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# *Report From the American Sleep Disorders Association*

# The Clinical Use of the Multiple Sleep Latency Test



## PART A: POSITION PAPER: GUIDELINES FOR THE USE OF THE MULTIPLE SLEEP LATENCY TEST (MSLT)

[This position paper is referenced by the square-bracketed numbers to the numbered paragraphs in the accompanying review.]

These practice guidelines represent the consensus opinion on the indications for use of the MSLT by the Standards of Practice Committee (SPC) of the American Sleep Disorders Association. The systematically developed consensus statements are produced to assist the clinician in making patient care decisions about the appropriate use of the MSLT for specific health care circumstances. The guidelines refer to adult clinical practice and were not developed to cover pediatric practice, although many of the consensus statements have relevance and are appropriate to pediatric health care.

Adherence to these guidelines is voluntary. These guidelines should not be considered inclusive of all proper methods of care or exclusive of other methods of care reasonably directed to obtaining the same results. The ultimate judgement regarding the propriety of any specific procedure must be made by the physician in light of the individual circumstances presented by the patient.

These are the first clinical guidelines as to the patient populations best assessed by the MSLT, the disorders for which the MSLT is indicated and the frequency of use of the MSLT. The Standards of Practice Committee of the American Sleep Disorders Association expect the guidelines to impact on professional behavior, patient outcomes and health care costs.

### I. General

1. Excessive sleepiness is a potentially life-threatening condition that can be associated with significant morbidity. [1.0,2.1]

2. Patient subjective reports of sleepiness can be unreliable and may not correlate with observer reports. [2.1]

3. The MSLT is the only scientifically validated objective test of excessive sleepiness. [2.2,2.3]

4. The MSLT is used to establish a diagnosis of specific sleep disorders or to determine the severity of sleepiness. [2.3,2.4,2.5]

5. The MSLT must be performed under appropriate conditions and with accurate technique and interpretation. [2.3]

### II. Indications for the MSLT

1. Narcolepsy: An MSLT is indicated for all patients suspected of narcolepsy to confirm the diagnosis and to determine the severity of sleepiness and should be performed before commencing treatment with stimulant medications. [3.1]

2. Obstructive sleep apnea syndrome: (a) The MSLT is indicated in patients with mild to moderate obstructive sleep apnea syndrome who complain of moderate to severe sleepiness. [3.2] (b) The MSLT may be indicated in patients with moderate to severe obstructive sleep apnea syndrome, especially if severe sleepiness is unappreciated or denied. [3.2]

3. Other causes of excessive sleepiness: An MSLT is indicated in the evaluation of patients suspected of having idiopathic hypersomnia, periodic limb movement disorder or when the cause of excessive sleepiness is unknown. [3.3]

4. Insomnia: The MSLT is indicated in the evaluation of the complaint of insomnia when the presence of moderate to severe excessive sleepiness is suspected. [3.4]

5. Circadian rhythm sleep disorders: The MSLT may be useful in documenting sleepiness in some circadian rhythm sleep disorders, but adequate scientific validation is not yet available. [3.5]

6. Assessment of treatment effects: The MSLT is indicated to assess the response to treatment following effective therapy for disorders that cause sleepiness when an additional sleep disorder that produces sleepiness is suspected, or if confirmation of relief of sleepiness is required to ensure occupational safety. [3.6]

7. Repeat MSLT testing: Repeat MSLT testing is indicated in the following situations: (a) when the initial test is believed to be an invalid representation of the patient's status; (b) when ambiguous or uninterpretable MSLT findings occur; (c) when the response to treatment needs to be ascertained; and (d) when more than one sleep disorder is suspected. [3.7]

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### III. Technology and methods

1. For correct interpretation the MSLT must be performed following an allnight polysomnogram. [2.4,3.1]
2. The MSLT consists of five nap opportunities to determine both severity of sleepiness and presence of two or more sleep onset rapid eye movement (REM) periods for the diagnosis of narcolepsy. [2.4]
3. A shorter four-nap test may be performed for determination of excessive sleepiness, but this test is not reliable for the diagnosis of narcolepsy unless at least two sleep onset REM periods (SOREMPs) have occurred. [2.4,3.1]
4. There are no validated computerized analysis systems for the MSLT. [2.3]

### IV. Scoring and interpretation

1. The MSLT technical report should include the onset and offset time of each nap, latency from lights out to the first epoch of sleep, amount of each sleep stage, total sleep time, mean latencies to sleep of all naps and number of sleep onset REM periods. [2.4]
2. Sleep onset is determined by the first epoch of any stage of sleep, including stage 1 sleep. [2.4]
3. The absence of sleep on any nap opportunity is recorded as a sleep latency of 20 minutes. This latency is included in the overall analysis of mean sleep latency. [2.4]

## PART B: SUPPORTING EVIDENCE FOR THE POSITION PAPER: A REVIEW

### 1.0 Introduction

Excessive sleepiness, defined as sleepiness that occurs at a time when the individual would usually be expected to be awake and alert, affects approximately 5% of the general population (1–4) and is associated with significant morbidity, in terms of loss of work hours, reduced productivity, increased work errors and impaired social and family relationships. There is an increased mortality risk to the patient and others when sleepiness affects motor vehicle drivers and those in crucial occupations, such as aircraft pilots, train drivers or nuclear power workers (5).

The most widely used objective test for the diagnostic evaluation of patients with excessive sleepiness is the multiple sleep latency test (MSLT). The appropriate use of the MSLT is required for sleep disorder center accreditation by the American Sleep Disorders Association (ASDA).

### 2.0 Background

#### *2.1 Methods*

This review and position paper were developed in the following way: 1) review position papers were provided by clinicians with clinical research and practice experience in the use of the MSLT; 2) a Medline search (1980–1990) and additional literature review were carried out and core position statements were developed; 3) the review and position statements were subjected to review by the SPC and by those who submitted the initial position papers, as well as by other clinicians involved in the practice of sleep medicine; and 4) the final document was agreed to by all members of the SPC and then approved for publication by the Executive Board of the American Sleep Disorders Association.

#### *2.2 Consequences of sleepiness*

Excessive sleepiness can impair the quality of life and is a major cause of morbidity and even mortality due to its role in industrial and transportation accidents (5–12). Excessive sleepiness causes impaired cognitive and intellectual functioning, which has serious psychosocial consequences such as impaired interpersonal, marital, work and social relationships (13–18). Education, recreation, motor vehicle driving, housework, employment, earning capacity and promotion can all be negatively affected by sleepiness (19,20). The severity of sleepiness can be defined in the following manner as detailed in the *International Classification of Sleep Disorders* (21):

*Mild sleepiness.* This term describes sleep episodes that are present only during times of rest or when little attention is required. Situations in which mild sleepiness can become evident include but are not limited to lying down in a quiet room, watching television or reading and when travelling as a passenger in a moving vehicle. Mild sleepiness may not be present every day. The symptoms of mild sleepiness produce a minor impairment of social or occupational function. This degree of sleepiness is usually associated with an MSLT mean sleep latency of 10–15 minutes and can often be found in otherwise normal, healthy control subjects (22).

*Moderate sleepiness.* This term describes sleep episodes that are present daily and that occur during very mild physical activities requiring, at most, a moderate degree of attention. Examples of situations in which moderate sleepiness may occur include driving and while attending concerts, movies, the theater or similar group meetings. The symptoms of moderate sleepiness produce a moderate impairment of social or occupational function. This degree of sleepiness is usually associated with an MSLT mean sleep latency of 5–10 minutes and has been referred to as the diagnostic "gray zone" (22,23).

*Severe sleepiness.* This term describes sleep episodes that are present daily and at times of physical activities that require mild to moderate attention. Examples of situations in which severe sleepiness may occur include eating, direct personal conversation, driving, walking and physical activities. The symptoms of severe sleepiness produce a marked impairment of social or occupational function. This degree of sleepiness is usually associated with an MSLT mean sleep latency of less than 5 minutes. Patients with mean sleep latencies below 5 minutes are regarded as having pathological sleepiness and should be warned about their potential for industrial or motor vehicle accidents (24).

Excessive sleepiness, which may be mild, moderate or severe sleepiness as defined above, is considered to be a contributing factor in major occupational catastrophies involving shift work, such as those at Chernobyl and Three Mile Island nuclear power plants and more recently the Exxon

Valdez oil spill accident (5,25). The Department of Transportation has made the inquiry into the role of sleepiness in transportation accidents an important new area of investigation (25,26).

Sleepiness is affected by age, circadian factors, medications and drugs and sleep disorders including insufficient nocturnal sleep (9,27–30). Alcohol and some medications can exacerbate the tendency for sleepiness, particularly in the elderly, and increase the incidence of home, industrial or motor vehicle accidents (31–33). The *International Classification of Sleep Disorders* (21) lists 33 medical and psychiatric disorders that produce excessive sleepiness. Of those, narcolepsy and obstructive sleep apnea syndrome are common, but still insufficiently recognized, causes of excessive sleepiness that can result in severe morbidity and increased mortality (8,10–12).

### 2.3 Tests of sleepiness

Sleepiness may need to be quantified because subjective reports of sleepiness can be unreliable (6,34). Typically, the severity of sleepiness is unappreciated by the patient; reports by patients and their spouses regarding the former's falling asleep when reading or watching television have only a 72% agreement owing to the patient's usual underestimation of the presence of sleepiness (34).

Sleepiness may be denied by the patient because of embarrassment or concern regarding punitive actions, such as loss of a driver's license or occupation. Occasionally, symptoms of fatigue and tiredness may be misconstrued as due to excessive sleepiness, particularly in patients suffering from psychiatric disorders such as depression. In addition, excessive sleepiness may be falsely reported in an effort to obtain restricted stimulant medications (35).

The MSLT is the most reliable test used in the assessment of sleepiness as other diagnostic means are either too insensitive and nonspecific or have not been adequately subjected to scientific evaluation. Observable indicators such as yawning, reduced activity, ptosis, lapses in attention and head drooping may be seen in patients who are sleepy but these signs are variably present and influenced by motivation and activity. The unreliability of such indicators precludes their usefulness in the assessment of excessive sleepiness. Subjective rating scales such as the Stanford Sleepiness Scale (SSS) provide a more reliable assessment of excessive sleepiness than behavioral indicators, but their precision is still inadequate due to individual differences in descriptions of subjective sleepiness (36–38). Subjects have been observed falling asleep while rating themselves fully alert.

Electrophysiological tests of sleepiness include pupillometry (39,40), evoked potential studies (41), performance tests such as the Wilkinson Vigilance Test (42), continuous ambulatory monitoring techniques (43) and actigraphy (44). The advantage of the MSLT over most other measures is that it directly measures the functional consequences of sleepiness, namely, falling asleep; and also measures sleepiness at two hourly intervals across the waking portion of the day. In addition, the MSLT is the only electrophysiological test that has been fully validated to be effective in detecting different degrees of sleepiness.

### 2.4 The MSLT technique

The MSLT is a four or five "nap opportunity" test in which the subject rests in a quiet darkened room and the latency to sleep is determined by standard electrophysiological means. Sleep latency is defined as the elapsed time from lights-out to the first epoch of any sleep stage (22,45). In addition to the sleep onset latency of each nap opportunity, the stages of sleep that occur provide information that is useful in the diagnosis of narcolepsy and other disorders of excessive sleepiness.

Detailed standard guidelines for the performance of the MSLT have been produced by the Task Force on Daytime Sleepiness of the Association of Sleep Disorders Centers, and published in both the medical journal *Sleep* (22), and the textbook *The Principles and Practice of Sleep Medicine* (45). For correct interpretation the MSLT must be performed under appropriate conditions and requires accurate technique.

The clinical MSLT assesses sleep latency by the initial appearance of any sleep stage, including stage 1 sleep (22). The test is continued for 15 minutes after the first epoch of sleep. If sleep does not occur the test is continued for 20 minutes after lights out and the sleep latency is scored as 20 minutes. Subjective reports of sleep duration and dreaming during naps can be helpful in the interpretation of both the clinical symptoms and the MSLT results. The scoring and interpretation of the MSLT must be performed carefully by an experienced person, especially when atypical, ambiguous or disrupted sleep stages occur as they can in obstructive sleep apnea syndrome and narcolepsy. It has been suggested that modified scoring criteria would improve accuracy in the determination of sleep latency in patients with the obstructive sleep apnea syndrome (46).

A variation of the MSLT, the Maintenance of Wakefulness Test (MWT), is performed under identical conditions as the MSLT but with the patient semireclining and instructed to attempt to remain awake (47,48). The MWT is used less commonly than the MSLT and mainly used to assess improved alertness following therapeutic interventions (49,50).

There are no significant safety hazards posed by the technology and its clinical use.

### 2.5 Reliability in detection of sleepiness

The sensitivity and specificity of the MSLT in detecting sleepiness have not been assessed; however, the test has been shown to reliably detect sleepiness that occurs following sleep disruption (51,52) and sleep loss (29,53,54), due to circadian effects (30) and hypnotic and alcohol effects (32,33,55–59). The test–retest reliability of the MSLT has been documented in normal subjects (60) and patients with narcolepsy (61).

The MSLT (including the MWT) has also been shown to be responsive to manipulations that reduce sleepiness such as sleep extension (62), caffeine ingestion (63) and the treatment of disorders of excessive sleepiness (64–66). However, MSLT studies of the effects of stimulant medications taken for narcolepsy have not reliably demonstrated reduction in sleepiness at clinically effective doses (23). The MWT, however, has been reported to be sensitive to the effects of stimulant medications (50). This lack of effect on sleep la-

tency in the MSLT and not the MWT may be due to improvement of the ability to remain awake rather than elimination of excessive sleepiness, which may undermine the usefulness of the MSLT for treatment outcome evaluations of analeptic medications.

*Limitations of the MSLT.* A patient's psychological and behavioral states affect the consequences of excessive sleepiness that leads to actual sleep, and, therefore, the interpretation of the MSLT results. In other words, the MSLT measures a subject's tendency to sleep rather than the likelihood of falling asleep. Accordingly, if mentally stimulated by psychological, behavioral or medicinal means, even a severely sleepy individual may have little evidence of sleepiness during the MSLT (51,67). This discrepancy between the underlying physiological drive for sleepiness and the overt manifest sleepiness limits the reliability of the MSLT in some patients and at some times (4,52). However, the MSLT has proven to be a reliable indicator of sleep tendency in most patients.

## 3.0 Indications for the MSLT

### *3.1 Narcolepsy*

The MSLT is both a sensitive and reliable test of the sleepiness due to narcolepsy (23,24,50,68–71). More than 80% of patients with narcolepsy have a mean MSLT sleep latency of less than 5 minutes (23). Of those who have longer latencies (greater than 5 minutes) on initial MSLT, some patients will demonstrate shorter latencies with subsequent retesting (72).

Two or more SOREMPs on MSLT testing are found in at least 80% of narcolepsy patients (23,24,68). Accordingly, this has been accepted as a diagnostic feature of narcolepsy, if other potential causes have been excluded, such as other sleep disorders, sleep deprivation and drugs, including alcohol, stimulants, tricyclic antidepressants and monoamine oxidase inhibitors (24,68,69,71). Occasional narcolepsy patients who do not have SOREMPs on initial MSLT testing will demonstrate two or more SOREMPs on subsequent testing (23).

The diagnosis of narcolepsy is dependent upon the presence of cataplexy or the presence of the characteristic features on the MSLT (21). Cataplexy is often subtle and can easily be overlooked by the patient and even by the physician. Unless cataplexy is witnessed by the clinician, which rarely occurs in clinical practice, the diagnosis of narcolepsy may remain in doubt. The diagnosis should be confirmed by the MSLT, especially if cataplexy is absent.

Because of the duration of treatment and potential for drug abuse, stimulant medications should not be prescribed until a definite diagnosis of narcolepsy is established. The patient may fabricate the symptoms to obtain stimulant medication (35).

Single nap studies to detect a SOREMP are not adequate to diagnose narcolepsy (73,74). A single SOREMP on one sleep opportunity is nonspecific for narcolepsy as it can occur in a variety of situations: healthy subjects (75–78), following sleep deprivation (79) and in association with other sleep disorders, such as obstructive sleep apnea syndrome (46,69,80,81). Because of improved specificity, the MSLT has now replaced the single nap test as the preferred diagnostic test for narcolepsy.

### *3.2 Obstructive sleep apnea syndrome*

Excessive sleepiness is reported to occur in at least 80% of patients with the obstructive sleep apnea syndrome (4,82). Although more variable than in narcolepsy, sleepiness can be equally severe and also associated with morbidity and mortality (64). Patients tend to underestimate the presence of sleepiness compared with spousal reports, and denial of symptoms is commonly reported (34).

Obstructive sleep apnea syndrome has been classified as mild, moderate or severe by the following criteria (21): (a) Mild; usually associated with mild sleepiness or mild insomnia. The majority of the habitual sleep episode is free of respiratory disturbance and the apneic episodes are associated with mild oxygen desaturation or benign cardiac arrhythmias. (b) Moderate; usually associated with moderate sleepiness or mild insomnia. The respiratory disturbance can be associated with moderate oxygen desaturation or mild cardiac arrhythmias. (c) Severe; usually associated with severe sleepiness. The majority of the habitual sleep episode is associated with respiratory disturbance, severe oxygen desaturation or moderate to severe cardiac arrhythmias. There can be evidence of associated cardiac or pulmonary failure.

The MSLT demonstrates excessive sleepiness in the majority of obstructive sleep apnea patients and typically shows mean sleep latencies below 10 minutes (23,46,51,64,66,81,83–85). Most patients with mild obstructive sleep apnea syndrome and few or no symptoms of sleepiness will show normal sleep latencies on the MSLT, whereas patients with moderate to severe sleep apnea syndrome can show severely reduced sleep latencies (86,87), sometimes despite few complaints of sleepiness. However, the relationship between severity of sleepiness and the respiratory disturbance index is not high, which suggests that some other factor such as sleep fragmentation or sleep deprivation may be contributing to the sleepiness (83,85). The presence of moderate to severe sleepiness on the MSLT can influence: (a) a clinician's recommendations regarding the need for any behavioral or occupational changes, (b) the decision to recommend a particular therapeutic intervention and (c) the decision for further investigations or additional follow-up assessment.

Occasionally SOREMPs are seen during the nap opportunities in patients with obstructive sleep apnea syndrome, and therefore, the concurrent presence of narcolepsy may be difficult to exclude on initial MSLT testing (45,83,87,88). Recurrent apneic episodes have been reported to occur in many narcolepsy patients (89). The presence of both narcolepsy and obstructive sleep apnea syndrome is estimated to occur in approximately 6% of patients with excessive sleepiness (90). In the absence of cataplexy, treatment of the sleep apnea should always occur before establishing a definitive diagnosis of narcolepsy.

Some specialists in sleep-related breathing disorders do not routinely perform the MSLT in patients with severe obstructive sleep apnea syndrome and base clinical decisions on overnight polysomnography, patient reports and subjective or objective [continuous positive airway pressure (CPAP) trial] response to treatment. Although there are no outcome studies that assess either the effect of this approach or that using objective documentation of sleepiness, the evidence

cited in this report indicates that some patients benefit from the objective assessment of sleepiness.

### 3.3 Other causes of hypersomnia

An MSLT is indicated when the excessive sleepiness is of unknown cause, not resolved spontaneously with behavioral treatment such as sleep hygiene recommendations or not due to transient circadian rhythm sleep disorders.

Idiopathic hypersomnia is a disorder of excessive sleepiness without REM sleep features such as cataplexy (90–92). An MSLT will typically show a sleep latency of less than 10 minutes and often less than 5 minutes (23,93). In contrast to narcolepsy, in idiopathic hypersomnia 80% of patients do not have a SOREMP on MSLT testing (93). REM sleep has been reported to occur more than once in less than 5% of patients in the five nap opportunities (93,94). Slow wave sleep may be detected on the MSLT. These MSLT features are not specific for idiopathic hypersomnia as they can be produced by chronic sleep deprivation, which needs to be excluded (67,95).

Periodic limb movement disorder, a disorder associated with periodic leg movements during the major sleep episode, can be associated with excessive sleepiness (23,96). Periodic leg movements occur with increased prevalence in narcolepsy, and therefore when sleepiness is suspected to be due to periodic leg movements the MSLT is indicated, not only to determine the severity of the excessive sleepiness but also to ensure that narcolepsy is not present (97).

Sleepiness due to mood disorders is rarely severe and usually does not require MSLT documentation; however, the MSLT may be useful if the cause or severity of the sleepiness is in doubt (97).

Some sedative medications, such as the prior use of hypnotics, antidepressants or anticonvulsants, can produce excessive sleepiness (23,55,98,99). An MSLT may occasionally be indicated to assess sleepiness in patients taking such medications who require full vigilance for home or occupational safety.

### 3.4 Insomnia

Many patients with insomnia have a state of hyperarousal that prevents the underlying physiological sleepiness from becoming manifest. Complaints of fatigue or tiredness are common but usually do not indicate an increased tendency to fall asleep (100,101). The MSLT mean sleep latencies are usually above 10 minutes (102–105). However, there is evidence that some patients with a complaint of insomnia, particularly those who have the disorder sleep state misperception, can have severe sleepiness on the MSLT of similar severity to that seen in idiopathic hypersomnia (100). The MSLT may be useful in the evaluation of the complaint of insomnia when excessive sleepiness is suspected.

### 3.5 Circadian rhythm sleep disorders

The MSLT has demonstrated excessive sleepiness during the desired waking/working hours following acute shifts of the sleep pattern, such as that seen in shift workers and following time zone change (jet lag syndrome) (106–108). However, the transient nature of shift work and jet lag as well as the widespread recognition of the presence of sleepiness in this group make the MSLT unnecessary in their evaluation. The MSLT is required if the degree of sleepiness or the occurrence of sleepiness-related work errors suggest that the patient may have an additional disorder, such as narcolepsy, that contributes to the sleepiness. Some patients with excessive sleepiness due, for example, to narcolepsy prefer a shift-work occupation under circumstances in which the sleepiness is less likely to be noticed as abnormal by the employer. There is a need to recognize, diagnose and treat such patients.

There is little information available on the use of the MSLT in persistent circadian rhythm sleep disorders. Excessive sleepiness, a common symptom of these disorders, can have severe morbidity and may produce occupational and educational problems (109,110). It may be necessary to document the presence or severity of sleepiness by an MSLT, but the timing of the MSLT is critical for correct interpretation. Guidelines for the performance of the MSLT in circadian rhythm sleep disorders have not yet been subjected to scientific evaluation. However, in some circumstances and in some circadian rhythm sleep disorders, such as the delayed sleep phase syndrome, the MSLT may be helpful (109,111). The MSLT in delayed sleep phase syndrome, when performed following a forced awakening at the desired but not usual time of awakening, can demonstrate sleepiness in the initial nap opportunities, when the patient would usually be sleeping, and can also show improved alertness in the late afternoon (109). Occasionally, another sleep disorder can be present with features suggestive of a circadian rhythm sleep disorder. For example, children with narcolepsy can exhibit features that suggest delayed sleep phase syndrome (112).

### 3.6 Assessment of treatment response

The use of the MSLT in the assessment of response to treatment is less clear than its use for diagnostic purposes (86,113). Some narcolepsy patients show little improvement in the MSLT latencies following subjectively effective treatment by stimulant medications (23). This lack of improvement may mean that stimulant medications in usual clinical doses increase the ability to remain awake but do not affect the ability to fall asleep. The MWT, a variant of the MSLT, may be a better indicator of beneficial treatment effects than the standard MSLT in patients with incurable sleepiness, such as narcolepsy or idiopathic hypersomnia (47,49).

The MSLT (including the MWT) has been shown to demonstrate improved alertness following treatment of obstructive sleep apnea syndrome by tracheostomy (64), uvulo-palato-pharyngoplasty (66) and CPAP therapy (65,114,115). However, following effective treatment for obstructive sleep apnea by tracheostomy or CPAP some patients continue to have excessive sleepiness (64,114,116,117). Posttreatment MSLT mean sleep latencies can be less than 10 minutes (64). The cause of the continued sleepiness is not known but may indicate the presence of concurrent narcolepsy, idiopathic

hypersomnia or other disorders of excessive sleepiness. Therefore, the MSLT may be indicated in some patients to determine whether treatment of the sleep apnea syndrome has been effective in eliminating excessive sleepiness. This is particularly important after treatments that are generally less efficacious than tracheostomy or CPAP, such as uvulo-palato-pharyngoplasty, dental appliances or use of respiratory stimulant medications.

Persistence of sleepiness after treatment of obstructive sleep apnea syndrome may be due to ineffective treatment, poor compliance to CPAP or other coincident sleep disorders (66,114,118–120). To determine the concurrent presence of narcolepsy, after treatment of obstructive sleep apnea syndrome by CPAP, the MSLT should be performed with the patient using the CPAP device.

Long-acting hypnotic medications will produce excessive sleepiness during the daytime in some patients treated for insomnia (56,57). Reduced sleep latencies have been demonstrated on the MSLT following patients' prior night's use of benzodiazepines (56). This drug-induced sleepiness may be a factor in the increased prevalence of hip fractures that has been reported in the elderly who use long-acting hypnotic medications (31).

Whether to assess sleep disorder treatment response by the MSLT is dependent upon the need to document full alertness for the purpose of assessing safety, including transportation or occupational safety. Most patients do not require MSLT testing after treatment so long as the treatment of the underlying disorder is considered to be maximally effective.

### *3.7 Repeat MSLT testing*

When the MSLT is indicated, either for diagnostic reasons or for determination of sleepiness severity, it usually needs to be performed only once in any particular patient. Repeat MSLT testing may be indicated in the following situations: (a) when the initial test is believed to be an invalid representation of the patient's status (68,72), such as that due to a stimulatory effect that reduces manifest sleepiness; (b) when ambiguous or uninterpretable MSLT findings occur; (c) when the response to treatment needs to be ascertained; and (d) when more than one sleep disorder is suspected.

## 4.0 Summary and future considerations

Excessive sleepiness is a potentially life-threatening condition that can be associated with significant morbidity and mortality. The MSLT is the only scientifically validated objective test that has been shown to be a sensitive test of excessive sleepiness. This test helps establish the diagnosis of disorders of excessive sleepiness, such as narcolepsy, and is useful for determining sleepiness severity. However, the test may produce false-negative results in a small number of patients who do not demonstrate manifest sleepiness during the test.

Normative data and sensitivity and specificity probabilities on the MSLT are needed, and further studies are required to define the MSLT's role in diagnostic considerations of some sleep disorders, such as idiopathic hypersomnia and sleep-related breathing disorders. The usefulness of the MSLT in patients with circadian rhythm sleep disorders requires further investigation as other types of alertness testing, such as continuous 24-hour monitoring, may be more useful. Outcome studies are essential to determine how the MSLT results affect treatment decisions and to determine the response to treatment.

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## REFERENCES

1. Bixler ED, Kales A, Soldatos CR, et al. Prevalence of sleep disorders in the Los Angeles metropolitan area. *Am J Psychiatry* 1979;136:1257–62.
2. Lavie P. Sleep habits and sleep disturbances in industrial workers in Israel: main findings and some characteristics of workers complaining of excessive daytime sleepiness. *Sleep* 1981;4:147–58.
3. Lugaresi E, Cirignotta F, Zucconi M, et al. Good and poor sleepers: an epidemiological survey of the San Marino population. In: Guilleminault C, Lugaresi E, eds. *Sleep/wake disorders: natural history. Epidemiology, and long-term evolution.* New York: Raven Press 1983:2–12.
4. Roth T, Roehrs T, Carskadon M, Dement W. Daytime sleepiness and alertness. In: Kryger MH, Roth T, Dement WC, eds. *Principles and practice of sleep medicine.* Philadelphia: Saunders, 1989:14–23.
5. Mitler MM, Carskadon MA, Czeisler CA, Dement WC, Dinges DF, Graeber RC. Catastrophes, sleep, and public policy: consensus report. *Sleep* 1988;11:100–9.
6. Aldrich CK, Aldrich MS, Aldrich TK, Aldrich RF. Asleep at the wheel. *Postgrad Med J* 1986;80:233–9.
7. Aldrich MS: Automobile accidents in patients with sleep disorders. *Sleep* 1989;12:487–94.
8. Aldrich MS: Narcolepsy. *N Engl J Med* 1990;323:389–94.
9. Carskadon MA, Dement WC. Cumulative effects of sleep restriction on daytime sleepiness. *Psychophysiology* 1981;18:107–13.
10. Findley LJ, Barth JT, Powers DC, Wilhoit SC, Boyd DG, Serat PM. Cognitive impairment in patients with obstructive sleep apnea and associated hypoxemia. *Chest* 1986;90:686–90.
11. Findley LJ, Unverzagt ME, Suratt PM. Automobile accidents involving patients with obstructive sleep apnea. *Am Rev Respir Dis* 1988;138:337–40.
12. Findley LJ. Automobile driving in sleep apnea. *Prog Clin Biol Res* 1990;345:337–43.
13. Beutler LE, Catsby Ware J, Karacan I, Thornby JI. Differentiating psychosocial characteristics of patients with sleep apnea and narcolepsy. *Sleep* 1981;4:39–47.
14. Broughton R, Ghanem Q. The impact of compound narcolepsy in the life of the patient. In: Guilleminault C, Dement WC, Passount P, eds. *Narcolepsy.* New York: Spectrum 1976:201–20.
15. Broughton RJ, Ghanem Q, Hishikawa Y, Sugita Y, Nevsimalova S, Roth B. Life effects of narcolepsy in 180 patients from North America, Asia and Europe compared to matched controls. *Can J Neurol Sci* 1981;8:299–304.

16. Broughton RJ, Guberman A, Roberts J. Comparison of the psychosocial effects of epilepsy and narcolepsy/cataplexy: a controlled study. *Epilepsia* 1984;25:423–33.
17. Kales A, Soldatos CR, Bixler EO, Caldwell A, Cadieux RJ, Verrechio JM, Kales JD. Narcolepsy–cataplexy. II. Psychosocial consequences and associated psychopathology. *Arch Neurol* 1982;39:169–71.
18. Krishnan RR, Volow MR, Miller PP, Carwile ST. Narcolepsy: preliminary retrospective study of psychiatric and psychosocial aspects. *Am J Psychiatry* 1984;141:428–31.
19. Goswami M, Glovinsky PB, Thorpy MJ. Need assessment and socio-demographic characteristics in narcolepsy. *Sleep Res* 1987; 16:339.
20. Thorpy MJ, Goswami M. Treatment of narcolepsy. In: Thorpy MJ, ed. *Handbook of sleep disorders.* New York: Marcel Dekker, 1990:235–58.
21. ICSD—Diagnostic Classification Committee; Thorpy MJ, Chairman. *International classification of sleep disorders: diagnostic and coding manual.* Rochester, Minnesota: American Sleep Disorders Association, 1990.
22. Carskadon MA, Dement WC, Mitler MM, Roth T, Westbrook PR, Keenan S. Guidelines for the multiple sleep latency test (MSLT): a standard measure of sleepiness. *Sleep* 1986;9:519–24.
23. Van den Hoed J, Kraemer H, Guilleminault C, Zarcone VP, Miles L, Dement WC, Mitler MM. Disorders of excessive daytime somnolence: polygraphic and clinical data for 100 patients. *Sleep* 1981;4:23–37.
24. Richardson GS, Carskadon MA, Flagg W, van den Hoed J, Dement WC, Mittler MM. Excessive daytime sleepiness in man: multiple sleep latency test measurement in narcoleptic and control subjects. *Electroencephalogr Clin Neurophysiol* 1978;45:621–7.
25. Dinges DF, Graeber RC, Carskadon MA, Czeisler CA, Dement WC. Attending to inattention. *Science* 1989;245:342.
26. Lauber JK, Kayten PJ. Sleepiness, circadian dysrhythmia, and fatigue in transportation system accidents. *Sleep* 1988;11(6): 503–12.
27. Carskadon MA, Harvey K, Duke P, Anders T, Litt IF, Dement WC. Pubertal changes in daytime sleepiness. *Sleep* 1980;2: 453–60.
28. Carskadon MA, Brown ED, Dement WC. Sleep fragmentation in the elderly: relationship to daytime sleep tendency. *Neurobiol Aging* 1982;3:321–7.
29. Carskadon MA, Harvey K, Dement WC. Sleep loss in young adolescents. *Sleep* 1981;4:299–312.
30. Richardson GS, Carskadon MA, Orav EJ, Dement WC. Circadian variation in sleep tendency in elderly and young adult subjects. *Sleep* 1982;5:S82–94.
31. Ray WA, Griffen MR, Downey W. Benzodiazepines of long and short elimination half-life and the risk of hip fracture. *JAMA* 1989;262:3303–7.
32. Mamelak M, Buck L, Csima A, Price V, Smiley A. Effects of flurazepam and zopiclone on the performance of chronic insomnia patients: a study of ethanol-drug interaction. *Sleep* 1987;10:S79–87.
33. Lumley M, Roehrs T, Asker D, Zorick F, Roth T. Ethanol and caffeine effects on daytime sleepiness/alertness. *Sleep* 1987;10: 306–12.
34. Wiggins CL, Schmidt-Nowara WW, Coultas DB, Samet JM: Comparison of self- and spouse reports of snoring and other symptoms associated with sleep apnea syndrome. *Sleep* 1990; 13(3):245–52.
35. Thorpy MJ, Wagner D, Spielman AJ, Weitzman ED. Polysomnographic documentation of narcolepsy. *Arch Neurol* 1983; 40:126–7.
36. Dement WC. Daytime sleepiness and sleep "attacks". In: Guilleminault C, Dement WC, Passouant P, eds. *Narcolepsy.* New York: Spectrum Publ., 1976:17–42.
37. Herscovitch J, Broughton R. Sensitivity of the Standford sleepiness scale to the effects of cumulative partial sleep deprivation and recovery oversleeping. *Sleep* 1981;4:83–92.
38. Hoddes E, Zarcone V, Smythe H, Phillips R, Dement WC. Quantification of sleepiness: a new approach. *Psychophysiology* 1973;10:431–6.
39. Lowenstein O, Loewenfeld I. Pupillary movements during acute and chronic fatigue: a new test for the objective evaluation of tiredness. *Invest Ophthalmol & Visual Sci* 1963;2:138–57.
40. Yoss RE, Mayer NJ, Ogle KN. The pupillogram and narcolepsy. *Neurology* 1969;19:921–8.
41. Aguirre M, Broughton R. Complex event-related potentials (P300 and CNV) and MSLT in the assessment of excessive daytime sleepiness in narcolepsy–cataplexy. *Electroencephalogr Clin Neurophysiol* 1987;67:298–316.
42. Wilkinson RT. Sleep deprivation. Performance tests for partial and selective sleep deprivation. *Prog Clin Psychol* 1968;8:28–43.
43. Broughton R, Dunham W, Newman J, et al. Ambulatory 24-hour sleep–wake monitoring narcolepsy–cataplexy compared to matched controls. *Electorencephalogr Clin Neurophysiol* 1988;70:473–81.
44. Newman J, Stampi C, Dunham DW, Broughton R. Does wrist-actigraphy approximate traditional polysomnographic detection of sleep and wakefulness in narcolepsy–cataplexy? *Sleep Res* 1988;17:343.
45. Carskadon MA, Dement WC. Normal human sleep: an overview. In: Kryger MH, Roth T, Dement WC, eds. *Principles and practice of sleep medicine.* Philadelphia: Saunders, 1989: 3–13.
46. Browman CP, Winslow DH. Determination of sleep latency in polysomnographic evaluation of daytime somnolence in patients with sleep apnea and patients with narcolepsy. *Clin Electroencephalogr* 1989;20:45–8.
47. Mitler MM, Gujavarty KS, Browman CP. Maintenance of wakefulness test: a polysomnographic technique for evaluating treatment in patients with excessive somnolence. *Electroencephalogr Clin Neurophysiol* 1982;53:648–61.
48. Mitler MM. Alerting drugs: do they really work? *Psychopharmacol Bull* 1987;23:435–9.
49. Mitler MM, Shafor R, Hajdukovich R, Timms RM, Browman CP. Treatment of narcolepsy: objective studies on methylphenidate, pemoline, and protriptyline. *Sleep* 1986;9:260–4.
50. Mitler MM, Hajdukovic R, Erman M, Koziol JA. Narcolepsy. *J Clin Neurophysiol* 1990;7:93–118.
51. Carskadon MA, Dement WC. The multiple sleep latency test: what does it measure? *Sleep* 1982;5:S67–72.
52. Stepanski E, Lamphere J, Badia P, Zorick F, Roth T. Sleep fragmentation and daytime sleepiness. *Sleep* 1984;7:18–26.
53. Carskadon MA, Dement WC. Effects of total sleep loss on sleep tendency. *Percept Mot Skills* 1979;48:495–506.
54. Carskadon MA, Dement WC. Midafternoon decline in MSLT scores on a constant routine. *Sleep Res* 1985;14:292.
55. Bliwise D, Seidel W, Karacan I, Mitler M, Roth T, Zorick F, Dement W. Daytime sleepiness as a criterion in hypnotic medication trials: comparison of triazolam and flurazepam. *Sleep* 1983;6:156–63.
56. Carskadon MA, Seidel WF, Greenblatt DJ, Dement WC. Daytime carry-over effects of triazolam and flurazepam in elderly insomniacs. *Sleep* 1982;5:361–71.
57. Dement WC, Seidel W, Carskadon M. Daytime alertness, insomnia, and benzodiazepines. *Sleep* 1982;5:528–45.
58. Roehrs T, Kribbs N, Zorick F, Roth T. Hypnotic residual effects of benzodiazepines with repeated administration. *Sleep* 1986;9:309–16.
59. Zwyghuizen-Doorenbos A, Roehrs T, Lamphere J, Zorick F, Roth T. Increased daytime sleepiness enhances ethanol's sedative effects. *Neuropsychopharmacology* 1988;1(4):279–86.
60. Zwyghuizen-Doorenbos A, Roehrs T, Schaefer M, Roth T. Test–retest reliability of the MSLT. *Sleep* 1988;11(6):562–5.
61. Scrima L, Hartman P, Johnson FH, Thomas EH, Hiller FC. The effects of gamma-hydroxybutyrate on the sleep of narcolepsy patients: a double blind study. *Sleep* 1990;13:479–90.
62. Roehrs T, Timms V, Zwyghuizen-Doorenbos A, Roth T. Sleep extension in sleepy and alert normals. *Sleep* 1989;12:449–57.
63. Zwyghuizen-Doorenbos A, Roehrs T, Lipschutz L, Timms V,

Roth T. Effects of caffeine on alertness. *Psychopharmacology (Berl)* 1990;100:36–9.
64. Dement WC, Carskadon MA, Richardson GS. Excessive daytime sleepiness in the sleep apnea syndrome. In: Guilleminault C, Dement WC, eds. *Sleep apnea syndromes.* Menlo Park: Alan Liss Inc., 1978:23–46.
65. Poceta JS, Ho S, Jeong D, Mitler MM. The maintenance of wakefulness test in obstructive sleep apnea syndrome. *Sleep Res* 1990;19:268.
66. Zorick F, Roehrs T, Conway W, Fujita S, Wittig R, Roth T. Effects of uvulopalatopharyngoplasty on the daytime sleepiness associated with sleep apnea syndrome. *Bull Europe Physiopathol Respir* 1983;19:600–3.
67. Jahnke B, Aldrich MS. The multiple sleep latency test (MSLT) is not infallible. *Sleep Res* 1990;19:240.
68. Amira SA, Johnson TS, Logowitz NB. Diagnosis of narcolepsy using the multiple sleep latency test: analysis of current laboratory criteria. *Sleep* 1985;8(4):325–31.
69. Browman CP, Gujavarty KS, Yolles SF, Mitler MM. Forty-eight-hour polysomnographic evaluation of narcolepsy. *Sleep* 1986;9:183–8.
70. Miara SA, Johnson TS, Logowitz NB. Diagnosis of narcolepsy using the multiple sleep latency test. *Sleep* 1985;8:325–31.
71. Mitler MM, van den Hoed J, Carskadon MA, Richardson GS, Park R, Guilleminault C, Dement WC. REM sleep episodes during the multiple sleep latency test in narcoleptic patients. *Electroencephalogr Clin Neurophysiol* 1979;45:479–81.
72. Kotagal S, Hartse KM, Walsh JK: Characteristics of narcolepsy in preteenaged children. *Pediatrics* 1990;85:205–9.
73. Roth B, Nevsimalova S, Sonka K, Docekal P. An alternative to the multiple sleep latency test for determining sleepiness in narcolepsy and hypersomnia: polygraphic score of sleepiness. *Sleep* 1986;9:243–5.
74. Wilson R, Raynal D, Guilleminault C, Zarcone V, Dement W. REM latencies in daytime sleep recordings of narcoleptics. *Sleep Res* 1972;6:200.
75. Carskadon M. The role of sleep-onset REM periods in narcolepsy. In: Guilleminault C, Dement WC, Passouant P, eds. *Narcolepsy.* New York: Spectrum, 1976:499–519.
76. Rechtschaffen A, Wolpert E, Dement W, Mitchell S, Fisher C. Nocturnal sleep of narcoleptics. *Electroencephalogr Clin Neurophysiol* 1963;15:599–609.
77. Webb W, Agnew H, Sternthal H. Sleep during the early morning. *Psychon Sci* 1966;6:277–8.
78. Weitzman E, Nogeire C, Perlow M, Fukushima D, Sassin J, McGregor P, Gallagher T, Hellman L. Effects of a prolonged 3-hour sleep–wakefulness cycle on sleep stages, plasma cortisol, growth hormone and body temperature in man. *J Clin Endocrinol Metab* 1974;38:1018–30.
79. Dement W, Rechtschaffen A. Narcolepsy: polygraphic aspects, experimental and theoretical concepts. In: Gastaut H, Lugaresi E, Berti-Ceroni G, Coccagna G, eds. *The abnormalities of sleep in man.* Gaggi: Bologna 1968:147–64.
80. Mitler MM, Gujavarty KS, Sampson MG, Browman CP. Multiple daytime nap approaches to evaluating the sleepy patient. *Sleep* 1982;5:119–27.
81. Zorick F, Roehrs T, Koshorek G, Sicklesteel J, Hartse K, Wittig R, Roth T. Patterns of sleepiness in various disorders of excessive daytime somnolence. *Sleep* 1982;5:S165–74.
82. Guilleminault C, Van den Hoed J, Mitler M. Clinical overview of the sleep apnea syndromes. In: Guilleminault C, Dement WC, eds. *Sleep apnea syndromes.* New York: Allan Liss Inc., 1978:1–12.
83. Guilleminault C, Partinen M, Quera-Salva MA, Hayes B, Dement WC, Nino-Murcia G. Determinants of daytime sleepiness in obstructive sleep apnea. *Chest* 1988;94:32–7.
84. Reynolds CF, Coble PA, Kupfer DJ, Holzer BC. Application of the multiple sleep latency test in disorders of excessive somnolence. *Electroencephalogr Clin Neurophysiol* 1982;53:443–52.
85. Roehrs T, Zorick F, Wittig R, Conway W, Roth T. Predictors of objective level of daytime sleepiness in patients with sleep-related breathing disorders. *Chest* 1989;95:1202–6.
86. Roth T, Hartse KM, Hartse F, Zorick F, Conway M. Multiple naps and the evaluation of daytime sleepiness in patients with upper airway sleep apnea. *Sleep* 1980;3:425–39.
87. Walsh JK, Smithson SA, Kramer M. Sleep onset REM sleep: comparison of narcoleptic and obstructive sleep apnea patients. *Clin EEG Neurophysiol* 1982;13:57–60.
88. Browman CP, Gujavarty KS, Sampson MG, Mitler MM. REM sleep episodes during the maintenance of wakefulness test in patients with sleep apnea syndrome and patients with narcolepsy. *Sleep* 1983;6:23–8.
89. Chokroverty S. Sleep apnea in narcolepsy. *Sleep* 1986;9(1): 250–3.
90. Guilleminault C, Dement W. 235 cases of excessive daytime sleepiness. *J Neurol Sci* 1977;31:13–27.
91. Billiard M. Other hypersomnias. In: Thorpy M, ed. *Handbook of sleep disorders.* New York: Marcel Dekker, 1990:353–74.
92. Roth B. *Narcolepsy and hypersomnia.* Basel: Karger, 1980.
93. Baker TL, Guilleminault C, Nino-Murcia G, Dement WC. Comparative polysomnographic study of narcolepsy and idiopathic central nervous system hypersomnia. *Sleep* 1986;9(1): 232–42.
94. Nahmias J, Karetzky M. Narcolepsy versus idiopathic CNS hypersomnolence: a comparison of patient and polysomnographic characteristics. *Sleep Res* 1989;18:275.
95. Roehrs T, Zorick F, Sicklesteel J, Wittig R, Roth T. Excessive daytime sleepiness associated with insufficient sleep. *Sleep* 1983; 6(4):319–25.
96. Bonnet MH, Arand DL. The use of triazolam in older patients with periodic leg movements, fragmented sleep and daytime sleepiness. *J Gerontol* 1990;45:139–44.
97. Coleman RM, Bliwise DL, Sajben N, Boomkamp A, de Bruyn LM, Dement WC. Daytime sleepiness in patients with periodic movements in sleep. *Sleep* 1982;5:S191–202.
98. Dement WC, Seidel W, Carskadon M, Bliwise D. Changes in daytime sleepiness/alertness with nighttime benzodiazepines. In: Usdin E, Skolnick P, Tallman J, Greenblatt D, Paul SM, eds. *Pharmacology of benzodiazepines.* London: MacMillan Press, 1982:219–28.
99. Johnson LC, Spinweber CL, Gomez SA, Matteson LT. Daytime sleepiness, performance, mood, nocturnal sleep: the effect of benzodiazepines and caffeine on their relationship. *Sleep* 1990;13:121–35.
100. Hauri P, Wisbey J. The MSLT in insomnia. *Sleep Res* 1990; 19:234.
101. Stepanski E, Zorick F, Roehrs T, Young D, Roth T. Daytime alertness in patients with chronic insomnia compared with asymptomatic control subjects. *Sleep* 1988;11:54–60.
102. Mendelson WB, James SP, Garnett D, Sack DA, Rosenthal NE. A psychophysiological study of insomnia. *Psychiatr Res* 1986;19:267–84.
103. Seidel WF, Dement WC. Sleepiness in insomnia: evaluation and treatment. *Sleep* 1982;5:S182–90.
104. Seidel WF, Ball S, Cohen S, Patterson N, Yost D, Dement WC. Daytime alertness in relation to mood, performance, and nocturnal sleep in chronic insomniacs and noncomplaining sleepers. *Sleep* 1984;7:230–8.
105. Sugerman JL, Stern JA, Walsh JK. Daytime alertness in subjective and objective insomnia: some preliminary findings. *Biol Psychiatry* 1985;20:741–50.
106. Nicholson AN, Pascoe PA, Spenser MB, et al. Sleep after transmeridian flights. *Lancet* 1986;2:1205–8.
107. Seidel WF, Roth T, Roehrs T, Zorick F, Dement WC. Treatment of a 12 hour shift of sleep schedule with benzodiazepines. *Science* 1984;224:1262–4.
108. Walsh JK, Sugerman JL, Muehlbach MJ, Schweitzer PK. Physiological sleep tendency on a simulated night shift: adaptation and effects of triazolam. *Sleep* 1988;11:251–64.
109. Thorpy MJ, Korman E, Spielman AJ, Glovinsky PB. Delayed sleep phase syndrome in adolescents. *J Adolesc Health Care* 1988;9:22–7.
110. Weitzman ED, Czeisler CA, Coleman RM, Spielman AJ, Zimmerman JC, Dement WC. Delayed sleep phase syndrome. *Arch Gen Psychiatry* 1981;38:737–46.

111. Rosenthal NE, Joseph-Vanderpool JR, Levendosky AA, et al. Phase shifting effects of bright morning light as treatment for delayed sleep phase syndrome. *Sleep* 1990;13:354–61.
112. Guilleminault C. Narcolepsy and its differential diagnosis. In: Guilleminault C, ed. *Sleep and its disorders in children.* New York: Raven Press, 1987:181–94.
113. Hartse KM, Roth T, Zorick FJ. Daytime sleepiness and daytime wakefulness: the effect of instruction. *Sleep* 1982;5:S107–1118.
114. Lamphere J, Roehrs T, Wittig R, Zorick F, Conway WA, Roth T. Recovery of alertness after CPCP in apnea. *Chest* 1989;96: 1364–7.
115. Poceta JS, Jeong D, Ho S, Timms RM, Mitler MM. Hypoxemia as a determinant of daytime sleepiness in obstructive sleep apnea. *Sleep Res* 1990;19:269.
116. Ledereich P, Thorpy MJ, Glovinsky PB, Burack B, McGregor P, Rozycki D, Sher AE. Five year follow-up of daytime sleepiness and snoring after tracheostomy in patients with obstructive sleep apneas. In: Chouard CH, ed. *Chronic rhonchopathy.* Paris: John Libbey Eurotext, 1988:354–7.
117. Thorpy MJ, Ledereich P, Glovinsky PB, Barnett M, Blumofe A, Burack B, Rozycki D, McGregor P, Sher AE. Follow-up in obstructive sleep apnea. *Jpn J Psychiatry Neurol* 1988;42:718–9.
118. Kreiger J, Kurtz D. Objective measurement of compliance with nasal CPAP treatment for obstructive sleep apnoea syndrome. *Eur Respir J* 1988;1(5):436–8.
119. Nino-Murcia G, McCann CC, Bliwise DL, Guilleminault C, Dement WC. Compliance and side effects in sleep apnea patients treated with nasal continuous positive airway pressure. *West J Med* 1989;150(2):165–9.
120. Norman SE, Fertil B, Judge E, Cohn MA. Compliance to nasal continuous positive airway pressure (NCPAP) four year follow-up. *Sleep Res* 1989;18:279.