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Cardiac Safety of Citalopram

ABSTRACT & COMMENTARY

Source: Rasmussen SL, et al. Cardiac safety of citalopram: Prospective trials and retrospective analyses. *J Clin Psychopharmacol* 1999;19:407-415.

Citalopram (celexa) is the newest available antidepressant in the class of selective serotonin reuptake inhibitors (SSRIs). While SSRIs are typically regarded as benign in terms of adverse cardiovascular effects, there has been some research in beagle dogs that suggests that a metabolite of citalopram, didemethylcitalopram (DDCT), may cause prolongation of the QT interval.

The current paper reports findings of a randomized, double-blind, placebo-controlled study of healthy volunteers (n = 23) designed to assess QTc interval changes during treatment with placebo or citalopram, and the correlation of such changes to plasma drug levels. The subjects, aged 21-41 years, had baseline ECGs performed, received placebo for three days as a run-in, and were then randomized to receive either placebo or citalopram 60 mg/d (maximum recommended daily dose) for four weeks. Repeat ECGs were performed several times during the placebo run-in period, at steady-state, and at the end of the treatment period. There were no statistically significant differences between the two arms at baseline, during run-in, or at steady-state. Further, there was no correlation between QTc intervals and serum concentrations of DDCT. Consistent with previous reports that beagle dogs have higher relative concentrations of DDCT due to species-specific differences in metabolism, DDCT was present in very low concentration in the healthy human subjects.

In addition to the study of healthy volunteers, the current paper reported the ECG findings of three randomized, double-blind, placebo- or active-controlled, fixed-dose trials in adult and elderly depressed or demented patients (n = 1460) in addition to more than 6000 ECGs performed in 1789 citalopram-treated patients collected from all clinical trials conducted from 1978 through 1996. In both the prospective and retrospective analyses, there were no significant effects on QTc intervals, indicating that citalopram has no effect on

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cardiac repolarization. Citalopram did cause a reduction in heart rate (~ ≤ 8 beats/min), which is consistent with some reports of fluoxetine-associated bradycardia.

COMMENT BY MICHAEL F. BARBER, PharmD

The importance of screening and treating depression in patients with cardiovascular disease has been emphasized previously. While it is apparent that SSRIs represent a much more favorable class of antidepressants compared to tricyclic antidepressants (TCAs) in terms of cardiac safety, the data on the magnitude of their effects on the QT interval have been somewhat sparse. Since there had been reports of citalopram-associated prolongation of the QT interval in beagle dogs, there was clearly a necessity for an investigation of such effects in humans. The current report suggests that citalopram is not likely to prolong the QT interval in humans. However, the results should be taken cautiously since a complete lack of QT effects cannot be ruled out in all patients. For instance, the data from the beagle dogs study seemed suggestive that animals that exhibit extensive metabolism of citalopram via CYP2D6 will achieve higher serum concentrations of DDCT, leading to QT prolongation. Since subjects in the current study were not phenotyped for CYP2D6, it is not clear whether humans who are extensive metabolizers of citalopram via CYP2D6 would display elevated concentrations of DDCT and may be at higher risk for QT prolongation. Further, since the patients in the trials were relatively free of cardiac disease, citalopram's safety for use in patients with cardiovascular disease has yet to be described. There has been at least one death reported in an overdose of citalopram; however, the exact cause of death was not established. Therefore, it would seem appropriate to use citalopram with caution in patients who are at high risk for developing arrhythmias due to QT prolongation (i.e., patients with prolonged QT at baseline, patients who are receiving medications known to prolong the QT interval such as quinidine, or patients with hypokalemia) until further data are available. ❖

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SSRIs and Breastfeeding

ABSTRACT & COMMENTARY

Source: Ohman R, et al. Excretion of paroxetine in breast milk. *J Clin Psychiatry* 1999;60:519-523.

Depression affects 13% of women after childbirth, a period with a four-fold higher risk for depression. Although appropriate treatment of depression in the post-partum period is important to both the mother and infant, many women and physicians shy away from medication because of concern about adverse events to infants who are exposed to antidepressant medications in breast milk.

The current article describes seven patients with depression or panic disorder, who received a constant 20 mg morning dose of paroxetine for eight days. In six subjects, trough and peak serum and breast milk levels were determined. The seventh subject had samples drawn at steady-state, 0, 1, 2, 3, 4, 6, 8, and 24 hours after paroxetine intake, on two occasions (at 20 mg/d and 40 mg/d) with an interval of seven weeks. A total of 58 milk/serum samples were analyzed. Concentrations varied in milk from 5.3-145 ng/mL and serum from 11-188 ng/mL. There was a mean increase of 61% in the paroxetine concentration from the time of the daily dose to six hours after the dose. The estimated individual mean relative dose to the infants was 1.4% (0.7%-2.9%) of the weight-adjusted maternal dose. Single milk/serum ratios varied from 0.31 to 2.00, with a mean of 0.69. Ten pairs of foremilk and hindmilk samples were collected; the concentration of paroxetine was 78% higher in hind-

milk compared to foremilk. In summary, the study demonstrated a considerable time-dependent, dose-dependent, and interindividual variability in the excretion of paroxetine in breast milk. Data on other antidepressants reveal that the relative dose to the infant is 1.2%-6.5% for fluoxetine, 0.7%-9% for citalopram, 0.5% for fluvoxamine, and 0.45%-1.04% for sertraline, compared to 1.4% for paroxetine in this study.

The small sample size of the current study limits definitive conclusion and precludes the ability to detect adverse events in the infants. In addition, the validity of the data is limited because serum levels were not measured in the infants.

COMMENT BY DONALD M. HILTY, MD

This preliminary study sets the stage for larger studies that may help us determine the safety of SSRIs for infants during breastfeeding. It is premature to conclude that one SSRI may be more safely used than another, given small sample sizes and the lack of head-to-head comparison. However, data regarding fluoxetine indicate that children followed for up to seven years following intrauterine exposure had no evidence of alteration in development or cognition. If a SSRI is clinically indicated in a mother who is breastfeeding, the overall risk of exposure to the infant may be reduced by not breastfeeding during the period of time in which peak levels of medication will be in the milk, or if possible, by maximizing the use of foremilk rather than hindmilk. ❖

Sexual Orientation and Suicidality

ABSTRACT & COMMENTARY

Source: Herrell R, et al. Sexual orientation and suicidality: A co-twin control study in adult men. *Arch Gen Psychiatry* 1999;56:867-874.

As herrell and associates point out, the american Psychiatric Association declassified homosexuality as a pathological condition more than 20 years ago. However, the experience of being gay—in particular, homosexual feelings in the context of minimal social support and possible stigmatization—has been previously associated with an increased risk of suicide attempts. However, most previous studies suffer from methodological difficulties.

The current study provides some of the most methodologically sound data to date on the topic. Herrell et al used co-twins, a technique that maximally controls for genetic and many environmental confounds. Specifically, the subjects consisted of twins from the Vietnam Era Twin (VET) Registry who were discordant for adult same-sex sexual behavior. The current study also used a widely used instrument in psychiatric epidemiology, the Diagnostic Interview Schedule via telephone interviews. Forty-eight monzygotic and 55 dizygotic male twin pairs, discordant for adult homosexual behavior, were identified from the registry. Specifically, the VET Registry twins were asked the following: "Have you ever had sexual relations with a man at any time since you were 18 years old?" Four lifetime symptoms of suicidality were analyzed: Thoughts about death, wanting to die, thoughts about committing suicide, and attempted suicide. Herrell et al concluded that men who had engaged in homosexual activities had a significantly increased incidence of each of the suicidality measures. Unadjusted matched-pair odds ratios were: 2.4 for thoughts about death; 4.4 for wanted to die; 4.1 for suicidal ideation; 6.5 for attempted suicide; and 5.1 for any of the suicidal symptoms. After adjustment for substance abuse and depressive symptoms (other than suicidality), all of the suicidality measures remain significantly associated with same-gender sexual orientation except for wanting to die (odds ratio, 2.5). Herrell et al conclude that the substantially increased lifetime risk of suicidal behaviors in homosexual men is unlikely to be due solely to substance abuse or other psychiatric comorbidity. Although the sample was too small to draw definitive conclusions, zygosity did not seem to be a relevant contributor to the results.

COMMENT BY LAUREN B. MARANGELL, MD

In many ways, this is a more representative sample than previous studies, particularly because this is a population-based registry, i.e., it was assembled without regard to sexual orientation. Several additional factors are worth noting. The sample was composed of Vietnam era veterans, most of whom were Caucasian. In addition, sexual orientation arguably involves feelings and desire, perhaps more than even behavior. Regrettably, the current available data only included a question about the behavioral dimension (i.e., a same-sex encounter). The presence or absence of same-sex sexual encounters may not accurately represent homosexuality, particularly in the absence of same sex fantasies. For example, a homosexual man may have only homosexual feelings and fantasies, but never act on these feelings. Similarly, a predominantly heterosexual man may have one same sex encounter on impulse. Addressing lifetime behavior provides only a limited characterization. Finally, although

homosexuality per se is not pathological, the burden of potential stigmatization and discrimination undoubtedly takes its toll. Another interesting, albeit theoretical hypothesis, put forth in an accompanying commentary by Bailey,[1] posits that intrauterine exposure to an altered prenatal endocrine environment may predispose to both male homosexuality and disorders that are more common in women, including depression and eating disorders. Further study appears warranted. ❖

Reference

1. Bailey JM. *Arch Gen Psychiatry* 1999;56:883-884.

A Mechanism of Confabulation Revealed

ABSTRACT & COMMENTARY

Source: Schneider A, Ptak R. Spontaneous confabulators fail to suppress currently irrelevant memory. *Nat Neurosc* 1999; 2(7):677-681.

Recognizing confabulation can be useful in the differential diagnosis of amnesia. A new study by Schneider and Ptak suggests that the mechanism underlying spontaneous confabulation may be a deficiency in the ability to suppress activated memory traces that are inappropriate to the current context. In essence, amnesiacs who confabulate may be activating too many memories rather than too few.

Schneider and Ptak examined six amnesiacs who acted according to invented stories (spontaneous confabulators) and compared them to 12 comparably amnesiac patients who did not confabulate, as well as 10 normal controls. Spontaneously confabulating patients had abnormalities in the basal forebrain or medical orbital frontal cortex. The subjects were shown various sets of pictures and asked to identify a target item that appeared recurrently among singly viewed distrators. The same sets of stimuli were used in multiple runs, with previous target items interchanged with distrators. Before each run, subjects were instructed to forget the pictures they had seen before, and to only identify recurrences within the given run.

On this continuous recognition task, all of the amnesiacs performed significantly worse than controls, making more false-positive responses. However, confabulating amnesiacs showed steeply increasing numbers of false-positive responses as the experiment progressed. When the interval between successive runs was increased to 30 minutes, the false-positive response rate of confabulators remained high. Interference by previously acquired information was most evident when the stimuli represented real world objects, but was also apparent when meaningless designs were used.

Schneider and Ptak concluded that confabulating and nonconfabulating amnesiacs did not differ in their ability to detect new target items, indicating that confabulation is not simply a consequence of failure to saliently represent incoming information. What did distinguish confabulators was their inability to suppress interference arising from previously acquired information. Confabulators may process information encountered 30 minutes ago as though it were part of their experience of the present moment.

COMMENT BY NORMAN R. RELKIN, MD

Schneider and Ptak provide a new slant on the memory-monitoring deficit hypothesis. Based on their findings, spontaneous confabulators may not be able to suppress mental associations that pertain to past events or fully distinguish them from those that arise in the present. Information acquired days, weeks, months, or even years before may intrude into their current thinking, leading to the bizarre, false ideas that constitute confabulations.

This appealing theory explains some but not all of the phenomenology observed in amnesiac confabulators. Perhaps the most famous bedside clinical test for confabulation is the "purple string test," in which the examiner pretends to stretch a string between their own hands, and asks the amnesiac whether they can see the nonexistent purple string. Confabulators, particularly those with acute Korsakoff's syndrome, often state that they see the string and that it appears purple. If suggestive statements and a beguiling hand position are all that one needs to convince the amnesiac patient that they are seeing something that isn't really there, it would seem that confabulation represents more than just a failure to distinguish past from present associations. Confusion about what has been seen in the past vs. the present could be invoked as a partial explanation.

The brain lesions identified in these confabulating amnesiacs were all located in fairly circumscribed locations, distinct from those found in amnesiacs who did not confabulate. The salient areas lie in or near midline structures comprising the anterior limbic system, including the medial orbital frontal cortex and hypothalamus. This confirms that confabulatory amnesia usually arises from dysfunction in midline brain structures, rather than medial temporal and lateral prefrontal areas involved in other forms of forgetfulness. (*Dr. Relkin is Assistant*

Professor of Neurology, New York Hospital-Cornell Medical Center.) ❖

Vascular Dementia Patterns and Protection by Antihypertensive Drugs

ABSTRACTS & COMMENTARY

Sources: Looi JC, Sachdef PS. Differentiation of vascular dementia from AD on neuropsychological tests. *Neurology* 1999;53:670-678; Guo Z, et al. Occurrence and progression of dementia in a community population aged 75 years and older. Relationship of antihypertensive medication use. *Arch Neurol* 1999;56:991-996.

Looi and sachdef extracted a meta-analysis of 27 acceptable studies on vascular dementia (VaD) directed at the above title. These, and a larger number of anecdotal references, provide a number of clinical factors that may help to separate these diagnoses, although many experienced neurologists already recognize the difference in at least the advanced forms of either.

Fundamentally, Alzheimer's disease (AD) undergoes an insidious but steadily progressive evolution, beginning mostly after 70 years of age. Often, the victim has one or more family members with similar memory losses. Short-term, episodic, verbal-event memory usually undergoes an early loss, followed by defects in semantic memory and eventually, long-term memory loss as well. Language degenerates, morsel by morsel, often developing into a progressive, general aphasia in later years. Motor dysfunction, if any, usually occurs late in the disease. Potential markers are the E3-4 alleles of the serum lipoproteins and an MRI brain scan that shows neither a large number of scattered white patches in the hemisphere nor consistently large ventricles. Hippocampal shrinkage may be defined by MRI in relatively late stages of the illness.

VaD occurs at roughly the same age or even younger than AD and usually develops one or more of four relatively consistent markers: similar VaD ancestral cases, but less consistently than with AD; a long history of even modest hypertension or other lipid-related arterial diseases; frontal lobe dysfunction in executive activities; and, early during the illness, a waxing and waning of memory as well as intentionality in behavior. As notedabove, MRI scanning usually defines multiple small and sometimes large demyelinated spots, mostly in the cerebrum, but consistent with focal ischemia, cerebral ventricular dilatation often occurs. Persons with vascular dementia tend to retain better language qualities than AD during the middle to late years of their problems. Many wander in and out of confusion, becoming incontinent and unsteady on their feet during the late stages.

Against the above background, Guo and colleagues describe a somewhat unexpected success in adding antihypertensive medication to the elderly dementia protection list.

Guo et al selected 1810 patients older than 74 years from the Stockholm area to conduct a dementia incidence study; 225 already had dementia, a prevalence of 12.4% at the time the study began. An additional 284 persons refused initial testing. This left 1307 without dementia for further testing after two years. Before the end of the two-year follow-up, 314 of the 1307 died, leaving 987 persons for detailed evaluation after 24 months. At the time of baseline assembly, subjects already taking diuretics with or without additional antihypertensive drugs had a higher score on the MMSE ($P = 0.006$) and a lower prevalence of dementia ($P < 0.004$). At the end of the two-year follow-up time another 199 of the baseline 987 had become demented (20%). Also, among a total of 314 deaths occurring before the full two years, 25 had become demented (8%). Overall, subjects taking antihypertension drugs had significantly less dementia than the remaining patients ($P = 0.03$).

Diuretics and other antihypertensive agents were taken by 484 persons at baseline. Despite the fact that they were a bit older and had more heart disease and stroke than persons not using antihypertensive drugs, the population receiving the drugs had significantly ($P < 03$) less dementia than did the remaining senescents.

In the 987 persons retested at two years (excluding deaths), subjects taking diuretics had an adjusted risk rate (RR) of 0.7 compared to the remaining population. Put another way, subjects not taking antihypertensive drugs declined into dementia at a rate of 17% per year. Persons taking the diuretics with or without other antihypertension agents had less than half that chance of becoming demented.

COMMENT BY FRED PLUM, MD

Looi and Sachdef crystallize the major four clinical markers (exclusive of MRI film results) that best characterize the presence of cerebral-vascular dementia. As they emphasize, indicants of possible VaD should lead to suspicion of impaired clinical function but they lack firm, high probability. AD, on the other hand, by its absence of frontal lobe dysfunction and early character-

istics of impaired short-term memory plus fractured language abilities, usually clinically characterizes itself. Identification of serum lipoprotein alleles E3 and/or E4 considerably increases the accuracy of diagnosing AD. As the following paragraph indicates, cerebral arterial sclerosis may have a lighter, treatable incidence than has previously been surmised.

Guo et al's report expresses a major point, namely that even mild systemic hypertension apparently can independently cause dementia or accelerate the destruction resulting from several degenerative neurological illnesses. The message is clear: physicians treating patients of any age who develop blood pressure greater than 150/85-90 mmHg should consider prescribing permanent treatment with a diuretic or equivalent antihypertensive drug. AD, however, is not to be ignored as a subtle, additional factor to vascular dementia. First of all, the disease has no inexpensive laboratory tests that firmly identify the illness, and second, your editor knows of laboratory animal experiments that have found that cholinergic stimulants such as donepezil may improve the behavior of laboratory rodents following experimental obstruction of cerebral arteries. (*Dr. Plum is Professor and Chairman of the Department of Neurology and Neuroscience at the Weill Medical College of Cornell University.*) ❖

Special Feature

Update in Palliative Care XI: Beyond Pain

By Thomas J. Smith, MD, FACP

George soros, the richest man in the world, watched his wife die of breast cancer. She was in pain, mostly ignored, or not ministered to by doctors who could have chosen to be there. There were no discussions about advanced directives, or prognosis and expectations. Having billions to give away, he started the Project on Death in America, in an attempt to change for the better how Americans die. He hired Kathy Foley to direct it, and he chose about 20 people a year for aid.

Now the American Medical Association has started the Education for Physicians on End of Life Care (EPEC), from which I just returned. The American Society of Clinical Oncology (ASCO) also plans a half-day symposium in 2000 at the annual meeting. Sign up for either, or both, if you get a chance.

I was initially unconvinced that palliative care was a specialty. After all, didn't we all know how to give morphine? Well, actually not, given that more than half of the patients in academic hospitals died with unrelieved pain—palliative care works, and some people do it better than others.

Think back over the last 10 patients you have had die at home or in the hospital, and what distressed them or their families. Here is some of what I have learned in my three years.

Delirium is Common and Treatable

Ask a hospice nurse, and you will find that "terminal delirium" is one of the most common and troubling problems that distresses families with relatives near death. Moaning and delirium is upsetting to watch and is often interpreted as pain. But it's not pain, and high accumulations of narcotics due to diminished renal clearance may be one of the most common causes. (*See Table 1.*)

Table 1

Troubleshooting in Terminal Delirium Cases

Cause	Solution
Too much narcotic	Don't stop it! Just cut it in half. If that does not work, then stop it.
Too little renal clearance	In the patient with weeks to live consider a trial of hydration, with a switch to another narcotic at equivalent doses.
No definable reason	Reassure the family that it is a common preterminal event.Reassure them that it is not pain. Don't treat unless it bothers someone. Who knows what they are seeing.*

**Haloperidol (0.25-1.0 mg syrup in the cheek, or rectal) works much better than benzodiazepines.*

Agitation is Common and Treatable, too

Again, haloperidol wins hands down over benzodiazepines, when studied. It has a good anti-emetic effect and probably accounts for fewer falls and fractures. Start low at 0.25 mg or so and work up. (Editor's note: Low doses of newer 'atypical' antipsychotics, such as risperidone, are also effective alternatives.)

Terminal Nausea is Common and Treatable

No one really knows why, but it is common. And the best solutions are not XYZ-tron at $60 a day. Use a dopamine-receptor drug, such as metaclopramide 10-30 mg q 6 h, and some dexamethasone. It has the added advantage of enhancing bowel motility, too. (These tablets can be added to a suppository.) If that fails, try haloperidol 0.5-1.0 mg q 6 h or droperidol. If that doesn't work, try another class of agents such as

the serotonin receptor drugs (granisetron, ondansetron, etc.) or a benzodiazepine.

Terminal Dyspnea is Treatable

No need to be fancy here. Any narcotic will do if given as for pain, around-the-clock, and at the dose that works. Morphine works to reduce the work of breathing by relaxing the lung smooth muscle. Start with 5-10 mg q 4 h and titrate up to comfort, just like with pain. There is typically little or no effect on respiratory rate. Don't start with pulse oximetry and ABGs since the goal is to relieve the suffering of labored breathing; it's easier to not start them than to take them away once established.

If that does not work, try a fan blowing air across the patient (I know it sounds too simple, but try it once—it works). Then, oxygen (but remember it's expensive and hard to stop), then try steroids (and next) benzodiazepines. For some patients, it takes a combo of morphine and benzodiazepines. Inhalers such as albuterol rarely work and are not worth trying.

Nebulized morphine/fentany/etc. had a brief run of popularity. And it works, but no better than nebulized saline in randomized clinical trials. It's the narcotic, and we might as well give it P.O. or P.R.

The "Death Rattles"

This is the number one reason panicked families call me or hospice nurses. The dying person is usually in no distress, but it's hard to watch. (*See Table 2.*)

Table 2

Alleviating the "Death Rattle"

Cause	Treatment
Ineffective clearance	Prop the patient up and to his/her side.
Too many secretions	Scopolamine patches, just like for seasickness. Rarely cause disorientation in the terminally ill (or hard to tell) but can cause problems in the frail elderly who are still mobile. Put them on before it gets too bad because it's easier to prevent.

Depression is the Norm, not the Exception

Depression can be hard to differentiate from the sadness and grief of dying. I have switched to the "Chochinov Test" of Dr. Harvey Chochinov:

"Are you Depressed?"

If the patient says no, or "Yes, this is terrible! I'm dying, and feel so hopeless, and I don't have the energy to get up and I can't sleep..." you have more than an 80% chance of being right in your diagnosis. Plus, you asked.

Typical antidepressants can work but may take weeks. Start them or get a psychiatric consultation (tough for the homebound). Consider starting a short course of methylphenidate (Ritalin) or some other stimulant at low doses (e.g., 2.5-5 mg in the morning and at noon) increasing to tolerance. This also works for the morphine patient who cannot wake up.

Dr. Kevorkian, the Pinata of Palliative Care

Dr. Chochinov noted in his sample of 200 dying patients that nearly one in 10 had clinical depression. Of those requesting euthanasia, six of 10 were depressed. Almost all the requests for physician-assisted suicide (PAS) came from the depressed group. Most of these requests changed after about two weeks. I won't go over the six-step program to manage PAS, but someone's clever phrase was too hard to pass up.

Hiccups

Antacid with simethicone (then try) metaclopramide 10-30 mg q 4-6 h (or cisapride 20 mg q 12 h, but try the low priced spread first) (and next) baclofen 5-10 mg q 6-12 h.

Chlorpromazine, the drug most often used, is too sedating and rarely effective. Don't use prochlorperazine (Compazine) with metaclopramide (Reglan), as the first will block the prokinetic movement of the second and will increase the chance of extra pyramidal side effects.

Myoclonic Jerks

"Well, doctor, you've finally gotten him comfortable but he's jerking all over as soon as he drifts off to sleep, do something about those seizures!"

Table 3

Treating Myoclonic Jerks

Cause	Solution
Normal reaction to narcotic	Reassurance. Benzodiazepine (e.g., lorazepam 0.5-1 mg q 12 h, clonazepam, or diazepam). Consider a switch to a new narcotic, which can give a "honeymoon" period of several weeks.
Hyponatremia or renal failure	Only treat if indicated.

Myoclonic jerks ("sleepstarts") are normal and can happen with any narcotic. I have listed some of the most common causes and solutions here. Reassure the patient/family that these are not seizures, and only require treatment if bothersome. These movements are so characteristic that no diagnostic testing is needed other than a good clinician! (*See Table 3.*)

This isn't Rocket Science, but there isn't a lot of Science being done about it

The ASCO abstracts on palliative care this year were heavily skewed to fatigue, vomiting, and pharmaceutical interventions. Most of the above-mentioned material has been known for about 10 years but had not trickled down to mainstream oncologists like me.

There are some great places to learn more about these programs. Check out the EPEC website (http://www.ama-assn.org/ethic/epec/) or address (The EPEC Project, Institute for Ethics, American Medical Association, 515 N. State Street, Chicago, IL 60610, 312-464-4979) and sign up for a course. Much of it is old hat to oncologists, but a great refresher, and a way to then teach our colleagues in medicine, family practice, and surgery to practice these procedures. After all, we want them here for us when we die.

Take Home Message

First, when someone is dying, call hospice six weeks before they are dead, not six hours.

Second, when someone is dying, there is a lot you can do about it. Yes, I know it's not reimbursed well, and it's harder than choosing between Taxol and Taxotere, but this is why most of us chose to become doctors.

You can make the difference between a peaceful planned death at home, or a traumatic death that includes uncontrolled pain, ER visits, late night phone calls, and disgruntled family members. Either will be long remembered—it's our choice. (*Dr. Smith is Director of Cancer Education for the Massey Cancer Center at the Medical College of Virginia in Richmond.*) ❖

Suggested Reading

1. McDonald N. *Palliative Medicine: A Case Based Manual*. Oxford, New York, 1998.
2. Eguchi I, Klastersky J, Feld R. *Current Perspectives and Future Directions in Palliative Medicine*. Springer, New York, 1998.
3. WHO. *Symptom Relief in Terminal Illness*. Geneva 1998.

CME Questions

43. The occurrence of confabulation may indicate:

a. diffuse rather than focal brain pathology.
b. the presence of medial temporal lobe lesion.
c. more severe amnesia than in nonconfabulating patients.
d. a deficit in distinguishing past from present associations.

44. Adult male homosexual behavior is associated with an increased risk of suicidality.

a. True
b. False

45. Which antidepressant is believed to expose the infant to the lowest percent of the weight-adjusted maternal dose?

a. Citalopram (Celexa)
b. Fluoxetine (Prozac)
c. Paroxetine (Paxil)
d. Sertraline (Zoloft)
e. Fluvoxamine (Luvox)

Readers Are Invited

Readers are invited to submit questions or comments on material seen in or relevant to *Psychiatric Medicine in Primary Care.*

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Fibromyalgia Update