Classic CSR usually occurs in patients with CHF or stroke and is, as mentioned previously, associated with a bad prognosis. However, idiopathic CSA is observed in those without any comorbidities and is not necessarily periodic in nature. Moreover, there are no data indicating that the presence of idiopathic or postarousal CSA by itself is a poor prognostic marker for patients, and no consensus on when and how it should be treated. In view of these differences, there is a definite need to develop a more precise definition of CSR-CSA to better define the relevance of CSR-CSA in CHF, and to foster comparisons of data across studies. We believe that studies examining CSR-CSA should, as a minimum, report on the cycle length, magnitude of oxyhemoglobin desaturation, the timing of arousals relative to the respiratory events, and frequency and duration of apneas and hypopneas of their study patients to distinguish classic CSR-CSA from idiopathic CSA.

Second, the data by Mansfield and colleagues indicate that sympathetic nervous activity, which is overexpressed in those with CSR-CSA, can be markedly attenuated by cardiac transplantation. Indeed, in the posttransplant setting, those with classic CSR-CSA before transplantation had urinary norepinephrine levels remarkably similar to those without CSR-CSA following surgery. Some may argue that these data support the contention that sympathetic nervous activity, which is overexpressed in those with CSR-CSA, can be markedly attenuated by cardiac transplantation. Indeed, in the posttransplant setting, those with classic CSR-CSA before transplantation had urinary norepinephrine levels remarkably similar to those without CSR-CSA following surgery. Some may argue that these data support the contention that CSR-CSA is responsible for the excess sympathetic nervous stimulation (beyond that observed in patients with CHF and no CSR-CSA) and that its abolition “caused” the fall in the excess sympathetic nervous activity in the post-transplant setting. This statement, however, largely ignores the fact that 20% of patients with CSR-CSA in the study by Mansfield and colleagues had “residual” CSA on posttransplant polysomnography. If CSR-CSA, indeed, contributes to the downward spiral of patients with CHF by perturbing the autonomic nervous system, one might have expected to see a higher urinary norepinephrine level in the CSR-CSA group than in the control group following transplantation (since none in the control group acquired CSR-CSA posttransplant). An alternative explanation of the data of Mansfield and colleagues was not designed to address this critically important question. Well-designed, larger prospective studies are needed to determine whether posttransplant CSA is associated with elevations in the sympathetic nervous activity and, more importantly, whether their presence leads to worse clinical outcomes in cardiac patients.

Don D. Sin, MD, FCCP
Godfrey C. W. Man, MD, FCCP
Edmonton, AB, Canada

Dr. Sin is Assistant Professor of Medicine and Dr. Man is Professor of Medicine, University of Alberta. Dr. Sin is supported by a New Investigator Award from the Canadian Institutes of Health Research, and a Population Health Investigator Award from Alberta Heritage Foundation for Medical Research.

Correspondence to: Don D. Sin, MD, FCCP, 2E4.29 Walter C. Mackenzie Centre, University of Alberta, Edmonton, AB, Canada T6G 2B7; e-mail: don.sin@ualberta.ca

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Getting the Most Out of Nocturnal Pulse Oximetry

D ue to its noninvasive nature and the convenient patient interface consisting of a small sensor clipped or taped onto the skin, pulse oximetry is widely used in pulmonary medicine, critical care, and anesthesia. In sleep medicine, pulse oximetry is an essential tool for tracking the rapid fluctuations in arterial oxygen saturation that are characteristic for the unstable ventilation in patients with sleep apnea. Pulse oximetry has provided early insights into sleep-related breathing disturbances, and has opened the way for subsequent systematic investigations of sleep apnea. Today, the technique is an integral component of polysomnography and, by itself, is commonly used as a simple tool in the evaluation of sleep apnea.
The clinical relevance of indexes derived from pulse oximetry alone or combined with other techniques that detect nocturnal breathing disturbances is emphasized by their correlation with impaired cognitive performance, cardiovascular diseases, and other consequences of sleep apnea.2,3

Over the last few years, the development of pulse oximetry has progressed in three major domains: first, the size and weight of the hardware have been reduced so that most devices are now portable, and some are even available in miniaturized form; second, paper chart recordings have been replaced by digital signal acquisition; third, novel mathematical and statistical techniques are being applied to computer-assisted analysis of pulse oximetry recordings. This is particularly useful and efficient for processing overnight recordings extending over several hours. While conventional analysis of nocturnal pulse oximetry has consisted of visual inspection of tracings for identification of desaturation events, baseline and minimal values, and patterns of fluctuations in oxygen saturation,4–6 this kind of subjective interpretation is being replaced by computerized scoring to automatically and objectively derive the variables previously obtained by tedious manual scoring. Additionally, computer analysis provides the opportunity to process pulse oximetry data by powerful statistical and mathematical methods. Several indexes derived by these techniques are listed in the Table 1, along with references to studies that have evaluated the diagnostic performance of nocturnal pulse oximetry with computer-assisted analysis for identification of patients with sleep apnea. The sensitivity of pulse oximetry ranged from 88 to 98%, and the specificity from 40 to 88% if an apnea/hypopnea index of 10/h or 15/h measured during polysomnography was defined as the upper limit of normal.5,7–15

The introduction of various novel approaches to quantify pulse oximetric information requires an evaluation of the relative utility of derived indexes in the diagnosis of sleep-disordered breathing. In the current issue of CHEST (see page 1694), Magalang and coworkers report a systematic comparison of several indexes derived from computer-assisted nocturnal pulse oximetry. Three large cohorts of patients with suspected sleep apnea were examined at two different institutions. The investigation confirms that the Delta index (Table 1), and desaturation indexes that reflect the variability of oxygen saturation, have a similarly high diagnostic accuracy for detection of patients with the obstructive sleep apnea syndrome. Moreover, the authors demonstrate that combining several pulse oximetry-derived variables to an aggregated model enhances the diagnostic yield over that of individual indexes. The study represents an important contribution to the validation of computerized analysis and interpretation of nocturnal pulse oximetry. Nevertheless, the efforts to get the most out of this fascinating noninvasive technique should continue. With further technical developments, and the advancement in our understanding of sleep apnea, new questions arise. For example, it is not clear to what extent the dynamic response characteristics,16 artifact detection and rejection algorithms,17 and other technical specifications that differ among various brands of pulse oximeters affect their performance in sleep apnea diagnosis. Finally, the principal discussion over the reference standard against which novel techniques in sleep apnea diagnostic should be evaluated is also relevant for pulse oximetry. It seems that future evaluations of pulse oximetry would be more meaningful if performed in regard to major clinical outcomes of sleep apnea rather than in comparisons with polysomnography,

<table>
<thead>
<tr>
<th>Index Description</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of dips or of rapid increases in oxygen saturation by a certain amount per hour</td>
<td>Vásques et al9 Taha et al10</td>
</tr>
<tr>
<td>Cumulative time spent below a certain threshold of oxygen saturation</td>
<td>Rauscher et al11</td>
</tr>
<tr>
<td>Index that integrates severity and duration of oxygen desaturation</td>
<td>Gyulay et al12</td>
</tr>
<tr>
<td>Variability of oxygen saturation quantified by the mean of the absolute differences in successive oxygen saturation values sampled at 12-s intervals</td>
<td>Chesson et al13</td>
</tr>
<tr>
<td>Spectral analysis of oxygen saturation and pulse rate is applied to evaluate the presence or absence of a peak in the periodogram within a period range of 30 to 70 s as a sign of periodic apnea/hypopnea</td>
<td>Lévy et al7 Pépin et al14</td>
</tr>
</tbody>
</table>

Table 1—Indices Derived From Computer-Assisted Nocturnal Pulse Oximetry\*
technique that has its own limitations, and depends itself on pulse oximetry for the detection of breathing disturbances.

**Konrad E. Bloch, MD, FCCP**
Zürich, Switzerland

Dr. Bloch is Director of the Sleep Laboratory, Pulmonary Division, University Hospital of Zürich.

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**Correspondence to:** Konrad E. Bloch, MD, FCCP, Pulmonary Division, University Hospital Zürich, Rämistrasse 100, CH-8091 Zürich, Switzerland; e-mail: pneubloc@usz.unizh.ch

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**REFERENCES**


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**Sputum Peroxynitrite Inhibitory Activity Is Stimulating**

In this issue of *CHEST* (see page 1755), Kanazawa and colleagues observe that low levels of a biochemical marker in sputum of asthmatics change toward normal in response to the administration of inhaled corticosteroid (beclomethasone dipropionate [BDP]). They extend the observations to demonstrate a strong relationship of structure (albeit biochemical) and airways function. Since these markers presumably reflect inflammation, reversal in the sputum suggests improvement of the intraluminal inflammatory changes of asthma. Superficially, this is a good work. Read the abstract, comment on the connection of asthma to inflammation, and remember sputum peroxynitrite inhibitory activity (PNI) for rounds tomorrow! But this is not the end of the story.

This work accomplishes much more because this translational clinical study fulfills a higher level of achievement. It is provocative at many levels. It promotes contemplation of questions—some old, some modified, some new. Hopefully, this response will lead to better understanding of the clinical problem, improved design of translational studies, and direction for basic pathogenic and pharmaceutical research.

The focused data of Kanazawa et al correlate the higher levels of oxidative products (nitrates and nitrites) found in the induced sputum of asthmatics with lower levels of an antioxidant marker, PNI, compared with normal control subjects. They further demonstrate the reversal of these perturbations and parallel improvement of airways function (FEV1) in response to inhaled corticosteroids (BDP). What can this mean? Clearly, it provides even more support for the first-line use of inhaled corticosteroids in the treatment of asthma. The obvious reasoning is that asthma is, in part, characterized by airway inflammation, steroids are anti-inflammatory, markers of inflammation revert toward normal with BDP use, and airways function improves. But what are the implications of this substantial yet only partial reversal toward normal? Does that mean that a higher BDP dose is likely to provide even more functional improvement? Or, alternatively, is the reversal in re-