Life Insurance Risk Assessment in Patients with Obstructive Sleep-Disordered Breathing

CLINICAL PRACTICE REVIEW COMMITTEE
AMERICAN ACADEMY OF SLEEP MEDICINE

EXECUTIVE SUMMARY

· Patients with obstructive sleep-disordered breathing have an increased risk cardiovascular morbidity and mortality.
· Current evidence suggests that patients who are compliant with proven effective therapy for sleep apnea should be expected to have little or no risk of excess mortality due to sleep apnea.
· Life insurance carriers should not assess excessive risk of mortality based on sleep-disordered breathing if the sleep-disordered breathing is effectively treated.

Every person applying for individual life insurance is subject to an analysis of their actuarial risk of death based upon evaluation of health status, risk factors, and certain behaviors (such as alcohol and tobacco use). The diagnosis of obstructive sleep-disordered breathing is now considered by the insurance industry to be a significant risk for mortality in this assessment and may be used to determine insurability. Evidence for excess mortality risk, effectiveness of treatment, compliance of patients with voluntary therapies, and documentation of both effectiveness and compliance are important ingredients in determining insurability. This position paper formulates recommendations for determining life insurance insurability based on available evidence on risk of excess mortality and morbidity and current understanding of the requirements for documenting effective therapy.

A. Evidence

A.1.1. Excess mortality

The National Commission on Sleep Disorders Research concluded in 1993 that sleep-disordered breathing was associated with an increased risk of morbidity and mortality [1]. Evidence for this conclusion continues to accumulate, and though it is primarily based on retrospective studies of patients with obstructive sleep disordered-breathing or matched comparisons of either patients or snorers and control subjects, the mass of evidence overwhelmingly favors an increased risk of mortality and a graded effect between the intensity of disordered breathing and mortality.

Retrospective studies of patients referred for clinical evaluation of the sleep apnea syndrome suggest that untreated patients have a higher mortality than treated patients and that the intensity of disordered breathing may influence the risk of mortality through indirect mechanisms such as body habitus or hypertension. Partinen [2] described an increase in vascular mortality rates of 127 patients treated conservatively as compared to 71 patients treated with tracheostomy. In another retrospective study, a 36% decrease in survival over eight years was noted in untreated patients with an apnea index of >20 as compared to untreated patients with an apnea index of < 20 [3]. Lavie noted a relative risk of mortality of 3:3 in males 40-60 years old during a similar 8 year follow-up [4]. Ancoli-Israel demonstrated a substantial gender-specific effect of RDI on mortality in the elderly [5]. A higher mortality was noted in men and a stronger relationship between RDI and mortality was evident in women. It is not surprising that sleep apnea interacts with other risk factors. In a study of 400 individuals matched for age, sex, and body mass index, snorers with risk factors had higher morbidity and mortality than snorers without risk factors [6]. Risk factors in this study included hypertension, diabetes, obesity, smoking, high serum cholesterol level. In a population survey of 3,100 males, subjects less than 60 years of age who had coexisting snoring and daytime sleepiness had a 2:7 fold risk of mortality over a 10 year period [7]. From these retrospective and matched-control studies, mortality appears to be related in a graded fashion to the intensity of sleep-disordered breathing and is highly influenced by co-morbidity. Prospective studies such as the Sleep Heart Health Study will be needed to determine whether sleep-disordered breathing causes excess mortality independent of comorbid conditions [8].

A.1.2. Excess Risk of Hypertension

Though there has been controversy regarding the risk of hypertension in snorers and patients with sleep-disordered breathing [9], substantial epidemiological and pathophysiological literature suggests that sleep-disordered breathing causes hypertension. In a random population survey of 7,699 individuals in Israel who underwent overnight monitoring, hypertensive subjects had a higher respiratory disturbance index and lower overnight saturation than non-hypertensive patients [10]. In a study of 147 male and female Wisconsin state workers, Hla found a dose-dependent effect of sleep-disordered breathing on risk of hypertension [11]. After controlling for age, obesity, and sex, subjects with a RDI of 5 had an odds ratio of 2 of having hypertension while subjects with an RDI of 25 had an odds ratio of 5. A subsequent study by these investigators found a linear relationship between the apnea-hypopnea index and both systolic and diastolic blood pressure [12]. The relationship was independent of known confounding variables. A similar dose-dependent effect has been observed in children with graded increases in diastolic hypertension associated with increasing respiratory disturbance index [13].

The evidence for a causal association between sleep apnea and hypertension is supported by known pathophysiological mechanisms. Excess sympathetic activity is a consistent finding in patients with sleep apnea syndrome and is presumed to contribute to the high incidence of hypertension in the disease [14-16]. Two studies have shown relationships between severity of sleep apnea and sympathetic activity or

blood pressure [15, 17]. Long-term use of CPAP results in a reduction in urinary catecholamine excretion [18]. A linear relationship has been found between compliance with CPAP and sympathetic activity [19]. Similarly, urinary catecholamine excretion decreased in patients following tracheostomy for sleep apnea [20].

A.1.3. Excess Risk of Myocardial Infarction

There is some preliminary evidence demonstrating increased risk of ischemic heart disease in patients with obstructive sleep-disordered breathing. One case-controlled study showed a 23 fold increased risk of recurrent myocardial infarction in patients presenting with acute MI [21]. This risk compared the highest and lowest quartile of the population according to apnea hypopnea index (>5.3 and <0.4 respectively). The design of the study does not exclude the possibility that myocardial infarction may have caused the sleep-disordered breathing. A similar retrospective study identified an increased risk of ventricular ectopy in patients with myocardial infarction complicated by sleep-disordered breathing [22]. Patients with sleep-disordered breathing were not at greater risk for mortality in the latter study. Given the known association between ventricular dysrhythmias and sleep-disordered breathing, it is not surprising the patients with myocardial infarction complicated by sleep-disordered breathing would have a higher prevalence of ectopic ventricular activity [23].

A.1.4 Excess Risk of Stroke

A review of six case-controlled studies suggested an odds ratio of stroke in patients with snoring or sleep-disordered breathing to be 1:6 to 3:2. These studies included one large study of 400 cases and 400 controls [24]. In a study using age and gender matched control subjects, 69-70% of patients had sleep-disordered breathing while only 15% of control subjects had sleep-disordered breathing [25]. Both groups had a similar frequency of habitual snoring. Similarly, Mohsenin found that 10 patients with hemispheric (but not brainstem) stroke had a respiratory disturbance index of 52 as compared to 3 in controls subjects matched for age, body mass index, and hypertension [26].

Much like studies of the association between sleep-disordered breathing and myocardial infarction, studies of stroke identify disordered breathing after the incident event which confounds the understanding of causation: does sleep-disordered breathing cause stroke/myocardial infarction or visa-versa? Potential mechanisms exist in both conditions to produce disordered breathing: depressed left ventricular function in myocardial infarction may contribute to periodicity in breathing and strokes may cause both periodicity and cyclical increases in upper airway resistance [27].

A.1.5 Risk of Vehicular Accidents.

Sleep disorders which produce excessive daytime sleepiness have been associated with increased risk of traffic accidents [12, 28]. In addition to human loss, there is substantial economic [29] and legal burden as well [30].

Both commercial and non-commercial drivers are at risk. Long haul truck drivers with OSAHS and EDS had a two fold increase in traffic accidents [31, 32]. In a case-controlled study of patients treated in emergency departments following traffic accidents, those with an AHI>10 had an odds ratio of 6:3 for having a traffic accident [33]. The type of accident tends to be specific. Single car accidents were seven-fold higher in patients with untreated sleep apnea; and if corrected for mileage driven, the incidence rose to 12-fold higher [34].

Questionnaires and vigilance testing such as Steer Clear [35-37] and the Divided Attention Driving Test [38] predict the increased risk and demonstrate improvement in risk with treatment. CPAP resolved the EDS associated with OSAHS as measured by MSLT and an 80 minute vigilance test. There was a significant decrease in the accident rate after treatment with CPAP as well.

Less attention has been placed on the non-driving accidents in the work place. However, diminished quality of life including social and work functioning have been linked to OSAHS and have been shown to improve with treatment [39, 40].

A.1.6 Conclusions

· Obstructive sleep-disordered breathing is likely to be a contributing cause of increased risk of vehicular accidents, hypertension, and excess mortality.
· Effective treatment of obstructive sleep-disordered breathing would be expected to reverse the excessive risk of vehicular accidents, hypertension, and excess mortality that are caused by sleep-disordered breathing.
· Though obstructive sleep apnea is associated with an increased relative risk of myocardial infarction and stroke, current studies do not provide sufficient evidence to determine causation.

B.2. Effective therapy

B.2.1. Non-surgical therapies

Non-surgical treatment of obstructive sleep apnea includes mechanical devices, position modification, and weight loss. Mechanical devices include dental appliances and positive airway pressure machines. Efficacies of non-surgical treatments vary with individual patient factors including severity of disease. Position modification may be useful for patients with obstructive respiratory events primarily occurring in the supine sleep position, but its use is hampered by the lack of data regarding efficacy of treatment over time. Weight loss in obese sleep apnea patients can decrease the occurrence of obstructive respiratory events in some patients. The high rate of rebound weight gain makes long-term dietary management of sleep apnea irrational for most morbidly obese patients.[41] Mandibular advancement dental appliances have variable efficacy for sleep-disordered breathing, neces-

sitating documentation of reversal by polysomnography with the appliance. Long-term compliance with dental devices is not well known.

Positive airway devices, including continuous positive airway pressure (CPAP) and bilevel positive airway pressure machines, are overall the most effective treatment for obstructive sleep apnea, with CPAP the most commonly prescribed. The effectiveness of CPAP in eliminating sleep-disordered breathing has been well documented.[42, 43] By correcting sleep-disordered breathing, normal sleep architecture and sleep continuity is restored. Nasal CPAP may work by reducing upper airway edema or increasing the area of the upper airways, likened to a "pneumatic splint".[44-48]

Many symptoms and consequences associated with obstructive sleep apnea are reversed by nasal CPAP. CPAP improves objective daytime sleepiness, mood, and quality of life.[39, 49-53] Effective CPAP therapy improves vigilance and driver performance.[36-38] CPAP lowers sympathetic activity associated with sleep apnea [19] and improves daytime blood pressure in sleep apnea patients who fail to have the normal nocturnal drop in blood pressure.[54] Patients treated with CPAP have lower mortality than untreated patients.[3] It is reasonable to assume that any therapy as effective as CPAP in eliminating obstructive respiratory events during sleep would lead to similar beneficial effects on morbidity and mortality. The correction of sleep disordered breathing and restoration of sleep continuity would be expected to correct excess morbidity and mortality with sleep apnea.

B.2.2. Surgical therapies

Surgery as an option to treat obstructive sleep apnea syndrome is steadily evolving since the uvulopalatopharyngoplasty was introduced in the late 1970's. Because of the short time that surgery has been used as a method of treatment and because of the constant evolution of treatment protocols, the long-term results for successful treatment of apnea have not been well documented until recently. Surgical treatment of sleep apnea can take one of two directions: either to bypass the point of obstruction or to try and reconstruct the airway at the point of obstruction. Tracheotomy is the procedure used to bypass the point of obstruction and this procedure is virtually 100% effective to control apnea; however, it is a considerable change in a person's lifestyle to have to deal with a tracheotomy and, indeed, this procedure is generally reserved for those patients with life-threatening sleep apnea who are unresponsive to either CPAP or reconstructive surgery on the upper airway.[55] Upper airway reconstructive surgery is more complex and there are many different procedures available to treat the various portions of the airway that may be closing during sleep.[56] The keys to success in upper airway reconstructive surgery are:

1. To be able to differentiate the points of obstruction, and
2. To be able to perform procedures to relieve this obstruction without affecting the functions of either swallowing or speech.

Unfortunately there are no objective tests that will pinpoint the areas of obstruction with 100% certainty. Therefore, we depend upon the surgeon's diagnostic skills and judgment in making these determinations. Because of this and because of the types of surgeries involved, a staged approach has been developed whereby the soft tissues are treated first and if unsuccessful, the next stage of therapy is skeletal surgery to open the airway.[57] This approach has been utilized since the mid 1980's and good long-term follow up results are reported with these procedures, specifically the uvulopalatopharyngoplasty with or without tonsillectomy and the genioglossus advancement and hyoid suspension procedures, along with bimaxillary advancement procedures when necessary.[58] Utilizing these surgical approaches in highly selected patients, may result in success rates of 65-97% . In unselected patients pharyngoplasty alone results in subtantial correction of obstructive sleep apnea (defined as a 50% drop in RDI and a post-operative RDI<20) in perhaps 40% patients [58a] with somewhat better results in highly selected series.

B.3. Compliance with Effective Therapy

The most commonly prescribed therapy for the obstructive sleep-apnea syndrome is continuous positive airway pressure therapy (CPAP). Effectiveness of CPAP in eliminating sleep-disordered breathing has been well documented. Since CPAP is effective in eliminating disordered breathing, criticisms of its effectiveness hinge on: a) whether evaluations for effective levels have been adequately determined in individual patients, and b) whether patients are consistently using treatment.

Two issues are involved in evaluating nasal CPAP use by patients, acceptance and compliance. Acceptance involves willingness to undergo nasal CPAP titration and take the equipment home. Compliance with nasal CPAP involves the consistency of use at home. Acceptance is obvious whereas compliance is a more complicated issue. Unfortunately, studies of CPAP compliance have not always made a distinction between the two.

Compliance studies are complicated by a lack of uniformity in defining and measuring compliance. Some studies define compliance by use of CPAP for a predetermined number of days and hours, while others define it by number of hours used divided by estimated number of hours slept. Additionally, methodologic variations exist concerning how effective CPAP level is determined and how patients are introduced to CPAP and followed for problems. Compliance with nasal CPAP has not been correlated in most studies with severity of apnea, though improvement in daytime somnolence may predict more consistent use of equipment [59, 60]. Patients experiencing mask interface problems or side effects of CPAP are less apt to use the device [61-63]. However, patient education and management of CPAP problems can improve compliance[64].

Most patients who were unwilling to use CPAP on a regular basis made their intentions known early after treatment started [65-67]. Kribbs, et al, found good correlation between CPAP use based on self-report and CPAP monitors [68].

Weaver, et al., reported that intermittent users of nasal CPAP therapy had residual symptoms of OSA compared to more consistent CPAP users [69]. Several studies suggest that most patients continuing CPAP therapy use it nightly for the majority of their sleep period [61] [59, 60, 62, 63, 66-68]. One difficulty in comparing compliance studies is the variation in how effective CPAP level was determined. Given that symptomatic improvement may reinforce compliance with CPAP, some studies may show lesser compliance because of suboptimal treatment or presence of additional sleep disorders.

The "optimal" compliance with nasal CPAP therapy is not known. Several studies suggest a slight protective effect for up to one night off CPAP even after a few days, though there is a large degree of individual variation[68, 70-72]. Decreased sleep fragmentation and increased arousal threshold may be the mechanism for this "carry-over" protection. Many articles assessing effect of treatment on morbidity and mortality associated with OSA have not formally evaluated compliance. Compliance as far as average nightly hours of use would not be expected to be significantly greater than that found by compliance studies. However, the threshold number of hours of nasal CPAP necessary to prevent or lessen long-term health consequences is not known. Data regarding optimal nightly duration of CPAP use to alleviate symptoms such as daytime sleepiness is also scarce.

At this point in time, compliance is best judged by a physician with training and experience in treating sleep disordered breathing. The most powerful studies of CPAP compliance utilize objective measurements of CPAP use and symptom resolution. However, these techniques are not always available to the clinical practitioner. The choice of CPAP equipment given to a patient is usually based on economic factors. Devices with "use meters" to confirm patient reported use are not readily available in all cases. Similarly, objective measurements of symptom resolution, such as the simulated driving test, Multiple Sleep Latency Test, or neuropsychological batteries, are not often utilized because of economic barriers and patient time constraints. Devices such as driving simulators are not affordable to many centers and measures such as the Multiple Sleep Latency Test may not be reimbursed by third party payers when used solely to confirm compliance and symptom resolution. In an era where the impetus is to keep health costs down and treatment decisions are not left only to the discretion of the medical practitioner, it is not equitable to penalize sleep apnea patients because objective compliance measures are not always available. Thus, the most practical means to assess compliance at this time is based upon the clinical judgment of the treating physician regarding patient report reliability and symptom resolution. Patients with other chronic diseases, such as hypertension, are not usually rated adversely in insurability due to lack of "pill counts" or objective determinants of compliance with prescribed therapy. Simply measuring a blood pressure on a given office visit does not ensure that the patient has taken their prescribed medications on other days. Deference in such instances is frequently given to the practitioner's judgment when favorable insurance ratings are granted based on documented blood pressure control on a specific day. In contrast, obstructive sleep apnea patients are not consistently given better insurance ratings if treated with CPAP because of the lack of objective documentation.

Patient education and treatment of problems arising during CPAP use have been shown to improve compliance. Patient follow-up under the direction of the treating physician is advisable for this reason as well as to ensure symptom resolution. Optimal timing of patient follow-up ultimately should be at the discretion of the treating physician, but generally should occur within the first one or two months of CPAP treatment. This allows adequate time for symptom resolution yet providing early intervention for problems which may limit compliance. Subsequent follow-up to determine long-term compliance and potential changes in treatment requirements is also advisable.

B.4.1 Conclusions

· Current evidence suggests that consistently used effective CPAP reverses morbidity and mortality associated with sleep apnea
· Any therapy that is demonstrated to reverse sleep-disordered breathing would be expected to reverse morbidity and mortality associated with the disease
· Effectiveness of therapies is best documented by polysomnography
· Patient compliance with CPAP can be assessed in the first two months of therapy and can be subsequently verified by periodic clinical evaluation and, when appropriate, recording of the time of CPAP machine operation.

C. Summary

C.1. The obstructive sleep-disordered breathing syndrome:

a) causes an increased risk of mortality and hypertension
b) is associated with an increased frequency in patients with strokes and heart attacks
c) causes an increased risk of automobile and work-related accidents

C.2. Effective and consistently used treatments of the obstructive sleep-disordered breathing syndrome with CPAP, dental devices, surgery, and weight loss results in normalization of these risks.

C.3. Effectiveness of treatment is best documented by polysomnograhic studies and periodic clinical evaluation demonstrating reversal of the symptoms and findings attributable to the obstructive sleep-disordered breathing syndrome.

D. Recommendations

D.1. Patients with sleep apnea requesting insurance should have:

· polysomnographic studies documenting effectiveness of treatment periodically.

· clinical evaluation to document consistent use of devices and correction of clinical symptoms and findings of obstructive sleep-disordered breathing.

D.2. Patients who are compliant with proven effective therapy for sleep apnea should be expected to have little or no risk of excess mortality due to sleep apnea.

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