The multiple sleep latency test in the diagnosis of sleep disorders

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Summary

The multiple sleep latency test provides an objective measure of a patient's daytime sleepiness. Sixteen tests were performed at Groote Schuur Hospital in 1987 and 1988 according to a fixed protocol. In 8 patients the test was definitely abnormal (mean sleep latency < 5 minutes) with 3 subjects diagnosed as having narcolepsy, 1 sleep apnoea syndrome, 1 idiopathic central nervous system hypersomnolence, 2 environment-related hypersomnolence and 1 psychophysiological hypersomnolence. In 2 patients the test results fell in the equivocal range (mean sleep latency 5 - 10 minutes), while in 5 the test revealed no evidence for a disorder of excessive sleep (mean sleep latency > 10 minutes). The test was uninterpretable for technical reasons in only 1 patient. In conclusion, the test — when performed in a standardised manner — is extremely helpful in the elucidation of possible disorders of excessive sleep.

The Groote Schuur Hospital experience with the MSLT in 1987 and 1988 is reviewed.

Patients and methods

Patients presenting to Groote Schuur Hospital with complaints of excessive sleep are initially assessed clinically, and thereafter undergo an MSLT if the diagnosis is uncertain or narcolepsy is suspected. If sleep apnoea is suspected, nocturnal polysomnography is performed usually without a preceding MSLT. Where considered clinically relevant other tests, such as computed tomography of the brain, pulmonary function tests, tests for HLA antigens and thyroid function tests, are requested. In 1987 and 1988 MSLTs were performed. The charts of the 16 patients were reviewed and the findings compiled and analysed. In each case a diagnosis in accordance with the Association of Sleep Disorders Centers Classification of Sleep Disorders was reached, based on both the results of the sleep studies and the clinical features.

The MSLT protocol was adapted from that recommended by the Association of Sleep Disorders Centers Task Force on Daytime Sleepiness. All sedative, antidepressant and stimulant medication was discontinued 2 weeks before the test. Patients were instructed to keep a record of their sleep behaviour for a week before the study. They were admitted to hospital the day before the test and retired to sleep at their usual bedtime. They slept in a quiet, dark, single-bed ward and were not disturbed until the following morning. No caffeine- or alcohol-containing beverages were permitted after supper. In tests performed in 1988 sleep was monitored during the night using an 8-channel Oxford Medilog 9000 ambulatory electro-encephalograph (EEG). Vertex and occipital EEGs, submental electromyograms (EMGs) and electro-oculograms (EOGs) were recorded on a cassette tape worn on a belt around the waist. Sleep staging was performed by visual analysis of the tape the following day.

The patients were awakened at 07h50 on the morning of the test. No caffeine-containing beverages were allowed during the day and the patients refrained from smoking 30 minutes before each test. The patients were given either four or five

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opportunities to sleep in a quiet, dark room with a comfortable bed in the EEG laboratory. If 5 tests were performed these took place at 09h30, 11h30, 13h30, 15h30 and 17h30. If four tests were performed these took place at 10h00, 12h00, 14h00 and 16h00. Sleep activity was monitored by standard polygraphic techniques using a conventional 8-channel EEG machine. EEG (referential central and occipital placements), submental EMG, EOG, and nasal airflow were recorded. Data were recorded at a paper speed of 10 mm/s, and thus the duration of each epoch was 30 seconds. A time constant of 0.3 s was used. At the start of each test patients were instructed to close their eyes, lie still and attempt to sleep. Each test was continued for 20 minutes if no sleep occurred or for 15 minutes after the first sleep epoch. Between tests the patients remained out of bed and were instructed to resist sleeping.

For each test sleep latency and rapid eye movement (REM) sleep latency were measured. The sleep latency is the time from onset of the test until the commencement of the first sleep epoch assessed by standard criteria. The mean sleep latency was calculated for the four or five tests performed. The REM sleep latency is the time from the onset of the first sleep epoch until the onset of the first REM sleep epoch. Normal values for the test are internationally recognised. A mean sleep latency of < 5 minutes is abnormal, both statistically and biologically, in that subjects with shorter mean sleep latencies demonstrate impaired performance and irresistible daytime naps. Mean sleep latencies of > 10 minutes are normal. Between 5 and 10 minutes lies a diagnostic grey area representing probable abnormal sleepiness. REM sleep occurring < 10 minutes after sleep onset (sleep onset REM: SOREM) in more than one of the tests is abnormal (Fig. 1).4

Fig. 1. Onset of REM sleep in a patient with sleep apnoea syndrome. Rapid eye movements are seen in the first 2 channels and submental muscle hypotonia in channel 7.

Results

Patients were divided into four groups based on MSLT results (Table 1). Results of other investigations were normal unless described below.

Group I — mean sleep latency < 5 minutes

The 8 patients in this group averaged a mean sleep latency of 3 minutes 14 seconds (range 43 seconds - 4 minutes 50 seconds). Their mean age was 36 years (range 23 - 52 years). Three patients were diagnosed as having narcolepsy. All 3 experienced SOREM periods in two or more of the tests (mean 3.75 SOREM periods per study), while none of the other 5 experienced more than one SOREM period in any of the tests. In addition, all 3 narcoleptic patients complained of cataplexy, 1 had experienced sleep paralysis and 2 had a family history of hypersomnolence.

One patient experienced apnoic periods during the test and subsequent nocturnal polysomnography confirmed a diagnosis of mixed central and peripheral sleep apnoea syndrome with significant hypoxia. Pulmonary function tests showed a mild restrictive pattern owing to obesity. One patient was diagnosed as having idiopathic central nervous system (CNS) hypersomnolence. Two patients had environment-related disorders of excessive somnolence — in 1 this was due to overcrowding at night; the symptoms resolved once the patient’s living conditions improved, and in the other the cause was an unusual shift work schedule. In 1 patient the diagnosis was that of transient psychophysiological hypersomnolence due to reactive depression as a result of marital difficulties. When these were resolved the patient’s sleep improved.

Group II — sleep latency 5 - 10 minutes

The 2 patients in this group averaged a mean sleep latency of 8 minutes (7 minutes 50 seconds and 8 minutes 55 seconds). No SOREM periods occurred. Their mean age was 27 years (23 and 30 years). In 1 patient the diagnosis was that of idiopathic CNS hypersomnolence and in the other an environment-related sleep disorder caused by working excessively long hours in association with an active social life. A change in his lifestyle resulted in improvement.

Group III — sleep latency > 10 minutes

The 5 patients in this group averaged a mean sleep latency of 14 minutes 6 seconds. No SOREM periods occurred. Their mean age was 27 years (range 13 - 45 years). In spite of complaining of excessive daytime sleepiness, there was no objective evidence for a disorder of excessive somnolence. In 2 of the patients a final assessment of occupation-related boredom was made. In 2 patients psychosocial problems resulted in feelings of fatigue. One patient, who complained of lifelong difficulty in waking in the morning, was assessed as being a ‘long sleeper’. A subsequent nocturnal sleep study allowing him to sleep as long as he wished showed a total sleep time of 11 hours 30 minutes with 6 complete sleep cycles.

### TABLE I. MSLT RESULTS AND FINAL DIAGNOSES

<table>
<thead>
<tr>
<th>Group</th>
<th>No. of patients</th>
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<tbody>
<tr>
<td>Group I</td>
<td>Narcolepsy: 3</td>
</tr>
<tr>
<td>Sleep apnoea syndrome: 1</td>
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</tr>
<tr>
<td>Idiopathic CNS hypersomnolence: 1</td>
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<tr>
<td>Environmental-related hypersomnolence: 2</td>
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<tr>
<td>Transient psychophysiological hypersomnolence: 1</td>
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<tr>
<td>Group II</td>
<td>Idiopathic CNS hypersomnolence: 1</td>
</tr>
<tr>
<td>Environment-related hypersomnolence: 1</td>
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<tr>
<td>Group III</td>
<td>No evidence for a disorder of excessive somnolence: 5</td>
</tr>
<tr>
<td>Group IV</td>
<td>No evidence for a disorder of excessive somnolence: 1</td>
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</table>
Group IV — technically unsatisfactory

The MSLT in 1 patient, aged 35 years, was technically unsatisfactory owing to a combination of the patient’s failure to discontinue stimulant medication until 48 hours before the test and an unusually high level of ambient noise during the study.

Discussion

The MSLT provides invaluable objective evidence of a patient’s degree of daytime sleepiness. The results reported here show that in the majority of patients a clear distinction can be made between those suffering from a disorder of excessive sleep and those with subjective complaints but no objective findings. The mean sleep latencies fell in the indeterminate range (5 - 10 minutes) in only 2 patients (12%) and a definitive clinical diagnosis was reached in both.

The test is relatively easy to perform, but strict attention must be paid to technical details. It is especially important that all drugs affecting sleep be stopped 2 weeks before the test. Most centres therefore perform formal nocturnal polysomnography on the preceding night in order to demonstrate that adequate sleep was obtained. However, the use of restricting multiple sensors, unfamiliar apparatus and the presence of a technician may result in disturbed sleep especially if a previous night of adaptation in the laboratory is not logistically possible. Initially our laboratory dispensed with monitoring sleep the preceding night but more recently we have used with success the Oxford Medilog 9000 ambulatory EEG system to assess total sleep time. This apparatus has been well tolerated by patients who sleep undisturbed in a single-bed hospital room. The use of this ambulatory system technically uninterpretable and this was owing to failure to observe two of these precautions.

In particular, it has been shown that disturbed sleep the night before the test and even up to a week earlier can result in false-positive results. Most centres therefore perform formal nocturnal polysomnography on the preceding night in order to demonstrate that adequate sleep was obtained. However, the use of restricting multiple sensors, unfamiliar apparatus and the presence of a technician may result in disturbed sleep especially if a previous night of adaptation in the laboratory is not logistically possible. Initially our laboratory dispensed with monitoring sleep the preceding night but more recently we have used with success the Oxford Medilog 9000 ambulatory EEG system to assess total sleep time. This apparatus has been well tolerated by patients who sleep undisturbed in a single-bed hospital room. The use of this ambulatory system has been acceptably validated for sleep monitoring.

The MSLT proved very useful in arriving at a final clinical diagnosis. SOREM periods are the classic neurophysiological marker of narcolepsy, but may also be seen in sleep deprivation, stimulant withdrawal and occasionally in sleep apnoea syndrome. More than one SOREM period was present in 3 of our patients all of whom were eventually diagnosed as suffering from narcolepsy. It might be argued that the diagnosis was obvious in the presence of cataplexy and sleep paralysis but the severe consequences of the condition and the need for lifelong medication makes it essential that the diagnosis be reached definitively as early as possible. In addition, 1 of these 3 patients had been refused unemployment insurance as his story of excessive sleep was disbelief before obtaining objective evidence.

Two patients were diagnosed as having idiopathic CNS hypersomnolence, an ill-defined condition distinct from narcolepsy, resulting in longer but less irresistible periods of daytime sleepiness and not associated with SOREM. One patient had sleep apnoea syndrome not suspected clinically, but the presence of nasal airflow obstruction in the MSLT led to the establishment of the diagnosis by nocturnal polysomnography. The remaining patients with objective hypersomnolence had varying causes of environmental or transient psychophysiological disorders of excessive sleep.

The group with normal sleep onset latencies is of special interest. These patients complained of excessive sleepiness but no objective evidence for this could be determined. Boredom accounted for the problem in 1 patient and neurasthenic symptoms of fatigue in another while the third fell into the category of a normal long sleeper at the far end of the Gaussian curve of total sleep times. This group of patients can give rise to much diagnostic confusion as many are inaccurately diagnosed as having narcolepsy and placed inappropriately on potentially toxic stimulant medication.

I am grateful to Mr Jan Top, senior neurophysiology technologist, for performing the MSLTs.

REFERENCES