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Preface

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Daytime sleepiness is a state that may be ignored for many years by affected subjects. Subjective scales have been developed for assessment of daytime sleepiness, but the controversies regarding their efficacy are many. Subjects with obstructive sleep apnea of similar apnea–hypopnea index may have an Epworth sleepiness scale (ESS) score that can be as low as 5 and as high as 15. Correlation between subjective scales and polysomnographic tests exploring daytime sleepiness are also often poor. Subjects with an ESS score of 6 have shown a mean sleep latency of 6 minutes as measured with the multiple sleep latency test (MSLT) and 8 minutes as measured with the maintenance of wakefulness test (MWT). Alternatively, subjects with an ESS score of 13 have shown a mean sleep latency of 14 minutes (by way of MSLT) and of 15 minutes (by way of MWT). Individuals may deny sleepiness but report "fatigue" or "tiredness" when they wake up that increases with time during the day. Many attempts have been made at defining sleepiness, and various tests have been used to measure attention and performance in sleepy patients with limited success.

Investigation of sleep deprivation or sleep restriction has shown that important variability exists in the way subjects respond to these challenges. In one of our studies with 7 days of sleep restriction to 4 hours per night, important individual differences in performance, subjective alertness scales, and polysomnographic tests were already evident by day 2. These differences persisted until the end of the experimental condition despite the fact that the flattening of the leptin secretion curve associated with sleep restriction and the increase in food intake were similar in all subjects [1]. We are far from understanding the biological basis of these individual differences. Our study on sleep restriction in young adults also showed dissociation between subjects in the decrements noted in different tests used over time; some were present in all subjects and some were present only in a subgroup of normal individuals. Additionally, the severity of the impairment, related to similar sleep restriction or deprivation, judged with specific performance tests or the MWT varied from mild to severe.

Clearly, we do not possess "the test" that can appropriately evaluate impairment due to daytime sleepiness, and we cannot effectively determine all impairments related to sleepiness in a given subject at all hours of the day. It is also difficult to properly assess the personal and societal risks that may arise when an individual is coping with sleep restriction, abnormal schedules, or a sleep-disturbing illness or mechanisms to maintain alertness. Finally, genetic and environmental factors, as always in medicine, may interact to produce excessive daytime sleepiness (EDS).

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Complaints of EDS exist, however, and its effects are clearly pronounced in everyday events such as decreased social/cognitive function and motor vehicle accidents. Epidemiologic studies have shown a very wide range of response to this matter. In the United States, many different methodologies have been applied to the study of EDS, making it difficult to compare studies; the reported prevalences vary from 0.3% to 16.3%. The Cardiovascular Health Study [2] found a 20% prevalence of participants being "usually sleepy in the daytime" in a sample of 4578 adults aged 65 and older. In Europe, the prevalence varied from 5% to 16% in adults aged 20 years and older. In addition, most investigations of the prevalence of EDS have been performed on Caucasians, and we lack information on the prevalence of the complaint in other ethnicities.

The notion that sleep restriction, shift work hours, and poor sleep hygiene—characteristics often linked with industrialization—are associated with EDS is recognized by many. Additionally, the fact that syndromes such as narcolepsy are associated with EDS—and that the symptoms can be a major element in the pathology of EDS—is well acknowledged. Systematic investigation of narcolepsy–cataplexy has lead to the discovery of the hypocretin/orexin system in the brain, and recognition of the importance of this system in the maintenance of alertness. However, we are still far from having a good grasp of the different neuronal systems involved in maintenance of normal alertness and the brain circuits associated and activated with EDS. Von Economo, studying encephalitica lethargica [3], identified brain regions critical for the maintenance of alertness, and since then many have tried to use the anatomic-pathology approach to investigate the problem. We have more tools at our disposal, but many points remain unclear. Recently, EDS was investigated in a large cohort study, the Honolulu-Asia Aging study cohort, and an increased risk of subsequent development of Parkinson disease was shown in men with EDS [4].

This issue of the *Sleep Medicine Clinics* covers many avenues, from evaluation of the sleepy patient to the many possible factors behind a complaint of EDS. Can we use the understanding of neurologic disorders to paint a clearer picture of EDS? What can we offer to subjects who complain of EDS? What is behind intermittent but recurrent hypersomnia? What are the risks associated with EDS in children and in adults? We are still far from finding responses to all these questions, but advances have been made during the past few years. These advances are outlined in this issue, as are the many remaining unanswered questions that our field needs to tackle.

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