MedSleep

Sleep Matters™

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ABOUT MEDSLEEP

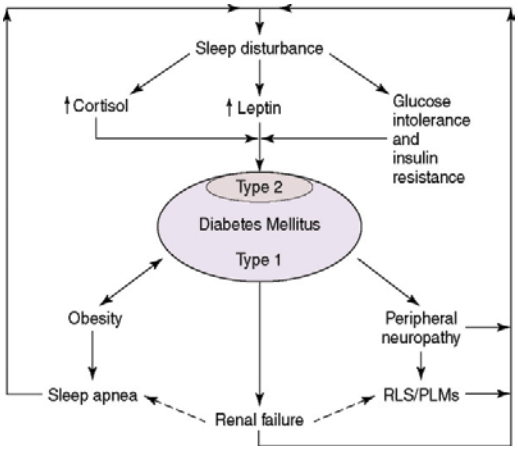
Founded in 2004, MedSleep is a network of Canadian sleep clinics committed to providing the highest quality sleep medicine services.

MedSleep clinics provide clinical consultation, diagnostic services (sleep testing) and treatment for the full spectrum of sleep disorders. Our management and technologist teams have had extensive experience providing sleep testing and training across Canada.

MedSleep Atlantic, formerly known as Atlantic Sleep Institute, was established in 2009 and has helped diagnose and treat individuals from communities across the Maritimes. We are proud to offer leading-edge technology and strive to provide the highest quality of sleep medicine services available. Level 3 testing is conducted at no fee.

Diabetes and Obstructive Sleep Apnea:
What have we learned about the relationship?

THE RELATIONSHIP between obstructive sleep apnea (OSA) and the spectrum of impaired glucose metabolism has received significant interest over the last few years. Several interventional studies have demonstrated improvements in glucose metabolism with the treatment of OSA, however the beneficial effect of OSA treatment on glucose metabolism has been lacking evidence of the improvement in long-term control. This may be because of complicating factors affecting this relationship, such as the severity of the impairment in glucose metabolism in the study population, poor treatment compliance to CPAP, and confounding factors such as obesity.



The Sleep Heart Health Study and the Wisconsin Sleep Cohort have offered the most convincing general population-based evidence of the association between poor glucose management and the development of OSA. In the Sleep Heart Health Study, Punjabi and colleagues demonstrated a 1.3-fold increased likelihood of impaired fasting glucose (IFG), a 1.2-fold increase likelihood for impaired glucose tolerance (IGT), a 1.4-fold increased likelihood for IFG and IGT, and a 1.7-fold increase likelihood for occult diabetes in those with OSA - even after adjusting for age, sex, BMI, race and neck circumference. They were also able to demonstrate an independent relationship between severity of OSA and degree of insulin resistance. The severity of hypoxemia was also associated with both insulin resistance and glucose intolerance.

Reichmuth and colleagues established a similar relationship in the analysis of the data from the Wisconsin Sleep Cohort. The study demonstrated a 2.3-fold increased likelihood of physician diagnosed diabetes among those with OSA. Furthermore, those with OSA had a 1.6-fold increased likelihood of developing diabetes after a 4-year period.

Theories about the mechanism of this relationship are complex and multi-factorial. There is evidence that the relationship exists due to the effect of intermittent hypoxemia on glucose management, derangement of the insulin counter-regulatory hormones epinephrine and cortisol, or from the effects on adipocyte-hormones such as leptin and ghrelin that regulate appetite and metabolism.

A number of subsequent studies were aimed at finding a causal relationship between the two conditions. Interventional trials have demonstrated benefits in enhancing insulin resistance, improvement in overnight glucose levels and even benefits to long-term control parameters such as hemoglobin A1c.

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What's inside?

What is CBT Insomnia Treatment?

COGNITIVE BEHAVIOURAL TREATMENT addresses maladaptive behaviours and dysfunctional thoughts that contribute to chronic insomnia. This treatment is particularly helpful in individuals who have developed a conditioned or psychophysiological insomnia.

Typically for these individuals, sleep has become a central concern with intense fear surrounding the consequences of not being able to sleep. As a result of this fear of not being able to sleep, patients enter the sleep environment already in a heightened state of physiological arousal and are quick to jump to anxiogenic thoughts – *i.e.,* "If I don't sleep tonight, I am going to be completely dysfunctional tomorrow!".

In addition, in an attempt to ensure a better sleep, they might go to bed earlier than usual despite not being sleepy, or take afternoon or evening naps. These catastrophic thoughts and compensatory sleep behaviour unfortunately results in a worsening of the insomnia (*see figure below*).

CBT is a structured therapy that can be provided on an individual or group basis. It typically involves 5 or 6 sessions and is presented in an educational format. Patients are instructed about normal sleep physiology, including homesostatic and circadian factors that promote healthy sleep. With this background, they are taught several behavioural techniques (*i.e.,* bed restriction, stimulus control therapy, relaxation training) to optimize these factors and avoid compensatory strategies that will aggravate their insomnia. In addition to learning behavioural techniques, they are taught how to constructively challenge the dysfunctional thinking that intensifies their nocturnal anxiety.

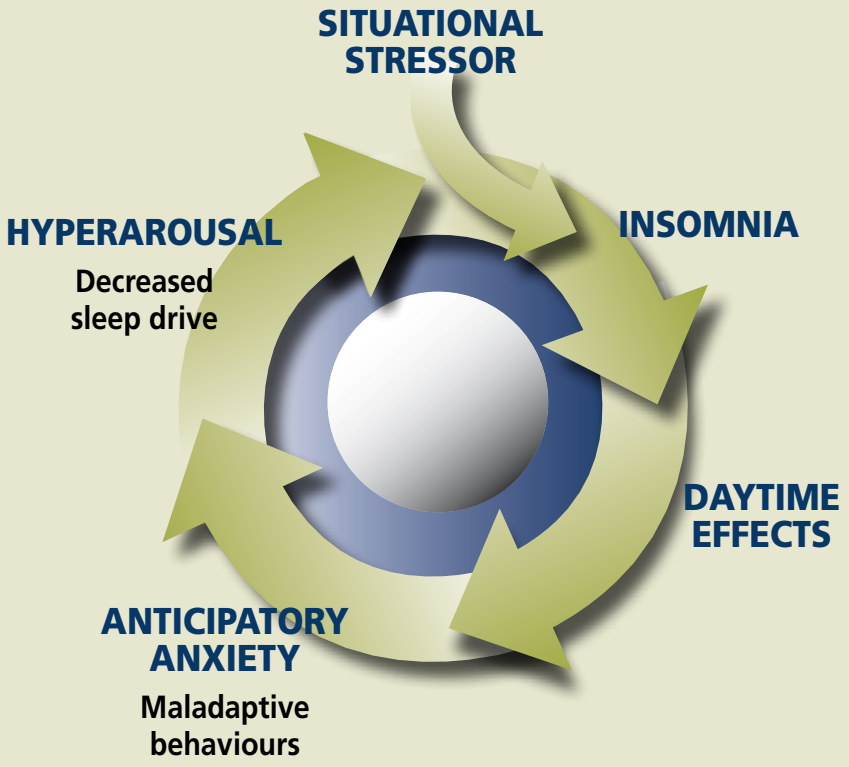
For example, a common scenario involves the tendency to catastrophize the impact of a bad night's sleep. Patients are gently challenged to examine whether in fact they have ever had satisfactory, productive days after a poor night of sleep.

Although short-term sleep restriction is unpleasant, it is usually time limited if one maintains optimal sleep scheduling, and it is not necessarily associated with catastrophic outcomes. As patients become increasingly aware of this, they are more readily able to manage their sleep anxiety and return to normal sleep patterns.

Patients are often surprised with the simplicity of the CBT strategies. However as with changing any behaviours, they often require the support, structure, and continuity provided by the program to sustain this new approach, especially during the initial phases of treatment when the insomnia can actually temporally worsen.

CBT for chronic insomnia has been well studied with several randomized, controlled trials showing a similar efficacy to hypnotic medications but with a more sustained effect. Current research is addressing optimal strategies to combine CBT with hypnotic medications. ❖

THE CYCLE OF CHRONIC INSOMNIA



A NEW PARASOMNIA

Sleep-Related Eating Disorder (SRED)

SLEEP-RELATED EATING DISORDER is a newly defined parasomnia which consists of recurrent episodes of involuntary eating and drinking during arousals from sleep with problematic consequences.

These episodes of eating are involuntary, with variable amounts of recall, similar to classic sleep walking. Usually, the eating involves high caloric foods although may in some cases involve raw foods or inedible substances. Some patients have full recall and alertness, however they report a compulsive quality to the sleep eating behaviour that they cannot control.

This is in contrast to Night Eating Syndrome, in which there is a voluntary consumption of excessive food before bed-time or during the night, and while fully awake. This diagnosis is also not made if this behaviour is an extension of an eating disorder such as Bulimia Nervosa. Patients present with insomnia and daytime fatigue as a result of these awakenings. In addition, they complain of morning anorexia and abdominal fullness, weight gain. Rarely, injuries may occur due to the sleep walking.

This disorder tends to occur more frequently in females with onset in their 20s. One study estimate a prevalence of 4.6% in university students (Winkelman JW et al. *Psychological Medicine* 1999;29:1461-1466). A co-morbid primary sleep disorder such as apnea or periodic limb movement disorder may be present in up to 40% of the cases.

Treatment starts with addressing any co-morbid primary sleep disorder which may be triggering or aggravating the night eating. Subsequently, initial small sampled studies have shown benefits from the use of dopamine agonists, SSRIs and/or clonazepam. Most recently there was a study demonstrating very good efficacy with the use of Topirimate (Winkelman JW, *Sleep Med* 2003). Further research is needed to further characterize this disorder and determine optimal treatment paradigms. ❖

Caffeine

DID YOU KNOW that after oil,
caffeine is the second-most traded commodity in the world?

- Coffee, brewed .....40 to 180 mg. per cup
- Coffee, instant .....30 to 120 mg. per cup
- Coffee, decaffeinated .....3 to 5 mg. per cup
- Tea, brewed American .....20 to 90 mg. per cup
- Tea, brewed imported .....25 to 110 mg. per cup
- Tea, instant ..... 28 mg. per cup
- Cocoa ..... 4 mg. per cup
- Chocolate, milk..... 3 to 6 mg. per ounce
- Chocolate - bittersweet .....25 mg. per ounce
- Cola and other soft drinks, containing caffeine .....36 to 90 mg. per 12 ounces
- Cola and other soft drinks, decaffeinated .....0 mg. per 12 ounces

- Caffeine amounts of 250 mg may cause intoxication in some individuals – with restlessness, nervousness, excitement, flushed face, gastrointestinal symptoms, and diuresis
- Doses above 1 g may be associated with more severe symptoms including insomnia, dyspnea, delirium and arrhythmias
- Due to tolerance, individuals may become accustomed to high doses without any symptoms
- Caffeine's half life is 3-7 hours, however the duration of effects may last up to 14 hours.

(from *Principles and Practice of Sleep Medicine*, 2005, Ch. 115)

Diabetes & OSA

Continued from page 1

However, there are also a number of trials that have come short of showing similar benefits with the treatment of obstructive sleep apnea. Two particular randomized control trials have drawn interest having failed to show improvement in insulin resistance or hemoglobin A1c.

Unfortunately, both studies failed to achieve good compliance on CPAP in their populations; good compliance being accepted as the usage of CPAP 70% of nights for an average usage of 4 hours a night. This is specifically important since Babu et al. observed in their study that improvements in glucose metabolism were isolated to those with good control.

Overall, there is evidence that treating sleep-disordered breathing may improve certain aspects of impaired glucose metabolism. Further research is needed to clearly define the strength of the treatment response and the factors that effect this relationship. However, both conditions have shown associations with increased cardiovascular risk and the treatment of the conditions generally benefits the patient. ❖

Treatment Algorithms for Restless Legs Syndrome (RLS)

RLS IS A CLINICAL SYNDROME characterized by unpleasant sensation and restlessness in the legs occurring when lying down, at sleep onset, or while resting in the later evening. The discomfort is relieved with movement and often leads to the complaint of insomnia. For many patients, this disorder goes undiagnosed for many years and may be associated with severe insomnia, secondary anxiety, and depression. With increasing awareness of the diagnosis, more patients are seeking treatment.

Prior to initiating treatment, secondary etiologies need to be excluded, such as uremia, peripheral neuropathy, and iron deficiency. In addition, aggravating factors such as excess caffeine consumption and/or the use of some medications (*i.e.,* tricyclic antidepressants and/or SSRIs) need to be addressed. Furthermore, it is important to exclude other co-morbid sleep disorders such as sleep apnea.

Current recommendations for first-line treatment are the use of dopamine agonists such as parimpexole and ropinirole. These agents are generally well tolerated and are used at doses much lower than when treating Parkinson's disease.

The most troublesome issue with the dopamine agonists is the development of "Augmentation". This refers to the development of either an earlier onset of symptoms, an increase in severity of symptoms, a faster onset of symptoms at rest, and/or the expansion of the symptoms to the arms, trunk or abdomen. This occurs most commonly with L-dopa, yet studies also have demonstrated this phenomenon in up to 50% of individuals using the other agents. Once augmentation develops it is recommended that one either change to another dopaminergic agent, lower the dose of medication, and/or initiate a 2nd line treatment.

Second-line treatments include the use of clonazepam, opioids and gabapentin. It is not uncommon for patients with severe cases to require combination treatment and/or periodic rotation of their medications. Once an orphan condition, RLS has now attracted much more attention of pharmaceutical companies. As a result, we anticipate that there will be more options available in the near future. ❖