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Who Will Experience a Recurrence of Depression after Recovery?

ABSTRACT & COMMENTARY

***Synopsis**: A total of 85% of patients who have recovered from an episode of major depression will suffer a recurrence of depression within 15 years. However, if a patient has remained in remission for at least five years, the rate of recurrence is reduced to 58%.*

Source: Mueller TI, et al. Recurrence after recovery from major depressive disorder during 15 years of observational follow-up. *Am J Psychiatry* 1999;156:1000-1006.

IT IS WELL-KNOWN THAT MAJOR DEPRESSION IS AN ILLNESS OF recurrence. In addition, despite effective antidepressant and psychotherapeutic treatments, major depression remains an illness with high morbidity and mortality. Mueller and associates have compiled a large and impressive set of data to examine the rate of recurrence of major depression after recovery from the index episode. The present analysis is part of the "NIMH Collaborative Program on the Psychobiology of Depression," a large multi-center study looking at many aspects of depressive illness. The present study used standard rating instruments and was naturalistic, in that Mueller et al did not control the treatment the subjects received. For the present analysis, the authors identified 431 subjects with a major depressive episode. They excluded subjects who had bipolar or schizoaffective disorder or dysthymia. The main criteria for patients entering the study was a recovery from their index episode of major depression at some point during the 15-year follow-up period of the study. A total of 380 eligible subjects participated in the study. Of the 380 subjects described above, Mueller et al also examined a subgroup of 105 subjects who remained well for at least a 5-year period during the 15-year follow-up period.

The results of the study are striking and unfortunate. By the end of the 15-year follow-up period, 323 of the original 380 (85%) recovered subjects had suffered a recurrence of major depression. The news was somewhat better for the 105 subjects who had sustained at least a 5-year period of remission during the follow-up

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period. Specifically, 61 of these 105 (58%) subjects had a recurrence of major depression during the 15-year duration of the study. The authors attempted to correlate various clinical variables to the risk of recurrence. Factors at the index episode that predicted recurrence of depression included female sex, longer duration of index episode, never marrying, and multiple prior depressive episodes. The authors note that neither these nor any other factors predicted recurrence in subjects who had a sustained remission of depressive symptoms during the follow-up period. Few patients appeared to receive antidepressant medication for what is considered an adequate duration at an adequate dosage.

COMMENT BY ANDREW L. STOLL, MD

This important and possibly landmark study of the natural history of major depression underscores the importance of further research into the causes and treatments for major depression. It also points toward the inadequate application of otherwise effective treatments for major depression. Specifically, antidepressant medications, and to a lesser extent psychotherapy, can markedly reduce the rates of recurrence of depressive episodes. We need to better educate our patients and ourselves of the importance of maintenance therapy in require close follow-up and adherence to effective therapies. ❖

Psychiatric Medicine in Primary Care,™ is published monthly by American Health Consultants, 3525 Piedmont Rd., NE, Bldg. 6, Suite 400, Atlanta, GA 30305.

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GST Registration Number: R128870672. Periodical postage pending at Atlanta, GA.

POSTMASTER: Send address changes to ***Psychiatric Medicine in Primary Care***, P.O. Box 740059, Atlanta, GA 30374.

Back issues: $15. Missing issues will be fulfilled by Customer Service free of charge when contacted within one month of the missing issue's date.

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World-Wide Web: http://www.ahcpub.com

Subscription Prices

United States

$189 per year (Student/Resident rate: $95).

Multiple Copies

1-9 additional copies: $170 each. 10-20 copies: $151 each.

Outside the United States

$219 per year (Student/Resident rate: $110 plus applicable GST).

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High Risk of Depressive Relapse in the Elderly

ABSTRACT & COMMENTARY

***Synopsis**: Following recovery and two years of successful antidepressant treatment after a first lifetime episode of major depression, this open prospective study found a 60.6 % recurrence rate in elderly patients who were tapered off antidepressant medication.*

Source: Flint AJ, Rifat SL. Recurrence of first-episode geriatric depression after discontinuation of maintenance antidepressants. *Am J Psych* 1999;156(6): 943-945.

SEVERAL NATURALISTIC STUDIES HAVE FOUND THAT older patients have a higher risk of suffering a recurrence of major depression following discontinuation of antidepressant medication. The current study prospectively examined the two-year outcomes of elderly patients with first-episode major depression following discontinuation of their maintenance antidepressant medication.

The study group consisted of 21 elderly patients (mean age, 74.4 years, SD = 6.6) who had recovered from a first lifetime episode of major depression with either nortriptyline (with or without adjunctive lithium) as a first line of treatment (n = 19) or phenelzine as a second line of treatment (n = 2). Following the two years of treatment without a return of symptoms, the antidepressant was then withdrawn over a period of eight weeks. Patients were then followed for another two years or until recurrence, whichever occurred first was used to estimate. The cumulative probability of a recurrence (Kaplan-Meier product limit method) of major depression was 60.6%. Fifty-eight percent of new episodes occurred within six months and 92% within 12 months from the start of discontinuation of medication. Other variables, such as age, gender, or severity of illness did not predict recurrence. If a patient had a recurrence, he or she restarted the discontinued antidepressant. Patients were treated with the same dose of antidepressant that they had previously responded to. Eleven (91.7%) of the 12 patients who had a recurrence agreed to restart antidepressant medication. Nine (81.8%) of the 11 patients responded to reintroduction of the antidepressant alone, and one (9.1%) of the 11 patients responded to the antidepressant and adjunctive lithium. The mean time for

response to treatment was 4.5 weeks (SD = 1.8), which was not significantly different from the 4.6 weeks (SD = 2.3) needed to respond to treatment of the index episode (t = -0.12, df = 9, P = 0.91).

COMMENT BY LAUREN B. MARANGELL, MD

These preliminary findings from this small open study certainly bear replication in a larger population with a double-blind, placebo-controlled design. However, the cohort was prospectively followed with structured assessments and the findings are concordant with naturalistic data and clinical experience. The high risk of recurrence in the elderly has led some to recommend that antidepressant medications be continued for a longer, perhaps indefinite, period of time. Flint and Rifat note that given the favorable response to reinstated treatment, an alternative strategy is to treat first-episode patients for a shorter period of time (i.e., 6 months), and then treat recurrences when they arise. However, re-response to medication is not guaranteed and the time to re-response in the current study was 4.5 weeks. As such, the risks of medication discontinuation must be carefully weighed against the benefits. Given that long-term treatment with appropriately selected antidepressant medication is safe and well tolerated in most elderly patients, continuation treatment may be preferred for high-risk patients. Other factors that increase the likelihood of relapse are a higher number of previous episodes and residual symptoms of depression. If a decision is made to discontinue antidepressants, the patient should be educated about recurrence and followed on a regular basis for at least one year. ❖

Apo E-4 and Memory Function in Non-Demented Middle-Aged Individuals

ABSTRACT & COMMENTARY

Synopsis: *Having the gene for apolipoprotein E-4, an important biological marker for Alzheimer's disease, is associated with subjective and objective mild memory difficulties in non-demented middle aged individuals.*

Source: Small GW et al. Memory self-appraisal in middle-aged and older adults with the apolipoprotein E-4 allele. *Am J Psychiatry* 1999;156:1035-1038.

ALZHEIMER'S DISEASE CONTINUES TO BE THE LEADING cause of late-life dementia. Recent research has revealed that the gene for Apo E-4 is a powerful predictor of both hereditary and sporadic Alzheimer's disease. Apolipoproteins are associated with B-A4 amyloid fibrils. Combining the Apo E-4 marker with clinical findings has improved diagnostic certainty in Alzheimer patients. The purpose of the present study was to examine whether the gene for apolipoprotein E-4 is associated with memory changes in younger patients with subjective memory complaints.

Small and colleagues recruited 39 subjects (22 women, 17 men; age range, 50-82) who responded to advertisements for a study of mild memory difficulties. They screened 180 subjects for treatable dementing illness, neurological or psychiatric disorders, and whether they were receiving psychotropic medications. Small et al excluded 141 subjects, leaving the study cohort of 39 subjects. Participating subjects then underwent neurocognitive testing and self-rating scales of memory function. Subjects with the Apo E-4 genotypes were identified through standard DNA screening methods.

Small et al then compared the findings for memory/cognitive functioning in the group with the allele for Apo E-4 vs. those without the allele. Statistically significant differences emerged for several measures of memory functioning, particularly in those 27 subjects between the ages of 55-74 (the association between Apo E-4 and Alzheimer's disease is highest in this age group). Most of the significant differences were of low magnitude, except for marked differences in "retrospective functioning," a subjective measure of a person's perceived loss of memory function over time. Small et al candidly described the several significant flaws in study design, and conclude their paper with the suggestion that combining measures of memory function with screening for Apo E-4 or other genetic predictors of Alzheimer's disease may have merit in predicting who will subsequently develop Alzheimer's disease.

COMMENT BY ANDREW L. STOLL, MD

Despite the considerable methodological limitations of the present study, the findings point to the potential value of combining clinical and even subjective measures of memory functioning with a simple genetic screening test. Longer term follow-up studies are underway by Small et al and other groups of investigators, and these studies may help pin down which aspect of neurocognitive functioning or subjective memory truly predicts which patients are at highest risk for developing Alzheimer's disease. Combining these data with even more sensitive and specific measures of which genotype(s) or phenotype(s) are at highest risk will also surely improve diagnostic accuracy. In the near future, emerging prophylactic therapies against Alzheimer's disease can target those

individuals identified as a high risk for developing this devastating dementing illness. ❖

Special Feature

Depression and Cardiovascular Disease: A Key Link?

By Michael F. Barber, PharmD

CARDIOVASCULAR DISEASE (CVD) IS THE LEADING cause of death and serious illness in the United States. The management of patients with CVD usually involves the identification and subsequent treatment of alterable risk factors for myocardial infarction (MI). However, recent research has identified a new possible risk factor, one that is often overlooked in primary care practices: depression. Depressive disorders may be present in nearly 40% of patients who have coronary heart disease and 45% in those who recently experienced an MI.[1] Further, a review of epidemiological evidence suggests that depression is an independent risk factor in CVD.[2]

This is based on striking evidence that associates the presence of depression with several markers of the development and progression of CVD. For instance, patients with depression exhibit markers of sympathoadrenal hyperactivity, such as elevated concentrations of corticotropin-releasing factor, resulting in elevated cortisol levels, increasing the risk for development and progression of CVD.[3] Also, patients with depression show diminished heart rate variability, which has been implicated as a risk factor for mortality in patients after MI.[4] Further, psychological stress (such as that present in depressed patients) increases the risk of life-threatening arrhythmias.[5] Depressed patients also exhibit enhanced platelet activation, which can cause or worsen CVD.[6] Thus, the presence of depression may be viewed as equally important as dyslipidemia with respect to CVD. Depression has been shown to be roughly equivalent to left ventricular dysfunction as a predictor of increased mortality within six and 18 months of an MI.[7,8]

The relationship between depression and CVD has long been recognized. However, the usual assumption is that patients are depressed or sad because they have cardiac disease. Such an assumption fails to recognize that depression is largely biologically based. Further, although it is conceivable that depression takes place in genetically vulnerable individuals in the context of a stressor (presence of a life-threatening disease), there are intriguing data that suggests a common pathophysiologic link between depression and CVD. For example, both disease states have been associated with a deficiency in omega-3 fatty acids, suggesting that the two may be etiologically linked, rather than depression resulting from CVD.[9] Unquestionably, the evidence to date necessitates the screening for depression in patients with CVD. After the diagnosis of depression is made, the clinician should consider initiating an antidepressant. The selection of an appropriate agent should be done on an individual basis, keeping in mind some general considerations regarding antidepressants and their adverse effects in patients with CVD. It is well established that tricyclic antidepressants (TCAs) have significant cardiovascular side effects. The two main cardiovascular effects of TCAs are arrhythmias and orthostatic hypotension. There are numerous published cases, several of which have been fatal, of torsades de pointes associated with the use of TCAs. Selective serotonin reuptake inhibitors (SSRIs) have largely replaced TCAs as front-line therapy for the treatment of depression as a result of their relatively benign side effects profile and more favorable safety profile in acute overdose situations. In addition, SSRIs have very few cardiovascular effects, making them more attractive for use in patients with CVD.

SSRIs and TCAs with CVD

There have been at least two reports of direct comparisons of cardiovascular effects of SSRIs and TCAs in patients with CVD. Roose et al.[10] found no conduction disturbances, arrhythmias, or orthostatic hypotension and only a 4% rate of cardiovascular effects (slight decreases on heart rate, increases in supine systolic pressure, and increases in ejection fraction) in fluoxetine-treated patients (n = 27), versus a 20% incidence of cardiovascular effects (increased heart rate and orthostatic hypotension) in nortriptyline-treated patients (n = 60). In a separate study, Roose et al.[11] found a 2% occurrence of adverse cardiac events in 41 patients treated with paroxetine versus 18% of 40 patients treated with nortriptyline. Sertraline has also been shown to be relatively devoid of adverse cardiovascular effects in an open trial of depressed patients who experienced an MI within the previous 30 days.[12] While these data are suggestive that SSRIs are safer to use in patients with CVD, it should be noted that these studies were small, open, and of relatively short duration. Bupropion (n = 58) was also found to be relatively safe with respect to cardiovascular side effects in depressed outpatients without CVD when compared to nortriptyline (n = 57), suggesting that it may be appropriate for patients with CVD.[13] Both groups had some orthostatic hypotension, but only the nortriptyline-

treated group showed a slowing of cardiac conduction. Presently, there is little known about the safety of venlafaxine or mirtazapine in this population. Venlafaxine causes dose-dependent increases in supine diastolic blood pressure, which may be significant only at doses above 300 mg/d.[14] Mirtazapine is relatively devoid of direct cardiovascular effects but has been associated with increases in lipid levels.[15] Consideration must also be given to the potential for an antidepressant to cause a clinically significant drug interaction with a patient's current medication. The most important drug interactions typically occur with "narrow therapeutic index" drugs.

Inhibition of Oxidative Metabolism

One of the most important mechanisms of clinically significant drug interactions caused by antidepressants involves the inhibition of oxidative metabolism of other drugs mediated through the cytochrome P450 (CYP) system. Within this enzyme system, the four most common isoenzymes which mediate antidepressant drug interactions are CYP2D6, CYP3A4, CYP1A2, and CYP2C.[16] Many medications commonly prescribed to patients with CVD are metabolized by one or more of these enzymes. Lipophilic beta blockers such as metoprolol are metabolized by CYP2D6, which can be inhibited by most SSRIs, most notably paroxetine and fluoxetine. Also notable is the fact that beta blockers do not necessarily cause depression. [17] Warfarin is metabolized by CYP2C and CYP1A2 and is inhibited by fluoxetine, fluvoxamine, and to a lesser extent, sertraline. Paroxetine and fluoxetine have also been reported to increase the likelihood of bleeding without an increase in clotting times, perhaps due to a serotonin-mediated decrease in platelet adherence.[18] This effect could theoretically take place with the other SSRIs as well. Inhibition of CYP3A4 by nefazodone, fluvoxamine, and to a lesser extent fluoxetine and sertraline may result in increased concentrations of calcium channel blockers as well as the antilipemic drug lovastatin, which may subsequently cause rhabdomyolysis.

Summary

In summary, given the effect of depression on patients with CVD, it is important to screen for and treat depression in this population. Despite the need for more long-term data, it is apparent that the newer antidepressants such as the SSRIs are relatively safer to use than TCAs with respect to CVD. Despite the improved safety, newer antidepressants may cause clinically significant drug interactions with medications commonly used in patients with CVD. Thus, the selection of an antidepressant should be done on an individual basis and with careful discrimination. ❖

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Special Feature

Management of Agitation and Aggression in the Elderly

By Lucy J. Puryear, MD

THE TREATMENT OF ELDERLY PATIENTS IS OFTEN COMplicated by the need to manage behavioral disturbances associated with dementia. As medical advances continue to extend life, a larger percentage of the population will be at risk for the behavioral sequelae that may accompany dementia of any cause. Behavioral disturbances can include verbal outbursts, physical aggression, and wandering. These behaviors can be extremely difficult for caregivers to manage and often require an increased level of care, including nursing home placement along with physical and/or chemical restraints.

Although it is often problematic to assess a patient whose cognitive abilities make productive conversation difficult, it is important to try and identify the cause of the agitation or aggression and to treat any underlying disorder. Pain, anxiety, psychosis, delirium, or even boredom may all account for agitation. Masking the symptoms with sedating drugs, as opposed to treating the underlying condition, may cause further complications. Behavioral treatments should be considered as first-line therapies. These interventions would include reorientation, decreasing extraneous stimuli, and providing environmental cues.[1] If these measures are inadequate, pharmacologic management should be added.

Typical Antipsychotics

Traditionally antipsychotic medications have been used to treat agitation and aggression. The 'typical' antipsychotics, such as haloperidol (Haldol) or thioridazine (Mellaril), have often been used in large doses to sedate patients and decrease yelling, screaming, and wandering. Typical antipsychotics are useful medications but, because of potentially disabling side effects, should be limited to those patients with manifest psychotic symptoms such as auditory or visual hallucinations, or paranoid delusions. In the elderly patient, typical antipsychotics can be difficult to use. They carry a large anticholinergic burden, which may worsen cognitive symptoms. The elderly are also more sensitive to the extrapyramidal side effects of these medications, including akathesia, parkinsonism, and dystonias. These patients are also at greater risk for developing tardive dyskinesia, a serious, permanent movement disorder.

Newer, 'atypical' antipsychotics are less likely to cause extrapyramidal symptoms and tardive dyskinesia. These newer medications include risperidone (Risperdal) and olanzapine (Zyprexa), and may be useful for the treatment of psychosis in the geriatric patient. Risperidone may be used in doses as low as 0.5 mg po at bedtime. There is the potential for orthostatic hypotension; blood pressure must be monitored and the dose should be increased slowly if necessary for symptom control. Above doses of 3-6 mg there is an increased incidence of EPS. Olanzapine should be used in doses of around 2.5 mg po at bedtime. This medication can cause marked sedation. Precautions must be taken to avoid falling at nighttime.

Benzodiazepines have been another frequent choice for the treatment of behavior problems in the elderly. In the geriatric patient, they pose particular problems and can increase memory impairment and confusion. They are also known to cause paradoxical disinhibition that may make aggressive behaviors worse. Benzodiazepines should be used in cases of acute agitation when quick control of the patient is essential. Low doses of 0.5-1.0 mg of lorazepam, po or IM, can be given every hour until symptoms remit. These medications should not be used for the control of chronic aggression.

Serotonergic Medications

Serotonergic medications have been used to treat agitation and aggression. Trazodone is a serotonergic antidepressant whose major side effect is sedation. Given at bedtime in the range of 25-50 mg po, it is a potent, nonaddicting hypnotic. Given in low doses throughout the day it may help control aggression.[2] Buspirone is another serotonergic medication that can be used to control behavioral disturbance. It is marketed as an anxiolytic and is non-addicting. It may be a useful medication for the patient with anxiety as a major component of their agitation. Given by mouth, 5 mg twice or three times a day may alleviate restlessness. Higher doses may be used; However, it is necessary to observe patients for dizziness and mental confusion. The serotonin reuptake inhibitors fluoxetine, sertraline, paroxetine, and citalopram may also be effective in treating anxiety and aggression.[3] These medications are typically well tolerated in the elderly, with starting doses of half the recommended initial dose.

Another class of medication, the anticonvulsants, has been used to control physical and verbal aggression in patients with dementia. Carbamazepine and sodium valproate (Depakote) have been shown to be safe in elderly

patients. Liver function tests must be monitored and white blood cell count followed for patients on carbamazepine. Depakote is well tolerated, does not cause cognitive disturbance and may markedly diminish hitting and wandering. Blood levels can be monitored but elderly patients may do well on levels below the standard 50 ng/ml.[4] Liver function tests and platelets must be followed when using this medication. These medications are obviously the drugs of choice when treating agitation or aggression due to the manic symptoms of bipolar disorder. They also may be particularly helpful in patients with impulsive aggressive behaviors such as striking out at caretakers.

Propanolol has been useful in high doses for the management of chronic aggression associated with brain damage.[5] Dosing begins at 10 mg twice or three times a day and may go as high as 200 mg a day in divided doses. The anti aggressive effect may take several weeks to develop even after therapeutic doses have been reached. This medication must be used cautiously in the elderly due to it's potential for causing hypotension and the potential for aggravating other medical conditions such as congestive heart failure and asthma. Its benefits are long-term behavioral control without cognitive side effects or the potential for addiction or abuse.

With careful consideration to the underlying reason for behavioral disturbances in the elderly, these behaviors can be successfully treated without additional side effect burden and potentially improve quality of life. Patients may be able to tolerate a less restrictive environment and decrease the risk of over sedation and other comorbidities of restraints.❖

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Special Feature

Telepsychiatry as a Method to Provide Psychiatric Care

By Donald M. Hilty, MD

ALTHOUGH NOT YET WIDELY AVAILABLE, TELEMEDICINE technology is one strategy to improve the accessibility of mental health care and consultation to patients and physicians in underserved areas. The format most applicable to psychiatry is live, two-way audio, two-way video transmission (interactive television).

Our University of California-Davis Health System uses telemedicine to link the Medical Center with 16 of its primary care clinics, which are 20-120 miles away. Telepsychiatric consultation is generally provided in weekly to monthly three-hour blocks to six clinics, via a consultation care model (i.e. the primary care physician is the principal provider of mental health services and calls the psychiatrist for advice or requests a one-time consultation).

A survey of a rural population revealed that over two-thirds of patients expected a less satisfactory doctor-patient interaction via telemedicine care than a traditional patient-doctor encounter.[1] To address this issue, we measured patients' preference for care, adherence to appointments, and satisfaction.[2] All patients received care at their primary care clinic, choosing between in-person care or telepsychiatric care. Respondents rated their ability to speak freely when using telemedicine, their preference for using telemedicine on subsequent visits, and their experience with the telemedicine physician on a 5-point Likert-like scale (1 = poor, 5 = excellent). A total of 42 patients made 118 visits consisting of a 60-minute evaluation (24 visits) or a 20-minute follow-up appointment (94 visits). The primary diagnosis for these patients was major depression, adjustment disorder with depressed mood, or panic disorder. For initial evaluations, 71% (17/24) chose in-person care and 29% (7/24) chose telepsychiatric care. For follow-up appointments, 65% chose in-person care and 35% chose telepsychiatric care. The appointment adherence rate for in-person evaluations was 71% and, for telepsychiatric evaluations, the rate was 86%. The appointment adherence rate for in-person follow-up appointments was 87% and for telepsychiatric follow-up appointments was 79%. Patient satisfaction data

were collected on 22 in-person visits and 31 visits via telemedicine. There was no significant difference found by t-test analysis on patients' rating between telepsychiatric care and in-person psychiatric care. These data indicate that when given an initial choice, patients' prefer in-person psychiatric care, but patient satisfaction was equivalent for in-person mental health care and telepsychiatric care on direct (satisfaction survey) and indirect (adherence rate with appointments) measures in this small sample.

Telemedicine appears to be a viable way of increasing access to mental health care in rural settings. Some patients have reservations about telemedicine, which may not fit their overall concept of seeing a doctor or patients' concerns about, or lack of familiarity with, technology in general. There are also subtle interactions that patients value in the doctor-patient relationship that are not possible via telemedicine (e.g., the significance of a handshake).

A potential pitfall to telemedicine consultation is that psychiatrists and primary care physicians do not develop a relationship by working "side-by-side" in the primary care clinic, even though joint sessions are possible. The importance of this can not be overstated, since referrals are usually made on the basis of the professional relationship. In addition, if psychiatrists are "present" only by telemedicine, they may not fully understand the complexities of primary care practice. ❖

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CME Questions

26. The risk of recurrence for a new episode of major depression is increased in which of the following circumstances?
a. Elderly patients
b. Patients with more prior episodes of depression
c. Longer duration of the index episode
d. All of the above

27. The risk of relapse for an elderly patient following successful treatment for a first episode of major depression is?
a. 20%
b. 30%
c. 40%
d. more than 60%

28. The percentage of patients who relapse during a 15-year observation period is?
a. 20%
b. 40%
c. 60%
d. 85%

29. Antidepressants with more significant cardiovascular effects are:
a. tricyclic antidepressants
b. serotonin reuptake inhibitors
c. nefazodone
d. bupropion

30. Adverse effects that occur when "typical"antipsychotics are used to treat agitation and aggression in the elderly include:
a. urinary retention
b. confusion
c. drug-induced Parkinsonism
d. All of the above

31. Depression has been shown to be roughly equivalent to left ventricular dysfunction as a predictor of increased mortality within six and eighteen months of myocardial infarction.
a. True
b. False

In Future Issues:

Management of Agitation