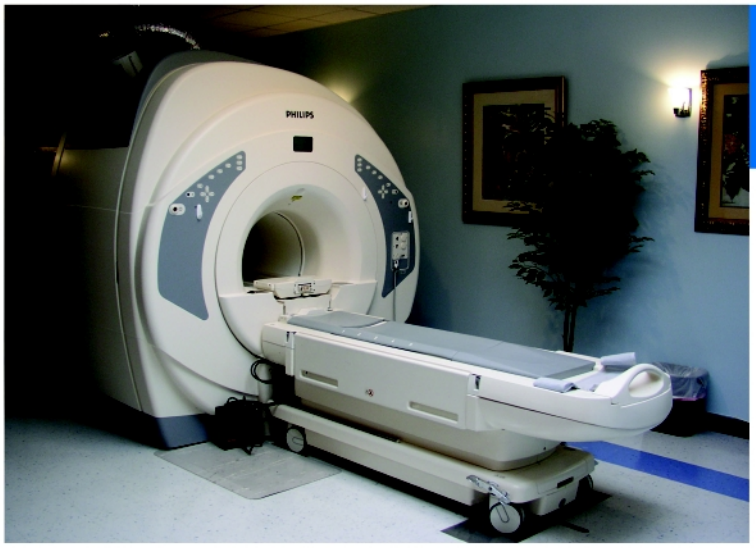
Twenty-Five Years Ago, March 1983

In Clinicopathological Conference Case Record: Rhode Island Hospital, Maurice A. Albala, MD, Thomas Wachtel, MD, George Meissner, MD, and David Williams, MD, discussed a 72 year-old hairdresser who initially presented with "shortness of breath, cough and sinusitis."

Frank M. D'Allessandro, MD, in "Diabetes Mellitis – Practical Aspects," noted that: "The determination of endogenous insulin levels has proved useful in difficult cases."

Edward W. Collins, MD, and John S. O'Shea, MD, in "Child Abuse and Neglect Update," discussed the increasingly important role of the physician. In 1979 the State of Rhode Island created the Department for Children and Families, and made "failure to report" a misdemeanor punishable by a fine up to $50 and/or one year in prison.

The Name of Choice in MRI



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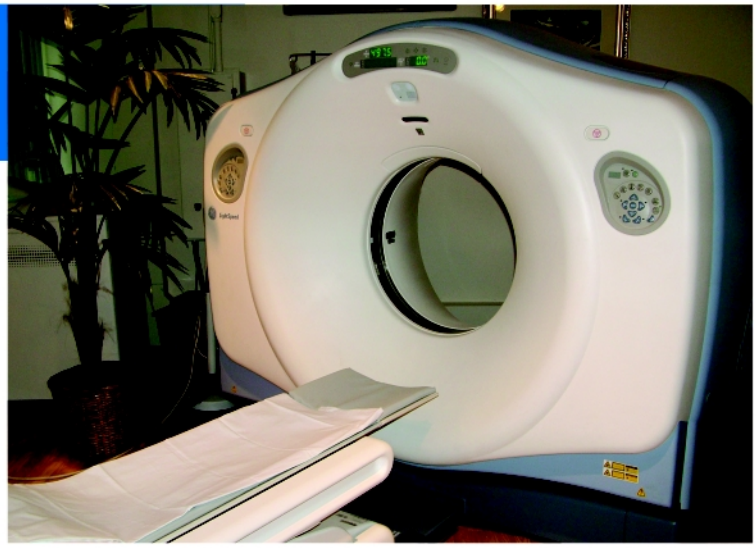

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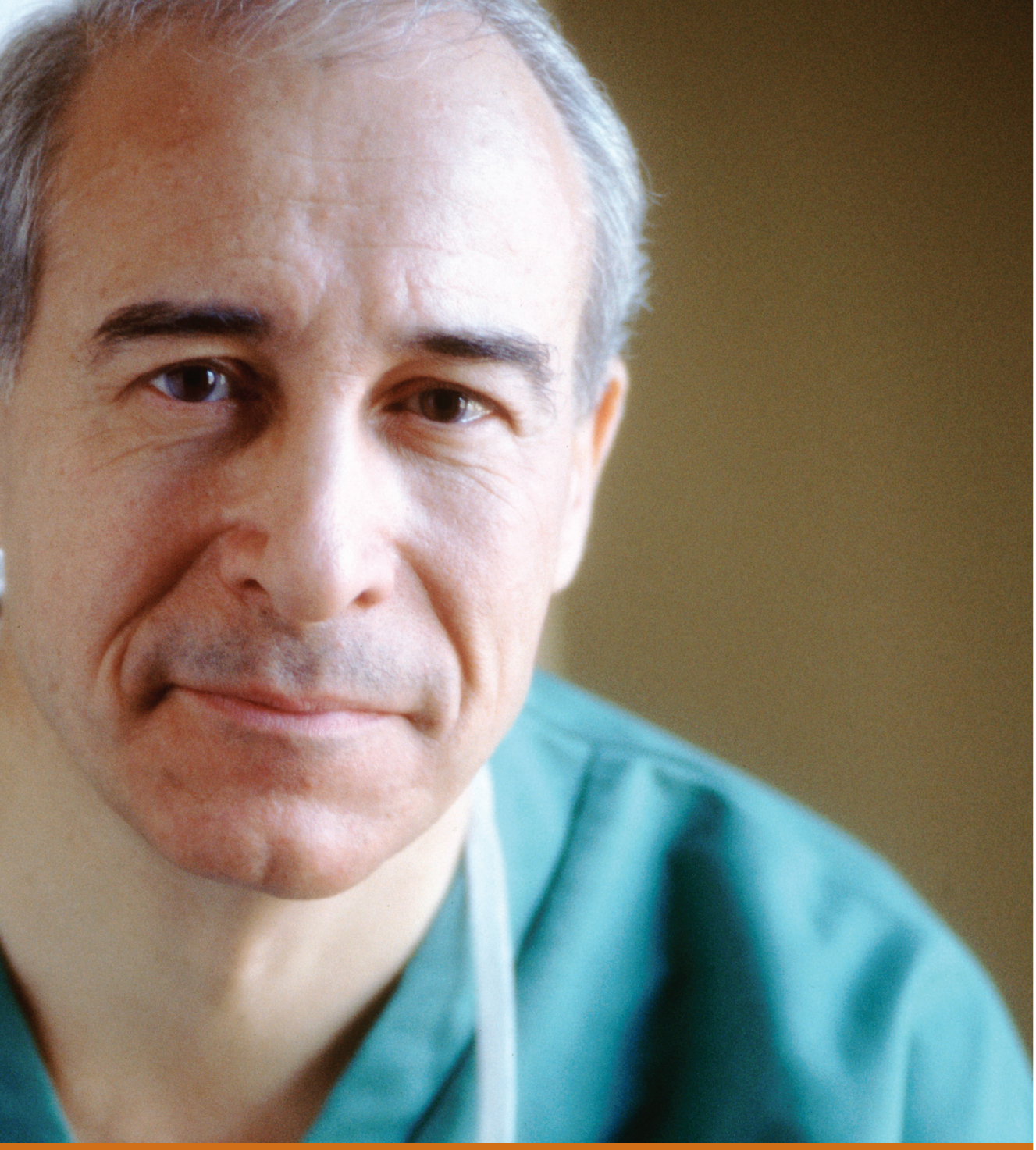

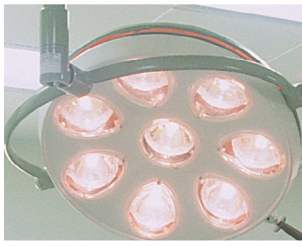

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