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Original article

Comparison of two methods of quantitative assessment of hypoxemia in patients with sleep disorders

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Abstract

Objective: To evaluate the validity of two methods of quantifying oxygen saturation during sleep: saturation impairment time (SIT) index and percentage of time spent below various levels of oxygen saturation (%T).

Background: Although many methods of reporting nocturnal hypoxemia in sleep have been utilized, no 'gold standard' has been identified to report in conjunction with frequency of breathing pauses. We compare two such methods.

Methods: Prospective, non-randomized, double-blind, controlled trial on 298 patients referred to a sleep-disorders center. In-laboratory recording of nocturnal polysomnography with the data from the pulse-oximetry channel stored in a computer for subsequent analysis. SIT index and %T data were compared for each patient, as were values between patient groups. Raw and logarithmic transformed data were analyzed using regression and non-parametric methods.

Results: SIT index and %T data correlate well, but deviations exist between the results of these tests in individual patients and between some patient groups.

Conclusion: SIT index, combining time and severity of desaturation, may provide additional useful data in the study of oxygen desaturation in sleep, compared to %T calculations. Such data may be important in future studies of physiological variables.

Keywords: Nocturnal hypoxemia; Hypoxia; Oximetry; Polysomnography; Sleep; Sleep apnea; Sleep disorders

1. Introduction

Sleep apnea syndrome is an increasingly recognized disorder in which patients experience repetitive apneas and hypopneas during sleep. Sleep apnea syndrome has been associated with physiologic consequences such as systemic and pulmonary hypertension, cardiac arrhythmias and congestive heart failure. The severity of sleep apnea has traditionally been described by the number of apneas (apnea index (AI)) or apnea and hypopneas (the apnea-hypopnea index (AHI)) as determined by the results of polysomnography. In order to ascertain the relationship of the physiologic consequences of the disorder, however, quantification of oxygen desaturation (SaO_2) is important.

Multiple techniques, including mean SaO_2 levels per epoch or per study, descriptions of various desaturation levels (e.g. highest, lowest, mean/epoch, numbers of drops), and time spent at or below differ-

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ent SaO_2 levels, have been used to report nocturnal hypoxemia [1–11]. We have previously published our findings using saturation impairment time (SIT), which is a computer-based technique for acquiring and analyzing cumulative nocturnal oxygen desaturation [11]. Since the publication of our information on SIT, computerized sleep-scoring systems that quantify hypoxemia as a percentage of the sleep-study time (%T) spent below various SaO_2 levels have become commonly commercially available. We have further refined our previous work on SIT to add %T values so we could make comparisons between these techniques. SIT index integrates time and degree of desaturation; %T directly reflects time but not specifically degree (severity) of desaturation. As can be seen in Fig. 1, by combining time with the degree of desaturation there is the potential for gaining additional information which may help correlating severity of desaturation with physiologic consequences. The data from the SIT index and %T are necessarily related because both use time of desaturation in their calculation. The question explored was to what extent SIT index and %T are predictive of one another in individual patients and in groups of patients with varying presence and severity of sleep apnea. Defining such details will permit appropriate use for further studies measuring physiological consequences of hypoxemia.

2. Methods

In order to perform the described comparison, we developed an additional analysis program to calculate

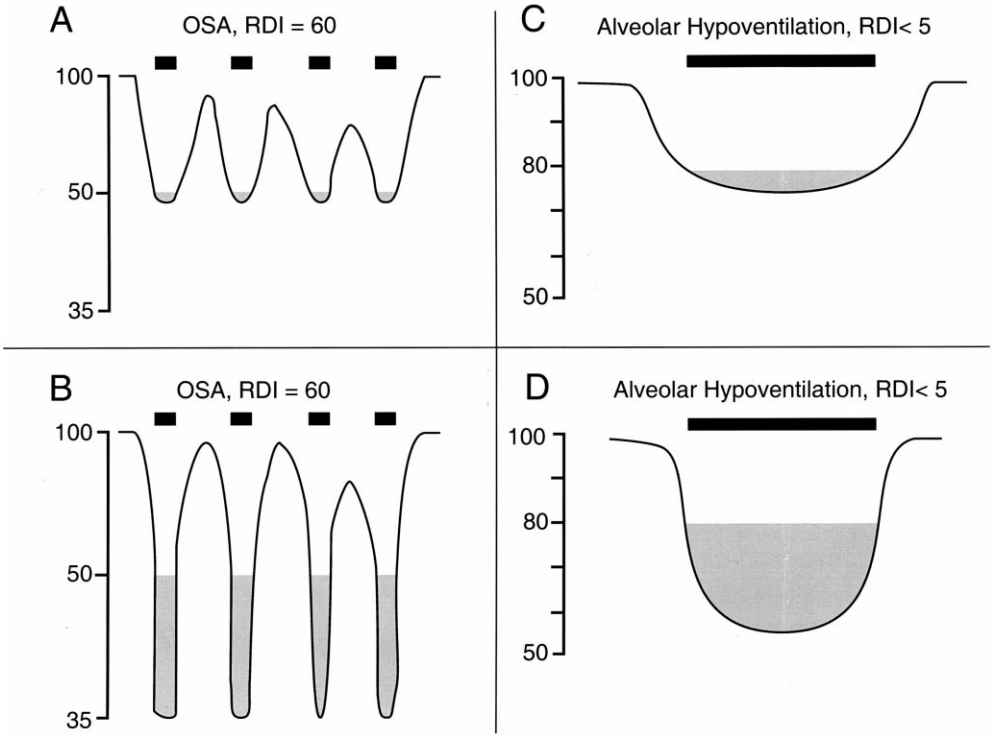

Fig. 1. (A) A theoretical patient shows frequent oxygen desaturations that extend to just below an SaO_2 of 50%. Patient B has desaturations well below that same threshold. Each patient in this illustration has the same time below their threshold for each apnea (%T), as represented by the black bars noted above the figures. However, patient in B has a considerably higher SIT index <50% as shown by the integrated area under the curve (light gray shaded area indicated within the oximetry curve). C and D depict other theoretical patients with prolonged durations of non-apneic desaturation due to hypoventilation. These two examples differ in degree of desaturation although the percent time of desaturation below 80% (see black bar) is the same for both. The SIT index <80% differs between them (light gray shaded area under the curve).

%T below the thresholds of baseline, 90, 80, 70, 60 and 50% SaO_2. Using this program, we determined the expected values and distributional properties for %T and then compare these to SIT index values from the patients to identify the relationship of these measurements to each other. Because both values reflect a measurement of time, we attempted to determine if the two methods are equivalent or if additional information may be obtained from the SIT index, which integrates the time and degree of desaturation.

2.1. Population

We reanalyzed data we had previously collected from polysomnographic studies of 298 consecutive patients who were self or physician referred to the Louisiana State University Medical Center Sleep Disorders Center. Inclusion criteria for data analysis included [11]: (1) obtaining at least 6 h of nocturnal polysomnography with simultaneous computerized analysis of oxygen saturation (and without major periods of signal loss due to probe off or persistent EEG signal loss which would make polysomnographic scoring and computerized acquisition incomplete); (2) having sufficient history, physical examination, and polysomnographic data to establish the presence of only one significant sleep-disorder diagnosis; and (3) finding no additional medical problems that would alter oximetry findings beyond the alterations expected for the patient's sleep-disorder diagnosis.

We used International Classification of Sleep Disorders Criteria [12] and on the basis of the final polysomnographic and clinical data, assigned each of the 298 patients to one of five groups. Group 1 included those patients with no sleep-related respiratory impairment (including such diagnosis as normal study, narcolepsy, CNS hypersomnia, periodic limb movement disorder, seizures, REM behavior disorder, bruxism or other non-respiratory related categories). Group 2 included patients who had abnormal sleep-related breathing but did not have sleep apnea syndrome (COPD, muscular dystrophy and obesity hypoventilation). Patients in groups 3, 4, and 5 had OSA which was classified according to their number of apneas and hypopneas (See Table 1).

2.2. Polysomnography

Polysomnography was performed at a paper speed of 10 mm/s on a Grass Model 78 polygraph or 8-20D EEG machine. Monitoring parameters included central and occipital electroencephalogram, elelectroculogram, chin and surface anterior tibialis electromyograms, and electrocardiogram. Respiratory monitoring included nasal and oral airflow recorded with thermistors, abdominal and respiratory movements using inductive plethysmography (Respitrace – Ambulatory Monitoring, Inc., Ardsley, NY), and oxygen saturation via pulse oximeter (Biox III, Ohmeda, Boulder, CO or Novametrix 500 or 505, Medical Systems, Inc., Wallingford, CT). Sampling settings were set to 6 and 8 s, respectively.

Sleep-stage scoring used 30-s epochs and followed standardized criteria [13]. Central, mixed, and obstructive apneas and hypopneas and desaturation were scored based upon respiratory effort, airflow measurements and oximetry. A respiratory disturbance was defined as a reduction of airflow amplitude

Table 1
Mean demographic data by group

	N	Mean (range)		Sex (M/F)	Mean (SD)	
		Age (years)	Weight (lbs)		AI	RDI
Group 1 (no respiratory impairment)	84	42 (15–79)	185 (121–338)	54/30	0.3 (0.8)	0.6 (1.1)
Group 2 (respiratory impairment, not OSA)	10	38 (21–57)	215 (182–270)	3/7	1.1 (1.0)	2.3 (1.6)
Group 3 (OSA, RDI = 5–19)	73	50 (14–79)	217 (123–369)	58/15	6.5 (4.2)	10.3 (4.5)
Group 4 (OSA, RDI = 20–39)	39	52 (27–69)	236 (133–430)	31/8	21.8 (7.3)	29.0 (5.6)
Group 5 (OSA, RDI ≥ 40)	92	47 (19–78)	255 (142–500)	81/11	64.6 (20)	72.7 (18.5)
Groups 3 + 4 + 5 (all OSA)	204	49 (14–79)	238 (123–500)	170/34	35.0 (30)	42.0 (31)

to less than 50% of the preceding baseline. It was then identified as either an apnea (airflow less than 20% of the preceding baseline for more than one half of the event) or a hypopnea (between 20 and 50% of the baseline). Review of the respiratory effort channels then provided the designation of event type: obstructive if effort amplitude was greater than 20% of baseline or chest-abdominal paradox was present, central if effort amplitude was less than 20% of the baseline state and without paradox, or mixed if at lease one fourth of the event showed obstructive and central components. Apneic Index (AI) was calculated as the number of apneas × 60/total sleep time. Respiratory disturbance index (RDI) was calculated as the number of apneas + hypopneas × 60/total sleep time.

2.3. Acquisition and analysis of oxygen saturation data

The details of the computerized data acquisition and analysis program for SIT are described in detail in our previous study [14]. Briefly, the acquisition program samples voltage output from an oximeter, at 0.5-Hz intervals, using an IBM PC or compatible computer. After the polysomnographic study is completed, a companion analysis program is used to integrate time and degree of desaturation for the entire night and calculate the SaO_2 values below various threshold saturation values, i.e. below the patient's pretest, supine and awake baseline level and below 90%, 80, 70, 60 and 50% SaO_2. The integral for desaturation below the 90% saturation level would be the SIT 90, for below the 80% saturation level would be SIT 80, etc. In order to be able to compare these integrals between different patients, or between the same patient but different studies, the integral is divided by total study time and referred to as the SIT index. The analysis program provides the two types of information used: SIT (the integration of the time and the degree of desaturation at below baseline, 90, 80, 70, 60 and 50% SaO_2); and %T (the percentage of study time spent below each identified level). Because multiple levels of saturation can be considered, the SIT index is also identified by threshold levels, thus the SIT index 90 is the integral below 90%, corrected for study time. SIT index values are 'unitless' in the presentation, but are actually a calculation of $SaO_2 \times$ min/h which is conceptually similar to other standard measures such as apnea index or RDI. This method (SIT) is harder to conceptualize however, because it combines *degree* and *duration* of desaturation (area under a curve) and is not simply a number of events per h (Fig. 1).

2.4. Statistical analysis

Initial review of our data revealed a highly skewed distribution of values, including many zero values, as would be expected for SIT index and %T at the lower threshold levels in patients without apnea or desaturations (most patients do not have desaturation to <50, <60, <70%, etc.). To avoid zero value, we therefore expressed the data using a format in which a logarithmic transformation was performed after adding a constant

$$\text{Transformed SIT} = \log_{10}(\text{SIT} + 1)$$

The %T values for the various saturations have a distribution typical of that commonly seen for percentages that span the range of 0–100 and an ARCSIN transformation was used for that data

$$\text{Transformed percent time} = \log_{10}\left[\text{ARCSIN}_{\text{radians}}(\%\text{time}/100)^{1/2} + 1\right]$$

This arcsin transformation was used to help linearize the relationship between the two variables and have the variance of deviations be of the same order of magnitude all along the curves, despite the fact that our data range from very large to very small numbers in this data set. The use of transformed data also provide better clarity in our figures that show conversion plots between SIT and %T data. Thus, our data provides both raw and transformed SIT index and %T reference values for the five patient groups. The data also allows examination of the conjecture that the SIT index and %T are not equivalent discriminator's among various clinically determined patient groups or individuals. For comparing SIT and %T measurements, least-square regression, with transformed SIT as the dependent variable and the linear and quadratic components of transformed time as the independent variables, was used. A *P*-value of less than 0.05 was deemed to be statistically significant.

Table 2
Mean raw and transformed SIT index and %T data for the listed groups of sleep disorder patients[a]

	Sit index						% Time below					
	Baseline	90%	80%	70%	60%	50%	Baseline	90%	80%	70%	60%	50%
Raw data: SIT AND % time, by groups												
Group 1 (no respiratory impairment)	46.7	0.4	0	0	0	0	50.7	0.3	0	0	0	0
	(47.3)	(1.6)					(36.4)	(1)				
Group 2 (respiratory impaired, not OSA)	185.7	137.7	21.5	1.8	0.3	0	71.6	29	9	0.8	0	0
	(209.2)	(223.2)	(36.5)	(3.7)	(0.7)		(27.8)	(40.1)	(18.0)	(1.6)		
Group 3 (OSA, RDI = 5–19)	123.5	53.7	16.4	4.7	1.3	0.4	60.9	10.9	4.5	1.3	0.3	0.1
	(183.6)	(174.5)	(74.1)	(27.7)	(9)	(3)	(34.7)	(26.2)	(15.3)	(5.8)	(1.5)	(0.7)
Group 4 (OSA, RDI = 20–39)	119.3	49.4	11.4	3.8	1.5	0.5	62.7	12.5	2.5	0.5	0.2	0.07
	(116.4)	(122.6)	(35.9)	(15.6)	(7.1)	(2.4)	(27.1)	(21.3)	(7.5)	(1.8)	(1)	(0.5)
Group 5 (OSA, RDI ≥ 40)	324.2	193.2	58.6	16.3	3.7	0.9	75.3	36.9	11.7	3.7	0.9	0.2
	(265.2)	(261.2)	(125.8)	(48.2)	(15.3)	(5.7)	(23.4)	(29.9)	(18.5)	(9.1)	(2.9)	(1)
Group 3 + 4 + 5 (all OSA combined)	213.2	115.8	34.5	9.8	2.4	0.6	67.8	23	7.4	2.2	0.56	0.13
	(237.1)	(221.7)	(98.8)	(37.4)	(12)	(4.4)	(29.3)	(29.8)	(16.2)	(7.2)	(2.2)	(0.8)
	1 vs. all	1 vs. all	1 vs. all	1 vs. all	1 vs. all	1 vs. 4,5	1 vs. 3,5	1 vs. all	1 vs. all	1 vs. all	1 vs. 3,4,5	1 vs. 5
	2 vs. 5	2 vs. 3					2 vs. 5	2 vs. 3				
	3 vs. 5	3 vs. 5	3 vs. 5	3 vs. 5	3 vs. 5	3 vs. 5	3 vs. 5	3 vs. 4,5	3 vs. 5	3 vs. 5	3 vs. 4	3 vs. 5
	4 vs. 5	4 vs. 5	4 vs. 5	4 vs. 5			4 vs. 5	4 vs. 5	4 vs. 5	4 vs. 5		
Transformed data: SIT AND % time below by groups												
Group 1 (no respiratory impairment)	1.4	0.1	0	0	0	0	0.24	0.01	0	0	0	0
	(0.6)	(0.2)					(0.12)	(0.02)				
Group 2 (respiratory impaired, not OSA)	2.0	1.4	0.7	0.2	0.1	0	0.31	0.15	0.06	0.02	0	0
	(0.5)	(1)	(0.8)	(0.4)	(0.2)		(0.08)	(0.14)	(0.09)	(0.03)		
Group 3 (OSA, RDI = 5–19)	1.8	0.6	0.3	0.1	0.1	0	0.27	0.07	0.03	0.01	0.005	0.002
	(0.6)	(0.8)	(0.6)	(0.4)	(0.3)	(0.2)	(0.11)	(0.11)	(0.07)	(0.04)	(0.020)	(0.012)
Group 4 (OSA, RDI = 20–39)	1.9	0.9	0.3	0.2	0.1	0.1	0.28	0.09	0.03	0.01	0.01	0.002
	(0.4)	(0.8)	(0.6)	(0.5)	(0.3)	(0.2)	(0.07)	(0.09)	(0.05)	(0.03)	(0.018)	(0.011)
Group 5 (OSA, RDI ≥ 40)	2.4	1.8	1.1	0.5	0.3	0.1	0.32	0.19	0.09	0.04	0.015	0.005
	(0.4)	(0.8)	(0.9)	(0.7)	(0.5)	(0.3)	(0.06)	(0.1)	(0.09)	(0.06)	(0.034)	(0.016)
Group 3 + 4 + 5 (all OSA combined)	2.1	1.2	0.6	0.3	0.1	0.1	0.29	0.13	0.06	0.02	0.01	0.003
	(0.6)	(1)	(0.8)	(0.6)	(0.4)	(0.2)	(0.08)	(0.12)	(0.08)	(0.05)	(0.027)	(0.014)

[a] Mean values (±SD). The last rows show, for each data grouping, the groups which are significantly different, ($P < 0.05$) on a percomparison basis using Mann–Whitney non-parametric tests with correction for ties. For both the SIT Index and %time data the combined Group (3 + 4 + 5, all OSA) was significantly different from Group 1 at all thresholds but not different from Group 2.

3. Results

3.1. SIT vs. %T

The mean and standard deviation (SD) values for raw and transformed SIT index (from our previous study) and %T, (current data) divided by groups, were compared to each other to establish whether %T could reliably predict SIT index and vice versa (Table 2). Figs. 2 and 3 represent examples of the relationship between SIT and %T values. The curvilinear component of the regression analysis was not statistically significant (0.05 level) at below 60 and 50% saturation but both linear and quadratic components were highly significant in all other equations ($P < 0.0001$ in all cases) (examples shown in Figs. 2 and 3). The squared multiple correlation coefficient indicates the percentage of variation in transformed SIT that is 'explained' by the corresponding time index. This value was 97% for the curves at 90 and 80% saturation, 93% for the curve at 70% saturation, 85% for the linear equation at 60% saturation, and 78% for the linear curve at 50% saturation.

Fig. 4 shows the relationship between SIT index and *transformed* SIT index values. The relationship between the %T index after the ARCSIN transformation has been performed (expressed as a logarithmic transformation) is seen in Fig. 5. Fig. 4 permits conversions to be made between SIT index and transformed SIT values, and Fig. 5 permits a similar conversion to be made between %T and transformed %T.

In the SIT vs. %T plots (Figs. 2 and 3), the deviations of individual SIT values from those predicted by these equations of 'time' appear, to the eye, to be random. In fact, the deviations are seemingly related

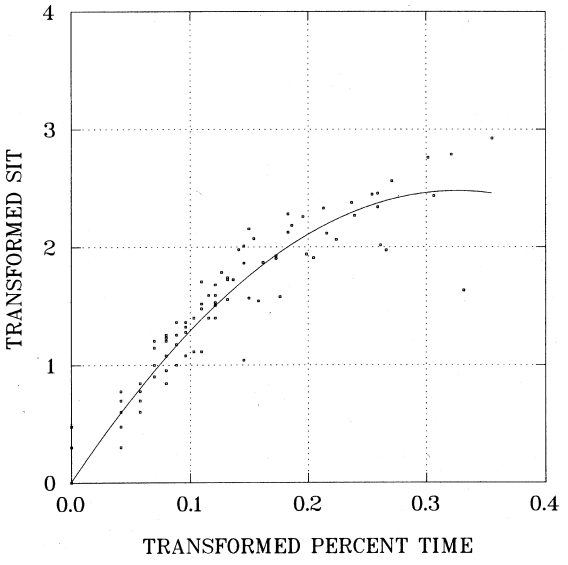

Fig. 2. Relationship of SIT to time at 80% saturation.

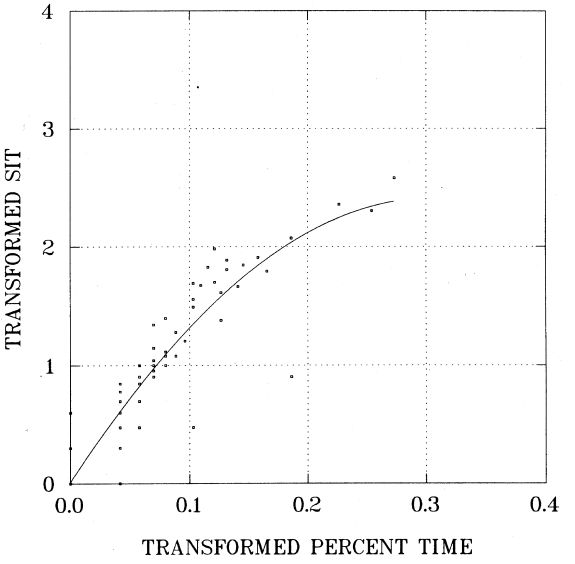

Fig. 3. Relationship of SIT to time at 70% saturation.

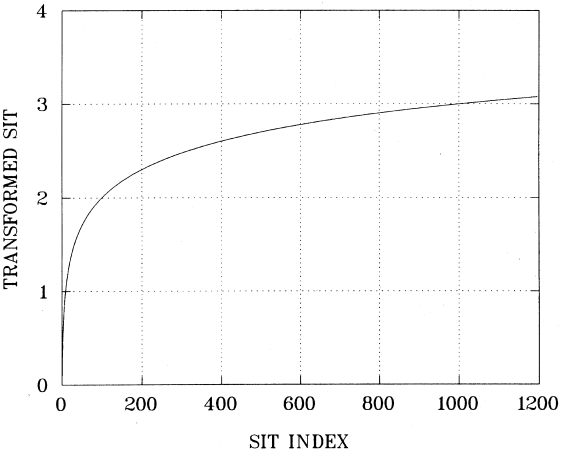

Fig. 4. Transformed SIT = $\log_{10}$(SIT + 1).

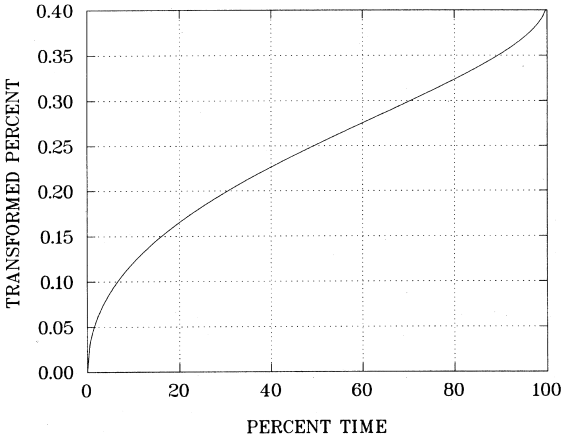

Fig. 5. Transformed percent time = $\log_{10}$[ARCSIN$_{radians}$(%time/100) + 1].

neither to SIT nor to time. However, there are several individuals exhibiting such departures from the typical curve values to warrant further study to determine if there may be physiologic reasons that might better explain their behavior.

3.2. SIT index and %T compared by patient groups

The SIT index (raw and transformed values) for each saturation's threshold level (baseline through 50%), and for each clinical group, are summarized in the left half of Table 2. The %T raw and transformed values for the same groups and % SaO_2 threshold levels are seen in the right half of Table 2.

Significant SIT index and %T differences are summarized in the sections below the top half of Table 2. Differences were considered to be significant if P was 0.05 or less on per-comparison basis using Mann–Whitney non-parametric tests with correction for ties. Protection against *P*-value inflation was provided by running Kruskel–Wallis non-parametric analysis of variance before the Mann–Whitney test and finding these results to be significant at $P <$ 0.005 at all thresholds.

For the groups, one can compare the performance of the SIT index with that of %T by comparing the pattern of statistically significant differences between groups at the various thresholds. For example, for SIT index baseline, groups 1 and 5 were statistically different from all other groups, but differences among groups 2, 3, and 4 were not significant. This finding can be contrasted to the %T less than baseline values, in which group 1 was statistically different from groups 3 and 5, but group 1 was not different from groups 2 or 4. Groups 2, 3, and 4 were not statistically different from one another. Similar comparisons can be viewed for each threshold level for these two techniques of assessing hypoxemia.

Not shown on the table, but also performed were SIT index and %T comparison of a combined group of all OSA cases (groups 3 + 4 + 5) compared to patients without apnea (to group 1) and to patients with non-apneic sleep related breathing disorders, (group 2) at each saturation threshold. At each threshold level, the combined OSA group was significantly different from group 1 but not from group 2. Tests on the transformed variables for SIT indices and %T, shown in the lower half of Table 2, are identical to those on the raw variables because the transformations preserve rank order and because we used non-parametric tests based upon ranks.

4. Discussion

The importance of the cumulative degree of hypoxia across the night, in relation to symptoms or physiologic changes in sleep apnea patients, has not been well studied, nor have important levels of nocturnal hypoxia been established in non-apneic patients with sleep related respiratory disease (SRRD), such as COPD, obesity hypoventilation syndrome, neuromuscular disorders, sickle cell anemia, etc. The severity of cumulative nocturnal hypoxemia that correlates with symptoms in humans has not been identified. Without that, it is not possible to compare SIT and %T techniques of hypoxemic quantitation to a gold standard to identify which technique is 'best'. Outcomes research on apnea pathophysiology and treatment effectiveness is in great demand. However, an important question remains; what method(s) should be used to try to gather and analyze desaturation data to allow comparisons among outcome studies. Percent time measurements look at only one parameter whereas the physiologic severity of hypoxemia is likely determined by both the time spent desaturated and the degree of desaturation. Both SIT or %T are readily able to be used with

modern computer analysis programs. As depicted in Fig. 1, the additional information provided by SIT can be appreciated when one considers the potential physiological consequences that might occur between patients 1 and 2 in each scenario identified.

4.1. SIT index, %T and hypoxemia measurements

Both SIT index and %T measurements are determined using time calculations, so we first investigated whether the two methods are so closely related as to make %T information always interchangeable with SIT index. For them to be truly interchangeable for clinical and research applications, SIT and %T should be able to predict one another for all types of patients and for individual patients within each patient group. Our data does show an expected statistical correlation between SIT index and %T but they do not always parallel one another. Differences are seen in the statistical correlations between SIT index and %T oxygen saturation values and group type in some of the *groups* (which will be discussed later) as well as when looking at the predictive ability of %T for SIT index (and vice versa) in *individual patients*. Figs. 2 and 3 do show deviations of individual SIT index values from those predicted by %T in some patients.

The transformed values in Table 2 were provided because means and standard deviations computed on the transformed scales will be more valid indicators of central tendency and variability (as compared to those calculated on the original scales of measurement) in the sense of the values being more nearly normally distributed over the whole range of observations values rather than being heavily skewed by many zero values in patients with mild apnea with little or no desaturation. Use of the transformed values provides a greater potential for plotting predicted conversions between SIT and %T, using figures, than does similar use of raw data. By reading across axes to the curves on these graphs, the predicted %T for specific SIT values can be obtained, or vice versa. Similar mathematical transformation can be applied to computer generated reports for conversion purposes.

4.2. SIT index and %T comparisons in different patient groups

If SIT and %T measure the same thing, they should produce an equivalent pattern of significant differences among the patient groups. The results in Table 2 do show a generally similar pattern; however, enough variation exists to suggest that important diagnostic or physiologic differences may be reflected in the SIT index but not in the %T measurements. For example, for the threshold less than baseline, when the SIT index is used, group 1 is different from all other groups; when using %T values, group 1 is only different from groups 3 and 5. At the low end of the saturation spectrum, at less than 60%, SIT identifies significant differences between group 1 and all other groups, but %T does not identify significant differences between groups 1 and 2. At thresholds less than 50%, SIT is statistically different between groups 1 and 4, and 3 and 5, but %T is not, however, %T shows a significant difference between groups 3 and 4.

We speculate that the failure of %T to identify differences between groups 1 and 2, at less than baseline thresholds, might be due to the extra power of SIT to add cumulative depth of desaturation to time of desaturation. For example, consider two patients with an SaO_2 baseline of 97% and equal numbers and lengths of apneas, but one with nadirs to 72% and the other to 79%. The two patients could have similar %T baseline, less than 90%, and less than 80% values but different SIT index baseline, less than 90% and less than 80% values because of SIT index's inclusion of integration of degree of desaturation. (Fig. 1, comparing panels a and b, demonstrates a similar point.) Depth of desaturation may also be of physiologic importance in disease states that cause continuous levels of hypoxemia, (i.e. a small % difference in levels of SaO_2 over a long period of the night accumulate to be a big difference in total desaturation). (The right half of Fig. 1 depicts this point.) This may be why group 2 differences exist between the two techniques.

We considered another explanation for group 2 differences may be the power of the calculations for the groups 2 data; group 2 is our smallest N, and so the possibility of an effect of the small N when comparisons involve group 2 %T values might also need to be considered. In an attempt to answer this question, we looked at a study by Timms et al. [15]. They used a modification of %T below selected SaO_2 levels that can serve to both support our findings and demonstrate the clinical utility of our approach. Their study looked at the effect of sleeping medication on SaO_2 in patients with COPD. They used an assessment of %T $<90\%$, $<80\%$ and

<70% SaO_2. If we consider their placebo night a representation of non-apneic, respiratory-impaired patients, the data shows values very close to our group 2 mean %T data. This finding is very encouraging because though both their and our groups had small Ns, the data shows very similar desaturation levels. Obviously their work does not support data about the differences between SIT and %T, but it does support the stability of our %T group 2 related values and suggests that a small N may not be the reason for the differences in the two techniques we studied.

Differences between SIT index and %T at <60% and <50% thresholds might also be related to SIT index's ability to additionally identify the depth of desaturation. At these levels, oximeters are less accurate, however, the data are representative of the same patient and the same time, so oximeter accuracy is not of concern comparing the two techniques. As actual time spent at these low SaO_2 levels is very small, SIT index's ability to focus also on how low the oxygen saturation actually goes may provide the extra information to discriminate between these groups. Quantifying the actual cumulative degree of desaturation appears potentially important to assist in understanding the role of desaturation in relation to the impairment of sleep, pulmonary function, cardiac function, hormone secretions, etc.

These data, along with the data from our previous study that compared RDI and SIT index [11] suggest that using SIT indexes may provide clinically relevant information that is separate from the information provided by RDI in sleep apnea patients. In addition, quantitating hypoxemia may better characterize the severity of disease in patients with SRRD. Furthermore, correction of RDI without correction of hypoxemia, as can occur with CPAP treatment in patients with sleep apnea but who also have pulmonary disease, could be readily monitored using cumulative desaturation techniques. We cannot yet answer which method best correlates with physiologic outcomes, but we find enough differences in some patient groups and in some individual patients to prompt further study and use of these techniques. Ongoing studies to identify SIT data in patients before and after treatment with CPAP, and looking at cardiovascular outcomes, may provide further validation of this technique.

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