Factors associated with maintenance of wakefulness test mean sleep latency in patients with mild to moderate obstructive sleep apnoea and normal subjects

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SUMMARY
This study investigated the possible factors related to the Maintenance of Wakefulness Test (MWT) mean sleep latency. A second analysis explored the characteristics of subjects who had discrepant Epworth Sleepiness Scale (ESS) and MWT scores. A total of 151 subjects (110 mild to moderate obstructive sleep apnoea (OSA) patients and 41 control subjects) were recruited for the study. The subjects completed an overnight Polysomnography (PSG), MWT, cognitive, performance and vigilance tasks and answered self-report questionnaires on mood and sleepiness. A forward stepwise multiple regression was performed on MWT mean sleep latency. The predictor variables age (r = 0.28), subjective sleep history for 1 week prior to MWT (sleep diary; r = 0.19) and number of >4% SaO2 Dips during the PSG (r = -0.21) best explained the MWT results, but only accounted for 12.8% of the variance in the test. It was found that 33% of subjects had discrepant ESS and MWT scores. A new variable was created to analyse these subjects (MWT/ESS discrepancy score; MED). A forward stepwise multiple regression analysis found that depression, performance errors and sleep disordered breathing explained 13.4% of the variance in MED scores. The MWT is a complex behavioural test whose scores do not seem to have a very robust relationship with potential predictors and co-correlates. Further comprehensive study is needed if the test is to be used in a diagnostically meaningful way.

KEYWORDS Maintenance of Wakefulness Test, Sleepiness and Mild OSA

INTRODUCTION
Objective tests of sleepiness such as the Multiple Sleep Latency Test (MSLT) and the Maintenance of Wakefulness Test (MWT) are used in research and clinical settings to assess the severity of sleep disorders and responses to treatment. The MSLT is the more widely used test. It measures sleep propensity when the subject is instructed to fall asleep. The MWT has more face validity as a test of sleep propensity in common behavioural situations such as driving, but it is used less frequently. In contrast to the MSLT, during the MWT subjects are instructed to try to stay awake while in a soporific environment, i.e. it tests the ability of the subject to resist sleep. Originally, the MWT used four evenly spaced 20-min trials (Mitler et al. 1982). The trial durations were subsequently lengthened to 40 min to try to eliminate the observed ceiling effect (Poceta et al. 1992). There is a comprehensive literature examining MSLT results in normal (Levine et al. 1988; Roehrs and Roth 1992; Zwyghuizen-Doorenbos et al. 1988) sleep deprived subjects (Bonnet and Arand 1995; Dinges et al. 1997; Levine et al. 1988; Martin et al. 1999; Roehrs et al. 1992; Zwyghuizen-Doorenbos et al. 1988) and patients with excessive daytime sleepiness (Aldrich et al. 1997; Chervin 2000; Guilleminault et al. 1988; Guilleminault et al. 1991). Based on
this literature it is agreed that an MSLT result <5 min indicates extreme sleepiness whereas a result between 5 and 10 min is borderline abnormal sleepiness. The MWT by comparison has been the subject of fewer investigations. Most research on the MWT has been in patients with obstructive sleep apnoea (Bennett et al. 1999; Tihonen and Partinen 1998) and narcolepsy (Harsh et al. 2000; Miller et al. 1998). One of the few studies that examined the normal ranges of the MWT suggests an abnormal MWT result is <19 min (Doghranji et al. 1997).

The factors that are related to average sleep latency in these tests are incompletely understood. A reasonable assumption would be that the severity of sleep disorders, such as OSA, which fragment sleep would be a major determinant. Previous investigation of the predictors of the MWT found that sleep fragmentation (Poceta et al. 1992) and electroencephalograph (EEG) arousal related body movements as measured by video monitoring (Bennett et al. 1998) were important variables. Sauter et al. (2000) and Kingshott et al. (1998) found that while MWT latencies tended to be shorter for patients with severe OSA, there was only a weak relationship between apnoea severity and MWT scores.

Bonnet and Arand (1997, 1998, 2000a,b) have shown that the MSLT and MWT mean sleep latencies are influenced considerably by individuals’ acute arousal level and that this can be moderated by exercise or music.

Psychological factors may also affect objective tests of daytime sleepiness. It has been found that in depressed patients the MWT sleep latency scores were inversely related to total sleep time (Kayumov et al. 2000). Motivation may also have an important influence on objective tests of sleepiness. Harrison et al. (1996) found that by simply offering subjects extra remuneration for a more rapid sleep onset caused a significant reduction in sleep onset latency on the MSLT. No studies to date have systematically examined MWT and motivation. Thus, objective tests of sleepiness seem to measure a combination of sleep propensity and underlying arousal.

One of the most frequently used measures of subjective daytime sleepiness is the Epworth Sleepiness Scale (ESS; Johns 1991). Studies have found that ESS scores are only modestly correlated with MSLT (Johns 1994) and MWT mean sleep latency (Sangal et al. 1999a,b). However, in a study of narcoleptic patients who received modafinil, mean scores on the ESS decreased while MWT and MSLT mean sleep latency increased compared with baseline, indicating that ESS scores follow changes in objective sleepiness (Fry 1998). In another study, narcoleptic patients with longer and higher quality nocturnal sleep had less sleepiness as measured by the ESS and MWT (Harsh et al. 2000). These studies suggest that subjective sleepiness as a trait on the ESS may be predictive of MWT and MSLT sleep latency as a state and vice versa.

The present study attempts to identify some of the variables that are predictive of MWT mean sleep latency and investigates the relationships amongst the co-correlates in a group of patients with mild–moderate OSA and normal controls. In a second analysis, the characteristics of those interesting individuals who show major discordance between subjective trait (ESS) and objective state (MWT) sleepiness scores are described in more detail. On the one hand, there are subjects who have little or no subjective sleepiness (ESS) yet are objectively sleepy (MWT). As they may not be able to readily perceive their sleep propensity, they may be particularly vulnerable to lapses while working or driving. At the other extreme are subjects who report high levels of subjective sleepiness (ESS) but are not objectively sleepy (MWT). These subjects may not be at risk of performance lapses because of sleepiness but symptoms of sleepiness may dominate the clinical presentation. We hypothesized that such subjects may have increased psychological complaints such as anxiety/depression that cause symptoms to be exaggerated and/or a higher level of physiological arousal that might counter the effects of sleepiness during the performance of the MWT.

METHODS

This study was approved by the research and ethics committees at the Repatriation General Hospital, Daw Park, Adelaide and Austin and Repatriation Medical Centre, Melbourne. All subjects gave written informed consent and received no remuneration for their participation in the study.

Participants

The patients for this study were recruited as part of a randomized, placebo-controlled trial investigating the treatment effects of CPAP and mandibular advancement splint. A total of 110 patients were invited to participate in the study after a clinical PSG that identified them as having mild–moderate OSA [categorized as an apnoea/hypopnoea index (AHI) between 10 and 30 per hour] but before any treatment was commenced. The patients had no significant co-morbidities (cardiovascular disease, respiratory disease or psychiatric disorders) and were not taking any regular medications (including hypnotic or anti-depressant medication).

Forty-one normal subjects also participated in this study. They were randomly selected from the telephone book within a 30-km radius of both the hospitals and were invited to participate via a telephone call. They answered a general health questionnaire, which screened for health and sleep difficulties. Normal volunteers were excluded from the study if they were <30 or >70 years of age, if English was not their first language, if they were heard to snore more than one night a week, or if they were on any medications for respiratory, cardiovascular or psychiatric disorders, or if they had a history of drug or alcohol abuse.

All subjects came to the laboratory for an introductory session to familiarize themselves with the laboratory environment. They were familiarized to the battery of neurobehavioural tests to be undertaken on the day of the MWT. The subjects then booked a time to come into the laboratory for an overnight PSG with an MWT the following day. The subjects were asked to keep a sleep diary for 1 week prior to the testing.
and to keep to their typical sleep–wake routine for that week. The participants’ subjective reports of nightly sleep duration during the week prior to testing time were averaged.

**Procedure**

*Overnight polysomnography*

The subjects arrived at the laboratory between 20.00 and 21.00 hours on the night of testing. Height and weight were measured to calculate BMI. The polysomnography included C4/A1 and C3/A2 EEG, electrocardiograph (ECG), submental electromyograph (EMG), ECG, airflow (nasal pressure cannula), respiratory effort (abdominal and thoracic bands), and SaO2. Sleep was scored according to standard criteria (Rechtschaffen and Kales 1968) and the EEG arousals were scored using ASDA (1992) guidelines. Sleep disordered breathing was defined according the following criteria: apnoea was defined as >80% reduction in airflow lasting at least 10 s and was classified as obstructive, central or mixed depending on whether or not there were ongoing respiratory efforts. Hypopnoea was defined as an event of at least 10 s duration, with a > 50% reduction from baseline in at least two of the following three signals: airflow, thoracic movement and abdominal movement. The apnoea plus hypopnoea index was computed by dividing the number of events by the hours of sleep. The AHI (respiratory events per hour asleep) arousal index (EEG arousals per hour asleep; ArI) and the number of >4% Dips in SaO2 during sleep (>4% SaO2 Dips) were the PSG variables used in the analysis.

**Maintenance of wakefulness test protocol**

On the day after the PSG, subjects were given four 40-min MWT trials at two hourly intervals with the first beginning between 08.00 and 10.00 hours. A quiet time of 15 min prior to each trial, where the subjects were restricted from excessive activity, reduced the effect of physiological variables on the test and smoking was prohibited during the 30 min before a trial. Subjects were required to abstain from caffeine beverages during the test day. Breakfast (cereal, milk and toast) was served at least 1 h before the first MWT trial and lunch (sandwich or roll, piece of fruit and non-caffeine beverage) immediately after the second trial (approximately 12 noon), 1 h before the next trial.

All trials were performed in a similar setting using a simplified recording montage (C3/A2, O1/A2, EMG, ECG and EOG). Bedrooms were insulated from external light and equipped with dimmer lights overhead. Ambient temperature in the room was approximately 22 °C. Bedroom doors were closed and all monitoring was performed external to the bedroom. This kept noise to a minimum. During each MWT trial, subjects sat semi-upright (10–30° back from vertical) in a comfortable lounge chair which had a high back to support the head and neck. Before each trial, subjects were instructed to ‘keep your eyes open and try not to fall asleep’ when the lights were dimmed. Subjects were asked not to use any extraordinary measures to stay awake, e.g. slapping their face, singing, whistling or moving about excessively in the chair. The recordings were then started and the lights dimmed to an illumination 10 lux. Each trial was terminated at the first occurrence of sustained sleep (three consecutive 30-s epochs of stage 1 sleep or one epoch of any other stage) or if sleep onset was not achieved after 40 min. The four sleep latencies were then averaged over the day to obtain the mean MWT sleep latency.

Heart rate also was monitored during the MWT as a measure of physiological arousal. The heart rate analysis was conducted on a subset of 52 subjects (32 patients and 20 control subjects) from Repatriation General Hospital, Daw Park who had ECG monitoring during the MWT trials. Resting HR was measured from the ECG (which was movement artefact-free) for 1 min approximately 5 min after the MWT trial started. Heart rate was also calculated approximately 5 min prior to sleep onset or approximately 5 min before the end of the test if no sleep was achieved. The mean of these two measures was then used for analysis.

**Neuropsychological testing**

Between the MWT trials, a trained neuropsychologist who was blinded to the subjects’ objective daytime sleepiness and AHI administered neuropsychological tests. The tests occurred at two hourly intervals beginning at 08.00 hours and lasting approximately 1 h. The neuropsychological tests included a 10-min visual psychomotor vigilance task [PVT median, PVT mean fastest 10% (PVTf10), PVT mean slowest 10% (PVTs10), PVT lapses and PVT unforced errors; Dinges and Powell 1985] and the Profile of Mood States (POMS; McNair et al. 1971) which were performed three times during the day. Scores were then averaged. ESS (Johns 1991) and the Beck Depression Inventory (BDI; Beck et al. 1961) were completed once during the day. Stanford Sleepiness Scale (SSS; Hoddes et al. 1973) was performed immediately before each MWT trial. The four scores were then averaged.

**Statistical analysis**

Patient and normal subject MWT mean sleep latency data was pooled for analysis (see Table 1 for patient and control group means and standard deviations of MWT results, PSG variables, PVT results and subjective mood and sleepiness questionnaires).

*MWT mean sleep latency predictors and co-correlates*

The MWT score is truncated at 40 min (ceiling effect). We considered several methods of trying to normalize the data but none were successful. A conditional (variables excluded if $P > 0.05$) stepwise multiple regression was conducted using the whole sample (patients and normal subjects combined) to
determine which were the independent predictors of MWT mean sleep latency. While it is not usual to perform regression analysis on non-normally distributed data, truncation in such analyses only lowers statistical power (e.g. reduces the magnitude of the relationships between variables). We therefore decided to continue with the analysis, acknowledging its limitations. Pearson correlations, with Bonferroni corrections for multiple comparisons, were conducted between MWT sleep latency and variables that could be best described as co-correlates rather than predictors.

Discrepancy between ESS and MWT mean sleep latency

It was noted during data collection that some subjects had objective (MWT mean sleep latency) and subjective sleepiness (ESS) results that were not concordant. There were a considerable number of subjects who had high ESS scores yet long MWT sleep latencies and vice versa. Therefore, we were interested in examining in more detail the subjects who had discordant scores.

Table 1 Descriptive statistics for the whole study population

<table>
<thead>
<tr>
<th></th>
<th>Total group</th>
<th>Patients</th>
<th>Control Subjects</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(n = 151)</td>
<td>(n = 110)</td>
<td>(n = 41)</td>
</tr>
<tr>
<td>MWT</td>
<td>32.4 ± 9.6</td>
<td>30.7 ± 10.2</td>
<td>36.7 ± 6.2</td>
</tr>
<tr>
<td>Age</td>
<td>47.8 ± 9.9</td>
<td>46.9 ± 10.1</td>
<td>49.9 ± 9.4</td>
</tr>
<tr>
<td>BMI</td>
<td>30.2 ± 5.3</td>
<td>31.1 ± 5.5</td>
<td>27.8 ± 4.1</td>
</tr>
<tr>
<td>SlpD</td>
<td>420.8 ± 61.5</td>
<td>419.7 ± 64.2</td>
<td>423.6 ± 54.9</td>
</tr>
<tr>
<td>AHI</td>
<td>15.5 ± 9.2</td>
<td>18.1 ± 7.4</td>
<td>6.1 ± 3.6</td>
</tr>
<tr>
<td>ArI</td>
<td>21.2 ± 9.3</td>
<td>22.7 ± 9.3</td>
<td>17.4 ± 7.8</td>
</tr>
<tr>
<td>TST</td>
<td>320.2 ± 63.5</td>
<td>320.2 ± 67.3</td>
<td>320.2 ± 53.2</td>
</tr>
<tr>
<td>&gt;4% SaO2 Dips</td>
<td>10.1 ± 15.1</td>
<td>12.22 ± 16.8</td>
<td>4.4 ± 6.6</td>
</tr>
<tr>
<td>Median PVT</td>
<td>241.8 ± 30.2</td>
<td>242.9 ± 30.8</td>
<td>239.0 ± 28.6</td>
</tr>
<tr>
<td>PVTt10</td>
<td>194.0 ± 20.9</td>
<td>193.7 ± 21.4</td>
<td>194.7 ± 19.7</td>
</tr>
<tr>
<td>PVTs10 (1/RT)</td>
<td>2.7 ± 0.5</td>
<td>2.6 ± 0.4</td>
<td>2.8 ± 0.4</td>
</tr>
<tr>
<td>PVT lapses</td>
<td>2.1 ± 2.8</td>
<td>2.5 ± 3.1</td>
<td>0.9 ± 0.9</td>
</tr>
<tr>
<td>PVT errors</td>
<td>7.2 ± 8.1</td>
<td>7.4 ± 8.6</td>
<td>6.5 ± 6.7</td>
</tr>
<tr>
<td>SSS</td>
<td>3.0 ± 1.0</td>
<td>3.1 ± 1.0</td>
<td>2.7 ± 1.0</td>
</tr>
<tr>
<td>ESS</td>
<td>9.3 ± 4.7</td>
<td>10.7 ± 4.5</td>
<td>5.44 ± 3.0</td>
</tr>
<tr>
<td>BDI</td>
<td>7.7 ± 5.9</td>
<td>9.2 ± 6.0</td>
<td>3.9 ± 3.7</td>
</tr>
<tr>
<td>POMSf</td>
<td>7.8 ± 5.5</td>
<td>8.8 ± 5.7</td>
<td>5.3 ± 4.1</td>
</tr>
<tr>
<td>POMSta</td>
<td>5.4 ± 4.4</td>
<td>5.7 ± 4.6</td>
<td>4.6 ± 3.9</td>
</tr>
<tr>
<td>POMSv</td>
<td>13.3 ± 8.3</td>
<td>12.1 ± 8.5</td>
<td>16.2 ± 6.8</td>
</tr>
<tr>
<td>POMSdd</td>
<td>3.2 ± 5.4</td>
<td>3.6 ± 5.6</td>
<td>2.2 ± 4.9</td>
</tr>
<tr>
<td>Total POMS</td>
<td>12.3 ± 22.4</td>
<td>15.5 ± 22.1</td>
<td>3.5 ± 20.9</td>
</tr>
</tbody>
</table>

MWT, Maintenance of Wakefulness Test (min); BMI, body mass index (kg m$^{-2}$); SlpD, prior subjective sleep history from the sleep diary (min); AHI, Apnoea/Hypopnoea Index (events per hour of sleep); ArI, Arousal Index (events per hour of sleep); TST, total sleep time from overnight PSG (min); >4% SaO2 Dips, number of oxygen desaturations >4% per hour of sleep during the overnight PSG; median PVT, median psychomotor vigilance task score (ms); PVTs10, the psychomotor vigilance task slowest scores [mean of the reciprocal slowest 10% scores (1/RT) on the psychomotor vigilance task, i.e. the smaller the score the slower the response; ms]; PVTt10, 10% fastest psychomotor vigilance task scores (ms); PVT lapses, number of RT ≥ 500 ms; PVT errors, number of psychomotor vigilance task errors; SSS, Stanford Sleepiness Scale; ESS, Epworth Sleepiness Scale; BDI, Beck Depression Inventory; POMSf, the Profile of Mood States – Fatigue scale; POMSta, the Profile of Mood States – Tension–Anxiety scale; POMSv, the Profile of Mood States – Vigor scale; POMSdd, the Profile of Mood States – Depression and Dejection scale; Total POMS, the Profile of Mood States – Total Mood Disorder Score.

To assess the discrepancy between objective and subjective measures of sleepiness, the MWT and ESS scores were standardized by transforming each score into a $z$-score. The $z$-score indicates the difference between a particular subject’s score and the mean score for the subject group as a whole. The mean for $z$-scores is always 0.0 and the standard deviation is 1.0. For example, the $z$-score is calculated as follows: ESS group mean (9.3) is taken away from the subject’s ESS score (15) and then divided by ESS group standard deviation (4.7) or $15–9.3/4.7=1.2$. The MWT results were treated the same way. For example, the MWT group mean (32.4) is taken away from the subject score (14) and then divided by the MWT group standard deviation (9.6) or $14–32.4/9.6=−1.9$. The resulting ESS and MWT $z$-scores were then added together to create a new continuous discrepancy variable $(1.2 ± 1.9 = 0.72)$. A score of 0 on the new variable MED score indicates concordance between the two scores. Low scores (below 1 or 1 SD below the mean) indicate subjects who are relatively less sleepy on the ESS than the MWT might suggest (or relatively more sleepy on the MWT than the ESS might suggest). High scores (above 1 or 1 SD above the mean)
indicate subjects who are relatively more sleepy on the ESS than the MWT might suggest (or relatively less sleepy on the MWT than the ESS might suggest).

A conditional stepwise multiple regression (variables excluded if \( P > 0.05 \)) was conducted with the MED score as the dependent variable. To explore for factors that might predict discrepancy between subjective (ESS) and objective (MWT) sleepiness, the variables of age, BMI, SlpD, AHI, ArI, TST, >4% SaO\(_2\) Dips, HR, PVTs10, PVTf10, PVT lapses, PVT errors, BDI and total POMS were entered into a regression model with MED score as the dependent variable.

**RESULTS**

There were 119 men (age range 24–68, median 48.0, mean 47.2 ± 9.6SD years) and 32 women (age range 23–79, median 49.5, mean 49.8 ± 11.1SD years) in the sample population.

**MWT mean sleep latency predictors and co-correlates**

Age, BMI, SlpD, AHI, ArI, TST, No. >4% SaO\(_2\) Dips and HR were entered into the regression model as potential predictor variables of MWT mean sleep latency. The regression model \((R = 0.36)\) was statistically significant \([F(3,134) = 6.5, P < 0.001]\), with age \((P = 0.006)\), SlpD \((P = 0.007)\), and >4%SaO\(_2\) Dips \((P = 0.03)\) contributing significantly to the model (see Table 2). They accounted for 4.8, 4.8 and 3.1%, respectively, for a total of 12.8% variance explained. That is, younger age, self-reported shorter sleep and sleep oxygen desaturations were all associated with a decrease in sleep latency on the MWT.

As age and MWT sleep latency were positively correlated (see Table 2), we wanted to determine if older individuals had more sleep than younger individuals. A one-way ANOVA was conducted [using a median age (48 years) split: group 1, <48 years; group 2, >48 years. Subjects whose age was 48 years were excluded from the analysis] on SlpD, TST, ArI and AHI. It was found that individuals aged <48 years had significantly more sleep on both their PSG TST \((P = 0.02)\) and SlpD \((P = 0.008)\) than older subjects. Subjects aged <48 years did not have more sleep disordered breathing or EEG arousals (see Table 3). Despite obtaining more sleep of similar quality to older subjects, younger subjects had shorter MWT latencies.

Testing for co-correlates of the MWT sleep latency found that the strongest relationship was with ESS followed by PVTS10 and PVT lapses (see Table 4). That is, short sleep latency on MWT was associated with increased behavioural sleepiness and with psychomotor slowing on a vigilance task.

**Discrepancy between ESS and MWT mean sleep latency**

Sixty-six subjects had a concordant MED scores (1SD from the mean, −1.0 and 1.0). Sixty-four percent of this group were patients and 36% were control subjects. There were 17% of the sample that had high MED scores (92% patients, 8% controls) and 16% that had low MED scores (88% patients, 12% controls). A chi-square analysis confirmed that there was a higher frequency of patients versus controls in the two groups of extreme scores \((P < 0.001)\).

The regression model used to explore possible predictors of the MED score was statistically significant \([F(3,147) = 4.3, P = <0.01]\), with PVT errors \((P = 0.003)\), BDI \((P = 0.004)\), and AHI \((P = 0.04)\) contributing significantly to the model (see Table 5). They accounted for 5.9, 5.0 and 2.5%, respectively for a total of 13.4% variance explained.

**DISCUSSION**

Very few studies have examined the factors associated with MWT sleep latency. It was our primary aim in this study to investigate some of the variables that may mediate MWT scores in patients with mild–moderate OSA and control subjects. First, the possible predictor variables of MWT mean sleep latency and its co-correlates were investigated; secondly those subjects who had discrepant MWT mean sleep latency and ESS scores were examined.

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**Table 2** Multiple linear regression modelling of MWT mean sleep latency

<table>
<thead>
<tr>
<th>Variables</th>
<th>MWT (DV)</th>
<th>Age</th>
<th>SlpD</th>
<th>&gt;4% desat</th>
<th>beta</th>
<th>% Unique variance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>0.24</td>
<td>–</td>
<td>0.23</td>
<td>4.8</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SlpD</td>
<td>0.19</td>
<td>−0.12</td>
<td>0.22</td>
<td>4.8</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&gt;4% SaO(_2) Dips</td>
<td>−0.21</td>
<td>−0.10</td>
<td>0.03</td>
<td>−0.18</td>
<td>3.1</td>
<td></td>
</tr>
</tbody>
</table>

\(R^2 = 0.13; \text{adjusted } R = 0.11; R = 0.36 (P < 0.01).\)

SlpD, prior subjective sleep history from the sleep diary (min); >4% SaO\(_2\) Dips, number of oxygen desaturations >4% per hour of sleep during the overnight PSG.

**Table 3** Subjects \((n = 146)\) divided into two age groups (median age split)

<table>
<thead>
<tr>
<th></th>
<th>Younger subjects</th>
<th>Older subjects</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>23–47 years</td>
<td>49–80 years</td>
</tr>
<tr>
<td>Mean SD</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SlpD*</td>
<td>434.6 66.5</td>
<td>407.1 53.0</td>
</tr>
<tr>
<td>TST**</td>
<td>332.6 59.0</td>
<td>308.6 66.0</td>
</tr>
<tr>
<td>ArI (NS)</td>
<td>20.6 8.7</td>
<td>21.8 9.8</td>
</tr>
<tr>
<td>AHI (NS)</td>
<td>15.5 8.1</td>
<td>15.4 8.7</td>
</tr>
</tbody>
</table>

\(*P < 0.05, **P < 0.001.\)

Subjects whose age was at the median (48 years) were omitted from the analysis.

NS, no significant difference; AHI, Apnoea/Hypopnoea Index; ArI, Arousal Index; TST, total sleep time from overnight PSG.
The score the slower the response; ms); PVT lapses, number of scores (1/RT) on the psychomotor vigilance task, i.e. the smaller vigilance task slowest scores (mean of the reciprocal slowest 10% psychomotor vigilance task scores (ms); PVTs10, the psychomotor median psychomotor vigilance task score (ms); PVTf10, 10% fastest theProfile of MoodStates–TotalMoodDisorderScore;medianPVT, Profile of Mood States – Depression and Dejection Scale; total POMS, Scale; POMSv, the Profile of MoodStates – Vigor Scale; POMSdd, the Fatigue Scale; POMSta, the Profile of Mood States – Tension–Anxiety BDI, BeckDepressionInventory; POMSf, the Profile of MoodStates– Fatigue Scale; POMSta, the Profile of Mood States – Tension–Anxiety Scale; POMSv, the Profile of Mood States – Vigor Scale; POMSdd, the Profile of Mood States – Depression and Dejection Scale; total POMS, the Profile of Mood States – Total Mood Disorder Score; median PVT, median psychomotor vigilance task score (ms); PVTf10, 10% fastest psychomotor vigilance task scores (ms); PVTs10, the psychomotor vigilance task slowest scores (mean of the reciprocal slowest 10% scores (1/RT) on the psychomotor vigilance task, i.e. the smaller the score the slower the response; ms); PVT lapses, number of RT ≥ 500 ms; PVT errors, number of psychomotor vigilance task errors.

MWT mean sleep latency predictors and co-correlates

The regression analysis, in first part of the study, found that age, previous sleep history (SlpD) and the number of > 4% SaO2 Dips were significant independent predictors of MWT sleep latency. We considered that sleep fragmentation would probably influence MWT sleep latency but interestingly, neither ArI or AHI were found to be independent predictors. A similar finding has been reported previously (Bennett et al. 1998).

Based on earlier reports (Bonnet and Arand 2000b), we considered that heart rate, a measure of physiological arousal, would likely be associated with MWT sleep latency (e.g. higher heart rate less objective sleepiness as measured by MWT). Heart rate did not, however, meet the statistical criteria for inclusion in the regression analysis. Indeed no relationship was found between MWT sleep latency and HR. At first this appeared surprising, as previous studies have shown that a higher heart rate is predictive of long sleep latency on MWT (Bonnet et al. 2000b) and MSLT (Bonnet et al. 2000a). However, the subjects in these earlier studies differed from ours in that they were younger and had insomnia, a condition associated with hyperarousal and long MWT and MSLT sleep latency.

Our findings are in agreement with an earlier study (Dohrmanji et al. 1997) which found a positive correlation between age and MWT sleep latency. In our study, older individuals appeared to be able to maintain wakefulness for a longer period and conversely, younger people appeared sleepier. Individuals aged < 48 years had significantly more sleep on both their PSG TST and sleep diary (subjective report of total sleep time in the week before the MWT) than did older subjects. Furthermore, younger individuals did not have more sleep disordered breathing or fragmented sleep. However, younger subjects, while obtaining more sleep might still not be meeting their sleep need and could therefore be sleep restricted. The fact that older individuals had longer mean sleep latencies may be the result of their difficulty in initiating sleep or that they are more able to maintain wakefulness in soporific conditions, whether because of experience or motivation.

It has long been established that the amount of prior sleep affects MWT sleep latency (Harma et al. 1998) and MSLT (Devoto et al. 1999). It is not surprising therefore, that in our study the subjective measure of the previous week’s sleep time was predictive of MWT sleep latency. It suggests that subjects who were sleep restricted found it more difficult to maintain wakefulness in a soporific environment.

The third variable found to independently predict MWT sleep latency was the number of > 4% Dips in SaO2 during sleep. Many studies have examined the effect of hypoxaemia on daytimes sleepiness and found conflicting results (e.g. Cheshire et al. 1992; Colt et al. 1991; Furuta et al. 1999; Kessler et al. 2001; Roehrs et al. 1989; Verstraeten et al. 1996). The relationship between hypoxaemia and MWT sleep latency in this study is small and only contributes 3% of unique variance. However, this finding adds weight to the argument that nocturnal hypoxaemia may play a small but significant role in the aetiology of daytime sleepiness.

These three variables explained less than one-sixth of the variability in MWT sleep latency scores leaving a large component of variance unexplained. This may in part be the result of the truncated nature of the MWT sleep latency score. When a variable’s range of scores is restricted in some way, the resulting magnitude of the correlation coefficient is reduced,

<table>
<thead>
<tr>
<th>Variables</th>
<th>MED (DV)</th>
<th>PVT Errors</th>
<th>BDI</th>
<th>AHI</th>
<th>beta</th>
<th>% Unique variance</th>
</tr>
</thead>
<tbody>
<tr>
<td>PVT errors</td>
<td>0.24</td>
<td>–</td>
<td>–</td>
<td>0.24</td>
<td>5.9</td>
<td></td>
</tr>
<tr>
<td>BDI</td>
<td>0.22</td>
<td>−0.01</td>
<td>–</td>
<td>0.22</td>
<td>5.0</td>
<td></td>
</tr>
<tr>
<td>AHI</td>
<td>0.21</td>
<td>0.05</td>
<td>0.18</td>
<td>–</td>
<td>0.16</td>
<td>2.5</td>
</tr>
</tbody>
</table>

Table 5 Multiple linear regression modelling of MED score

\[ R^2 = 0.13; \] adjusted \[ R = 0.12; \] \[ R = 0.37 \ (P < 0.01). \]

PVT errors, number of psychomotor vigilance task errors; BDI, the Beck Depression Inventory; AHI, Apnoea/Hypopnoea Index.
underestimating possible relationships between factors. It is a limitation of all research using MWT sleep latency and would only be resolved if the subject population were exceptionally sleepy (thus lowering sleep latency scores) or if there were no time specific end point to the test. While only a small amount of the MWT mean sleep latency is associated with the factors investigated here, it does not mean that the test has any less validity. The list of variables measured in this study are by no means exhaustive and other factors, such as motivation, personality or individual physiological arousal, are very possibly important determinants of MWT sleep latency. In addition, individual differences in our vulnerability to sleepiness and effects of sleepiness (Van Dongen et al. 2003), which may be caused by a genetic predisposition, could indeed affect our ability to maintain wakefulness. Therefore, rather than dismissing the MWT as a test lacking in validity, more research is needed to pinpoint the factors affecting sleep latency.

The investigation of the potential co-correlates with the MWT sleep latency found that self-reported sleepiness and psychomotor vigilance had the highest correlations. Both trait and state daytime sleepiness was measured in this study. ESS (trait sleepiness) correlated with MWT sleep latency. Several previous studies have found a relationship between ESS and MWT (Johns 2000; Sangal et al. 1999a,b). It was not entirely surprising that psychomotor vigilance task was a significant correlate of MWT sleep latency. Dinges et al. (1997), in their extensive examination of PVT during a sleep restriction protocol, compared results with Carskadon and Dement (1981) MSLT mean sleep latency scores and found they correlated at \( r = -0.95 \) (\( P < 0.001 \)). Such a relationship was likely to also exist with MWT sleep latency and in fact we have shown that PVT and MWT sleep latency scores were moderately correlated. In general, performance tasks are considered a probe of central nervous system capability and therefore reflect the slowing of processing which occurs when an individual experiences sleepiness (Dinges et al. 1997). It therefore follows that slower responses on the PVT would indicate sleepiness and give an indication of potential MWT performance.

**Discrepancy between ESS and MWT mean sleep latency**

The second part of this study examined the subjects who had discrepant MWT results and ESS scores. A new ‘discrepancy’ variable (MED) was created to investigate potential relationships to other variables. Subjects with low MED scores may potentially be at greater risk of sleep episodes while driving or at work because of a relative inability to detect sleepiness and thus take countermeasures. Subjects with high MED scores may present clinically with major symptoms of sleepiness that might be a safety concern and lead to unnecessary testing.

In our study population 35% of the ESS and MWT scores were discordant. The multiple regression found, that depression, more severe sleep apnoea and a tendency to errors on a psychomotor vigilance task (impulsive, unprompted button presses) were associated with a higher MED score. While the level of depression reported here is of marginal clinical significance, these scores are higher than would be expected in the general population. Depression and ESS have previously been found to be moderately correlated (Whitney et al. 1998). Thus, in patients with high MED scores, self-reported sleepiness may be exaggerated because of the subjects’ overall feelings of dissatisfaction regarding their health. The same depression may also increase MWT sleep latency. It has been previously found that individuals suffering depression have increased daytime alertness although they have more disturbed sleep (Kayumov et al. 2000).

The tendency for subjects with high MED scores to have increased PVT errors may indicate an underlying increased physiological arousal and drive to maintain performance in the presence of sleepiness. This has been observed previously in subjects undergoing experimental sleep deprivation protocols (Doran et al. 2001). There was no association between markers of arousal (i.e. heart rate, PVT reaction time) and MED scores. It would seem unlikely therefore, that high MED scores were because of a relative prolongation of MWT sleep latency because of hyperarousal.

In conclusion, the MWT is a complex behavioural test. We have shown that age, prior sleep history, and >4% SaO2 Dips are significant predictors of MWT sleep latency, and account for 12.8% of the variance in the test. Most of the variance in the test remains unexplained and further study is needed to identify factors mediating MWT sleep latency. Approximately, one-third of the subjects had discordant ESS and MWT results. Subjects with high MED scores appear to have more sleep disordered breathing, more depression, and make more psychomotor errors than subjects with concordant results.

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**REFERENCES**


