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Original article

Effect of the new Medicare guideline on patient qualification for positive airway pressure therapy

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Abstract

Background: New Medicare criteria for prescribing continuous positive airway pressure (CPAP) recognize hypopnea as a sleep disordered breathing event. In so doing, hypopnea was redefined as requiring a 4% oxygen desaturation. The criteria omit electroencephalogram (EEG) arousals from the definition. This study was designed to assess how the new Medicare guideline changes CPAP eligibility.

Methods: Polysomnograms from 113 consecutive patients with obstructive sleep apnea were scored using both a definition for hypopnea that considered EEG arousals and the new Medicare definition that does not consider EEG arousal. CPAP eligibility was evaluated and compared.

Results: Sixteen percent of all patients and 41% of patients apnea + hypopnea index $\leq$20 did not qualify for CPAP under the new Medicare guidelines.

Conclusions: The new Medicare guidelines may underestimate OSA event occurrence and thereby deny CPAP therapy to many patients. Published by Elsevier Science B.V.

Keywords: Medicare guidelines; Obstructive sleep apnea; Hypopnea; Arousals; Desaturation

1. Introduction

The new Medicare guideline for prescribing continuous positive airway pressure (CPAP) to treat obstructive sleep apnea (OSA) went into effect on April 1st 2002. One crucial change involved recognizing hypopnea as a sleep-disordered breathing event. Unfortunately, operational definitions for hypopnea vary [1,2]. Hypopnea in its simplest form is a decrease in ventilation. However, the term is generally used to denote a pathophysiological sleep event resulting from partial airway occlusion. The resultant flow limitation can produce oxyhemoglobin desaturation, central nervous system arousal, or both. The new Medicare guideline defined hypopnea as 'an abnormal respiratory event lasting as least 10 s with at least a 30% reduction in thoracoabdominal movement or airflow as compared to baseline, and with at least 4% desaturation'. This definition was based on recommendations made by the Clinical Practice Review Committee of the American Academy of Sleep Medicine [3].

Other major changes in the Medicare guideline include: (1) changing the CPAP treatment threshold from a simple count total of 30 apnea episodes to a sleep normalized parameter; and (2) considering patient comorbid conditions in setting the treatment threshold. Thus, patients with sleepiness, cardiopulmonary disease, or other apnea-related comorbidities are eligible for treatment when five or more apnea and/or hypopnea events occur per hour of sleep. By contrast, the non-sleepy, otherwise healthy patient must have 15 apnea and/or hypopnea events per hour of sleep.

Our clinical impression was that more than half of the hypopnea events resulting from flow limitation ended with arousal and/or desaturations of less than 4%. Therefore, applying the 4% desaturation requirement for defining hypopnea would significantly decrease the number of events scored. As a result, the new Medicare guideline, which does not include arousals as part of the definition of hypopnea, would underestimate important sleep-disordered breathing events. Consequently OSA would be under-diagnosed and treatment denied to a subgroup of patients. The purpose of

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the present study was to assess how the new Medicare guidelines change CPAP eligibility.

2. Methods

2.1. Design

Medical records of 113 consecutive patients recently diagnosed with obstructive sleep apnea at the Houston VAMC Sleep Disorders Center (August 2001–March 2002) were reviewed. Appropriate IRB committees approved this protocol. Optical disk copies of polysomnographic records were scored using three different hypopnea definitions. Common to all definitions was that hypopnea involved a 10 s or more decrease in thoracoabdominal effort or airflow (from thermistor) by at least 30%, and was associated with:

A 4% desaturation or electroencephalogram (EEG) evidence of arousal;
B 4% desaturation;
C EEG evidence of arousal.

Definition A accords with how hypopnea events are traditionally scored in our clinical practice. The overall summary statistic combining apnea and hypopnea scored in this manner is apnea + hypopnea index (AHI). AHI was used as the benchmark for OSA disease severity. *Definition B* accords with how hypopnea is scored by the new Medicare guideline. Essentially, these events are hypopnea events associated with desaturation, designated as desaturating hypopneas (DH) and summarized with an apnea + DH index (ADI). *Definition C*, which was applied only for purposes of comparison, is not used clinically. Nonetheless it can be useful to compare ratios of non-desaturating hypopnea with arousals to DH events. These non-desaturating hypopnea (NDH) events can be summarized with an apnea + NDH index (ANI). All arousals were scored according to the American Sleep Disorders Association (ASDA) guidelines [4].

Definition of comorbid conditions was chosen in the context of new Medicare guidelines, with comorbidity defined as the presence of hypertension, coronary artery disease, congestive heart failure, cerebrovascular accident, clinical depression or self reported sleepiness (Epworth sleepiness scale score >10).

2.2. Materials

The Grass Heritage (Astromed, RI) computerized polysomnographic systems were used to record electroencephalograms (C3-A2, O1-A2), left and right electro-oculograms, sub-mentalis electromyograms (EMGs), anterior tibialis EMGs, airflow [with oral and nasal thermistors (ProTech, WA)], nasal pressure (in three patients suspected of having Upper Airway Resistance Syndrome), electrocardiograms (using modified precordial lead), and respiratory effort [thoracic and abdominal movements using double buckle piezoelectric belt system (Sleepmate Technologies, VA)]. Oxygen saturation was measured using ear probes with Ohmeda pulse oximeters (Ohmeda, CO). Polysomnographic records were stored on optical disks and all polysomnograms were scored manually by one of the authors. The scorer was not blinded with regard to events identified using the alternative definition.

2.3. Statistics

Data were analyzed using Statistical Package for Social Sciences V.9.0.0 (SPSS, Chicago, IL). Non-parametric tests were used because data were not normally distributed. Mann–Whitney rank-sum test was used to assess the group differences for continuous variables and Chi Square tests were used for categorical variables. Spearman rank correlation was used to assess correlations.

3. Results

We evaluated polysomnograms from 113 consecutive patients with obstructive sleep apnea undergoing diagnostic sleep evaluations. Age ranged from 31 to 81 years (mean = 57 years) and 96% were male. The sample population was 65% Caucasian, 28% African-American, 6% Hispanic, and 1% Native American. Mean AHI was 50.3 events per hour of sleep. Using the clinical convention according to Aldrich [5] and Chockroverty [6], 34 patients (30%) had mild OSA (AHI $\leq$20), 31 patients (27%) has moderately severe OSA (AHI $>$20 and $\leq$50) and 48 patients (43%) had severe OSA (AHI $>$50). Table 1 shows additional characteristics of patients assessed.

Overall, 90% of the patients had one or more comorbid condition, with no statistically significant difference across varying AHI groupings.

ADI was calculated for each patient. Each patient was classified according to their eligibility for CPAP under the new Medicare criteria; that is, a patient is eligible if ADI $\geq$ 15 (for any patient), or if ADI $\geq$ 5 (in a patient with comorbidity).

Overall, 16% of patients in this sample did not qualify for CPAP under the new Medicare guideline. Of all the patients who did not qualify for CPAP, most (78%) had AHI $\leq$20. Among patients with mild OSA, 14 (41%) did not qualify. The corresponding disqualification rates for patients with moderate or severe OSA were 10% ($n = 3$) and 2% ($n = 1$), respectively (see Fig. 1). Table 2 shows a comparison of the percentage of patients who would qualify for CPAP therapy under the old (30 or more apnea episodes) and new Medicare Guideline. Patients who did not qualify for CPAP had lower apnea indexes (AIs, 3 versus 29, $P < 0.001$), lower AHIs (19 versus 56, $P < 0.001$), lower apnea: hypopnea ratios (0.57 versus 1.42, $P < 0.001$), lower DHs as a percentage of hypopnea (12 versus 38%,

Table 1
Summary of patient characteristics[a]

Variable	Mean	Minimum	Maximum	Standard deviation
Age (years)	57	31	81	10.9
BMI (kg/m^2)	35.9	25.1	64.2	8.1
Epworth sleepiness scale score	12.4	0	23	5.7
AI (events/hour)	25.1	0	104	28.3
AHI (events/hour)	50.3	6	142.4	35.4
ADI (events/hour)	34.5	0.9	115.2	31.0

[a] Notation: AI, apnea index; AHI, apnea + hypopnea index; and ADI, apnea + DH index.

$P < 0.001$), and a lower Epworth sleepiness scale scores (10 versus 13, $P = 0.007$). Age, sex, BMI, comorbidity prevalence, baseline oxygen saturation, and minimum oxygen saturation did not differ between qualifiers and non-qualifiers.

Furthermore, within the group with AHI ≤20, qualifiers did not differ from non-qualifiers in age, race, BMI, AI, AHI, comorbidity presence, apnea: hypopnea ratio, baseline oxygen saturation, or minimum oxygen saturation. By contrast, non-qualifiers had a significantly lower proportion of hypopneas associated with 4% or more desaturation (14 versus 40%, $P < 0.001$) than those patients who would qualify for CPAP under the new Medicare guideline.

In our sample, approximately one third (34%) of all hypopnea events were associated with 4% or more desaturation. This fraction did not change across groups varying by AHI grouping. Consequently, the AHI–ADI difference increased linearly with increasing AHI. This invariance applied to groupings but not to individual patients. The ratio of AHI to ADI results varied widely, making it difficult to predict the proportion of hypopneas with 4% desaturation for an individual patient.

Ratio of number of apneas to the number of hypopneas for a given AHI increased in a statistically significant manner with increasing severity of OSA (0.41 for mild OSA, 0.64 for moderately severe OSA, and 2.64 for severe OSA; $P < 0.001$, Mann–Whitney U). Mean Epworth score was 13 and did not differ significantly between OSA severity groups. ADI correlated well with AHI (0.919, $P < 0.01$; Spearman Rho) (see Fig. 2). AHI correlated better with the ANI (0.953, $P < 0.001$; Spearman Rho) when compared to ADI.

4. Discussion

Overall, 16% of patients in this sample did not qualify for CPAP treatment under the new Medicare guideline. Of all the patients who did not qualify for CPAP, most (78%) had AHI ≤20. In the group with AHI ≤20, 41% of patients would be denied CPAP treatment. However, the new definition compares favorably with the old Medicare definition that requires 30 or more apnea episodes as the cutoff point (26% additional patients would qualify for treatment with the new versus old Medicare definitions).

Clinically, the new Medicare guidelines will deny CPAP

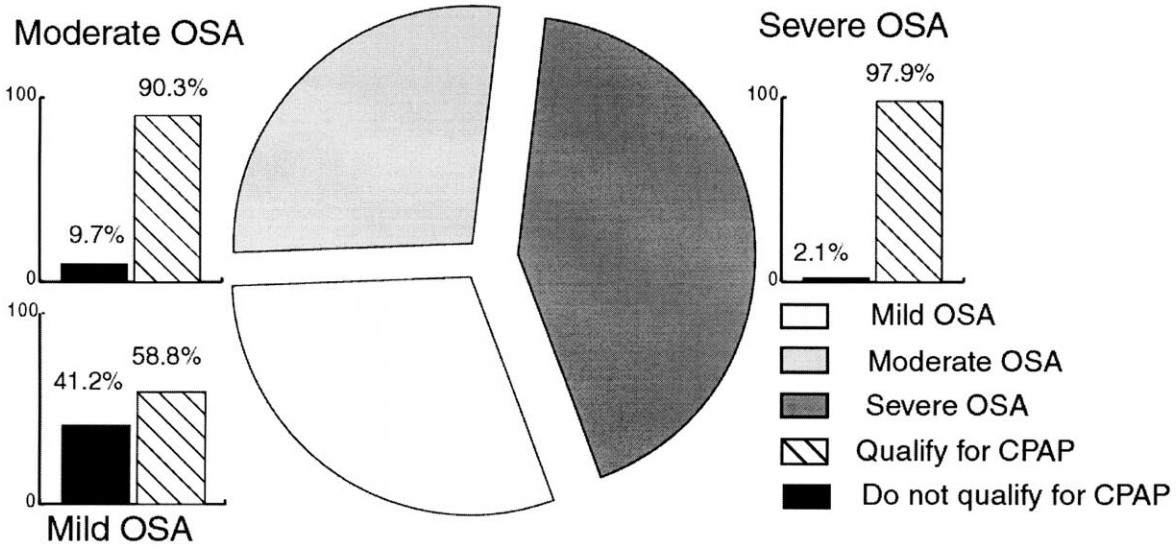

Fig. 1. Pie chart representing patients with mild OSA (30%), moderate OSA (27%) and severe OSA (43%). Accompanying bar charts represent proportion of these subgroups that qualify/do not qualify for CPAP under the Medicare guidelines.

Table 2
Comparison of percentages of patients qualifying for CPAP under the old and new Medicare guidelines

Patients qualifying for CPAP therapy	AHI ≤20 ($N = 34$)	20 < AHI ≤ 50 ($N = 31$)	AHI >50 ($N = 48$)	Total ($N = 113$)
Old Medicare guideline (%)	15	61	88	58
New Medicare guideline (%)	59	90	98	84

treatment to many patients with mild OSA but will have little effect on most patients with moderate or severe OSA. Whether or not treating patients with low AHIs will affect long-term health outcome will require additional research because the benefits of treating these patients with CPAP remain controversial. However patients with Upper Airway Resistance Syndrome (who would not qualify under the new guideline) objectively benefit from CPAP [7]. Epidemiologically, the estimated prevalence and severity of OSA in a given population derived from the Medicare definition will be lower than that derived from a definition that considers arousals in scoring hypopnea events. This lower estimate may or may not just be scientifically misleading but the new guidelines could lead to decreased resource allocation relative to the definition of hypopnea that has been used in our laboratory (which considers sleep disturbance in the form of arousal). For example, in our sample, 30% of patients had AHI ≤20, 27% had AHI between 20 and 50, and 43% had AHI >50 based on AHI where hypopnea considered the presence of EEG arousal. Using the new Medicare criteria, 43, 30, and 27% would have ADIs in these ranges.

A recent peer-reviewed, evidence-based medicine literature review concerning sleep disturbances, increased wakefulness, and arousals concluded that 'It is clear from this literature that fragmented sleep is less restorative than consolidated sleep, and leads to sleepiness-related daytime impairment [8].' Untreated OSA contributes significantly to mortality [9,10]. Motor vehicle accident rate in patients with OSA is seven times higher than general the population [11], and excessive daytime somnolence is a likely contributor in a significant proportion of these accidents.

Published evidence also indicates that arousals clearly produce adverse cardiovascular consequences [12–14]. Some studies suggest increased arousals are more important than sleep hypoxemia as a risk factor for increased sympathetic tone and consequent hypertension. Yoon and colleagues (2001) found EEG arousals correlated better with the sympathetic activity than the mean arterial saturation [15]. Ringler and coworkers [16] compared blood pressure elevation in four groups of patients: OSA with desaturation, OSA with nocturnal oxygen supplementation and no desaturation, OSA treated with no arousal or hypoxemia, and OSA treated with no arousals but hypoxemia induced by supplemental nitrogen. The first three groups had equivalent blood pressure (BP) elevation; by contrast, the fourth group did not. Thus, BP elevation was linked primarily to arousals rather than desaturation events. Catcheside and colleagues (2001) found sympathetic activation accompanying arousals from sleep was unaffected by experimentally induced hypoxia in healthy men [12]. Arousals and arterial desaturation contribute to the increased sympathetic tone and blood pressure, with possible interaction between the two factors [17,18].

The Sleep Heart Health Study (SHHS) defined hypopnea as 4% desaturation associated with 30% reduction in thoracoabdominal excursion or flow limitation. Thus, AHI in SHHS differs from what is labeled AHI in clinical practice that considers arousal in the definition of hypopnea but accords with ADI as defined in the present study. SHHS demonstrated that increased cardiovascular morbidity correlated with ADI [19]. What remains unclear is whether long-term outcome and cardiovascular morbidity correlate with AHI defined in a manner that considers disturbance of the sleep process.

In general, approximately one third of all hypopnea events were associated with 4% or more desaturation. Furthermore, this was constant across AHI groupings. Desaturation is determined by interaction of several factors: (1) baseline SaO_2; (2) duration of event; (3) degree of flow

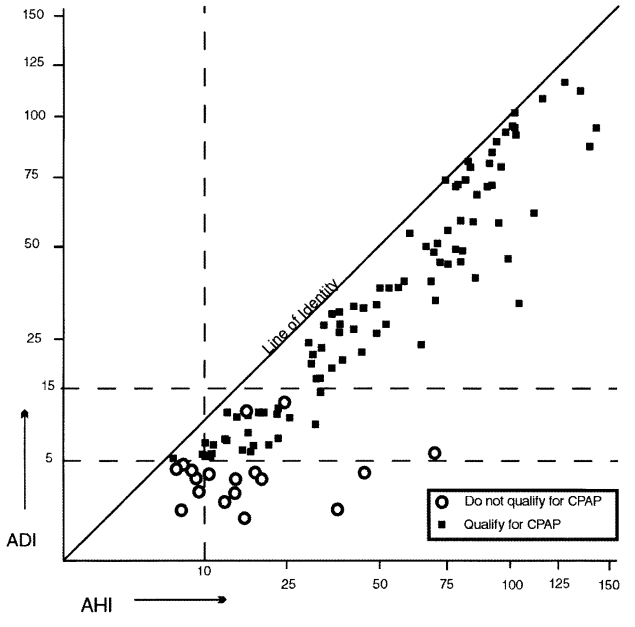

Fig. 2. Correlation between apnea + hypopnea index (AHI) (events/hour) and apnea + desaturating hypopnea (DH) index (ADI) (events/hour); ($r = 0.919$). The line of identity is shown to emphasize that ADI is usually a partial fraction of AHI (below the line). Solid squares represent patients qualifying for CPAP under the new guidelines, and hollow circles represent patients who will be denied CPAP under the new guidelines.

limitation; and (4) underlying cardiopulmonary function. These factors vary significantly between individual patients; however, they may be evenly distributed across severity groups within a given population.

In our population, which has a high prevalence of cardiopulmonary disease, the proportion of hypoxia-inducing hypopnea to all hypopnea was 34%; however, this would likely differ in other patient populations. That is, in clinics drawing from a healthier population base, CPAP denial based on the new criteria may be significantly higher; however, further study is needed.

This constant fraction translates into an increasing difference between AHI and ADI as the AHI increases (i.e. the AHI-ADI increases as the AHI increases). The mean difference between AHI and ADI varied from 7 for patients with mild OSA, to 23 for patients with severe OSA. This contrasts with the near perfect correlation reported by Tsai and colleagues (1999) between AHIs calculated using different definition for hypopnea events, and the lack of impact of exclusion of arousals on this correlation [20]. The discrepancy between these results and our data likely stems from differences in the definitions for arousal. In the present study we used the published ASDA standard for scoring arousals, whereas in the other study an EMG amplitude criteria was added for scoring arousals even in NREM sleep. This likely led to exclusion of an unknown number of hypopnea events associated with arousals.

In summary, the current Medicare guideline that requires 4% desaturation as part of the definition for hypopnea will classify fewer events than a definition that considers desaturations and/or arousals. This may lead to a lower OSA prevalence estimate in a given population. More important clinically, the guideline may deny CPAP therapy to a significant proportion of patients with mild OSA. However, further study is required to determine whether changing CPAP treatment threshold has any long-term health consequence. Nonetheless, arousals have adverse physiologic consequences and thus represent an important sleep pathophysiology. To ignore arousals induced by flow limitations ignores a basic tenant of sleep medicine. Sleep is a brain process and sleep disorders are disturbances of this process.

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