Post-Stroke Depression

Henry Olders, MD, FRCPC
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I want to thank you for inviting me to this conference. When Claudette Fletcher asked me to be a presenter, I protested that I didn't know a great deal about post-stroke depression. However, I began looking at the literature, and I think I'm not alone. It seems even the PSD experts don't know a great deal that's definite about this subject. With that said, let's look at what we do know:

Demographics of PSD (Slide 2)

Prevalence ranges from 20% to 60%, depending on the study. Compare this to a depression rate of 15% in elderly.

However, the prevalence of depression in medical-surgical inpatients ranges from 20 to 30%.

It has not been proven that depression after stroke is more common than depression after other physical illnesses in the elderly. The implication is that post-stroke depression may not be a separate entity; it may just be depression following any severe illness.

Underrecognition of PSD in clinical settings (slide 3)

In one study, only one of 113 patients with post-stroke depression was referred for psychiatric consultation and only one received antidepressant medication.

In a large rehabilitation hospital with over 200 stroke admissions per year, less than 10 on average received a psychiatric consultation for depression.

A study of stroke patients on a rehabilitation ward found that 68% of the patients were identified as depressed by a standard psychiatric interview, while 50% were depressed according to the Beck Depression Inventory. However, none of these patients was recognized as depressed by the rehabilitation team.

Two common errors contributing to under-recognition:

1. Dismissing depressive symptoms, such as anorexia, weight loss, fatigue, and insomnia, as part of the stroke.
2. recognizing that the patient is depressed, but viewing it as a normal reaction to the underlying illness: "You'd be depressed too if you had just had a stroke!" Since many, if not most, stroke patients are not depressed, it is simply not logical to view depression as a "normal" reaction to the illness.

So, who cares? Negative effects of PSD (slide 4)

- Increases morbidity and mortality
- PSD patients are less cooperative with treatment and more difficult to manage
- Adversely affects recovery of functional status and cognitive performance
- Worsens quality of life of patients and caregivers
- PSD may produce a true dementia by itself
- Treatment of PSD may benefit cognitive function

Risk factors for developing PSD (slide 5)

- Female gender
- History of previous depression
- History of previous stroke
- Living alone
- Social distress prior to the stroke
- Social inactivity
- Stroke-related risk factors
    - Intellectual impairment
    - Decrease in social activity
    - Pathological crying
    - Functional impairment

Prognosis

What happens if we don't treat post-stroke depression adequately or aggressively enough? Some people believe that, when cognitive impairment is associated with depression (what is called "pseudodementia"), the cognitive impairment will become irreversible if left too long. This is based on the principle, "If you don't use it, you lose it."

When stroke victims are assessed at 3 months, we find dementia in 30%. An important question is whether some of these are depressive pseudodementias that have gone unrecognized. Differentiating the two is very difficult.

Computerized EEG, evoked potentials, regional cerebral blood flow as measured by PET, or SPECT may help to distinguish Alzheimer's or vascular dementia from pseudodementia due to depression. However, in a stroke patient, the stroke may cause EEG or SPECT changes that mask the usefulness of these tests, which aren't very readily available anyway. So a frequent approach is to try an

antidepressant. A positive response, that is, an improvement in cognition, means it was a pseudodementia.

However, even when we suspect a pseudodementia due to post-stroke depression, chances are high that the depression will not respond to treatment, as antidepressant treatments are effective in only 60 to 70% of patients. ECT can bring about remission in 80 to 90% of depressed patients, but it is less likely to be used than medication. In those cases where the depression fails to respond to treatment, we are likely to say that it wasn't pseudodementia at all, but instead a depression of dementia. Is there a possibility that we just haven't been aggressive enough, or innovative enough with our treatments?

A further difficulty is that it often takes weeks for antidepressants to work. Luckily, there is a test for depression which works rapidly and will pick up 60 to 70% of depressions. It is also readily available and easy to use. That test is total sleep deprivation, which has been used to distinguish pseudodementia from true dementia. Simply keep someone awake all night; by afternoon of the next day, you'll see a response.

Diagnosis

The standard psychiatric interview remains the best diagnostic method. These days, psychiatrists are supposed to be applying a set of diagnostic criteria, such as the Diagnostic and Statistical Manual, version 4 (DSM-IV) of the American Psychiatric Association. This slide (slide 6) shows the diagnostic criteria for a major depressive episode.

Can you diagnose depression even when the patient cannot tell you his or her mood state? Yes; however, the clinical assessment must include information from family members, caregivers, nurses, the rehabilitation team and family physicians, along with observation of behaviour.

Are standard depression rating scales useful in PSD? Yes, for patients who do not have severe neurological or cognitive deficits that may alter the signs and symptoms of depression.

What about patients who deny their illness?

5% of post-stroke patients deny depressed mood even though they fulfilled the criteria for depression.

Does location of the lesion matter?

Early studies showed that left anterior lesions were more often associated with depression.

Other studies showed right hemisphere lesions were more likely to result in PSD

Still other studies found no association with right or left cerebral pathology.

What makes studies difficult to perform and to interpret

(slide 7) I've talked about difficulties in diagnosis, and about our lack of definitive knowledge about such things as the influence of lesion laocation. Why the lack of definitive knowledge? Well, studies on post-stroke depression are difficult to carry out and the results are hard to interpret. This slide lists some reasons why.

- What should be included in "stroke"? Atherothrombotic brain infarction, cerebral embolism, intracerebral hemorrhage, subarachnoid hemorrhage, transient ischemic attack, all of the above?
- What kinds of "depression" should be included? Eg "major depression" as per criteria in DSM-IV; "minor depression" otherwise called dysthymic disorder; depression as defined by a cutoff point on a rating scale? Does the sadness experienced by any individual who experiences an insult to their physical and mental integrity, which we call mourning and is labelled as an "adjustment disorder" in the official nosology, qualify as depression?
- We have no laboratory test to diagnose depression.
- Many strokes may be undiagnosed; for example, of 280 patients in the Framingham heart study cohort who had autopsy-proven stroke, 113 had no mention of stroke on the death certificate.
- The vegetative symptoms of depression, such as apathy, decreased energy, poor appetite, weight loss, or sleep disturbance, are mimicked by the physical sequelae of stroke.
- In those studies where verbal reports of depressed mood are important in diagnosis, depression may be underestimated because aphasic patients, or patients who deny depressed mood, will not be picked up. It has been shown that patients with right hemisphere lesions are more likely to deny depressed mood; this may bias studies towards finding more depression with left hemisphere lesions even when there is no underlying laterality.
- Studies which exclude patients with severe comprehension impairments may also underestimate PSD.

Treatment

(slide 8) I was unable to find studies on the use of psychotherapy in post-stroke depression. However, at least two psychotherapy modalities, cognitive-behavioral therapy and interpersonal therapy, have been shown effective in so-called "functional" depression, and they may also be effective in PSD, unless you believe that the brain lesion itself causes the depression.

Support and social activities

Stroke rehab programs with emphasis on support and social activities: only 41% rate of depression at 3 months, vs 54% for rehab programs focussing only on physical and speech therapy

Henry Olders
Comment [1]: PSD articles to get 12 oct 98.

Cyclic antidepressants

A study using nortriptyline found it was effective; however, 35% of patients failed to complete the study because of side effects.

Because stroke pts are usually elderly, they are poorly tolerant of the anticholinergic side effects; orthostatic hypotension can conceivably contribute to ischemic strokes.

Since stroke pts may be predisposed to seizures, avoid maprotiline because it lowers seizure threshold

MAOIs

Difficult to use because of effects on blood pressure

SSRIs

Likely to be better tolerated than TCAs

Avoid bupropion which can lower seizure threshold

Some can inhibit metabolism of cardiac drugs, such as coumadin and digoxin; levels of these other drugs must therefore be carefully monitored

Psychostimulants

Both amphetamine and methylphenidate have been found effective

In a restrospective chart review comparing methylphenidate to nortriptyline in PSD, 53% of the methylphenidate group experienced complete remission of symptoms, compared to 43% of the nortriptyline group. Methylphenidate acted in 2.4 days compared to 27 days for nortriptyline.

In general, methylphenidate acts rapidly, & has few side effects (typically increase in BP; subjective feelings of restlessness or agitation). There does not seem to be any problem with seizures in patients with neurological illness. None of the several studies seemed to express much concern about the possibility that an increase in blood pressure might cause problems in patients at risk of hemorrhagic stroke, but I'd be careful just the same.

A recent study, reported in the Archives of Physical Medicine and Rehabilitation, looked at 21 stroke patients, consecutive admissions to a community-based rehab unit. Methylphenidate at 15 mg bid, at 8 am & noon, or placebo, were given for 3 weeks, in double-blind fashion, along with physical rehab. Hamilton Depression Rating Scale and Zung Self-Rating Depression Scale scores improved, as did motor functioning and functional independence ratings, more with methylphenidate than with placebo.

Electro-Convulsive Therapy

Indicated for patients who do not respond to adequate trials of antidepressants; for those with high suicide risk; for psychotic depressions; or for patients who are not eating or drinking because of depression.

Considerably more effective than medication: response in 80-90% of patients (compare to 60-70% for antidepressants)

Stroke is not a contra-indication, although it is recommended to wait 3 months after the stroke

Exercise caution in people at risk of hemorrhagic stroke, as there is an acute spike of intracerebral blood pressure during the induced seizure.

In a retrospective study of ECT in 14 PSD patients, 12 had a marked improvement in depression. Of the 6 patients who had cognitive impairment, ECT resulted in improvement of both depression and cognitive function in 5 of the 6.

Relationship with sleep

I want to mention two studies which imply a connection between sleep and PSD. The significance will become apparent later on in my talk.

1. Fedoroff et al[1] found that morning depression, delayed sleep, subjective anergia were strongly associated with low mood and were relatively uncommon among stroke patients without a mood disturbance.
2. Stern & Bachman[2] found that sleep disturbance correlated well with dysphoric mood.

What we don't know about PSD

While we know a little about PSD, there's a lot we don't know. Some of the important questions still to be answered include (slide 9):

- Is lesion location important in determining whether a stroke patient will become depressed?
- How much of the cognitive impairment seen with PSD is reversible?
- Is PSD qualitatively different from depression associated with other medical illnesses in the elderly?

The bottom line: the experts don't know what causes post-stroke depression. But this shouldn't be a surprise: the experts don't know what causes depression!

Here's what I think. I believe that depression may be caused by too much sleep.

Not just any sleep, but too much REM sleep.

How can a person possibly have too much sleep? Isn't sleep good for you? And isn't it stress that causes depression? Don't a lot of depressed people have difficulty sleeping? For them, it sure doesn't seem to be too much sleep!

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[1] Fedoroff JP, Starkstein SE, Parkh FM, Price TR, Robinson RG. Are depressive symptoms nonspecific in patients with acute stroke? Am J Psychiat 1991 sep:148(9); 1172-6

[2] Stern RA, Bachman DL. Depressive symptoms following stroke. Am J Psychiat 1991 mar: 148(3); 351-6

Development of Post-Stroke Depression

Let's consider two scenarios: in the first scenario, an elderly person suffers a stroke resulting in severe impairment, requiring hospitalization. In the second scenario, the stroke results in only mild deficits, and the hospital stay is brief before the patient returns home.

Scenario 1: severe impairment

In the hospital, our patient spends most of the day and night in bed, at least initially. Under these conditions, it's easy to sleep longer than usual. Cytokines such as the interleukins which are produced during an Acute Phase Inflammatory Response, cause drowsiness, thus contributing to sleeping more. Some patients attempt to avoid the painful feelings they experience after a stroke as they become aware of their deficits, by escaping into sleep. If the person manifests anxiety or agitation, it is likely they will be sedated with benzodiazepines, such as ativan, and/or antipsychotics, such as haldol; these medications increase drowsiness and cause the patient to sleep more.

Now we have a patient who is sleeping more than usual, almost certainly getting more sleep than he or she needs. What happens when we get more sleep than we need? We develop difficulty falling asleep or staying asleep; this is called insomnia. And what do we doctors do when the nursing staff or the patient himself or herself reports difficulty sleeping? We prescribe sleeping pills, usually benzodiazepines, such as ativan, rivotril, serax, halcion, dalmane, and others. And of course, sleeping pills cause the individual to sleep longer.

Does too much sleep have any bad effects other than insomnia? It's not a simple question, because sleep has a number of components or stages, which can be identified by recording the electrical activity of the brain. An electroencephalogram recorded during sleep, called a polysomnogram when it also records the electrical activity of the muscles which move the eyes, shows that there are 5 distinct sleep stages, stages I thru IV of successively deeper sleep, and Rapid Eye Movement Sleep, REM sleep for short. This slide shows how sleep is organized, at 3 different stages of life (slide 10).

REM sleep typically takes up 20 to 25% of total sleep time. The first period of REM sleep usually occurs 90 minutes after falling asleep, and subsequent REM sleep episodes occur at 90 minute intervals. REM sleep seems to be the only component of sleep which is necessary for normal functioning of the brain, probably for long-term memory. It seems that REM sleep is also the only component of sleep where too much is bad for you. This shouldn't come as a surprise; consider all the other things that our bodies need: food, water, vitamins and minerals, sunshine, oxygen, and so on. Too much of any of these necessities of life is bad for us. Too much food leads to obesity, diabetes, high blood pressure, heart disease, etc. Drinking too much water can cause seizures. Too much of certain vitamins is dangerous. Too much oxygen in premature infants causes blindness. Too much sun causes cataracts and skin cancer.

In the same way, even though we must have some REM sleep, too much is bad for us. In what way? It seems that too much REM sleep can cause a clinical depression, in people who are genetically predisposed. In the rest of us, too

much REM sleep seems to cause minor depressive symptoms, especially fatigue or lack of energy or lack of motivation.

So, the average elderly person will function well with perhaps 6 hours of sleep at night. When they suffer a stroke and are hospitalized, the number of hours spent sleeping increases to 8 or 10 or even more, per day. Since REM sleep takes up say 25% of total sleep time, adding 4 hours of sleep will add 1 hour of REM sleep.

But it's not quite so simple. It turns out that the amount of REM sleep a person gets increases throughout the night, from a minimum in the early evening to a maximum in the morning around 8 am, but the actual timing of the peak is probably tied to the time of sunrise. REM sleep follows a diurnal rhythm, and we know that the signal which most strongly programs our diurnal rhythms is light.

Thus, if you were to sleep 6 hours, from midnight to 6 am, about 25%, or 1.5 hours, of your sleep might be REM sleep. If your 6 hours of total sleep were taken earlier in the night, say from 9 pm to 3 am, the percent REM would be less, say 15%, for a total of 0.9 hours of REM sleep. But if you took the same 6 hours of sleep later in the morning, from 5 am to 11 am, the percent REM would be considerably higher, for example 40%, giving 2.4 hours of REM sleep. Thus, "sleeping in", staying in bed past sunrise, may lead to excessive REM sleep. For example, many teenagers tend to sleep very late on weekends; when they finally get up, they have little energy or motivation, until late at night when they get into a party mood.

Our stroke patient may be not only sleeping more hours than usual, it is also highly likely that some of those sleep hours occur after sunrise, when REM sleep is at its peak. For example, many hospitalized patients nap after breakfast.

Scenario 2: mild impairment

The patient who is able to return home shortly after the acute event is much less likely to be spending all day in bed than the hospitalized patient. However, there is still a risk of getting too much REM sleep. During the acute phase, the patient will be sleeping more; it is easy to get into this habit, and also to get into the habit of sleeping late, when the usual routine of work, hobbies, volunteer work, Golden Age, and so on is disrupted by the stroke.

As we saw earlier, sleeping longer and sleeping late both increase the amount of REM sleep. Sleeping longer may lead to insomnia, and excessive REM sleep causes, at the least, fatigue.

Many people assume, incorrectly, that their insomnia means that they're not getting enough sleep. And they further assume, again incorrectly, that their fatigue is proof that they're not getting enough sleep. So what do they do? They increase their time in bed, they stay in bed later, and they take sleeping pills! All of these interventions increase sleep time, making the insomnia worse; and they increase REM sleep, making the fatigue worse. Thus, a vicious cycle sets in. If the person is genetically predisposed to depression, the excessive REM sleep brings on a clinical depression.

The following slides are from a study I did with cancer outpatients, who received a questionnaire asking them about sleep habits, fatigue, and depression. This

slide (slide 11) shows how the people who got up later, in this case after 7 am, had significantly higher scores on the Beck Depression Inventory.

This slide (slide 12) looks at two aspects of fatigue. Again, as people get up later, their self-rated fatigue goes up. Now, this study simply picked up on an association between sleeping later and depression and fatigue; it can't tell us anything about cause and effect. You could argue that the depression and fatigue are responsible for the patient getting up later.

We also looked at attitudes about sleep. Here (slide 13) are some results. I suggest that many of the people who get up late, do so because of their beliefs about sleep and what they should do if they don't sleep well.

Treating Depression by Reducing REM Sleep

If too much REM sleep can cause depression, could we treat depressions by reducing REM sleep? A lot of evidence suggests that this works. For example, almost all antidepressant medications strongly suppress REM sleep. So do other antidepressant treatments, including electroconvulsive therapy, stimulants such as methylphenidate, and even exercise, which in some studies is as effective as medication against depression. But by far the simplest way to reduce REM sleep is simply to sleep less, or to sleep earlier in the night. It turns out that total sleep deprivation is a very effective treatment for depression, as is partial sleep deprivation when the person gets up earlier than usual. Unfortunately, even though staying awake all night leads to a total remission of depressive symptoms in about 70% of patients the very next afternoon, when the person has a sleep which includes REM sleep, the depression returns.

Practical issues in treating PSD

So, how can we apply this business of excessive REM sleep to our patients with PSD?

Evaluation

Evaluate for depression

First, evaluate for depression, ideally with a psychiatric interview, supplemented possibly by standard scales, whether completed by the patient himself or herself, such as the Beck Depression Inventory, or by a health professional, such as the Hamilton Depression Rating Scale.

This slide (slide 14) shows important diagnostic categories based on the degree of depression, and important features which must be evaluated and documented.

- Major depression
- Dysthymia
- Subsyndromal Depressive Disorder
- Bipolar Affective Disorder, Depressed Phase

- Presence of psychosis
- Presence of agitation
- Presence of suicidality
- Presence of self-neglect
- Presence of homicidality

Sleep history

The next step is to obtain a careful sleep history. This is important even when there is little or no depression, but there is significant fatigue, lack of energy, or lack of motivation.

This slide shows the sort of questions you want to ask (slide 15)

Quality of sleep

Time of going to bed

How long to fall asleep

Waking during the night

- Reasons for waking
- Number of times
- How quickly to fall asleep again

Time of waking in the morning

What does the person do after waking?

Time of getting out of bed for the day

Daytime drowsiness/sleepiness

Very important; failure to identify and appropriately refer cases who subsequently have accidents can engender civil, possibly criminal liability.

- How often
- What time of day or evening
- How distressing
- Accidents/near-accidents due to drowsiness

Fatigue/tiredness (differentiated from drowsiness/sleepiness)

- How often
- What time of day or evening

- How distressing
- Impact on life (work, play, relationships)

Daytime naps

- What time of day
- How long
- How often
- How long to fall asleep

Use of sleeping aids

- Prescribed medications (including somebody else's pills)
- OTC drugs
- Alcohol or other substances

Frequency of sleep problems

- Difficulty falling asleep
- Difficulty staying asleep
- Waking too early
- Inability to get up

How distressed by their sleep problem

Sleep habits when working and doing well

Attitudes towards sleep

- If you feel sleepy, does that always mean you're not getting enough sleep?
- If you feel fatigued, does that mean you need more sleep?
- If you sleep poorly at night, do you make up for it by sleeping late or by taking a long nap?
- If you've slept poorly, would you go in to work late or call in sick?

I particularly want to emphasize a couple of things: first, the arising time. Many patients with depressive symptoms will tell you that they wake up very early. It's important to persist, to ask what the person does when they wake up, and to ask if they go back to bed. I can't give you numbers, but many people with chronic illness will tell you if you are persistent, that they go back to bed, perhaps after breakfast, and they may not get up until 9 or 10 am, or even later.

A second important question has to do with their typical sleep habits when they were well, not depressed, and working.

Intervention

Fatigue or mild depression

If the patient experiences fatigue, lack of energy, or lack of motivation, or is mildly depressed:

Early rising

Symptoms of fatigue or depression can be treated by reducing REM sleep.

Although antidepressants, exercise, psychostimulants, and ECT suppress REM sleep, avoiding sleep after sunrise is an effective, inexpensive, and safe method to decrease REM sleep.

What I ask patients to do is to get up at 6 am, and to not sleep or take naps until at least after lunch. To prevent what are called microsleeps, they should avoid even lying down during this same time.

This is also a worthwhile first intervention for the stroke victim who is fearful of taking antidepressant medication, or refuses it.

People who are taking sleeping pills may find it impossible to get up earlier. In this case, it is essential to gradually taper the sleep medication dose.

Daytime sleep

Controlling daytime sleep is essential to making this approach work. Research shows that short naps are refreshing, relieve drowsiness, and increase alertness and feelings of wellbeing. Longer naps can induce sluggishness and torpor, and may also impair nighttime sleep.

Approach: when patients feel drowsy or sleepy, they should have a brief nap, ie lie down for not more than 15 or 20 minutes (a kitchen timer may be useful for waking up). If the person has not fallen asleep after 15 minutes, they do not need a nap; if they fall asleep after 5 or 10 minutes and thus sleep for only a few minutes, they will be able to get going again easily.

Major depression

If the patient is suicidal, homicidal, severely agitated, psychotic, or neglecting self, then hospitalize; commitment if dangerous to self or others

Most depressed patients can be started on an SSRI, such as paroxetine, starting at 10 mg in the morning, ideally at 6 am, and taken with food. Increase to 20 mg per day after one week. If severe side effects at 10 mg, decrease to 5 mg, increase to 10 mg after one week.

Reassess paroxetine after 3 weeks: increase dose of paxil by 10 mg in the morning if poor response and side effects are low or tolerable.

Split dose (eg 30 mg am and 10 mg pm at supper) for daily doses above 30 mg.

After an adequate trial of paroxetine (typically 6 to 8 weeks at 40 or 50 mg daily), consider augmentation therapy such as adding desipramine 25 mg, lithium, triiodothyroxine, or pindolol. Alternatively, switch to another antidepressant.

For psychosis or agitation, add olanzapine starting at 2.5 mg qhs; increase to 5 mg qhs after 1 week (sooner for inpatients).

For elderly or frail patients: use lower doses, possibly only half of usual adult dose.

For bipolar patients, if there is a risk of an antidepressant-induced switch into mania, use low doses of antidepressants, monitor very closely for symptoms of mania, consider starting a mood stabilizer prior to or along with the antidepressant.

Associated hypersomnia

When your evaluation reveals excessive sleep, particularly morning sleep, or lethargy, fatigue, or lack of drive, consider adding methylphenidate. This can be helpful all by itself if the usual antidepressants cannot be used. Avoid psychostimulants if anxiety or agitation are problems.

Methylphenidate is also useful in insomnia. By inhibiting daytime sleep, the patient may experience better nighttime sleep.

Usual starting dose is 5 mg bid, in the morning and at noon, again, ideally the first dose is taken at 6 am; increase at weekly intervals up to maybe 15 or 20 mg bid (some people reportedly go up to 70 or even 90 mg daily).

Associated insomnia:

When the sleep history identifies significant insomnia, I recommend (slide 16):

Sleep hygiene measures

- Arise at the same time each day.
- Limit daily in-bed time to “normal” amount.
- Discontinue use of drugs that act on the central nervous system; eg, caffeine, nicotine, alcohol, and stimulants.
- Avoid daytime napping except when sleep diary indicates a better night’s sleep as a result.
- Establish physical fitness with a routine of exercise early in the day, followed by other activity.
- Avoid evening stimulation; substitute either listening to the radio or leisure reading for watching television.
- Try a warm 20-minute body bath or soak near bedtime.
- Eat on a regular schedule; avoid large meals near bedtime.
- Practice an evening relaxation routine.

- Maintain comfortable sleeping conditions.
- Spend no longer than 20 minutes awake in the bed.
- Adjust sleep hours and routine to optimize daily schedule and living situation.

Sleep restriction/sleep compression

In addition to the usual sleep hygiene measures, I recommend an approach which has been shown by a number of studies to be the most effective treatment for what is called psychophysiological, or primary, insomnia. This intervention is called sleep restriction by some, and sleep constriction by others, but the technique is the same:

Start with the person's own estimate of how many hours of sleep they get in 24 hours; they should then spend only that many hours in bed each night.

Once they have been sleeping well for a week at a given stage, increase the time in bed by a half-hour.

Daytime drowsiness can be relieved by short naps, as I talked about earlier.

Light

The most important cue to adjusting the biological clock is the length of the day/night cycle.

Individuals who are insufficiently exposed to light, especially morning light, may develop delayed sleep phase syndrome. This can occur if people keep their bedrooms dark, or possibly even if windows are small or views obstructed with trees, fences, walls, etc.

Approach: encourage patients to keep their drapes/blinds open at night while they're asleep. When buying or renting housing, look for bedrooms that receive plenty of natural light.

Caffeine

Caffeine may induce its own metabolism, thus individuals who use small amounts, or who use it irregularly, may have more severe and longer-lasting effects, such as insomnia or anxiety, than those who consume larger quantities.

Caffeine acts as a mood elevator: in the ongoing Nurses' Health Study involving about 90,000 nurses in the U.S., those who drank no coffee committed suicide at 2 1/2 times the rate of those who drank 2 or more cups daily.

Approach: encourage consistent caffeine consumption, eg 2 cups coffee daily.

Exercise

Vigorous cardiovascular (ie aerobic) exercise of at least 30 minutes stimulates endorphin production, which makes the person feel good, and suppresses REM sleep, which has an antidepressant and antifatigue effect. For patients who are capable, walking, jogging, aerobic routines or aerobic dance, swimming, cross-

country skiing, or cycling. Aquafitness is often useful for people with functional limitations. Use your imagination!

If not engaged in too near bedtime, exercise can also improve sleep.

Hypnotic medications

Benzodiazepines can be dangerous in elderly patients: risk of falls, automobile accidents, cognitive impairment, disinhibition, depression, and dependence.

For dependent individuals, taper very gradually.

For those people who have difficulty sleeping and are unable or unwilling to comply with sleep hygiene and sleep restriction, trazodone (25 or 50 mg hs) is an effective hypnotic, even though relatively ineffective as an antidepressant.

That's the end of my talk. Thank you for being such a great audience.