The SaO$_2$/t Diagram as a Useful Means To Express Nocturnal Hypoxemia*

Pierre Aubry, M.D.; Vincent Jouineaux, M.D.; Dominique Rose, M.D., Ph.D.; Alain Duran, M.D.; and Pierre Levi-Valensi, M.D.

The computerization of SaO$_2$ recording during polysomnographic monitoring allows the construction of a diagram expressing the percentage of TIB spent at different steps in saturation. We studied the value of this diagram in three groups of male patients: (1) nine healthy subjects (all volunteers); (2) 25 patients with COPD who had a mean daily SaO$_2$ of 92.3 ± 1.3 percent; and (3) 25 patients with SAS who had a mean daily SaO$_2$ of 92.1 ± 1.4 percent. The results show the existence of a discriminating quality in the diagram's morphology, the existence of strong correlations (p<0.01) between the percentage of TIB spent at SaO$_2$ less than 85 percent, and the total duration of the desaturation dips. (Chest 1989; 96:1341-45)

SaO$_2$ = arterial oxygen saturation; SAS = sleep apnea syndrome; TIB = time in bed

A method of summarizing the SaO$_2$ recording during polysomnographic monitoring is not yet standardized. The construction of a diagram expressing the percentage of TIB spent at different steps of saturation is possible with the emergence of software allowing the computerized analysis of data. The potential advantage of such a diagram was suggested by many authors.1-3 We tried to estimate its physiologic and diagnostic value in patients with a COPD and in patients with a SAS, both with a daily SaO$_2$ on the shoulder of the oxyhemoglobin dissociation curve, in comparison to normal volunteer subjects.

**MATERIALS AND METHODS**

We have studied three groups of subjects: (1) one group of 25 male patients with COPD who had a mean age of 65.5 years (range, 61 to 74 years) and a mean daily SaO$_2$ of 92.3 ± 1.3 percent; (2) one group of 25 male patients with SAS, noticeably younger, with a mean age of 53 years (range 36 to 75 years) and with a mean daily SaO$_2$ of 92.1 ± 1.4 percent, which is not statistically different from the patients with COPD; these patients were obese for the most part; and (3) one group of nine volunteer male subjects, all in good health, without any disease. Their mean age was 60.6 years (range, 55 to 67 years), with a mean daily SaO$_2$ of 97.6 ± 0.7 percent.

The subjects were all examined while in a stable state, without any other complaint. Drugs having some influence on the quality of sleep or acting on the respiratory nervous centers were withheld for one week before study. None received almitrine bisemilate. The patients with COPD and SAS gave their consent before the study, and the healthy subjects were all volunteers. Their anthropometric data appear in Table 1.

Some investigations were conducted during a 24-hour hospitalization. They included the following:

**Diurnal Investigations**

Respiratory function tests were performed in all patients and normal subjects, including spirometry and body plethysmographic determination of the total lung capacity. The results are expressed as a percentage of the predicted values (CECA norms). Analysis of the blood gas tensions was performed at rest, in the recumbent patient (Table 1).

**Nocturnal Investigations**

All patients and normal subjects were submitted to a whole night's polysomnographic monitoring, breathing room air and including the following: (1) continuous SaO$_2$ recording (Hewlett-Packard 47201 A); (2) nasobuccal airflow recording by thermistors; (3) thoracoabdominal movements recorded by impedance plethysmography (Respitrace) without any quantitative measurements; and (4) the EEG, EOG, and submental muscles EMG recorded according to the classic method.

All of these signals were recorded on (1) an ink-writer recorder (Beck Instrument Recorder) with five channels for the respiratory data (speed, 6 cm/min) and (2) an eight-channel recorder (Alvar) for the neurophysiologic data (speed, 60 cm/min).

Respiratory data such as the SaO$_2$, nasobuccal airflow, and thoracic movements were also analyzed by a computer (Apple IIIE) in real time according to software that we use regularly and have previously validated.* The selection frequency of data is 2 Hz for oximetry and 7 Hz for detection of apnea.

The computerized results included the following:

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**Table 1—Anthropometric and Functional Data**

<table>
<thead>
<tr>
<th></th>
<th>Normal Subjects</th>
<th>COPD Patients</th>
<th>SAS Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Anthropometric data</strong></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Age (years)</td>
<td>60.6 ± 4.7</td>
<td>65.5 ± 7.3</td>
<td>53.0 ± 9.4</td>
</tr>
<tr>
<td>Weight (% pred)</td>
<td>109 ± 7</td>
<td>123 ± 21</td>
<td>171 ± 27</td>
</tr>
<tr>
<td><strong>Pulmonary function tests</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>TLC (% pred)</td>
<td>102 ± 22</td>
<td>103 ± 17</td>
<td>89 ± 12</td>
</tr>
<tr>
<td>VC (% pred)</td>
<td>109 ± 19</td>
<td>69 ± 16</td>
<td>73 ± 19</td>
</tr>
<tr>
<td>FEV/VC (% pred)</td>
<td>102 ± 7</td>
<td>51 ± 14</td>
<td>82 ± 19</td>
</tr>
<tr>
<td><strong>Arterial blood gases</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diurnal PaO$_2$ (mm Hg)</td>
<td>93.5 ± 5.8</td>
<td>64.1 ± 3.6</td>
<td>63.5 ± 3.6</td>
</tr>
<tr>
<td>Diurnal PaCO$_2$ (mm Hg)</td>
<td>38.2 ± 1.8</td>
<td>43.2 ± 6.6</td>
<td>47 ± 5.35</td>
</tr>
<tr>
<td>Diurnal SaO$_2$ (mm Hg)</td>
<td>97.6 ± 0.7</td>
<td>92.3 ± 1.3</td>
<td>92.1 ± 1.4</td>
</tr>
</tbody>
</table>

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Table 2—Polysomnography Results

<table>
<thead>
<tr>
<th>Sleep variables</th>
<th>Normal Subjects</th>
<th>COPD Patients</th>
<th>SAS Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total duration of recording</td>
<td>413 ± 27</td>
<td>394 ± 33</td>
<td>384 ± 29</td>
</tr>
<tr>
<td>(min)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Wakefulness (% of the TIB)</td>
<td>23 ± 18</td>
<td>35 ± 14</td>
<td>20 ± 15</td>
</tr>
<tr>
<td>NREM Sleep (% of the TIB)</td>
<td>67 ± 13</td>
<td>57 ± 13.9</td>
<td>68 ± 3</td>
</tr>
<tr>
<td>REM Sleep (% of the TIB)</td>
<td>0 ± 8</td>
<td>7.7 ± 6.7</td>
<td>12.1 ± 6.2</td>
</tr>
<tr>
<td>Respiratory events</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean nocturnal SaO₂ (%)</td>
<td>96.3 ± 1.0</td>
<td>90.7 ± 2.5</td>
<td>84.0 ± 6.7</td>
</tr>
<tr>
<td>Desaturation dips (no/h of TIB)</td>
<td>1.3 ± 2.3</td>
<td>8.3 ± 9.6</td>
<td>37.9 ± 18.7</td>
</tr>
<tr>
<td>Time spent in desaturation (% of TIB)</td>
<td>1.0 ± 1.8</td>
<td>13.4 ± 16.0</td>
<td>47.6 ± 23.8</td>
</tr>
<tr>
<td>Apnea Index</td>
<td>1.8 ± 2.1</td>
<td>2.6 ± 2</td>
<td>34.0 ± 20.0</td>
</tr>
<tr>
<td>Time spent in anea (% of TIB)</td>
<td>0.7 ± 0.8</td>
<td>2.6 ± 0.5</td>
<td>32.5 ± 25.0</td>
</tr>
</tbody>
</table>

Episodes of desaturation were counted in terms of number, total duration, and mean duration. An episode of desaturation was recorded when the SaO₂ decreased more than 4 percent from the baseline saturation, defined as the mean SaO₂ computed at the start of recording, during the first 30 minutes, with the patient resting in bed and still awake. The duration of the desaturation episodes was expressed as a percentage of the total recording time. Indeed, the saturation level during wakefulness was also one component of the nocturnal oxygenation.

Computerized results included the SaO₂/t diagram, which shows the percentage of the TIB spent at different steps of saturation. The saturation scale is divided into 2.5-percent steps, and the time scale is divided into percentage of the TIB. An arithmetic scale was chosen here. The recordings of SaO₂ less than 40 percent were gathered into the step of 40 to 42.5 percent.

Mean nightly SaO₂ was included in the computerized results, as were apneas. Apneas were counted in terms of number, total duration, and apnea index. An episode of apnea was defined as a complete cessation of airflow at the nose and mouth for at least 10 seconds. It should be noted that hypopneas were not computed due to absence of accurate quantification of ventilation.

The different recording devices were synchronized by a time signal transmitted every minute to each recorder by the computer. Each ink writer recording was reexamined by a specialized practitioner in parallel to the computerized analysis.

RESULTS

Sleep

All patients showed a total sleep time greater than two hours with REM sleep. The quality of sleep was very variable from one patient to another, with generally a total REM period decreased and wakefulness increased. The mean values appear in Table 2.

SaO₂/t Diagram

The mean values of the percentage of the TIB spent for each step of saturation were calculated in each group (Fig 1). Pathology induces great differences in the diagram's morphology; a more open distribution of time data was noticed in patients with COPD compared to normal subjects, and this even in SAS. The statistical analysis by Student's t-test for unpaired data reveals some significant differences between the separate groups (Table 3).

Between normal subjects and patients with COPD,
differences are very significant for saturations greater than 95 percent (characterizing normal subjects) and for saturations between 85 and 90 percent (characterizing patients with COPD).

Patients with COPD and those with SAS significantly differ in saturation levels from 42.5 to 85 percent, where the patients with SAS spend more time than those with COPD, and in levels from 87.5 to 95 percent, where patients with COPD spend more time than those with SAS; however, the two groups spend about the same time in saturation levels between 85 and 87.5 percent.

**SaO₂/t Diagram and Respiratory Events**

We tried to display some correlations between SaO₂/t data and the respiratory events recorded, and especially for the percentage of the TIB spent under certain SaO₂ values.

*SaO₂/t Diagram and Desaturation Episodes.* We examined the data in each group for correlations between the following: (1) between the number of desaturation dips per hour (compare with the TIB) and the total duration spent at different steps of saturation (expressed in percentage of the TIB); and (2) between the total duration of these episodes (expressed also as a percentage of the TIB) and the total duration spent at different steps of saturation.

In the control group a strong correlation was found between the number of desaturation dips and the total duration spent below 92.5 percent (r = 0.71; p<0.02) and also with the total duration spent below 90 percent (r = 0.87; p<0.01). There was also a significant correlation between the duration of these episodes and the total duration spent below 92.5 percent (r = 0.7; p <0.05).

In the group with COPD, we found significant correlations between the number of episodes and the total duration spent below 85 percent (r = 0.63; p<0.01) and between the duration of the episodes and the total duration spent below 85 percent (r = 0.84; p<0.01).

In the group with SAS, there was no correlation between the number of the episodes and the total duration spent below 85 percent, below 80 percent, or below 60 percent with even a negative coefficient. That could be explained as we shall see in the discussion. Nevertheless, a strong correlation existed between the duration of the episodes and the total duration spent below 85 percent (r = 0.94; p<0.01).

*SaO₂/t Diagram and the Mean Nightly SaO₂.* In the groups with COPD and SAS, a significant correlation was found between the mean SaO₂ and the total duration spent below 85 percent (r = -0.79 for COPD and r = -0.95 for SAS; p<0.01). No correlation was found in normal subjects.

*SaO₂/t Diagram and Apneas.* No significant corre-

**DISCUSSION**

This study shows the usefulness of the SaO₂/t diagram in expressing the nocturnal hypoxemia in patients whose daily SaO₂ is situated on the shoulder of the oxyhemoglobin dissociation curve. Such a diagram offers a real discriminative quality in its morphology and allows a fast and global approach to hypoxemia and its gravity in these patients. Many points must be argued.

**Conditions of Validity**

An accurate interpretation of the nocturnal variations in SaO₂ through this diagram implies sensitive oximeters with a low time delay, devices currently available.** It remains absolutely necessary to make sure of sleep, to estimate its quality and composition. So the recording of sleep variables is indispensable.
and also allows the correlation of respiratory events to different sleep stages.

The morphology of the SaO₂/t diagram could be altered by the use of different scales in its graphic presentation. Some authors used a semilogarithmic scale, and we made sure that our SaO₂/t diagram keeps all its qualities even with the scale (Fig 2).

**SaO₂/t Diagram and Respiratory Events**

**Episodes of Desaturation.** It may be difficult to understand that there is no correlation found between the number of desaturation dips and the total duration spent below 90 percent, below 85 percent, below 90 percent, or below 60 percent in patients with SAS, when the total duration of these episodes is correlated with the total duration spent below 85 percent. This could be explained if we consider an episode of desaturation as a saturation decrease of 4 percent and more from the baseline saturation. Thus, many successive decreases of saturation, caused, for example, by several close apneas, will be computed as only one episode if the level of SaO₂ does not increase enough between each apnea. That is often noticed in patients with SAS and also explains why we found negative coefficients of correlation (although not significant). The number of desaturation episodes is not enough to describe hypoxemia in these patients, which also depends on their duration, on their closeness, and on the lowest saturation reached. All of these factors are taken into account by the SaO₂/t diagram, and we consider it a suitable means to express the hypoxemia of patients with SAS.

**Apneas.** It is not surprising to find no correlation between the different total durations and the number of apneas in normal subjects and patients with COPD. In these cases, the desaturation episodes are induced more by mechanical changes in the thoracic cage or in the ventilation-perfusion ratio than by apneas, especially in REM sleep.

Nevertheless, it will be obvious that in patients with SAS, the total duration for the lowest saturations are tied to apneas. Thus, it seems wrong to find no correlation between the apnea index and the total duration spent below 85 percent, below 80 percent, or below 60 percent. We can give some possible explanations here:

From a physiologic point of view, we have found, as have other authors, variable levels of nocturnal hypoxemia for a nearly equal number of apneas in patients with SAS. The determinant factors of hypoxemia are multiple, including duration of sleep spent in apneas, the baseline PaO₂ in the awake patient, the expiratory reserve volume, and also probably the eventual degree of bronchial obstruction. It should be noted that some authors have found significant correlations between airflow and oxygenation parameters.

From a methodologic point of view, we had not the means to quantify accurately the tidal volume during the night, and for this reason, we did not compute hypopneas. These hypopneas should play a major role in some patients with SAS.
To end the discussion, we must emphasize that in practice, for patients with SAS who have diurnal SaO$_2$ on the shoulder of Barcroft's curve, we verified in all cases that apneas in high number induce a typical morphology to the SaO$_2$/t diagram. This morphology should be completely normalized when therapy removes apneas (for example, nasal CPAP) (Fig 3).

CONCLUSION

The SaO$_2$/t diagram expressing the total duration (as a percentage of the TIB) spent at different steps of saturation is a useful means to describe nocturnal hypoxemia; however, it is not a substitute for analytic data, which remain necessary to understand the underlying mechanisms of hypoxemia and their relation to sleep stages. From this study, it appears that this diagram allows the following: (1) an easy and global estimation of nocturnal hypoxemia and of its gravity (indeed, the definition of criteria in order to consider desaturation episodes or "baseline SaO$_2$" or any reference value is artificial; one of the interesting points of the SaO$_2$/t diagram is that we do not need any arbitrary definition to express nocturnal SaO$_2$); (2) the computation of total durations spent below certain levels of saturation; and (3) an easy estimation of the results of therapy on nocturnal saturation.

Finally, the morphology of the SaO$_2$/t diagram seems to discriminate patients whose daily SaO$_2$ is on the shoulder of the oxyhemoglobin dissociation curve. It must appear with analytic results in the final report of polysomnographic study.

REFERENCES

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