interventricular septum bowed into the left ventricle during diastole, although it continued to curve into the right ventricle during systole when left ventricular pressure was higher than right ventricular pressure. The leftward shift in the septum during diastole led to slower filling rates in the left ventricle as compared with the right. As expected, right ventricular volumes were increased compared with a normal population while left ventricular end-diastolic volume was decreased. Right ventricular ejection fraction was markedly depressed while left ventricular ejection fraction declined modestly. Stroke volume from both ventricles was depressed. The normal rightward bowing of the septum during systole was halved in the pulmonary hypertensive patients as compared with normal individuals.

The authors concluded that right ventricular pressure overload caused an alteration in septal bowing that encroached on the left ventricle thereby reducing its diastolic filling. This led to a decrease in stroke volume by the Frank-Starling mechanism.

Similar effects have been observed in patients with severe pulmonary disease with resultant pulmonary hypertension. Vizza et al observed a direct correlation between right and left ventricular ejection fraction in a large cohort of patients with severe pulmonary disease. A subset of their patient population underwent lung transplantation with a consequent reduction in pulmonary hypertension. In these latter patients, right and left ventricular ejection fraction improved. Schena et al studied 30 patients with advanced COPD. The results in this study were similar to those reported by Marcus et al: increased right ventricular volume, a leftward shift in the interventricular septum, and reduced left ventricular volume and diastolic function.

In conclusion, experimental and clinical observations support the concept of the reverse Bernheim phenomenon, ie, a leftward shift of the interventricular septum secondary to right ventricular pressure and volume overload. This septal shift alters left ventricular volume as well as diastolic and, to a lesser extent, systolic function. Patients with a wide array of severe pulmonary diseases are prone to develop the reverse Bernheim phenomenon.

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To Sleep Deeply, Perchance to Wake Refreshed

The deepest nonrapid-eye-movement (NREM) sleep stages in humans, stages 3 and 4, are also called slow-wave sleep (SWS) and are defined by the appearance of a substantial amount of large amplitude (≥ 75 μV) slow waves in the cortical EEG, with a frequency of 0 to 2 cycles/s (cps). In man, SWS is at a maximum towards the beginning of the night and progressively declines across the sleep period. In all mammalian species studied to date, slow waves increase dramatically during sleep following sleep deprivation. Experimental evidence from human and animal studies suggests that slow waves reflect the homeostatic component of NREM. The report in this issue of CHEST by Heinzer et al (see page 1807) details an intriguing hypothesis, namely, that daytime sleepiness, as measured by the multiple sleep latency test (MSLT) in the obstructive sleep apnea syndrome (SAS), is the result of a decrease in slow wave activity (SWA), defined as cortical EEG waves of 0.75 to 4.5 cps (compared with the 0 to 2 cps of SWS) during the first NREM period during sleep. The approach uses spectral analysis, which does not involve an amplitude criteria. The authors conclude that SWA, but not SWS, correlates with the MSLT.

In traditional sleep staging, visual inspection of the EEG is used to determine the presence or absence of SWS and to determine the proportion of the scoring epoch during which SWS is present. Continuous quantification of cortical SWA during sleep, as performed by the authors, can be achieved through computerized spectral analysis. For this analysis, the digitized EEG is subjected to a fast Fourier transformation to determine the frequency composition of the waveform. SWA in this study was defined as the sum of the power (in microvolts squared) between 0.75 and 4.5 cps. As spectral analysis is valid only for stationary waveforms, short segments of the EEG (4-s epochs, in this study) are analyzed in sequence to approximate a stationary wave.

To better understand the study, the reader should be aware of several factors in the design: (1) as
detailed above, SWS and SWA are related but not the same; in addition, the method of measuring SWA is not standard or necessarily easily applied in clinical practice; (2) the MSLT is only one measure of daytime sleepiness and may not correlate highly with other outcomes, such as neuropsychological tests, quality of life, simulated driving tests, and subjective feelings of sleepiness; (3) the MSLT as used by the authors is not standard but is a modification of the recommended experimental MSLT; specifically, the 20-s epochs are not recommended, and the definition of sleep onset and the use of 10 min of sleep are not standard; and (4) the study number is small, with 7 to 10 subjects with SAS and 10 control subjects being analyzed.

How the design features affect the conclusions or application of the results is speculative, but the reader should be cautious in attempting to apply the results to current clinical practice.

To confirm the current observations, a larger study should be done using not only the MSLT but also other measures of daytime sleepiness and function such as the Epworth Sleepiness Scale, the maintenance of wakefulness test, and quality-of-life measures. In addition, the concept that SWA (but not SWS) during the first NREM period is critical in daytime function needs to be confirmed in different settings. For example, if the hypothesis is correct, then treatment of SAS to reduce daytime sleepiness may only require treatment of the first 2 h of sleep with minimal benefit of longer treatment. In general, it may be possible to sleep for 2 h and not suffer most of the consequences of sleep deprivation. If this concept can be applied not only to the SAS but to other conditions, this may have major implications for shift workers, soldiers, and others.

If the observations of this study are confirmed, sleep medicine practitioners may need to revise the current standard scoring system for staging NREM sleep. Speculatively, SWA may be staged as a substitute for SWS using criteria of 0.75 to 4.5 cps with no amplitude requirements. This may require modifying the approach to handling sleep recordings since it may be difficult to score SWA by visual inspection. Instead, spectral analysis may become the preferred approach. In addition, scoring of the first NREM-REM cycle for SWA may provide predictive value for the degree of daytime sleepiness. Improvement in SWA of the first NREM-REM cycle with treatment in SAS may provide predictive value for successful management of daytime sleepiness. In addition, this study provides a greater rationale for staging sleep, even in patients with obvious obstructive sleep apnea.

It is also possible that the observations of this study may not be applicable to clinical sleep staging. Instead, the observations may help design further research to understand sleep factors that influence daytime function. Ultimately, this research may help design better diagnostic and treatment strategies for patients with daytime sleepiness from various sleep disorders.

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