RESTLESS LEGS SYNDROME:
GUIDELINES FOR DIAGNOSING AND TREATMENT

By
KATHY SCAMMELL MORTON

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To the Faculty of Washington State University:

The members of the Committee to the thesis of Kathy S. Morton find it satisfactory and recommend that it be accepted.

[Signatures]

Chair

[Signatures]
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Abstract

Restless legs syndrome is a sleep disorder characterized by unpleasant, deep-seated paresthesis in the legs. Patients have an irresistible urge to walk or move their legs to relieve the discomfort. Symptoms occur primarily at night and become more intense after the patient goes to bed. Patients describe these sensations as tingling, crawling, burning or aching. The myoclonic jerking associated with Restless Legs Syndrome (RLS) causes the patient to be aroused several times during the course of a night’s sleep, resulting in decreased quality of sleep. In most cases, onset of symptoms is not associated with any neurologic, metabolic or circulatory disturbance. A positive family history exists in 30% to 50% of the patients with RLS. Secondary causes of RLS include iron deficiency anemia, renal insufficiency and folate deficiency. Treatment is aimed at relieving the symptoms or ameliorating them. Dopaminergic drugs are the agents-of-choice due to their efficacy in treating the symptoms of RLS. Nurse practitioners can effectively diagnose and treat RLS by recognizing the signs and symptoms. The goal of treatment is aimed at improving the patients overall quality-of-sleep and alleviating anxiety associated with RLS.
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Sleep is an active state essential for mental and physical restoration. More than 100 million Americans of all ages regularly fail to get a good night’s sleep (American Sleep Disorders Association, 1994). Approximately, 40 million Americans suffer from sleep disorders. Sleep disorders, sleep deprivation and sleepiness adds approximately $15.9 billion to America’s health care bill each year. The consequences of sleep disorders include reduced productivity, lowered cognitive performance, increased likelihood of accidents, higher morbidity and mortality and decreased quality-of-life. A few examples of sleep disorders include narcolepsy, sleep apnea, restless leg syndrome and insomnia (SleepNet, 1997).

Restless leg syndrome is a neurosensimotor sleep disorder that has been documented as early as the 17th century. Restless Leg syndrome (RLS) is the fourth leading cause of insomnia (Brodeur, Montplaisir, Godbout & Marinier, 1988). It is characterized by unpleasant, deep-seated paresthesia in the legs and rarely affects the arms. Involvement can either be unilateral or bilateral and symmetrical. The most unusual, but distinctive feature of RLS is that the sensory and motor symptoms are worse when lying down or sitting. Patients have an over-whelming urge to move their legs to relieve the discomfort. These symptoms make it
difficult for patients to get to sleep or stay asleep. RLS leads to significant insomnia with subsequent daytime sleepiness, anxiety and depression.

RLS occurs in approximately 10-15% of the population, but it appears that these numbers are on the rise (Lavigne & Montplaiser, 1994). RLS occurs in all ages with equal gender distribution. The disease is usually progressive with symptoms beginning at any time from infancy to extreme old age. For the elderly, the condition can be tormenting, because their inability to pace or even stand to relieve these restless sensations.

The diagnosis of RLS is based on clinical signs and symptoms. For practitioners unaware of the disease, initial misdiagnosis or lack of diagnosis is a common problem. Most patients reported symptoms before the age of 20 years (Walters, Picchietti, Ehrenberg & Wagner, 1994). Patients typically did not seek medical attention until the fourth decade of life. A survey by the Restless Leg Syndrome Foundation revealed that sufferers had tried an average of four to five physicians over a period of 15 years before finding adequate treatment. A correct diagnosis was made on an average of two years after seeking medical attention and 22% of patients reported that they did not believe they had a treatable condition (Walters et al., 1996). When patients sought medical attention,
misdiagnosis included skin irritation, arthritis or malingering. In the younger patient, misdiagnosis included “growing pains” and attention deficit hyperactivity disorder. In most cases, no neurologic, metabolic, psychogenic or circulatory disturbance could be found that would explain the onset of symptoms (Coccagna, Lugaresi, 1981).

Treatment is aimed at relieving the symptoms of idiopathic RLS since there is no cure for the disease. Treatment should be considered when patients are functionally impaired by symptoms and complain of sleep disturbances or excessive daytime sleepiness. The large number of treatments available, is a testimony to the difficulty in treating RLS. Treatments range from mild stretching exercises to the use of several pharmaceutical agents. A treatment that is a miracle for one patient may have no effect or increase the severity of the symptoms in another. The best treatment may be arrived at, only after active experimental cooperation between the practitioner and patient (Hening, 1995). The subjective nature of the symptoms contributes to the difficulty in formulating a treatment plan for the patient.

The purpose of this article is to analyze and synthesize current research on diagnosis and treatment of RLS. Nurse practitioners need to be aware of symptoms of RLS associated with this sleep disorder.
Patients may be reluctant to talk about their sleep patterns. They may have already been told that their symptoms are due to psychogenic causes. Many patients are unaware of their disruptive sleep patterns until their partner complains of their frequent leg jerking during sleep. Many RLS patients suffer from significant insomnia that results in a disrupted lifestyle.

Pathophysiology

To understand RLS, a brief overview of sleep will help pinpoint how RLS affects sleep. Sleep like wakefulness is an active, complex process involving specific brain regions. Those parts include the thalamus, the medulla oblongata and the pons. Another important area of the brain is the suprachiasmatic nuclei, located in the hypothalamus. The reticular formation, located in the pons and upper brain stem, contains several neurotransmitters that play an important role in behavioral arousal (noradrenaline, dopamine, acetylcholine, histamine, glutamate and aspartate). Certain other neurotransmitters have been identified that actively inhibit the reticular activating system (GABA and adenosine). In addition, there are other "sleep substances" that can induce, modify or influence sleep. Certain peptides such as opiate peptides, alpha-acetyl-MSH (melanocyte stimulating hormone) and somatostatin may have a role
in promoting sleep onset. Such substances as insulin, cholecystokinin, prostaglandins, interleukins, growth hormone and prolactin when released into the blood also have sleep promoting properties (Basics of Sleep Behavior, 1993). These substances are important in the treatment of RLS, especially when the treatment involves different pharmaceutical agents and their pharmacodynamics.

Sleep can be separated into two specific types: rapid eye movement (REM) and non-REM sleep. Non-REM sleep consists of four broad stages. One method to describe these stages is through electroencephalogram (EEG) monitoring. Stage 1 is characterized as light sleep, the person is not responsive, but is aware of their surroundings. Stage 2 is K-spindle sleep. Stages 3 and 4 are characterized by high voltage slow brain wave activity on the EEG. A larger stimulus is usually required to produce an arousal for these stages. Stages 3 and 4 are also known as slow wave sleep or delta sleep (Kruger, Roth, & Demert, 1989).

The cyclic alteration of non-REM and REM sleep constitutes the basic sleep pattern (Richards, 1996). During an early cycle of sleep, approximately 80 minutes is spent in non-REM sleep followed by 10 minutes in REM sleep. This cycle is repeated 3-6 times during the night. As the night progresses, individuals spend less time in Stages 3 and 4,
with these stages disappearing altogether in later cycles. Stage 2 sleep expands and occupies a greater portion of non-REM cycle. Generally, REM cycles become progressively longer (Kryger, Roth & Dement, 1989). Sleep latency refers to the time it takes to fall asleep while sleep efficiency is the total time spent asleep. In a study of 133 patients with RLS, a high correlation was found between what the patients noted on a questionnaire regarding their symptoms of RLS and polysomnographic (PSG) recordings (Montplaisir et al., 1996). The PSG recordings demonstrated that these patients did indeed have prolonged sleep latencies and increased number of awakenings when compared to the control group. RLS and periodic leg movement in sleep, appear to affect Stages 1 and 2 of non-REM sleep and absent in Stages 3 and 4 and REM sleep (Kryger et. al. 1989).

Etiology

Since the 17th century, sleep researchers have sought to pinpoint the exact etiology of RLS. Unfortunately, the etiology remains unknown. Sleep researchers have formulated several hypotheses on the mechanism behind RLS. Today most researchers agree that RLS and periodic leg movement in sleep (PLMS) originate in the central nervous system. High resolution functional magnetic resonance imaging (fMRI) has identified
cerebral generators associated with periodic leg movement and sensory leg discomfort in 19 patients with RLS (Bucher et al., 1997). When these patients complained of sensory leg discomfort, there was bilateral activation of the cerebellum and contralateral activation of the thalamus. High resolution functional magnetic resonance imaging demonstrated periodic leg movements in sleep in RLS patients, which were associated with overactivity in the cerebellum, the red nuclei and the brainstem. Reticular structures in the brainstem appeared to be the primary generators of RLS (Bucher, 1997).

A second hypothesis stems from the observation that patients with RLS symptoms improve in response to certain centrally acting substances (e.g. levodopa and opioids). The clinical response to these dopaminergic agents suggests involvement of the dopaminergic, adrenergic and opiate transmitter system (Wetter, Pollmacher, 1997).

Finally, the circadian rhythm influences patients sensory symptoms. Both sensory symptoms and PLMS increased in the evening and nighttime hours with the majority occurring between midnight and 3 am. The sensory and motor symptoms decreased in the later morning hours. RLS/PLMS symptoms appear more severe during the falling phase of the
circadian temperature cycle with symptoms decreasing as the temperature increases in the morning (Trenkwalder, 1996).

Clinical Features

Restless leg syndrome is characterized by unpleasant sensations in the legs, mostly inside the calves, which induce an irresistible urge to move the legs. These sensations generally involve the legs but occasionally affect the feet, thighs, arms and hands. Patients report that these symptoms occur either on the right or left side of the body. Rarely, are the symptoms bilateral (Montplaisir et al., 1996). Patients describe these sensations as tingling, crawling, aching, or painful. The distinctive feature of RLS is that the symptoms appear at rest. Symptoms occur predominately at bedtime interfering with a patient’s quality of sleep. Other triggering activities include movies, meetings, car trips, long-distance flights and even relaxation exercises. Relief is attained by frequent movement or stretching of the legs. Walking provides the most effective relief, followed by kicking, flexing or massaging the legs. Due to the decrease in the quality of sleep, patient’s primary complaints are excessive daytime sleepiness (EDS), anxiety, depression, confusion and slowed thought processes.
RLS is also characterized by motor disturbances, which include periodic leg movements in sleep (PLMS), brief myoclonic jerks and dyskinesias. PLMS are regular, jerky, unilateral or bilateral movements that cause involuntary repetitive extension of the big toe and are associated with flexion of the hips, knees and ankles. A diagnosis of PLMS is significant because almost 100% of the patients diagnosed with RLS also have PLMS (Wetter & Pollmacher, 1997). Patients may be unaware of these movements until their bed partner complains of these kicking movements. PLMS occur primarily in Stages 1 and 2, decreasing in Stages 3 and 4. It is the duration of these movements that causes significant problems with sleep.

Differential Diagnosis

Diagnosis of RLS is based on a thorough patient history and physical examination. The sleep partner should also be included in the clinical history with emphasis on leg or body jerks, restless sleep, insomnia or excessive daytime sleepiness. The International Restless Legs Syndrome Study Group, composed of 28 investigators from seven countries in 1995, established the four main criteria necessary to establish a diagnosis of RLS (Walters, 1995). The first, is a desire to move the limbs, usually associated with paresthesias/dysesthesias. The second is
motor restlessness, (involuntary movements) because the patient feels compelled to move to relieve the symptoms. These movements are voluntary however, because the patient chooses which movements to make. Thirdly, symptoms are worse or exclusively present at rest with at least partial and/or temporary relief with activity. Fourth, symptoms are worse in the evening or at night.

Additional clinical features include sleep disturbances such as prolonged sleep latency, difficulty maintaining sleep and daytime fatigue. Next are involuntary movements. These may involve periodic leg movements either while awake or asleep. Neurologic examination reveals no abnormalities. Routine electromyogram (EMG) and nerve conduction studies are normal. Family history may reveal that the affected individual may have first-degree relatives with the same symptoms. Parent-child transmission and occurrence in both genders suggests an autosomal-dominant inherited condition. In families with idiopathic RLS, 40 to 60% of the patients have at least one first-degree relative affected with RLS (Lavigne & Montplaisir, 1994). Though the condition can occur at any age yet it is more prevalent in the elderly (Walters, Picchietti, Ehrenberg & Wagner, 1994).
There are several substances that can trigger or worsen RLS. These include the D2 receptor antagonists (classical neuroleptics) and tricyclic antidepressants (Wetter & Pollmacher, 1997). Finally, secondary causes of RLS need to be ruled out. These include end-stage renal disease, iron deficiency anemia, folic acid deficiency, peripheral neuropathy, venous insufficiency, rheumatoid arthritis, diabetes, hypothyroidism, hyperthyroidism, elevation of parathyroid hormone and pregnancy.

RLS occurs in 12% to 15% of all pregnant women. Although the condition is reversible after the delivery, some women will go on to develop idiopathic RLS later in life (Trenkwalder, Walters, Hening, 1996). Other women will only have this condition during pregnancy.

Diagnostic Studies

Once a thorough clinical history has been obtained from the patient, the next step is to rule out secondary causes of RLS. A polysomnography is indicated when a diagnosis of periodic limb movement is considered, due to patients or bed partners complaints of jerky legs, difficulty sleeping and excessive daytime sleepiness. Laboratory tests include renal function panel, serum iron, ferritin, complete blood count, fasting blood sugar, TSH and folic acid. Needle electromyography and nerve conduction studies
need to be ordered if polyneuropathy is suspected. Finally, the diagnostic work-up should include techniques to rule out vein disease. Kanter (1995) acknowledges that vein disease can be rule out by a knowledgeable examiner using continuous-wave Doppler. He states that the symptoms of RLS and venous insufficiency often overlap, making the diagnosis difficult.

Treatment

Treatment of RLS is generally symptomatic and should be initiated when the patient complains of excessive daytime sleepiness, sleep disturbances and severe symptoms while awake. Primary treatment for RLS currently includes the use of dopaminergic agents, benzodiazepines, opioids, anti-seizure agents and hypertensive agents. Dopaminergic agents are the drugs of choice due to their efficacy in treating the symptoms of RLS. Dopaminergic drugs include levodopa in conjunction with a dopa decarboxylase inhibitor (DDCI), such as carbidopa or benzerazine.

Brodeur et al. (1988) in a study of six patients with RLS reported a noticeable decrease of sleep latency, leg and armparethesia when giving a daily dose of 100 mg levodopa/25 mg benzerazine. Patients reported an
improved quality of life with fewer nocturnal awakenings during clinical interviews.

Trenkwalder et al. (1995), in a study of 28 patients diagnosed with RLS, administered a single dose of 100-200 mg levodopa/25-50 mg benserazide at bedtime. He noted a reduction in the number of periodic leg movement arousals in the first four hours after intake. After four hours, the efficacy of the drug disappeared.

Several side effects occur with the administration of levodopa. The first is rebound, defined as an increase in symptoms in the morning or after awakening. Guilleminault, Cetel and Philip (1993) monitored 20 patients taking a nighttime dose of Sinemet CR 50/200 over a 7-week period. He noted the emergence of symptoms in the morning hours after awakening. Administering a dose of sustained-release medication during the daytime alleviated these symptoms. A second side effect, augmentation, is defined as the appearance of RLS symptoms starting earlier than they did prior to treatment. Augmentation differs from rebound in that augmentation occurs in a retrograde fashion rather than an extension pattern as seen in rebound. As augmentation becomes worse, the symptoms started earlier in the day. These symptoms persist throughout the day, unless treated with medication or patient activity (Allen & Earley, 1996). As the dosage of
carbidopa/levodopa is increased, the symptoms are noted earlier in the day. If this becomes problematic for the patient, either using a lower dosage of carbidopa/levodopa or alternating medications may be indicated. Finally, side effects such as dry mouth, headaches, gastrointestinal symptoms and increased wakefulness have been documented.

For patients who do not respond to levodopa or carbidopa/levodopa, bromocriptine (Parlodel) may be an alternative choice. In a double blind, randomized crossover trial of bromocriptine and placebo, bromocriptine was shown to be effective in decreasing periodic leg movements (Walters et al., 1988). This study consisted of six patients, who were given a total dose of 7.5 mg of bromocriptine in divided doses over a period of 30 days. The number of periodic leg movements per hour of sleep decreased 57% from those who received placebo. Patients spent an extra 10-15% of their sleep period in stages 1 and 2 (light sleep). Bromocriptine may decrease periodic leg movements in sleep and parethesias (Walters, 1988).

Earley and Allen (1996) studied the use of pergolide, another dopamine agonist, for managing RLS. Pergolide had three major advantages over bromocriptine, (1) pergolide had a longer duration of
efficacy, (about 10-12 hours) than bromocriptine (about 5-6 hours) (Walters et. al. 1988), (2) bromocriptine statistically failed to relieve the symptoms of RLS and (3) bromocriptine cost more than pergolide. Seventy three percent of the patients were successfully treated with pergolide despite the fact that these same subjects failed to achieve satisfactory treatment with carbidopa/levodopa. Patients with more severe symptoms showed a 94% success rate compared to 23% for those on carbidopa/levodopa regimen. A common side effect, restless leg augmentation, occurred in only 15% of the patients on pergolide compared to 59% on carbidopa/levodopa (Earley, 1996).

Benzodiazepines were among the first medications to be used in the treatment of RLS (Matthews, 1979). Clonazepam is the most commonly prescribed drug for treating RLS, especially in milder cases and in younger patients. Mitler, Browman, Menn, Gujavarty and Timms (1986), in a study of ten patients over a three year period, reported that the total number of leg movements were unaffected by clonazepam and temazepam. However, sleep was improved by reducing the tendency to arouse after a leg movement. Read, Feest and Nassim (1981) found that patients with RLS due to uremia, reported decreased symptoms when benzodiazepines were used. Out of fifteen patients studied, six reported
complete relief of symptoms after the first dose and eight others responded following an increase in dosage. The most common side effect noted with the benzodiazepines was daytime somnolence along with ataxia and confusion in the elderly patient.

Opioids offer another alternative in the treatment of RLS. Walters et. al. (1993), in a randomized double-blind study of 11 patients, compared the effects of oxycodone with a placebo. The average dose was 15.9 mg, given in divided doses over a 2-week period. The patients then were studied polysomnographically under double-blind conditions. Results showed a decrease in paresthesias, motor restlessness, nighttime arousal’s and periodic leg movements in sleep. These differences were attributed to utilizing dosages that produced a therapeutic effect. Hening et. al. (1989) found that eight of his 10 patients continued treatment with opioids without significant side effects from 6 months to 15 years. Montplaisir et. al. (1992) recommends using opioids when other drugs fail to control the symptoms or when the side effects are intolerable. Start with the lowest dosage possible and monitor for any development of tolerance or dependency (Montplaisir, 1992).

Alternative drug choices include carbamazepine and clonidine. Telstad et al. (1984) in a double-blind study with 174 patients, found both
carbamazepine and placebo had a significant effect on restless legs ($p<0.01$). A positive correlation was found between the reduction in severity of symptoms and serum concentration of carbamazepine. The researchers noted the syndrome to be more common in middle aged women with low systolic blood pressure. Carbamazepine did not achieve the relief documented by dopaminergic or opioid therapy (Telstad, 1984). Further double-blind, controlled studies need to be done to document the efficacy of carbamazepine.

Clonidine can be used in patients with idiopathic RLS. Nine patients diagnosed with RLS were monitored over a 4-week period (Wagner et al., 1994). Patients reported improvement in leg sensations ($p<0.05$) and motor restlessness ($p<0.03$) while on clonidine (Wagner et al.). On polysomnography, sleep onset occurred faster (12 minutes) than placebo (30 minutes). REM sleep was decreased in the clonidine group. No significant changes were noted in sleep stages 3 and 4, sleep efficiency, awakenings, arousals or periodic leg movements during sleep.

**Conclusion**

The diagnosis of RLS remains a clinical one. The use of the four main criteria to diagnose RLS will help guide the nurse practitioner in making a correct diagnosis. If the nurse practitioner suspects other sleep
related disturbances along with RLS, a sleep study should be ordered. In secondary causes, the underlying condition should be treated first and further follow-up should be done to rule out idiopathic disease.

Treatment with dopaminergic agents is the most effective means of alleviating RLS symptoms. Brodeur et. al. (1988) suggested using a low dose carbidopa/levodopa agent, such as Sinemet SR for patients with mild symptoms of RLS. Patients may need a change in their dosage or the time of administration to relieve symptoms. Patients may also experience periods of remission where no medication is needed. If patients do not obtain any relief of their symptoms with a carbidopa/levodopa regimen, nurse practitioners can choose from other pharmaceutical options. These include the benzodiazepines, opioids and clonidine. How the nurse practitioner treats the symptoms of RLS will depend on the patient’s response to the treatment and adverse effects from these medications.
REFERENCES


The Sleep Research Society. (1993). Basics of sleep behavior. Los Angeles, California; UCLA.


Table 1

PHARMACEUTICALS TO TREAT RESTLESS LEGS SYNDROME

<table>
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<th>DRUGS</th>
<th>DOSAGE</th>
<th>SIDE EFFECTS</th>
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<tr>
<td><strong>DOPAMINERGIC</strong></td>
<td></td>
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<tr>
<td>Sinemet</td>
<td>Comes in tablets of 25/100, 10/100, 25/250. Start with 25/100, one tablet at night, may increase to 3 tablets/day.</td>
<td>Nausea, confusion, dizziness, hallucinations, dyskinesia.</td>
</tr>
<tr>
<td>Sinemet CR</td>
<td>Comes in tablets of 25/100, 50/200. Start with one dose of 25/100, 2 hours before bedtime. May take another dose during the daytime to control daytime symptoms</td>
<td>Rebound and augmentation. Keeping the dosage low and not more than 2-3 tablets per day will help eliminate these effects.</td>
</tr>
<tr>
<td>Bromocriptine</td>
<td>Comes in 2.5mg and 5mg tablets. Usual dosage is 5-15mg per day in divided doses. Start with 2.5mg at bedtime. Slowly increase to avoid side effects.</td>
<td>Nausea, hallucinations, confusion, hypotension, syncope, nasal stuffiness, headache.</td>
</tr>
<tr>
<td>Pergolide</td>
<td>Comes in 0.05mg, 0.25mg, 1.0mg. Start at 0.05mg daily. Increase by 1/2 to 1 tablet every 3 to 7 days till you reach a maximum dose of 1.5mg per day. Increase slowly to avoid side effects. Take one hour prior to onset of symptoms.</td>
<td>Hallucinations, confusion, hypotension, syncope, nasal stuffiness, nausea, headache.</td>
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<tr>
<td>DRUGS</td>
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<td>SIDE EFFECTS</td>
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<tr>
<td>BENZODIAZEPENES</td>
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<td>Clonazepam (Klonopin)</td>
<td>Comes in 0.5mg, 1 mg, 2mg tablets. Usual dosage range is 0.5mg to 2mg at bedtime.</td>
<td>Persistent daytime somnolence, confusion.</td>
</tr>
<tr>
<td>Triazolam (Halcion)</td>
<td>Available in 0.125mg and 0.25mg tablets. Usual dosage is 0.125 mg to 0.5mg at bedtime.</td>
<td>Rebound insomnias, short term amnesia.</td>
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<td>OPIOIDS</td>
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<td>Oxycodone (Percodan, Percocet.)</td>
<td>Available in 5mg tablets. Usual dose is 2.5mg to 20mg per day, divided into doses every 4 to 6 hours.</td>
<td>Constipation, nausea</td>
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<tr>
<td>Propoxyphene (Darvon, Darvocet)</td>
<td>Usual dose is 3 to 4 tablets per day.</td>
<td>Constipation, nausea</td>
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<td>ANTI-SEIZURE DRUGS</td>
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<td>Carbamazepine (Tegretol)</td>
<td>Comes in 100mg and 200mg tablets. Start at 100mg per day and slowly increase. Dosages range from 100mg to 300mg per day.</td>
<td>Dizziness, drowsiness, unsteadiness, nausea. Check CBC, liver function test and urinalysis for drug toxicity.</td>
</tr>
<tr>
<td>HYPERTENSION DRUGS</td>
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<td></td>
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<tr>
<td>Clonidine (Catapres)</td>
<td>Comes in 0.1mg, 0.2mg, 0.3mg tablets. Usual dosage is 0.1mg to 0.3mg at bedtime. May increase to 0.9mg per day in divided doses.</td>
<td>Dry mouth, drowsiness, dizziness and constipation</td>
</tr>
</tbody>
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