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Hypopnea, a Floating Metric: Implications for Prevalence, Morbidity Estimates, and Case Finding

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Summary: The respiratory disturbance index (RDI) is the most frequently used metric to characterize sleep-disordered breathing. Clinically, the RDI is used to classify disease status and guide treatment decisions. For research purposes, the RDI is used to describe population distributions of sleep-disordered breathing. Its popularity as a cardinal disease-defining measure, however, may not be justified given that standardized criteria do not exist for defining hypopnea, a key component of the RDI. This paper reviews sources of variability in identifying hypopneas, including: the magnitude of changes in breathing amplitude necessary to describe breathing as "reduced" (from "discernible" to >50%), variations in the utilization of sensors with different sensitivities to detect airflow/ventilation (i.e. thermocouples, thermistors, and pressure transducers), and differential use of data on oxygen saturation and arousals to discriminate normal breathing from hypopneas. The extent to which disparate approaches influence the overall RDI and population estimates of disease also is discussed. **Key Words:** Hypopnea—Sleep-disordered breathing—Sleep apnea–hypopnea syndrome—Measurement.

Over the last two decades, enhanced appreciation of the prevalence and morbidity of sleep apnea–hypopnea syndrome (SAHS), coupled with increasingly available, sophisticated technology, has led to the widespread use of polysomnography to diagnose this and other sleep related breathing disturbances. The number of sleep laboratories has proliferated from a total of three accredited centers in 1977 to 337 centers and laboratories in 1996 (Torgrimson, personal communication). With the increase in number of patients diagnosed, there has been an increase in number of therapeutic interventions—medical, surgical, and most recently, dental. The increase in number of sleep laboratories that has paralleled the growing recognition of SAHS in the community has been driven by clinical lore, which dictates that a diagnosis of SAHS must be established on the basis of a specified frequency of sleep-related respiratory disturbances (traditionally termed "apneas" and "hypopneas") recorded during in-laboratory polysomnography or unattended home studies in patients with suggestive symptoms. Implicit in this practice is the notion that there is a threshold number of respiratory disturbances that defines the presence or absence of "disease" and that the severity of this "disease" can be gauged by the frequency of respiratory disturbances. Right or wrong, these assumptions are reinforced by third-party payers whose reimbursement policies for SAHS treatments require documentation of a minimum number of sleep-related respiratory events. In this regard, the apnea plus hypopnea index (AHI) has been used analogously to measure blood pressure values inasmuch as it is assumed that as a threshold value is progressively exceeded, the risk of adverse health sequel linearly increases. Treatment is therefore offered after the threshold level is exceeded. The similarities between blood pressure measurement and AHI are far overshadowed by conceptual differences as well as limited validation by outcome measures. These issues are described below.

A prerequisite to defining the threshold value of event frequency is establishment of a valid, physiologically and clinically meaningful definition of abnormal breathing events that is universally accepted and reproducibly measurable. This is relatively simple for apneas that are identified in absolute terms (i.e. absence of airflow). The challenge is greatest for establishing a "gold standard" for defining and measuring hypopneas, which conceptually reflect a quantitative spectrum of airflow (or volume) from just above zero

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to just below "normal". Thus, identifying hypopnea requires recognition of subtle, semiquantitative reductions in the amplitude of a respiratory signal for a discrete period without indication that airflow has ceased. Controversies exist regarding which sensors and combinations of sensors best detect and distinguish changes in breathing patterns, and what definitions of changes in breathing patterns constitute evidence of clinically significant upper airway obstruction. Comparisons to blood pressure measurement are useful. Published methods for standardizing blood pressure measurement exist, and they include specification of equipment standards and criteria for recording distinct Kortokoff sounds (1). In contrast, there are no guidelines regarding the use of appropriate instrumentation for detecting hypopnea (2). Identification of a numerical value (e.g. systolic blood pressure level) also is clear and is universally understood to represent the same physiological variable. However, hypopnea is detected on the basis of a change in one aspect of breathing pattern. In the absence of numerically quantitative values, the distinction between a normal and an abnormal pattern can be much less clear and difficult to operationalize. By relating outcomes to discrete numerical representations of blood pressure, prospective studies provide solid evidence of which levels of blood pressure confer morbidity (3). This contrasts with sleep medicine, for which, because of substantial uncertainty regarding the relationship between physiological "exposures" (and indeed, uncertainty as to what constitutes an exposure) and health outcomes, a small amount of data exists upon which to base definitions for identification of threshold levels from which risk or disease status are determined.

The absence of established guidelines for standardizing measurement and assessment of SAHS has, in turn, influenced clinical case finding, pathogenesis research, and estimates of the public health impact of SAHS. Problems include variable consideration of both apneas and hypopneas in characterizing disease, as well as variable identification of hypopneas. Early studies that attempted to estimate the prevalence of sleep apnea in clinical (4) and industrial (worker) populations (5) based estimates exclusively on identification of apneas. This yielded a prevalence estimate of sleep apnea as low as 1% (4). More recently, with inclusion of hypopnea in the measurement of the respiratory disturbance frequency (and defining a sleep-related breathing disorder as an AHI $\geq$ 5), prevalence estimates have increased to 9–25% (in middle-aged individuals) (6), and >25% in the elderly (7). Differences between earlier and later studies likely relate both to differences in sampling and study methodology, as well as to differences in how respiratory disturbances were defined and identified. Reports that have provided data on apnea indices, in addition to a combined apnea plus hypopnea frequency, suggest that the frequency of respiratory disturbances is at least twofold higher when hypopneas are considered as contrasted to consideration of only apneas (7). The extent to which a pure "apnea index" differs from an apnea–hypopnea index is itself variable and highly dependent on how airflow is recorded and hypopnea defined. Specifically, between-laboratory differences in the techniques and criteria used to characterize hypopnea contribute to the variability in the frequency of hypopneas identified across laboratories and studies. In this commentary, we review sources of variability in hypopnea and how this may affect our understanding of SAHS.

HISTORICAL OVERVIEW

Clinicians have simultaneously recorded sleep and breathing variables for over two decades. In 1978, Guilleminault described apneic activity, defined as the absence of airflow for >10 seconds, as rare occurrences in normal men and women, and suggested a threshold level to distinguish pathological from normal states (8). The importance of measuring hypopnea (identified as a reduction in the amplitude of airflow, recorded from a nasal/oral thermistor, and chest wall excursion, recorded with inductance plethysmography, occurring in association with a >4% decrease in oxygen saturation), in addition to apnea, was emphasized by Block et al. (9). They described approximately 40% more hypopneas than apneas (105 vs. 60 hypopneas and apneas, respectively) during sleep in 49 volunteers who were "normal". In this study, the potential importance of identifying hypopneas was underscored by the observation that hypopneas frequently were associated with oxygen desaturation, a phenomenon described as "abnormal breathing". However, the presence and wide range of numbers of hypopneas in their putatively normal volunteer sample also highlighted the fact that assumptions, and not data, were employed in applying any given cutoff level to identify clinical impairment.

The importance of quantifying hypopneas in clinical assessments was further emphasized by Gould et al. in 1988 (10). These investigators reported that 36% of a consecutive series of 50 patients with features of sleep apnea syndrome demonstrated recurrent hypopneas (defined as a 50% reduction in the sum of thoracoabdominal excursion signal obtained from inductive plethysmography, lasting for >10 seconds) but few apneas on overnight polysomnography (10). Because these patients did not differ clinically from those who demonstrated frequent apneas, they proposed that the frequency of hypopneas be considered in characterizing abnormalities of breathing during sleep and that

the term "sleep apnea syndrome" be abandoned in favor of "sleep hypopnea syndrome". Following this report, there has been an increasing trend to include assessment of hypopneas in clinical and in research studies. AHI thus is the most common metric employed in describing the level of respiratory disturbances across clinical and research laboratories. The physiologic definition of hypopnea, however, remains to be standardized. Even more troubling is that, even if comparable criteria are applied to a given variable reflecting breathing pattern, a hypopnea in one laboratory may not be physiologically the same as in another due to differences in recording techniques and sensor sensitivity. Thus, the AHI is currently a rather fluid metric and one that does not lend itself readily to uniform interpretation.

TABLE 1. *Results of a survey of interlaboratory variability in hypopnea definition [adapted from (11)]*

	% Laboratories
Minimum amplitude reduction	
Any	9%
20–30%	23%
33–40%	14%
50%	54%
Associated oxygen saturation	
Not considered	18%
Any	34%
≤2%	5%
>2 ≤ 4%	18%
>4%	25%
Associated arousal[a]	
Required	73%

[a] No consistent definition of arousal noted; both electroencephalogram-based, movement, sound.

ISSUES RELATED TO MEASURING CHANGES IN BREATHING PATTERN

Although temporal trends have been to measure and report both hypopneas and apneas, the growth in reporting hypopneas has not been accompanied by standardization of their measurement. By definition, identifying meaningful variations of persistent airflow limitation (hypopneas) is more subtle or open to individual interpretation than absence of airflow (apneas), especially when qualitative recording techniques are employed. Clinicians and investigators have developed diverse approaches for interpreting which of these more subtle changes in breathing represent "clinically significant" respiratory disturbances. Additionally, the availability of numerous sensors for estimating changes in flow and volume has contributed to different approaches for gauging airflow and respiratory effort among laboratories. An American Thoracic Society Consensus Report in 1989 acknowledged the variability both in sensors used to characterize airflow and measure respiratory effort and the variability in popular definitions of hypopnea (2). The panel wrote, "While methods of monitoring sleep stage are standard, the optimal techniques for monitoring respiration are uncertain . . . there are differences in the criteria used to define hypopnea. The field would be advanced if standard definitions of indices used to quantify abnormality were developed". The extent of this problem was highlighted in a survey of 44 American Sleep Disorders Association-accredited sleep laboratories published in 1994 (11). This survey showed that no two laboratories used the same equipment and definition of hypopnea! The sources of variation included: criteria for amplitude reduction; inclusion of oxygen desaturation as part of hypopnea definition; inclusion of arousal as part of hypopnea definition; and minimal duration for hypopneas (Table 1). Although the majority of laboratories used a thermistor to detect airflow, it is likely that several kinds of thermistors were used. Great variability was noted in the sensors used to detect respiratory effort. Arousals, required to identify hypopneas in some laboratories, also, were defined by diverse criteria, including evidence of snoring, movement, and/or electroencephalogram (EEG) changes.

Quantitative measurements of either airflow or ventilation during sleep are usually impractical, requiring use of a face mask with a pneumotachograph. Therefore, qualitative assessments usually are made from changes in the relationship between inspired and expired carbon dioxide or temperature (thermistors or thermocouples) or from measures of rib cage and abdominal motion [inductive plethysmography, strain gauges, piezo sensors, electromyogram (EMG) electrodes] to make inferences about airflow and ventilation, respectively. Several sources of between-laboratory variation relate to the use of different sensors and combinations of sensors for characterizing ventilation during sleep, each of which may vary dramatically in its ability to accurately gauge ventilatory changes. Hypopneas will be detected differently according to the extent to which changes in airflow as compared to changes in ventilatory excursions are emphasized—an area for which there is no consensus. Furthermore, since various sensors detect ventilatory changes with different sensitivities, the choice of which of the more than five commercially available thermistors and/or more than two inductive plethysmography sensors and/or more than three strain gauges or piezo electric sensors, etc., are selected for measuring airflow and/or ventilation will influence hypopnea detection. Even among thermistors, often assumed to have similar measurement properties, variations in sensitivity exist because of differences in frequency responses, which are in

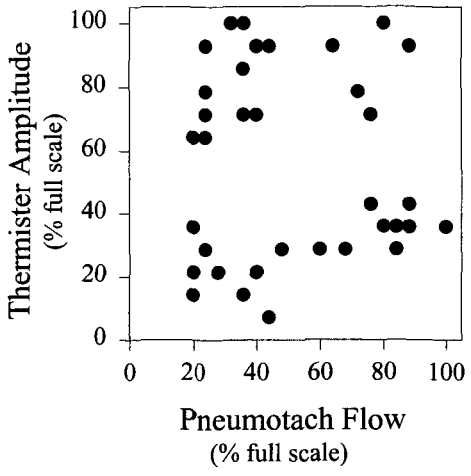

FIG. 1. Relationship between breath amplitude, measured by a thermistor (y axis) and by a pneumotachograph. No relationship is demonstrated (provided courtesy of David Rapoport, M.D.).

turn related to differences in surface area and mass differences among available products. A dramatic example of this was provided by the Toronto Sleep Laboratory, where poor and variable levels of agreement were reported for hypopneas detected by three different thermoelements and inductive plethysmography, each compared to hypopnea detection by direct measurement of ventilation with body plethysmography (kappa statistics: 0–0.3) (12). This suggests that variability in characterizing ventilation related to use of different sensors may be greater than has been appreciated previously. The potentially very poor correlation for data on breathing amplitude obtained with different sensors is shown also in Fig. 1. These data were obtained while simultaneously recording peak flow from a commercially available thermistor and a pneumotachograph (Fig. 1). Essentially, no relationship is seen between the amplitudes as measured by each sensor. Concern regarding the poor levels of agreement between these ventilatory measurements has been recognized by some and has encouraged the development of unobtrusive techniques to more directly assess airflow. In particular, assessment of ventilation from a nasal cannula connected to a simple pneumotachograph has been advocated as a more direct and sensitive means to quantify changes in airflow (13). However, this technique may be limited because of problems maintaining the patency of the cannula during the study, problems distinguishing apneic from hypopneic activity, and errors due to mouth breathing. It is not clear whether newer methods of assessing airflow will enhance diagnostic accuracy.

Although data on breathing obtained during clinical polysmonography are almost invariably obtained from noncalibrated sensors, they are usually interpreted quantitatively. Thus, hypopneas are usually identified based on arbitrary criteria for changes in the amplitude of the measured signal that are applied rigorously within any given laboratory. As shown in Table 1, these criteria vary widely among laboratories. Clearly, differences in the frequency of reported events will vary as a function of the magnitude of the change in signal required to identify any cluster of breaths as "normal" versus "hypopneic" (according to the definition used for hypopnea).

It is evident that even if there were universal agreement regarding the definition of hypopnea, differences in sensor characteristics and properties would likely lead to application of the same label to what, in reality, may be a wide range of physiologic events. Further complicating this issue, even when using a standard amplitude criterion, inconsistencies in characterizing event frequencies will result from numerous technical (e.g. precise placement of sensors) and biological (e.g. body habitus, breathing route) factors that vary between individuals. Thus, even within laboratories using a standard set of instruments and definitions, hypopnea frequencies may provide information regarding ventilatory impairment that is noncomparable between and even within individuals (across studies). The recommendation made by George and Kryger in 1985 that ". . .we should stop using the term hypopnea and instead describe the reductions in tidal volume, or frequency, or both" (14) reflects these frustrations; however, even this fairly drastic statement does not address the problems of variable quantification of tidal volume that result from use of different commonly used sensors and the vagaries introduced by variations in sensor placement and patterns of breathing between individuals.

A number of special clinical circumstances present further ambiguities in measuring and interpreting hypopneas. The variability in breathing patterns in rapid eye movement (REM) sleep often makes it impossible to establish the "normal" amplitude of breathing in this state, and thus difficult to quantify the amplitude changes of potentially "hypopneic" breaths. Particularly difficult clinical conditions include chronic obstructive pulmonary disease, in which ventilatory changes may be difficult to quantify from inductive plethysmography because of paradoxical movements of the thorax and because of the occurrence of desaturations unrelated to upper airway obstruction. Identifying hypopneas is also problematic in periodic movement disorders (or other arousal disorders), in which periodic arousals may cause recurrent hyperpneas, obfuscating the distinction between breaths of normal versus reduced amplitude. Even less is known about monitoring partial upper airway obstruction in the pediatric population (15). The importance of recognizing hypopneas has been further challenged with

TABLE 2. *Variation of prevalence with hypopnea definition*

Study	Sample	Sensors	Hypopnea definition			% AHI > 5
			Amplitude	Desaturation	Arousal	
Gislason et al. 1988 (17)	61 Swedish men, 39–69 years	Thermistor/Sound	"Marked"	≥4%	+	1.3%
Ancoli-Israel et al. 1991 (7)	427 elderly, >65 years	Inductive bands	>50% reduction	–	–	81%
Dickel and Mosko 1990 (18)	100 community volunteers, >70 years	Thermistor	"Decreased"	+/–	++	34%
Phillips et al. 1992 (19)	92 volunteers, 50–80 years	CO_2/Inductive bands	NI	NI	NI	15%
Jennum and Sjol 1992 (20)	748 Danes, 30–60 years	Inductive bands	NI	NI	NI	M: 11% F: 6%
Young et al. 1993 (6)	602 Wisconsin employees, 30–60 years	Thermocouple/CO_2	"Discernible"	≥4%	–	M: 24% F: 9%
Olson et al. 1995 (21)	441 Australian community residents, 35–69 years	Sound/Chest movement	Deviation of sound/excursion	–	–	M: 78% F: 57%
Redline et al. 1997 (22)	385 Cleveland community residents, 3–84 years	Thermistor/Impedance	"Discernible"	>2.5%	–	M: 40% F: 20%

AHI, apnea plus hypopnea index; NI, not indicated directly in methods section; M, male; F, female.

the recent description of the "upper airway resistance syndrome" (16). In this syndrome, daytime fatigue is thought to occur secondary to sleep fragmentation (arousals or microarousals) associated with increased respiratory effort, often unassociated with clear changes in respiratory amplitude or oxygen saturation.

IMPLICATIONS

What are the implications of the variability in defining and measuring hypopneas? Differences in measurement of ventilation and definition of hypopnea most clearly affect case finding (clinically) and population prevalence estimates (epidemiological studies), in which cutoff values are used to identify disease or affection status. Table 2 demonstrates the range of prevalence estimates for sleep-disordered breathing based on an AHI > 5 (17–22). As can be seen, prevalence rates vary widely. Some of the differences clearly relate to the age and gender composition of the samples studied, as well as to the extent to which snorers were oversampled in recruitment and/or analyses were adjusted for sampling frequencies. Interestingly, this table shows that, in contrast to common clinical practice, most epidemiological studies have not used a defined amplitude criterion to define hypopneas (usually using changes described as "any" or "discernible"). Whether the lack of clear criteria for amplitude changes has negatively affected the reliability or validity of such measurements is not clear. Additionally, research studies have used various combinations of sensors to detect breathing changes and have utilized different criteria for corroborative data (desaturation and arousal) to identify hypopneas. These differences limit the ability to compare studies; thus, it is difficult to use such data to confirm findings across populations or to identify whether study differences relate to demographic and geographic factors. In analyzing our data (the Cleveland family study), it is apparent that, when using an amplitude criteria for hypopnea defined as "discernible", marked differences in prevalence estimates result as the desaturation criteria change from 2.5 to 5% (reducing prevalence estimates at least threefold). The inability to confirm prevalence rates across populations makes estimating the potential public health impact of sleep-disordered breathing (SDB) almost impossible.

Similar methodological problems plague studies of pathogenesis, where the intent of the investigation is to relate sleep-disordered breathing (the "exposure") to the occurrence of hypertension, cardiovascular or cerebrovascular events, and neuropsychological impairment. Establishing a potentially pathogenetic role of SAHS in the development of chronic disease requires demonstration of consistent "dose–response" relationships between the exposure (e.g. AHI) and the outcome (e.g. hypertension). However, such risk relationships may be discrepant as classification of exposure varies (e.g. with inconsistent definitions of hypopnea). For example, in studies of neuropsychological impairment, stronger relationships may be observed between impairment and "exposure" when exposure is based on an AHI that incorporates only hypopneas associated with arousals, as compared to studies that define exposure with an AHI that is calculated without consideration of associated sleep fragmentation. Similarly, if it is the exposure to hypoxemia that is involved with vascular responses to SAHS, then one may expect stronger observed relationships for studies utilizing definitions of hypopnea that require

TABLE 3. *Comparison of study findings[a] examining the relationship between SAHS activity and hypertension*

Study	Sensor	Corroborative Data	Crude OR	Adjusted OR
Jennum and Sjol 1993 (23)	Inductive bands	None	2.0	ns
Hla et al. 1994 (24)	Thermistor/CO_2	>4% desaturation	—	2.0–5.0
Olson et al. 1995 (25)	Sound/Chest movement	None	3.7	1.5

SAHS, sleep apnea–hypopnea syndrome; OR, orienting response; ns, not significant.
[a] Community-based studies reviewed.

desaturation for hypopnea identification, as compared to studies that consider amplitude changes only. Could this explanation account for the widely divergent conclusions of different investigators regarding the relationship between hypertension and SAHS? As seen in Table 3 (23–25), of the three recent population-based studies of hypertension and SDB, the strongest relationship with AHI was demonstrated in the study by Hla et al. in which hypopnea was based on the observation of any change in airflow associated with a 4% desaturation (24).

Extrapolating data from epidemiologic or other research studies to clinical practice requires an understanding of the research methods used to identify and describe AHI activity and a comparison of these methods to those used individual practices. However, inadequate descriptions of methods used, and a poor appreciation of how differences in methods could affect disease identification, limit the ability to make such assessments.

As with many other relatively new areas of investigation, the challenges are many. Among these is the need to identify the relevant physiological parameters for characterizing SAHS and identifying high risk subjects (or those who would benefit from intervention). Identifying the essential mediators of SAHS outcomes (i.e. desaturation, sleep fragmentation, autonomic disturbances, etc.) would facilitate construction of appropriate measures for monitoring and quantifying "exposure". Until recently, it had been assumed that oxygen saturation data best identified physiologically important breathing changes. Thus, the occurrence of desaturation has been used to evaluate the clinical relevance of hypopneas and to appraise the merits of various monitoring approaches. One of the first examples of the former can be found in the landmark report by Block et al., who first suggested the importance in measuring hypopneas (9). In this report, hypopneas were interpreted as "abnormal" because they were frequently associated with desaturation. Thus, the ultimate arbiter of "normality" versus "abnormality" was desaturation. One may argue that if this is appropriate, one may need to measure only oxyhemoglobin saturation. An example of the use of oxygen saturation data to evaluate measurement approaches for hypopneas is provided in the work of Gould et al. (10). These investigators compared hypopneas identified by changes in airflow to hypopneas identified by changes in thoracoabdominal motion (10). They interpreted the stronger correlations for desaturation frequency and the arousal index with a hypopnea index based on changes in thoracoabdominal movement, as compared to a hypopnea index defined by changes in thermistry, as evidence by which to recommend measurement of thoracoabdominal movement over thermistry. Interpreting the differences between such correlations (with desaturation and arousal indices) assumes that the object of measuring hypopneas is to predict desaturation or arousal. In fact, the rationale for measuring breathing is that information on breathing pattern complements information obtained from other physiological signals (or provides insight regarding the mechanisms for the desaturations/arousals on which a therapeutic plan may be based). If hypoxemia is a cardinal marker and outcome determinant of SAHS, why measure breathing pattern (rather than oxygen saturation alone)? As has been more recently recognized, sleepiness (16) and acute elevations of blood pressure (26) (two potential consequences of SAHS) may occur in the absence of desaturation. Any assessment of the relative merits of one measurement procedure versus another is incomplete without data addressing which methods for identifying hypopneas best predict short- or long-term outcomes (e.g. cardiovascular morbidity) or treatment responses. To date, we are aware of no study that has addressed this. Indeed, without standardized measurement and terminology, such data cannot be reliably obtained and meaningfully interpreted.

Figure 2 depicts the multiple (and overlapping) areas that require definition (of which hypopnea is only one area). The key challenge is to identify the relevant pathogenic exposures in any causal pathway between SAHS and adverse health outcomes (e.g. vascular disease, pathologic sleepiness, neuropsychological impairment). This requires identifying which physiologic events (exposures) are relevant, determining which can be feasibly and reliably measured, deciding which technology can be uniformly used across laboratories, deciding how to define or summarize discrete values from the physiological patterns measured, and, finally, determining which physiologic value, or set of values, best identifies high risk subjects (or subjects who could

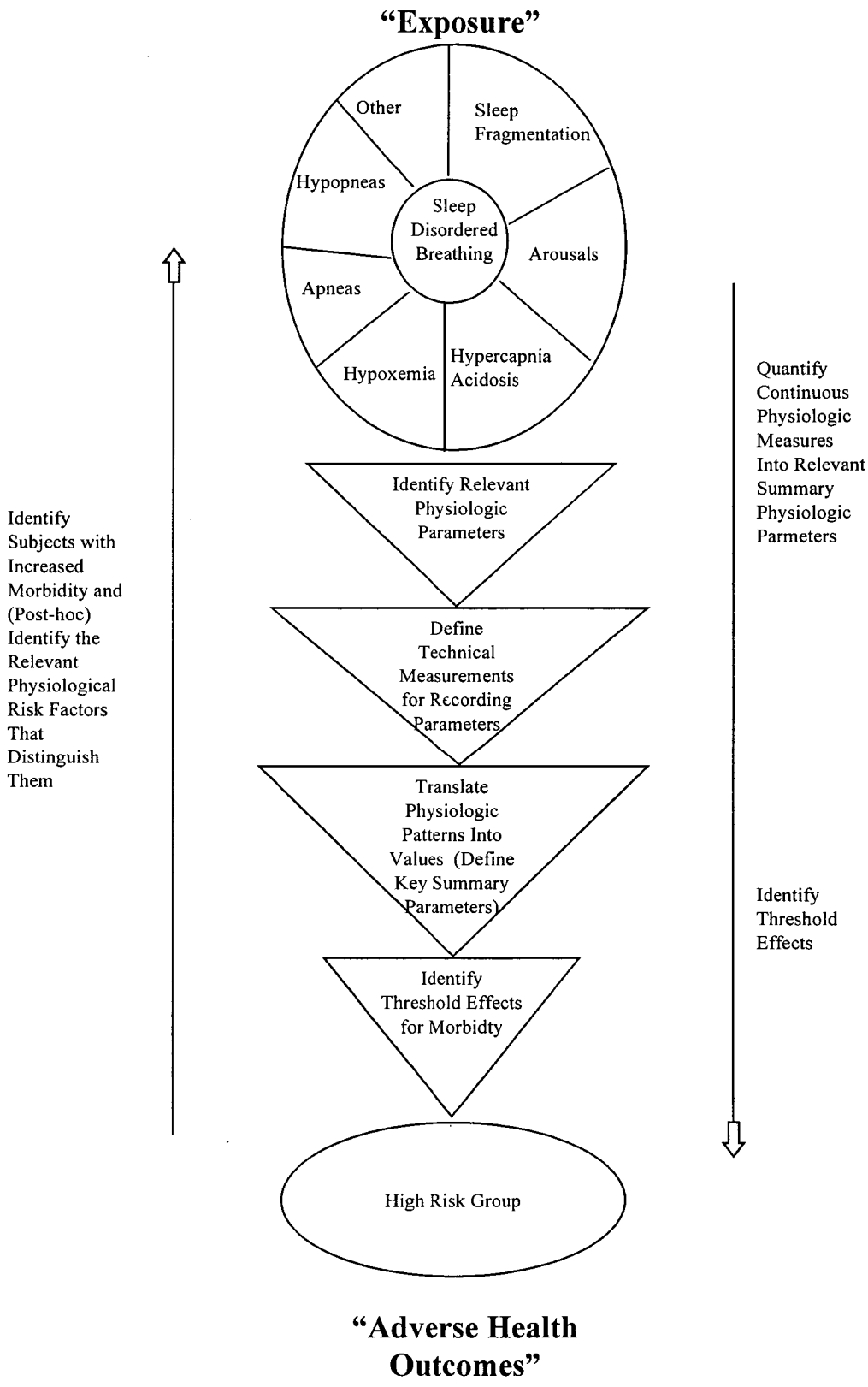

FIG. 2. Conceptual schema, illustrating problems in characterizing sleep disturbed breathing patterns as a potential "exposure".

benefit from intervention). However, identification of high risk subjects may require availability of a sufficiently robust database to, on a post hoc basis, identify which parameters best identify adverse outcomes. In turn, the availability of such a database requires some degree of agreement as to what should be measured and how such measurements should be standardized. The crucial element is to figure out how many assumptions can be made at each point in this rather complex pathway.

In 1995, the first large-scale, prospective multicenter study of the cardiovascular consequences of SAHS was initiated by the National Institutes of Health in the U.S. [the Sleep Heart Health Study (SHHS)]. One challenge has been to choose methods for measuring SAHS that would be widely acceptable to the clinical community. A major interest also was to choose methods that would be sufficiently flexible to allow a post hoc evaluation of various measures of SAHS, including which components of hypopnea (amplitude change, desaturation, arousal) are most predictive of (and possibly pathogenetically linked to) cardiovascular diseases. This study provides the opportunity to answer questions on defining sleep apnea–hypopnea activity based on prospective data rather than to perpetuate the use of arbitrary criteria to choose among different monitoring approaches. However, in this study, the limitations associated with the need to maintain a single standardized monitoring protocol did not provide the opportunity to design a study to evaluate differences in the sensitivities of different sensors or utility of different amplitude criteria in defining hypopnea. Thus, not all areas that require standardization will be addressed with this study.

Will data from the SHHS and other studies justify continued measurement of hypopnea? Will measurements of hypopnea be linked in a dose–response fashion to incident disease? Will data demonstrate a threshold effect for AHI, and, if so, is that threshold at all close to the arbitrary definitions used in clinical practice? Will data obtained from polysomnography be sufficiently reliable to adequately describe risk characteristics across the population? Will the results allow standardization of measurement of SAHS activity analogously to measured blood pressure? Will these data justify the continued use of the AHI as the metric for characterizing SAHS severity? Will data on respiratory activity provide better information on exposure than snoring or other symptom reports, or oximetry used alone? These questions reflect the tremendous amount of work that lies ahead—work that should result in improved identification and treatment of patients and a healthier, less sleepy society.

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REFERENCES

1. Kirkendall WM, Feinleib M, Freis ED, et al. Recommendations for human blood pressure determination by syhygmomanometers. SubCommittee of the Human AHA Postgraduate Education Committee. *Circulation* 1980;1145A–55A.
2. Indications and standards for cardiopulmonary sleep studies. American Thoracic Society Consensus Report. *Am Rev Respir Dis* 1989;139:559–68.
3. WHO-ISH guidelines for the treatment of hypertension. *J Hypertens* 1989;7:689–93.
4. Franceschi M, Zamproni P, Crippa O, Smirne S. Excessive daytime sleepiness: a 1-year study in an unselected inpatient population. *Sleep* 1982;5:239–47.
5. Lavie P. Incidence of sleep apnea in a presumably healthy working population: a significant relationship with excessive daytime sleepiness. *Sleep* 1983;6:312–8.
6. Young T, Palta M, Dempsey J, Skatrud J, Weber S, Bader S. The occurrence of sleep disordered breathing in middle-aged adults. *N Engl J Med* 1993;328:1230–5.
7. Ancoli-Israel S, Kripke DF, Klauber MR, Mason WJ, Fell R, Kaplan O. Sleep-disordered breathing in community dwelling elderly. *Sleep* 1991;14:486–95.
8. Guilleminault C. State of the art: sleep and control of breathing. *Chest* 1978;73:293–9.
9. Block AJ, Boysen PG, Wynne JW, Hunt LA. Sleep apnea, hypopnea, and oxygen desaturation in normal subjects. *N Engl J Med* 1979;300:513–7.
10. Gould GA, Whyte KF, Rhind GB, et al. The sleep hypopnea syndrome. *Am Rev Respir Dis* 1988;137:895–8.
11. Moser NJ, Phillips B, Berry DTR, Harbison L. What is hypopnea, anyway? *Chest* 1994;105:426–8.
12. Berg S, Haight JSJ, Yap V, Hoffstein V, Cole P. Respiratory airflow measurement techniques for sleep studies. *Sleep* 1997; 20:1157–61.
13. Ahmed MM, Norman RG, Walsleben JA, Rapoport DM. Nasal cannula pressure sensor vs thermistor: detection of respiratory events during NPSG. *Am J Respir Crit Care Med* 1996;153: A715.
14. George CF, Kryger MH. When is an apnea not an apnea? *Am Rev Respir Dis* 1985;131:485–6.
15. Rosen CL, D'Andrea L, Haddad GG. Adult criteria for obstructive sleep apnea do not identify children with serious obstruction. *Am Rev Respir Dis* 1992;146:1231–4.
16. Guilleminault C, Stoohs R, Clerk A, Cetel M, Maistros P. A cause of excessive daytime sleepiness: the upper airway resistance syndrome. *Chest* 1993;104:781–7.
17. Gislason T, Almqvist M, Eriksson G. Prevalence of sleep apnea syndrome among Swedish men. *J Clin Epidemiol* 1988;41:571–6.
18. Dickel MJ, Mosko SS. Morbidity cut-offs for sleep apnea and periodic leg movements in predicting subjective complaints in seniors. *Sleep* 1990;13:155–66.
19. Phillips BA, Berry DTR, Schmitt FA, Magan LK, Gerhardstein DC, Cook YR. Sleep-disordered breathing in the healthy elderly. Clinically significant? *Chest* 1992;101:345–9.
20. Jennum P, Sjol A. Epidemiology of snoring and obstructive sleep apnea in a Danish population, age 30–60. *Eur Sleep Res* 1992;1:240–4.
21. Olson LG, King MT, Hensley MJ, Saunders NA. A community study of snoring and sleep disordered breathing. *Am J Respir Crit Care Med* 1995;152:711–6.

22. Redline S, Tishler PV, Hans M, Tosteson TD, Strohl KP, Spry K. Racial differences in sleep disordered breathing in African Americans and Caucasians. *Am J Respir Crit Care Med* 1997; 155:186–92.
23. Jennum P, Sjol A. Snoring, sleep apnoea and cardiovascular risk factors: the MONICA II study. *Int J Epidemiol* 1993;22:439–44.
24. Hla KM, Young TB, Bidwell T, Palta M, Skatrud JB, Dempsey J. Sleep apnea and hypertension. A population-based study. *Ann Intern Med* 1994;120:382–8.
25. Olson LG, King MT, Hensley MJ, Saunders NA. A community study of snoring and sleep disordered breathing: health outcomes. *Am J Respir Crit Care Med* 1995;12:717–20.
26. Ringler J, Basner RC, Shannon R, et al. Hypoxemia alone does not explain blood pressure elevations after obstructive apneas. *J Appl Physiol* 1990;69:2143–8.