MEDICAL OPINION Regarding Obstructive Sleep Apnea with CPAP

Date: October 31, 2020

Note: For Medical Opinion and Nexus formulation purposes and establishment of reasonable medical certainty, according to the Code of Federal Regulations, and upheld by the BVA Administrative Law Judge, the following terminology and definitions were recognized and utilized:
"is due to" (100% probability)
"is more likely than not" (greater than a 50% probability)
"is at least as likely as not (a 50%/50% probability); consistent with "reasonable doubt"
"is less likely as not" (less than a 50% probability)
"is not due to" (0% probability)

***Rationale as below are based upon a review of the available medical records and VA documentation which may include the following**: Military service medical records and personnel records (C-File), Civilian medical records, Documentation in progress notes to include physical exams and diagnostic impressions, Radiological study reports, Laboratory studies, Claims file paperwork, VARO correspondence, Veteran lay statements, Previous C&P exams, Clinical experience and expertise, and Medical literature review.

MEDICAL OPINION #1

It is at least as likely as not (a 50%/50% probability) that the veteran's weight gain is due to and/or related to his service connected Left Knee and Right Knee.

MEDICAL OPINION #2

It is at least as likely as not (a 50%/50% probability) that the veteran's Obstructive Sleep Apnea with CPAP is secondary to, related to, and/or aggravated by the weight gain from the service connected Left Knee and Right Knee.

MEDICAL OPINION #3

It is at least as likely as not (a 50%/50% probability) that without the veteran's weight gain his Obstructive Sleep Apnea with CPAP would not have occurred.

Current Diagnosis of Obstructive Sleep Apnea

The veteran has a current, formal diagnosis of Obstructive Sleep Apnea (OSA) as substantiated by his current VA medical records. **The veteran is prescribed a CPAP for treatment of his OSA due to reported symptomatology to include: trouble sleeping with frequent awakenings, daytime fatigue, witnessed loud snoring, difficulty completing task, poor concentration, headaches upon awakening, and pauses in respirations while sleeping.**

Current Service Connection for Musculoskeletal Disability Condition

The veteran has a current, formal diagnosis of Left Knee and Right Knee which is service connected inclusive of commonly known symptomatology to include pain, painful motion, and an associated sleep disturbance (insomnia and frequent awakenings) related to the pain.

Veteran's Progressive Weight Gain

The veteran has reported that he weighed 170lbs upon military entrance and has a current weight of 195lbs with a BMI of 28, meeting the national standard by the CDC for being overweight. This is evidence of progressive weight gain and a current overweight condition which chronologically developed ***after*** the musculoskeletal symptomatology related to his service-connected condition.

Sleep Disturbance and Weight Gain/Obesity

Multiple studies within the medical literature, including one by Cauter and Knutson, document evidence from both laboratory and epidemiological studies to indicate that chronic partial sleep loss, to include insomnia and frequent night-time awakenings, increase the risk of obesity and weight gain. Sleep loss results in metabolic and endocrine alterations, including decreased glucose tolerance, decreased insulin sensitivity with loss in beta cell function (leading in an elevated risk of diabetes), elevated sympathovagal balance, increased evening concentrations of cortisol, increased levels of ghrelin which signal caloric need, decreased levels of leptin causing a reduction in satiety, and increased hunger and appetite related to internal misperception of insufficient energy availability. The adverse impact of sleep deprivation and dysregulation of the neuroendocrine appetite regulation is likely to be driven by increased activity in neuronal populations expressing the excitatory peptides orexins that promote hunger signals. These effects led to an increased body mass index (BMI) and obesity.

Musculoskeletal Conditions Associated with Weight Gain/Obesity

This veteran is currently service connected for his Left Knee and Right Knee. This veteran was awarded service connection with consideration of the following symptomology: pain, painful motion and associated sleep disturbances with insomnia and frequent awakenings. Due to

the persistent pain of his service-connected Left Knee and Right Knee, he was unable to be as active as he was before his musculoskeletal conditions and subsequently began to progressively gain weight. Weight gain has been established as secondary sequelae of a more sedentary lifestyle related to "joint pain, knee stiffness, and muscle weakness." (Zeni & Snyder-Mackler) Veterans with chronic lower back pain have reduced aerobic fitness levels for fear of re-injury which would lead to obvious weight gain. (*See Medical Literature below)

Pathophysiology of Obstructive Sleep Apnea Related to Obesity

Genta et al. found in their study that the upper airway anatomy plays a major role in obstructive sleep apnea (OSA) pathogenesis. An inferiorly displaced hyoid as measured by the mandibular plane to hyoid distance (MPH) has been consistently associated with OSA. The hyoid is also a common landmark for pharyngeal length, upper airway volume, and tongue base. Tongue dimensions, pharyngeal length, and obesity are associated with OSA severity, although the link between these anatomical variables and pharyngeal collapsibility is less well known. Obesity as measured by body mass index (BMI), neck and waist circumferences, and variables associated with hyoid position (pharyngeal length, upper airway volume, and tongue dimensions) have been found in the medical literature to be associated with passive pharyngeal critical closing pressure (Pcrit).

Eckert and Malhotra documented the pathophysiological processes that occur in OSA due to obesity. They reported that the deposition of fat around the pharyngeal airway is likely to increase the collapsibility of the pharyngeal airway. Fat deposition around the abdomen leads to reductions in functional residual capacity which would be predicted to reduce lung volume tethering effects on the upper airway. Low lung volumes are also associated with diminished oxygen stores, which would contribute to ventilatory control instability (high loop gain). Finally, obesity has been associated with functional impairment and weakening in the upper airway muscles.

Effects of Chronic Musculoskeletal Pain on Sleep

The medical literature, including a study by Brennan and Lieberman documents that chronic musculoskeletal pain causes sleep disturbances such as difficulty falling and staying asleep and less restful sleep. Evidence suggests that pain and sleep exist in a bidirectional relationship in which pain causes sleep disturbance and sleep disturbance intensifies pain. This association can impair a patient's daily function and decrease quality of life. Nadeem et al. documented in their study that patients with "chronic musculoskeletal pain sleep not only significantly less but also with inferior sleep quality. Their REM sleep is also less in duration and its onset is delayed."

Pathophysiology of Sleep Disturbance related to Obesity

Sleep is an important modulator of neuroendocrine function and glucose metabolism in adults. There is rapidly accumulating evidence from both laboratory and epidemiological studies to indicate that chronic partial sleep loss may increase the risk of obesity and weight gain. Laboratory evidence is indicating that sleep curtailment in adults results in a constellation of metabolic and endocrine alterations, including decreased glucose tolerance, decreased insulin

sensitivity, elevated sympathovagal balance, increased evening concentrations of cortisol, increased levels of ghrelin, decreased levels of leptin, and increased hunger and appetite. Review of cross-sectional epidemiological studies associating short sleep with increased body mass index and prospective epidemiological studies that have shown an increased risk of weight gain and obesity in adults who have sleep disturbances to include insomnia and frequent awakenings. Altogether, the evidence points to a possible role of decreased sleep duration in the current epidemic of obesity.

Weight Gain and Obesity a Major Risk Factor Associated with Obstructive Sleep Apnea

Multiple studies have documented that obesity is known to predispose patients to Obstructive Sleep Apnea (OSA). Phillips BG found in his study that male patients with Obstructive Sleep Apnea had a history of significant weight gain (6.8 +/- 2.3 kg) over the year preceding the study. Specifically, Newman AB documented that men were more likely to have an increase in Respiratory Disturbance Index (RDI) with a given increase in weight than were women, and this was not explained by differences in starting weight, waist circumference, age, or ethnicity. Babson et al reported in their study that "obesity is one of the leading risk-factors for OSA." Obesity is associated with mood, anxiety, and somatoform disorders as well as elevations in psychological distress. OSA was associated with an increased risk for psychological diagnosis including MDD (21.8%), anxiety (16.7%), PTSD (11.9%), and bipolar disorder (3.3%). As outlined above, this is related to a multitude of complex inter-related pathopsychophysiological mechanisms and processes that occur directly from these mental health disorders and result in weight gain and/or obesity due to and/or are associated with neuro-endocrine dysfunction processes associated with altered consumptive behaviors, sleep disturbances, and treatment medications.

Obesity is such a high risk factor for OSA, that even a one standard deviation increase in body mass index (BMI) has been associated with a 4-fold increase in the prevalence of OSA. Indeed, to further make the point, with severe obesity (BMI > 40), the prevalence of OSA ranges between 40% and 90%.

The medical literature has described from a pathophysiological perspective how Obstructive Sleep Apnea is associated with obesity, exacerbates hyperglycemia and other neuroendocrine chemicals and processes, and promotes further weight gain in a cyclical fashion due to an induced metabolic syndrome, with subsequent progression of weight gain and worsening of the OSA condition.

To confirm this opinion, the Acting General Counsel (022) of the Department of Veterans Affairs released a Memorandum, VAOPGCPREC 1-2017, dated January 6, 2017 covering the subject: **Service Connection Based on Obesity which confirms that obesity may be an intermediate step between a veteran's service connected conditions and the veteran's current disability condition with entitlement to service connection on a secondary basis**. The Memorandum Discussion number 14, "Finally, we address whether obesity could qualify as an "intermediate step" between a service-connected disability and a current disability, for example, whether a veteran could establish entitlement to service connection for hypertension if a veteran's service-connected back disability causes obesity due to lack of exercise, which leads

to hypertension…With regard to the hypothetical presented in the previous paragraph, adjudicators would have to resolve the following issues: (1) whether the service-connected back disability caused the veteran to become obese; (2) if so, whether the obesity as a result of the service-connected disability was a substantial factor in causing hypertension; and (3) whether the hypertension would not have occurred but for obesity caused by the service-connected back disability. If these questions are answered in the affirmative, the hypertension may be service connected on a secondary basis." Thus, if the evidence shows that the veteran's service-connected condition has ***caused*** weight gain and that weight gain has ***caused or contributed*** to his current condition, without evidence of another causal factor, the veteran is eligible for service connection. This veteran has had a total weight gain of approximately **25 pounds** from active duty to present time. This medical opinion does in fact support the conclusion that the veteran's sleep apnea "is at least as likely as not" secondary to the weight gain from his service-connected condition, substantiated by medical literature, therefore, he should be service-connected on this basis. Furthermore, obesity is the number one risk factor of developing Obstructive Sleep Apnea. Finally, the veteran at least as likely as not would not have developed Obstructive Sleep Apnea if not for his weight gain. All three are affirmative in this veteran's case which should result in his service-connection for his Obstructive Sleep Apnea.

Summary and Conclusion

Therefore, per the below *Evidence of Consideration in this Medical Opinion, it is at least as likely as not (a 50%/50% probability) that the veteran's weight gain is due to and/or related to his service connected Left Knee and Right Knee; it is at least as likely as not (a 50%/50% probability) that the veteran's Obstructive Sleep Apnea with CPAP is secondary to, related to, and/or aggravated by the weight gain from the service connected Left Knee and Right Knee; and it is at least as likely as not (a 50%/50% probability) that without the veteran's weight gain his Obstructive Sleep Apnea would not have occurred.

***Evidence of Consideration and Nexus Rationale that is outlined in this Medical Opinion:**

1) **The 38CFR and Department of Veterans Affairs Memorandum dated January 6, 2017 VAOPGCPREC 1-2017 regarding Service Connection Based on Obesity from Acting General Counsel**
2) **Thorough review of Service and/or Civilian Medical Records**
3) **Current diagnosis of Obstructive Sleep Apnea with CPAP which chronologically developed, manifested, and was diagnosed after the service-connected musculoskeletal condition**
4) **Veteran's credible Lay Statement with reports of trouble sleeping with frequent awakenings, daytime fatigue, witnessed loud snoring, difficulty completing task, poor concentration, headaches upon awakening, and pauses in respirations while sleeping**
5) **Service-connected Left Knee and Right Knee condition with pain/painful motion**
6) **A history of a marked weight gain condition which chronologically developed after the musculoskeletal symptomatology related to his service-connected condition with an associated sleep disturbance.**
7) **Medical Literature in support of the nexus rationale based upon a multitude of complex inter-related pathopsychophysiological mechanisms and processes that occur directly**

from these musculoskeletal disorders and result in weight gain and/or obesity due to and/or are associated with neuro-endocrine dysfunction processes

MEDICAL LITERATURE IN SUPPORT OF THE NEXUS RATIONALE

Patil, S. P., Schneider, H., Schwartz, A. R., & Smith, P. L. (2007). Adult Obstructive Sleep Apnea: Pathophysiology and Diagnosis. Chest, 132(1), 325. http://doi.org/10.1378/chest.07-0040

Adult Obstructive Sleep Apnea - Pathophysiology and Diagnosis

Abstract

Obstructive sleep apnea (OSA) is a highly prevalent disease characterized by recurrent episodes of upper airway obstruction that result in recurrent arousals and episodic oxyhemoglobin desaturations during sleep. Significant clinical consequences of the disorder cover a wide spectrum, including daytime hypersomnolence, neurocognitive dysfunction, cardiovascular disease, metabolic dysfunction, and cor pulmonale. The major risk factors for the disorder include obesity, male gender, and age. Current understanding of the pathophysiologic basis of the disorder suggests that a balance of anatomically imposed mechanical loads and compensatory neuromuscular responses are important in maintaining upper airway patency during sleep. OSA develops in the presence of both elevated mechanical loads on the upper airway and defects in compensatory neuromuscular responses. A sleep history and physical examination is important in identification of patients and appropriate referral for polysomnography. Understanding nuances in the spectrum of presenting complaints and polysomnography correlates are important for diagnostic and therapeutic approaches. Knowledge of common patterns of OSA may help to identify patients and guide therapy.

Obstructive sleep apnea (OSA) is a highly prevalent disease, affecting 4% of men and 2% of women,1 and strongly linked to the current obesity epidemic. The disorder is characterized by recurrent episodes of upper airway obstruction, and is associated with reductions in ventilation, resulting in recurrent arousals and episodic oxyhemoglobin desaturations during sleep.2 Significant clinical consequences of the disorder cover a wide spectrum including daytime hypersomnolence,3,4 neurocognitive dysfunction,5 cardiovascular disease (hypertension, stroke, myocardial infarction, heart failure),6,7 metabolic dysfunction,8–10 respiratory failure, and cor pulmonale.11 The major risk factors for the disorder include obesity, male gender, postmenopausal status, and age, and are discussed in detail elsewhere.12–14 The rising prevalence of obesity in the United States suggests that OSA will represent an escalating public health burden.15 Therefore, knowledge and understanding of the pathogenic basis, clinical presentation, and diagnosis of OSA are essential for the development of preventive, screening, and therapeutic strategies to reduce the public health burden of the disorder. In this review, we will review the current state of knowledge regarding the pathophysiologic basis of OSA and its clinical presentation and diagnosis.

[redacted]

Danny J. Eckert and Atul Malhotra "Pathophysiology of Adult Obstructive Sleep Apnea", Proceedings of the American Thoracic Society, Vol. 5, No. 2 (2008), pp. 144-153.

Pathophysiology of Adult Obstructive Sleep Apnea

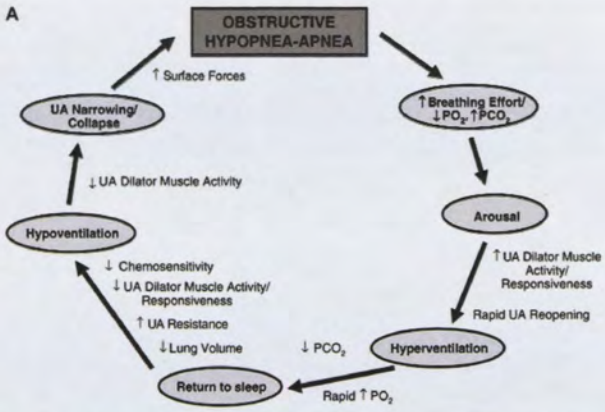

Figure 2. (*A*) Schematic representation of the typical pathophysiological sequence that occurs in obstructive sleep apnea (OSA) (*shown in gray*) and the associated physiological processes that occur throughout the cycle that are either protective/restorative (*outside the circle*) or perpetuating (*inside the circle*). UA = upper airway. (*B*) Schematic representation of the possible sites where each of the various pathophysiological traits would either predispose or tend to worsen OSA (*inside the main circle*). Note that some of the effects of these traits may be interrelated (i.e., a high loop gain may increase the ventilatory response to arousal and the propensity for cyclical breathing, and also lead to periods of decreased upper airway dilator muscle activity). Refer to the text for further detail. Some of the potential factors that may alleviate OSA at various points throughout the typical physiological cycle (*dashed arrows* and *ovals*) are located outside the main circle. Note that some factors are theoretical and largely untested whereas others are more proven therapies (i.e., continuous positive airway pressure [CPAP]).

Proc Am Thorac Soc,
http://www.atsjournals.org/doi/abs/10.1513/pats.200707-114MG

Published in: Danny J. Eckert; Atul Malhotra; *Proc Am Thorac Soc* **2008,** 5, 144-153.
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Sleep Disturbance and Obesity Medical Literature

Van Cauter, E., & Knutson, K. L. (2008). Sleep and the epidemic of obesity in children and adults. European Journal of Endocrinology, 159(S1), S59–S66. http://doi.org/10.1530/EJE-08-0298

Sleep and the epidemic of obesity in children and adults

Abstract

Sleep is an important modulator of neuroendocrine function and glucose metabolism in children as well as in adults. In recent years, sleep curtailment has become a hallmark of modern society with both children and adults having shorter bedtimes than a few decades ago. This trend for shorter sleep duration has developed over the same time period as the dramatic increase in the prevalence of obesity. There is rapidly accumulating evidence from both laboratory and epidemiological studies to indicate that chronic partial sleep loss may increase the risk of obesity and weight gain. The present article reviews laboratory evidence indicating that sleep curtailment in young adults results in a constellation of metabolic and endocrine alterations, including decreased glucose tolerance, decreased insulin sensitivity, elevated sympathovagal balance,

increased evening concentrations of cortisol, increased levels of ghrelin, decreased levels of leptin, and increased hunger and appetite. We also review cross-sectional epidemiological studies associating short sleep with increased body mass index and prospective epidemiological studies that have shown an increased risk of weight gain and obesity in children and young adults who are short sleepers. Altogether, the evidence points to a possible role of decreased sleep duration in the current epidemic of obesity.

Beccuti, G., & Pannain, S. (2011). Sleep and obesity. Current Opinion in Clinical Nutrition and Metabolic Care, 14(4), 402–412. http://doi.org/10.1097/MCO.0b013e3283479109

Sleep and obesity

Purpose of review: This review summarizes the most recent evidence linking decreased sleep duration and poor sleep quality to obesity, focusing upon studies in adults.

Recent findings: Published and unpublished health examination surveys and epidemiological studies suggest that the worldwide prevalence of obesity has doubled since 1980. In 2008, 1 in 10 adults was obese, with women more likely to be obese than men. This obesity epidemic has been paralleled by a trend of reduced sleep duration. Poor sleep quality, which leads to overall sleep loss has also become a frequent complaint. Growing evidence from both laboratory and epidemiological studies points to short sleep duration and poor sleep quality as new risk factors for the development of obesity.

Summary: Sleep is an important modulator of neuroendocrine function and glucose metabolism and sleep loss has been shown to result in metabolic and endocrine alterations, including decreased glucose tolerance, decreased insulin sensitivity, increased evening concentrations of cortisol, increased levels of ghrelin, decreased levels of leptin, and increased hunger and appetite. Recent epidemiological and laboratory evidence confirm previous findings of an association between sleep loss and increased risk of obesity.

Prinz, P. (2004). Sleep, Appetite, and Obesity—What Is the Link? PLoS Medicine, 1(3), e61. http://doi.org/10.1371/journal.pmed.0010061

Sleep, Appetite, and Obesity—What Is the Link?

There is a well-documented relationship between short sleep duration and high body mass index (BMI). In the largest study, a survey on sleep duration and frequency of insomnia in more than 1.1 million participants, increasing BMI occurred for habitual sleep amounts below 7–8 hours [1]. A recent prospective study found an association between sleep curtailment and future weight gain [2]. The mechanism linking short sleep with weight gain is unknown, but Mignot and colleagues' study in this month's PLoS Medicine [3] adds to the growing evidence implicating leptin and ghrelin, the two key opposing hormones involved in appetite regulation.

Knutson, K. L., & Van Cauter, E. (2008). Associations between sleep loss and increased risk of obesity and diabetes. Annals of the New York Academy of Sciences, 1129, 287–304. http://doi.org/10.1196/annals.1417.033

Associations between sleep loss and increased risk of obesity and diabetes

Abstract

During the past few decades, sleep curtailment has become a very common behavior in industrialized countries. This trend for shorter sleep duration has developed over the same time period as the dramatic increase in the prevalence of obesity and diabetes. There is rapidly accumulating evidence to indicate that chronic partial sleep loss may increase the risk of obesity and diabetes. Laboratory studies in healthy volunteers have shown that experimental sleep restriction is associated with an adverse impact on glucose homeostasis. Insulin sensitivity decreases rapidly and markedly without adequate compensation in beta cell function, resulting in an elevated risk of diabetes. Prospective epidemiologic studies in both children and adults are consistent with a causative role of short sleep in the increased risk of diabetes. Sleep curtailment is also associated with a dysregulation of the neuroendocrine control of appetite, with a reduction of the satiety factor leptin and an increase in the hunger-promoting hormone ghrelin. Thus, sleep loss may alter the ability of leptin and ghrelin to accurately signal caloric need, acting in concert to produce an internal misperception of insufficient energy availability. The adverse impact of sleep deprivation on appetite regulation is likely to be driven by increased activity in neuronal populations expressing the excitatory peptides orexins that promote both waling and feeding. Consistent with the laboratory evidence, multiple epidemiologic studies have shown an association between short sleep and higher body mass index after controlling for a variety of possible confounders.

Effects of Chronic Pain on Sleep

Brennan M. J., Lieberman J. A., III. (2009). Sleep disturbances in patients with chronic pain: effectively managing opioid analgesia to improve outcomes. Curr. Med. Res. Opin. 25 1045–1055. 10.1185/03007990902797790

Sleep disturbances in patients with chronic pain: effectively managing opioid analgesia to improve outcomes.

Abstract

Background: Many patients with chronic pain experience pain-related sleep disturbances, such as difficulty falling and staying asleep and less restful sleep. Evidence suggests that pain and sleep exist in a bidirectional relationship in which pain causes sleep disturbance and sleep disturbance intensifies pain. This association can impair a patient's daily function and decrease quality of life. Evidence suggests that patients with chronic pain can use opioid analgesics or other pain medications to control their pain and, in turn, improve some measures of sleep. This may include subjective sleep measures such as increased sleep time, and, as evidenced in recent

studies, objective sleep measures such as sleep efficiency.

Scope: The role of effective analgesia in the improvement of pain-related sleep disturbance is discussed herein, specifically the risks and benefits of opioid therapy for the treatment of patients with chronic pain and disturbed sleep. MEDLINE and PubMed searches were conducted to locate relevant studies dated from January 1975 to April 2008. English-only randomized controlled trials and nonrandomized studies were considered.

Findings: Numerous studies support the benefits of effective analgesia with opioid therapy on sleep.

Conclusion: Pain control achieved with pharmacotherapy, specifically opioid therapy, may help to improve sleep in patients for which opioid therapy is appropriate.

Chronic Pain and OSA Effects on Sleep

Nadeem R, Bawaadam H, Asif A, et al.: Effect of musculoskeletal pain on sleep architecture in patients with obstructive sleep apnea. Sleep Breath, 2014, 18: 571–577

Effect of musculoskeletal pain on sleep architecture in patients with obstructive sleep apnea.

Abstract

Rationale: Obstructive sleep apnea and chronic musculoskeletal pain both affect sleep. Sleep architecture of patients suffering from both is largely unknown.

Objectives: This study seeks to define the sleep architecture of patients with chronic musculoskeletal pain and obstructive sleep apnea.

Methods: Patients with obstructive sleep apnea diagnosed by sleep study during the past 3 years were included. Patients with clinical documentation of chronic musculoskeletal pain constituted cases, while others were classified as controls.

Measurements: Demographics, clinical factors affecting sleep, medications affecting sleep, Epworth sleepiness scores, and polysomnographic parameters; total sleep time, sleep efficiency, sleep stages, rapid eye movement (REM) sleep onset, apnea-hypopnea index, arousal index, and periodic leg movements were recorded.

Results: There were 393 subjects: 200 cases (obstructive sleep apnea and chronic musculoskeletal pain) and 193 controls (obstructive sleep apnea alone). There was significant difference in total sleep time (274.5 ± 62.5 vs. 302.2 ± 60.1 min, $p = 0.0001$), sleep efficiency (73.54 ± 15.8 vs. $78.76 \pm 14.3\%$, $p = 0.0003$), and REM sleep onset (148.18 ± 80.5 vs. 124.8 ± 70.9 min, $p = 0.006$). Subgroup analysis within the obstructive sleep apnea with chronic musculoskeletal pain group revealed that subjects had better total sleep time and sleep efficiency

if they were on REM sleep affecting medications (suppressants and stimulants). Those on REM sleep suppressants slept 25.7 min longer and had 6.4% more efficient sleep than those not on REM suppressants ($p = 0.0034$ and $p = 0.0037$).

Conclusion: Patients with obstructive sleep apnea and chronic musculoskeletal pain sleep not only significantly less but also with inferior sleep quality. Their REM sleep is also less in duration and its onset is delayed. Despite low TST and SE, these patients may not exhibit sleepiness.

Obesity and Obstructive Sleep Apnea Medical Literature

Gami AS, Caples SM, Somers VK. Obesity and obstructive sleep apnea. Endocrinol Metab Clin North Am. 2003;32(4):869–894.

Obesity and obstructive sleep apnea.

Abstract

There is a very high prevalence of OSA in obese individuals and a high prevalence of obesity in patients with OSA. The pathophysiology of OSA is intimately linked to obesity. Anatomic and functional considerations of the pharyngeal airway, the CNS, central obesity, and leptin likely interact in the development of OSA in obese individuals. OSA may itself predispose individuals to worsening obesity because of sleep deprivation, daytime somnolence, and disrupted metabolism. The diagnosis of OSA requires the clinician's awareness of its potential to cause a spectrum of acute and chronic neurocognitive, psychiatric, and nonspecific symptoms in patients who may be unaware that their sleep is disturbed. Symptoms and examination findings help predict which obese individuals have OSA, and polysomnography is the gold standard by which to make the diagnosis and assess the effects of treatment. Numerous disease states are associated with both OSA and obesity, and it is becoming clear that the relationships are mediated by complex interrelated mechanisms. Common diseases and disease mechanisms in OSA and obesity suggest that conditions related to obesity may be better managed if patients, particularly those who are morbidly obese, are evaluated and treated for previously undiagnosed OSA. OSA is cured in only specific cases with craniofacial or upper airway surgery, and the general application of UVP is not efficacious. OSA also can be cured with sufficient lifestyle-mediated or surgical weight loss; however, in the absence of long-term weight maintenance, OSA returns with weight gain. Although not curative, nasal CPAP is the initial treatment of choice for most patients because of its noninvasive approach and technical efficacy. It is limited, however, by patient acceptance and long-term compliance. Advances in mask comfort and use of humidified air should increase its acceptance. Future management strategies include newer generations of positive airway devices that automatically titrate pressures (which are not yet recommended by expert organizations) and multidisciplinary approaches to managing the care of patients with OSA.

Shah N, Roux F. The relationship of obesity and obstructive sleep apnea. Clin Chest Med. 2009;30:455–65, vii.

The relationship of obesity and obstructive sleep apnea.

Abstract

Obstructive sleep apnea is a common disorder, and obesity is a known risk factor for its development. The prevalence of obesity is increasing worldwide, and a corresponding increase in the prevalence of obstructive sleep apnea and its cardiovascular and non-cardiovascular consequences is likely. This article reviews the established evidence supporting obesity as a risk factor for obstructive sleep apnea and discusses the evidence suggesting that obesity is also a consequence of obstructive sleep apnea. There is evidence that treating obesity reduces the severity of obstructive sleep apnea and that treating obstructive sleep apnea decreases obesity. However, the evidence does not support a sustained correlation between weight loss and improvement in sleep-disordered breathing

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Quilliot D1, Petit FX, Cornette A, Ziegler O. [How do you really know if the obese patient has sleep apnea?]. Ann Endocrinol (Paris). 2002 Dec;63(6 Pt 2):S38-51

[How do you really know if the obese patient has sleep apnea?]

Abstract

Obesity is known to predispose to obstructive sleep apnea (OSA), a condition characterized by repeated episodes of apnea or hypopnea during sleep, due to the interruption of airflow through the nose and mouth. These episodes lead to the fragmentation of sleep and to decrease in oxyhaemoglobin saturation. Patients with massive obesity, with or without daytime hypersomnolence should be systematically screened for OSA, because many of them appear to be asymptomatic and unaware of their breathing abnormalities during sleep. Polysomnography (PSG) in an attended hospital laboratory setting is the gold standard for the diagnosis of OSA. However portable recording devices can be used for screening with good sensibility and specificity and even for diagnosis when the apnea-hypopnea index is high. However the final diagnosis can only be carried out in a sleep laboratory using PSG by highly-qualified personnel, because of the limitations of the portable recording device. There is a strong association between OSA and the risk of traffic accidents. It has been established that OSA affects quality of life. There is also increasing evidence that OSA is an independent risk factor for cardio-vascular diseases. This has been successfully demonstrated for hypertension by prospective studies. But the evidence remains weak for myocardial infarction, stroke or mortality. Treating OSA with continuous positive airway pressure (CPAP) is the treatment of choice. CPAP improves quality of life, driving simulator performance, blood pressure and sleepiness, as demonstrated by randomised placebo controlled trials. The majority of obese OSA patients are currently not being offered diagnosis testing and treatment. It's a real challenge due to the epidemic increase of obesity prevalence. Portable recording devices could be available outside the sleep laboratory in nutrition department, where morbid obesity is treated. This emphasizes the need for a real

collaboration between these departments and sleep experts.

Newman AB, Foster G, Givelber R, Nieto FJ, Redline S, Young T. Progression and regression of sleep-disordered breathing with changes in weight: the Sleep Heart Health Study. Arch Intern Med. 2005;165:2408–2413. doi: 10.1001/archinte.165.20.2408.

Progression and regression of sleep-disordered breathing with changes in weight: the Sleep Heart Health Study.

Abstract

Background: The relationship of weight changes to the incidence, progression, and remission of sleep-disordered breathing (SDB) is not well defined. This study aims to determine the relationship between change in weight and progression or remission of SDB by polysomnography.

Methods: We performed a longitudinal cohort study of the cardiovascular consequences of sleep apnea in diverse US communities. Sleep apnea and polysomnographic indicators of SDB were assessed 5 years apart.

Results: A total of 2968 men and women (mean age, 62 years) participated in the study. Men were more likely to have an increase in Respiratory Disturbance Index (RDI) with a given increase in weight than were women, and this was not explained by differences in starting weight, waist circumference, age, or ethnicity. In a linear regression analysis, both men and women had a greater increase in RDI with weight gain than a decrease in RDI with weight loss. In a categorical analysis of larger degrees of change, this sex difference was also evident. Associations were similar in diverse ethnic groups. However, SDB progressed over time, even in those with stable weight.

Conclusion: Modest changes in weight were related to an increase or decrease in SDB, and this association was stronger in men than in women.

Phillips B.G., Hisel T.M., Kato M., Pesek C.A., Dyken M.E., Narkiewicz K., Somers V.K. (1999) Recent weight gain in patients with newly diagnosed obstructive sleep apnea. J. Hypertens. 17, 1297–1300.

Recent weight gain in patients with newly diagnosed obstructive sleep apnea.

Abstract

Objective: Patients with obstructive sleep apnea are often obese. Obesity may contribute to both sleep apnea itself and to the cardiovascular risk associated with sleep apnea. Weight loss in obese patients with sleep apnea may alleviate symptoms and decrease the severity of sleep apnea. Whether patients with obstructive sleep apnea are indeed predisposed to recent weight gain, as

compared with similarly obese subjects without sleep apnea, is not known.

Patients and Methods: We compared 1-year weight histories in 53 male and female patients newly diagnosed with obstructive sleep apnea, compared with 24 controls matched for gender, age, body mass index, and percent body fat. Sleep apnea patients had never been treated. Control subjects were proven to be free of sleep-disordered breathing by overnight polysomnography.

Results: Patients with obstructive sleep apnea (n = 53) had a significant recent weight gain of 7.4 +/- 1.5 kg compared with a weight loss of 0.5 +/- 1.7 kg (P = 0.001) in similarly obese controls (n = 24). Male patients with obstructive sleep apnea (n = 28) had a history of significant weight gain (6.8 +/- 2.3 kg) over the year preceding the study compared with male control subjects (n = 13), in whom average weight fell by 0.58 +/- 2.4 kg (P = 0.03). Female patients (n = 25) with obstructive sleep apnea had an 8.0 +/- 1.9 kg weight gain compared with female controls (n = 11) who had a history of weight loss of 0.46 +/- 2.6 kg (P = 0.02).

Conclusion: These findings support the concept that patients with obstructive sleep apnea may be susceptible to increasing obesity in the period preceding the diagnosis of obstructive sleep apnea.

Genta, P. R., Schorr, F., Eckert, D. J., Gebrim, E., Kayamori, F., Moriya, H. T., … Lorenzi-Filho, G. (2014). Upper Airway Collapsibility is Associated with Obesity and Hyoid Position. Sleep, 37(10), 1673–1678. http://doi.org/10.5665/sleep.4078

Upper airway collapsibility is associated with obesity and hyoid position

Abstract

Study Objectives: Upper airway anatomy plays a major role in obstructive sleep apnea (OSA) pathogenesis. An inferiorly displaced hyoid as measured by the mandibular plane to hyoid distance (MPH) has been consistently associated with OSA. The hyoid is also a common landmark for pharyngeal length, upper airway volume, and tongue base. Tongue dimensions, pharyngeal length, and obesity are associated with OSA severity, although the link between these anatomical variables and pharyngeal collapsibility is less well known. We hypothesized that obesity as measured by body mass index (BMI), neck and waist circumferences, and variables associated with hyoid position (pharyngeal length, upper airway volume, and tongue dimensions) would be associated with passive pharyngeal critical closing pressure (Pcrit).

Design: Cross-sectional. SETTING: Academic hospital.

Patients: 34 Japanese-Brazilian males age 21 to 70 y.

Interventions: N/A.

Measurements and Results: We performed computed tomography scans of the upper airway, overnight polysomnography, and Pcrit measurements in all subjects. On average, subjects were

overweight (BMI = 28 ± 4 kg/m(2)) and OSA was moderately severe (apnea-hypopnea index = 29 [13-51], range 1-90 events/h). Factor analysis identified two factors among the studied variables: obesity (extracted from BMI, neck and waist circumferences) and hyoid position (MPH, pharyngeal length, tongue length, tongue volume, and upper airway volume). Both obesity and hyoid position correlated with Pcrit (r = 0.470 and 0.630, respectively) (P < 0.01). In addition, tongue volume, tongue length, pharyngeal length, and MPH correlated with waist and neck circumferences (P < 0.05).

Conclusions: Pharyngeal critical closing pressure is associated with obesity and hyoid position. Tongue dimensions, pharyngeal length, and the mandibular plane to hyoid distance are associated with obesity variables. These findings provide novel insight into the potential factors mediating upper airway collapse in obstructive sleep apnea.

Chronic Pain (from spine/joint) and Obesity

Knees

Vincent HK, Lamb KM, Day TI, Tillman SM, Vincent KR, George SZ. Morbid obesity is associated with fear of movement and lower quality of life in patients with knee pain-related diagnoses. PM&R. 2010;2:713–22. doi: 10.1016/j.pmrj.2010.04.027.

Morbid obesity is associated with fear of movement and lower quality of life in patients with knee pain-related diagnoses.

Abstract

Objective: To compare fear of movement in patients with different body mass index (BMI) values referred for rehabilitative care of the knee and to examine whether this fear contributed to self-reported knee-related function. We hypothesized that fear of movement would be elevated with increasing BMI and that fear would correspond with lower self-report knee-related function and lower quality of life (QOL).

Design: Retrospective cross-sectional study.

Setting: Outpatient therapy clinic affiliated with a tertiary care hospital.

Patients: Patients with knee pain diagnoses (n = 278) were stratified into 4 BMI groups (in < or =25 kg/m(2) non-obese; 25-29.9 kg/m(2) overweight; 30-39.9 kg/m(2) obese; > or =40 kg/m(2) morbidly obese).

Main Outcome Measurements: The Tampa Scale of Kinesiophobia (TSK; fear of movement), International Knee Documentation (IKDC; knee function), and Short-Form 8 (SF-8; QOL) scores were main outcomes. Pain and straight leg raise test scores also were collected.

Methods: After review of the medical records, descriptive statistics and nonparametric tests

were performed, and TSK, QOL, and SF-8 scores were compared. Hierarchical regression modeling determined the contribution of TSK scores to the variance of IKDC scores.

Results: Pain scores were greatest in the non-obese group and lowest in the morbidly obese group (7.5 +/- 2.6 points vs 4.8 +/- 3.1 points; P < .05). TSK scores in morbidly obese patients were greater than in nonobese patients (27.1 +/- 7.7 points vs 22.0 +/- 6.6 points; P = .002). The SF-8 mental-physical subscores were 27% to 32% lower in the morbidly obese than non-obese patients (both P < .0001). IKDC scores were lower in the morbidly obese than non-obese patients (32.1 +/- 19.2 points vs 50.9 +/- 23.8 points; P = .0001). Pain severity and TSK scores contributed 28.6% and 7.1% to the variance of the IKDC scores (overall R(2) = 68.6).

Conclusions: Morbid obesity is associated with elevated fear of movement. Pain was the strongest predictor of IKDC scores, and fear of movement enhanced this predictive value of the regression model. Despite lower absolute pain severity in the morbidly obese group, this fear may influence IKDC scores in this population. Morbidly obese patients might benefit from rehabilitation activities that reduce fear of movement to optimize participation in rehabilitation activity.

Marcus D.A. Obesity and the impact of chronic pain. Clin. J. Pain. 2004;20:186–191. doi: 10.1097/00002508-200405000-00009.

Obesity and the impact of chronic pain.

Abstract

Objective: To evaluate obesity as a marker for increased pain severity, disability, and psychologic distress in treatment-seeking, mixed chronic pain patients.

Methods: Three hundred seventy-two consecutive chronic pain patients seeking evaluation at a university pain clinic were divided into 3 weight categories, based on body mass index (BMI): normal (BMI < 25 kg/m2), overweight (BMI between 25 kg/m and 30 kg/m2), and obese (BMI > or = 30 kg/m2). Patients completed questionnaires to identify pain severity, disability, depression, anxiety, and quality of life.

Results: Pain severity and days per week with pain were similar among the weight groups. Disability was related to increasing weight status, with increased BMI associated with more days per week with both reduced activity and complete disability. Depressive symptoms were also related to weight category, with an average Beck Depression Inventory score of 11.81 +/- 7.55 in normal, 12.88 +/- 11.64 in overweight, and 15.78 +/- 9.88 in obese patients. Anxiety scores were similar among the weight categories. Physical function domain of quality of life was also reduced in relation to weight.

Conclusions: Weight is associated with co-morbid disability, depression, and reduced quality of life for physical function in chronic pain patients. Calculation of the BMI should become a routine part of the screening evaluation for chronic pain patients, with additional screening for

disability and psychologic distress in patients with elevated BMIs.