

ASSESSING THE DROWSINESS OF DRIVERS

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(Commissioned by VicRoads, Australia)

2001

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1.0 Summary

This is a review of psychophysiological evidence relevant to the problem of drowsy drivers who “fall asleep at the wheel”. Although wide-reaching, it is not an exhaustive review. It does not include biochemical and hormonal changes at sleep-onset. It focuses rather on several points of view that I believe are potentially helpful, as well as others that have been tried in the past and which I believe are not very helpful. Several conceptual and methodological problems are identified that are impeding our progress in this field. They relate particularly to the currently accepted model of sleep and wakefulness, and to the definitions of sleep stages by Rechtschaffen and Kales (1968) that are quite inadequate to describe drowsiness as a fluctuating state between alert wakefulness and R&K stage-1 sleep.

An understanding of the anatomy, physiology, and movements (both normal and abnormal) of the eyes and eyelids can explain their role in intentional and unintentional sleep-onset in a way that has not been described before. The concept of a microsleep, which is a brief period of stage-1 sleep with theta-waves dominating the EEG, is useful but does not explain many episodes of performance failure by drowsy subjects. Many such “lapses” are associated with alpha-waves in the EEG when the eyes are open. These waves would normally be blocked when the eyes are open in an alert subject. The “lapses” evidently involve the impairment of visual perception, especially at the periphery of the visual field, and an inability to maintain attention. Most cognitive processes are slowed down by drowsiness.

In the drowsy state, a driver who is striving to stay awake can fall asleep with his eyes open. This poses a potential problem for methods of monitoring drowsiness that rely solely on the duration of eyelid closures and blinks, such as PERCLOS. The recognition of slow eye movements that are unique to the drowsy state is a promising new method. However, much more research is needed.

2.0 Introduction

In order to understand and then to solve the problem of the drowsy driver who crashes his vehicle because he has “fallen asleep at the wheel,” it is necessary first to understand the whole process of falling asleep. For something we all do at least once a day on average, and which is so important to our health and well-being, there has been relatively little research on falling asleep compared with other aspects of sleep. One cause of this neglect may be that we have not have a word for this process in the English language. I introduced the term sleepening in 1990 but that term has not been widely recognised (Johns, 1990). Perhaps we ignore something for which we do not have a name. However, some other languages such as French, Spanish and Italian have always had such a word.

In this review I have not outlined the evidence that shows how important drowsy driving is as a public health and safety issue. That is assumed. Rather, I have gathered the evidence that relates to the problem of how best to assess the behavioural state of an active person such as a truck driver who intends to stay alert so that he can drive safely, but whose drowsiness varies involuntarily along a continuum of states between alert wakefulness and sleep. This has been an objective of a variety of researchers for at least 25 years. Many would advocate, as I do, that we need new methods and a device for monitoring some drivers such as long-haul truck drivers, particularly those driving at night. However, the same methods would potentially be much more widely applicable among other drivers, as well as aeroplane pilots, train drivers, ship’s captains and sedentary industrial workers.

3.0 Current Problems with the Assessment of Drowsiness in Drivers.

The fact that there is no monitoring device commercially available yet to monitor drowsiness is testament to the difficulties involved. There are several important conceptual and methodological problems that have not been adequately addressed and which, in my opinion, are impeding our progress. The major problems are summarised below.

3.1 The conceptual framework that is needed to understand sleepening is currently very muddled (Johns, 1998) (see appendix). There is confusion among such terms as sleepiness, drowsiness and fatigue. This is due in part to the fact that separate research disciplines have investigated these problems from different points of view that are not entirely compatible. The distinction between drowsiness and fatigue is discussed further in Section 4.0.

3.2 There have been rapid improvements in our understanding of sleep and its disorders over the past few years, particularly since polysomnography (overnight sleep monitoring) became readily available for routine clinical use, not just for research. This produced a focus of interest on what we might call intentional sleepening that involves lying down purposely in bed with the intention of sleeping. An important review of intentional sleep onset by Ogilvie (2001) appeared after this report was drafted. It has been assumed by some people that intentional sleeping is the same as unintentional sleepening, as can occur while sitting and watching TV or driving a vehicle. There are important differences, as we shall see.

3.3 The definitions of sleep stages by Rechtschaffen and Kales (1968), called R&K stages hereafter, were based on the electroencephalogram (EEG), the electro-oculogram (EOG) and the electromyogram (EMG), and were almost universally

adopted by sleep researchers after 1968. They have been very useful for assessing the structure of a night's sleep, particularly from the point of view of the differences between REM and non-REM sleep. However, by the R&K definitions, stage-1 follows wakefulness. Consequently, many people have been led to believe that sleep onset begins with Stage-1. Brief periods with theta waves in the EEG that may only last for a few seconds have been called microsleeps. Within the conceptual framework of the R&K definitions an EEG dominated by alpha-waves shows the subject to be awake, and there is assumed to be a strong dichotomy between being awake and being asleep. This is far from true, as we shall see. For example, slow or rolling eyes movements (SEMs) usually begin in the drowsy state several minutes before R&K stage-1 sleep. I agree with Ogilvie (2001), whose work is so important in this area of sleep research, that wakefulness and sleep are separated by a sleep onset period (SOP) that involves a continuum of drowsy states, not a single or unvarying state. In the context of the drowsy driver, we are more interested in when wakefulness that is compatible with safe driving ends rather than when sleep begins.

3.4 The objective assessment of sleepiness, in the sense of sleep propensity, has been dominated for more than 20 years by the Multiple Sleep Latency Test (MSLT) which the American Sleep Disorders Association (later the American Academy of Sleep Medicine) has endorsed (Thorpy, 1992). Sleep is said to begin in the MSLT with the first 30 sec of R&K stage-1 or any other sleep stage. Until then, the subject is said to be awake. The MSLT has been promoted as a test of a subject's general level of sleepiness, not just of sleepiness in the particular test situation (Chervin et al, 1995). This has led many people to believe that the mean sleep-onset latency in the MSLT is an accurate measure of a subject's sleepiness in daily life, including sleepiness while driving. This belief is based on an assumption that I have argued against and which I believe is wrong (Johns, 2000c). There is evidence that the mean sleep latency in the MSLT is but one of many situational sleep propensities. Each has one component that is specific to the test situation and another that is specific to the subject's response to that situation. In fact, assessments of the sensitivity and specificity of different methods for measuring sleepiness have shown that responses to a questionnaire, the Epworth Sleepiness Scale, are more accurate than the MSLT in distinguishing the long-term excessive daytime sleepiness of narcoleptics from the sleepiness of normal subjects (Johns, 2000a). I have also shown that a subject's sleep propensity measured in one situation cannot be relied upon as an accurate predictor of sleepiness measured in a different situation (Johns, 1994; 2001). This is a reason for believing that if a driver's drowsiness is to be measured at all, it should be monitored continuously, not just measured at other times when he is not driving.

3.5 For more than a decade the most widely accepted model of sleep and wakefulness has been that of Borbély and his colleagues (Borbély et al, 1989). This involves two independent processes, called process-C and process-S. Process-C provides a circadian influence on sleepiness (i.e. of sleep propensity). The phase of the intrinsic rhythm of its activity is set mainly by the timing of daylight and dark, mediated by the optic nerve and the suprachiasmatic nucleus in the hypothalamus. As a result, melatonin is secreted by the pineal gland, beginning in the early evening and continuing until about dawn. Process-S reflects the duration of prior wakefulness at any particular time. It increases rapidly at first and then more slowly during the period of wakefulness. It is discharged during sleep, quickly at first, then slower. While this model has been very useful, its promotion has been such that other important influences on sleepiness have been largely ignored. I have attempted to redress this situation by pointing out the added importance of postural, behavioural and situational influences on sleep propensity (Johns, 1994; 2001). These are so important that we cannot accurately measure sleepiness without reference to the

subject's posture, activity and situation in which it is being measured. I have called this characteristic of a situation its somnificity (Johns, 2001). In addition, there are substantial differences in sleep propensity between individual subjects under the same circumstances that are not predicted either from processes-C or S, or from the somnificity of the situation in which sleepiness is measured, or from any sleep disorders such as obstructive sleep apnea or narcolepsy that the subject may have. These individual differences between subjects may be partly inherited as a psychophysiological trait. Because of such differences, data that enable fairly accurate predictions of sleepiness at a particular time to be made for groups of subjects do not enable accurate predictions to be made for individual subjects. I have proposed a new 4-process model of sleep and wakefulness that addresses these issues as no other model does (see Appendix).

3.6 Despite these difficulties, there are methods available now that have been available for the past 25 years for monitoring a driver's sleepiness continuously. However, they are for research rather than routine use, and have not been adequately validated. Some that involve the attachment of electrodes to the driver to monitor the EEG or EOG etc are unlikely ever to be acceptable for routine use in the community. I believe the most promising methods involve new techniques for monitoring eye and eyelid movements. They have been the focus of my recent research. However, as we shall see, we simply do not yet know enough about episodes of "falling asleep at the wheel" or of sleeping in general. More research is urgently needed. In particular, we need an objectively measured scale that can tell us whether or not a driver is alert enough to drive safely at a particular time. The absence of such a scale is a major impediment to our progress.

4.0 Some Definitions

It is very important that we define what we think we are talking about in this context. It is regrettable that many researchers in this field have failed to do that, thereby confusing themselves and others.

Drowsiness or the drowsy state is an intermediate state between alert wakefulness and sleep as defined electro-physiologically by the pattern of brain waves (EEG), eye movements (EOG) and muscle activity (EMG), typically as measured under the chin. The level of drowsiness usually fluctuates in the drowsy state. It begins before the onset of Stage-1 sleep, but exactly at what point is yet to be established. The hypnagogic state is the same as the drowsy state.

The term sleepiness is used here in the sense of sleep propensity, or the probability of falling asleep at a particular time, i.e the chances of making the transition from wakefulness through the drowsy state and to Stage-1 sleep, whether or not sleep progresses to other stages subsequently. By this definition, sleepiness is neither a state nor a process. It is analogous to a position along a continuum of arousal states from alert wakefulness to sleep. Some researchers may refer to this as objective sleepiness. That is because, for some purposes, sleep propensity is measured by an objective test such as the Multiple Sleep Latency Test, and some people confuse the method of measurement with the nature of what is to be measured. By contrast, the Epworth Sleepiness Scale measures sleep propensity by subjective reports (Johns, 1991). However, subjective sleepiness is different, it is a measure of the presence and intensity of a set of feelings and symptoms that accompany the drowsy state. This can be measured by self-reports, for example by the Karolinska Sleepiness Scale (Åkerstedt et al, 1990).

The term fatigue is used here in two senses, one as a state involving the “subjective experience of tiredness and a disinclination to continue performing the current task”, as defined by Brown (1994). The second is as a process involving “those determinable changes in the expression of an activity which can be traced to the continued exercise of that activity”, as defined by Bartlett (1953). Or, put another way, the changes in performance of a task related to the duration of its continuous performance. We should also distinguish physiological or muscular fatigue from mental fatigue. These concepts arose from the psychology of work and performance, whereas sleepiness and drowsiness have arisen from a different discipline, the psychophysiological study of sleep. Unfortunately, many people still equate fatigue with sleepiness.

To help quantify sleepiness under different circumstances and within different time frames, I have introduced some new terms, as follows (Johns, 2000b):

Instantaneous Sleep Propensity (ISP): a subject’s sleep propensity at a particular time and place. This can vary widely over periods of seconds to minutes depending on the subject’s posture and activity, both physical and mental, the time of day, the duration of prior wakefulness, the effects of stimulant or sedative drugs, and the effects of sleep disorders such as obstructive sleep apnea or narcolepsy. There are important individual differences also that may be inherited. When normal people are active, going about their usual daily activities, their ISP will not be zero but it will be less than the critical ISP at which sleeping begins. However, many normal people will reach their critical ISP when they sit down at night, relax and watch TV. If the critical ISP is reached while driving, the driver will doze off or fall asleep, with the potential for catastrophic consequences.

Situational Sleep Propensity (SSP): a subject’s usual or habitual sleep propensity when in the same situation repeatedly. This depends to some extent on the somnificity of the situation in which it is measured. It includes a learned component, depending on the subject’s response to that situation. This tends to remain fairly constant over time. The MSLT measures one particular SSP in terms of the mean sleep onset latency in several naps under the same circumstances.

Average Sleep Propensity (ASP): a subject’s average sleep propensity across a variety of specified daily activities and situations. This remains fairly constant over periods of months, if not years. The Epworth Sleepiness Scale measures a subject’s ASP. A subject with a chronically excessive ASP will tend to doze under circumstances in which normal people usually do not.

I have shown that a measurement of a subject’s ASP or of a particular SSP is not very good as a predictor of his ISP in another situation, except among the minority of subjects such as severe narcoleptics who have an extremely high ASP. We cannot accurately predict the ISP of most people when they are driving at a particular time and place. However, such predictions are reasonably accurate for groups of subjects in whom the individual and situation-specific components of their sleepiness are averaged. This review is mainly concerned with the ISP of an active individual, such as a truck driver at work.

5.0 The Psychophysiology of Sleepening.

Sleep onset involves a wide variety of physiological and psychological changes that are anathema to safe driving. We can summarise much of the relevant knowledge about them under several headings. However, biochemical and hormonal changes in relation to sleep-onset are not included here. Then we shall probably be in a better position to consider various methods for assessing drowsiness in drivers.

5.1 The EEG and Microsleeps.

Changes in the EEG recorded continuously during sleep have been the mainstay of sleep research for more than 50 years. It is no wonder, therefore, that the EEG should be used to investigate sleepening.

When an alert subject closes his eyes the EEG changes and becomes dominated by alpha-waves with a frequency of 8-12 Hz, particularly at posterior (occipital) electrode sites (Fig. 1b). However, not all subjects generate obvious alpha-waves then. If sleepening progresses with the eyes closed, the occipital alpha-waves are joined or replaced by waves of similar or slightly lower frequency (7-11 Hz) that are generated more frontally and centrally, not at the occiput. These are then relaxed by theta-waves (4-7 Hz). Initially the theta-waves may be present in short bursts, lasting only a few seconds, alternating with periods of alpha-waves (Fig. 1c). Each short period of theta-waves lasting 1-10 secs is called a microsleep. R&K stage-1 is present when an epoch of the EEG (typically 20-30 seconds) is made up of more than 50% of theta-waves (Fig. 2a). In fact, R&K stage-1 is also characterised by low-amplitude delta-waves (0.5-4.0 Hz). It is not until the amplitude of the delta-waves increases to greater than about 50 microvolt, peak to peak, and their occurrence increases, to be joined by spindles and K-complexes, that R&K stage-2 is present (Fig. 2b). Then the proportion of high-amplitude delta-waves increases in R&K stage-3, and further again in R&K stage-4. REM-sleep involves the return of the EEG to the R&K stage-1 pattern, but with the appearance of rapid eye movements and the abolition of skeletal muscle tone as measured by the submental EMG (Fig. 2c). Recordings are usually scored in the sleep laboratory with epochs of 20 or 30 secs. However, in the context of a rapidly fluctuating drowsy state, much shorter epochs are preferred. (e.g 5 sec). We shall not be concerned further with the sleep stages after R&K stage-1.

The alpha-waves that appear when the eyes are closed usually disappear as soon as the eyes are opened. This disappearance is called alpha blocking. However, in a very drowsy subject, alpha-waves can appear in the EEG when the eyes are open. Alternatively, alpha waves may appear in an alert subject whose eyes are open but whose focus of attention has changed from the visual to a non-visual mode e.g. doing mental arithmetic or listening carefully to a conversation on a mobile phone. Under those circumstances, alpha-blocking may mean either that the subject has become more drowsy again, with theta-waves and a microsleep or more persistent sleep ensuing, or that he has become less drowsy (more alert), or alternatively that the focus of his attention has changed back from the non-visual to the visual mode, with the waking pattern of the EEG appearing (low amplitude, mixed frequencies). Thus, the context within which alpha-waves appear in the EEG and are then blocked is very important for their interpretation, particularly whether the eyes are open or closed at the time.

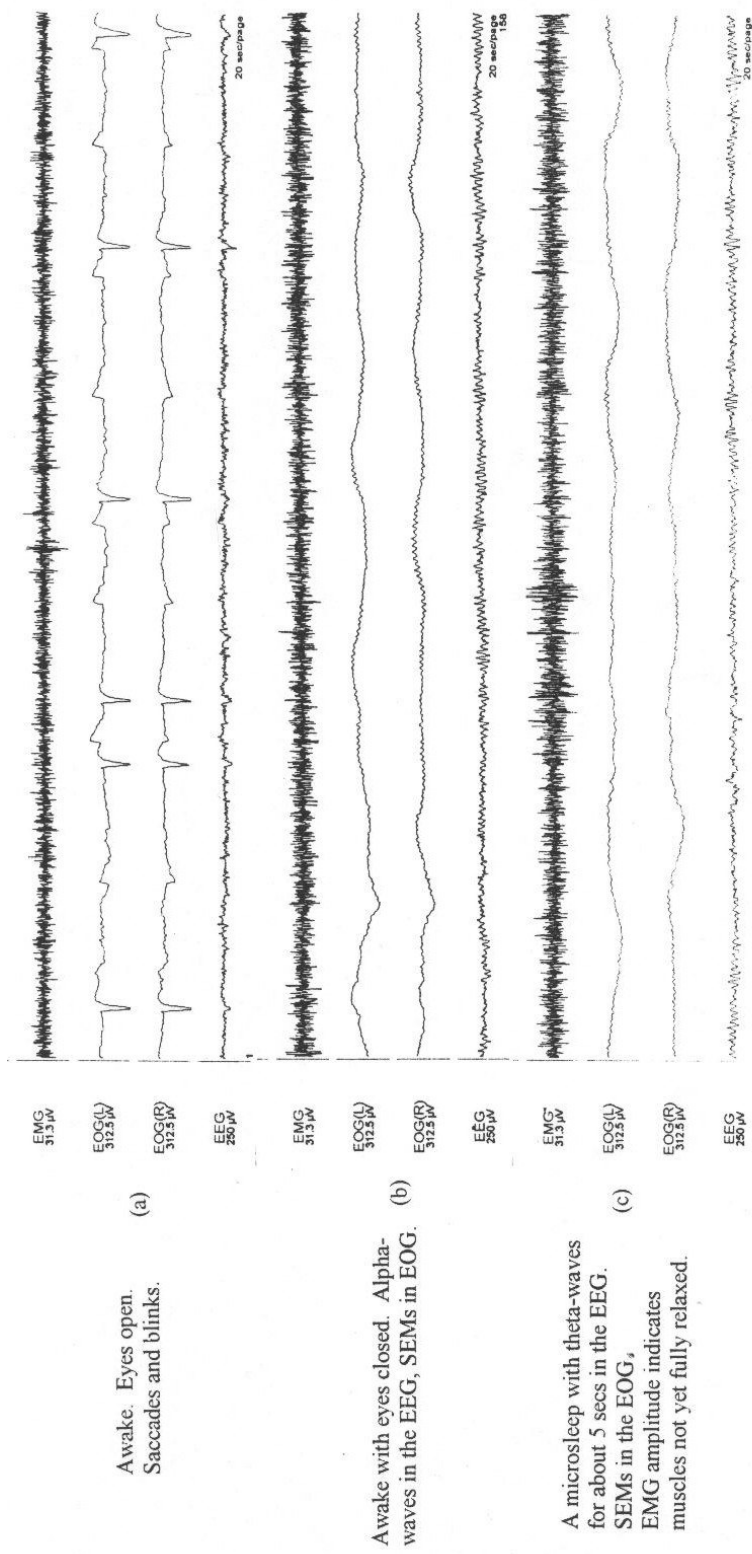


Fig 1 (a) (b) (c). The submental EMG, the left and right EOG's, and the EEG (C4-A1) (a) awake with eyes open (b) awake with eyes closed (c) drowsy with a microsleep. 20 sec. epochs.

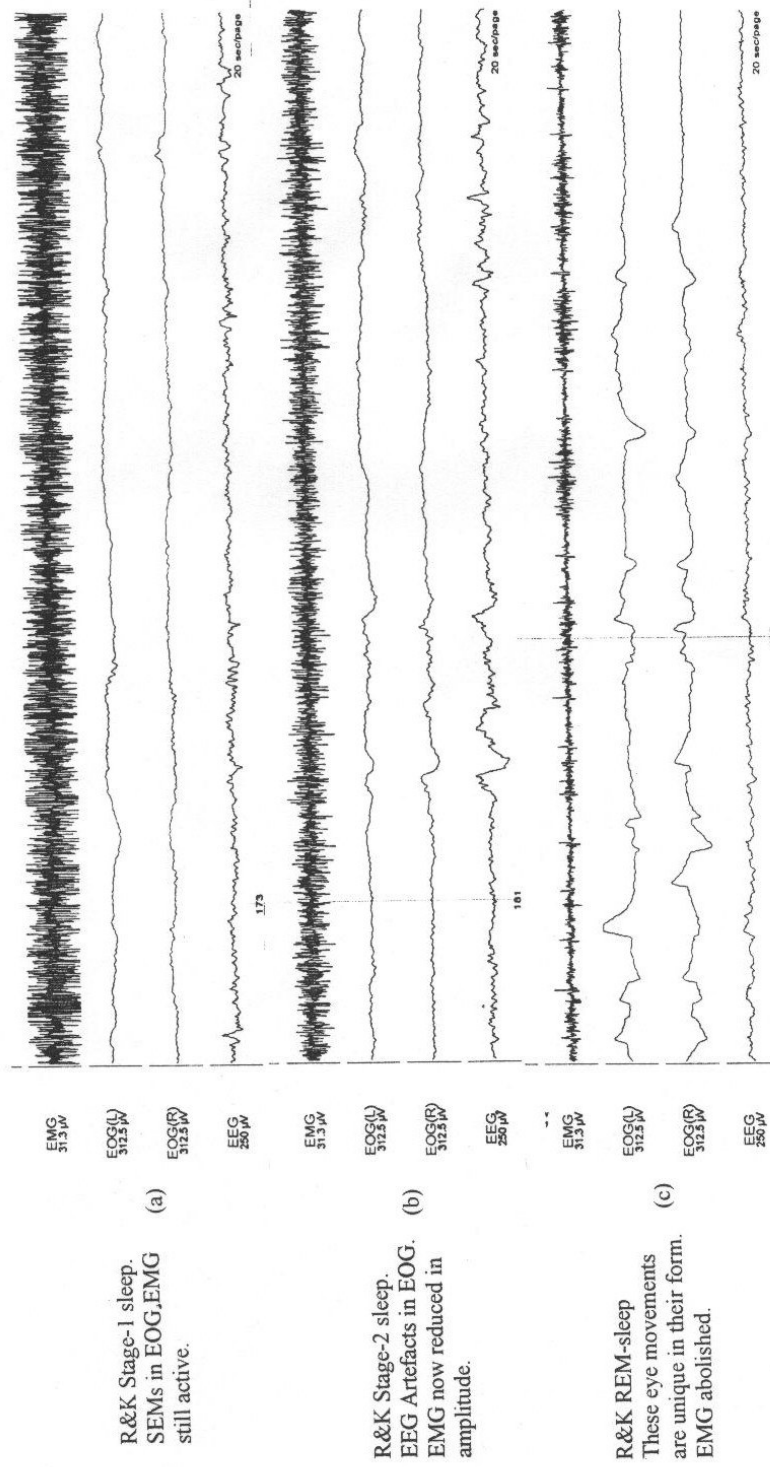


Fig. 2 a,b,c. The submental EMG, the left and right EOG's and the EEG (C4-A1) during (a) R&K Stage-1, (b) Stage-2, (c) REM-sleep. 20 secs epochs.

Valley and Broughton (1983) were among the first to investigate the EEG of the drowsy state in detail. Their experimental subjects were patients suffering from narcolepsy and chronic excessive sleepiness who were sitting down, trying to keep their eyes open while performing an auditory vigilance test. The experimenters subdivided Stage-1 sleep into two parts. Stage-1A involved slowing of the dominant frequency of the EEG by at least 1 Hz and the presence of a “fragmenting alpha rhythm intermixed with a medium voltage, mixed frequency pattern” and with “partial or definite slow rolling eye movements”. Stage-1B was dominated by theta-waves, with less than 20% of the time taken up by alpha-waves, but with vertex sharp waves and either slow rolling eye movements, or little or no ocular movement. The subjects’ performance in responding as quickly as possible to an auditory signal (a buzzer) by pushing a button was impaired in Stage-1A compared with the alert state, with slower responses and more frequent lapses with no response, compared with alert wakefulness. Their performance was much worse again in Stage-1B when there were few responses. Thus, performance was impaired in the drowsy state even before the occurrence of microsleeps with theta-waves in Stage-1B.

Hori and his colleagues (1994) pursued this EEG analysis in more detail. They distinguished 9 “stages” (referred to here as H-stages) between alert wakefulness (with eyes closed) and sleep (Fig. 3). H-stages 1 and 2 are equivalent to R&K -stage 0 (wakefulness). H-stages 3-8 are all part of R&K stage-1, and H-stage 9 is equivalent to R&K stage-2. There is usually a steady progression from one H-stage to the next during intentional sleep onset. The time taken to push a button after an auditory stimulus (the response time) increases progressively with these H-stages (Hori et al, 1991). By contrast, reports of visual imagery (hypnagogic imagery) increase in frequency from H-stage-1 to H-stage-5 but then decrease again in H-stage-9 (Hori et al 1994). The relevance of Hori’s analysis to drowsy driving is uncertain because his subjects had intentional sleep onset after lying down with their eyes closed. In addition, eye movements were not included in Hori’s analysis. The use of an auditory stimulus to elicit a behavioural response, both in Hori’s and in Valley and Broughton’s experiments may not be relevant to the driving task which relies mainly on visual, not auditory vigilance (see below). Nevertheless, Hori’s classification of EEG stages during the sleep onset period represents a major step forward in our knowledge.

Changes in the EEG during sleep onset have been investigated by power spectrum analysis with 1-Hz resolution using the Fast Fourier Transform (FFT) (Makeig et al 1993; Jung et al, 1997). The changes described visually in the past with traditional frequency bands (delta, theta, alpha, sigma, beta) have been refined in detail, but otherwise confirmed. The spectral power of the EEG at all frequencies increases progressively with prolonged wakefulness and sleep deprivation. The ratio of the spectral power of the EEG within the alpha frequency range (8-12 Hz) recorded from anterior (frontal) electrode sites compared with posterior (occipital) sites, is a useful new measure of a subject’s ISP (Hori et al, 1994).

The relationship between changes in the EEG and lapses in performance, whether during a psychomotor vigilance test or while actually driving, is discussed in more detail below.

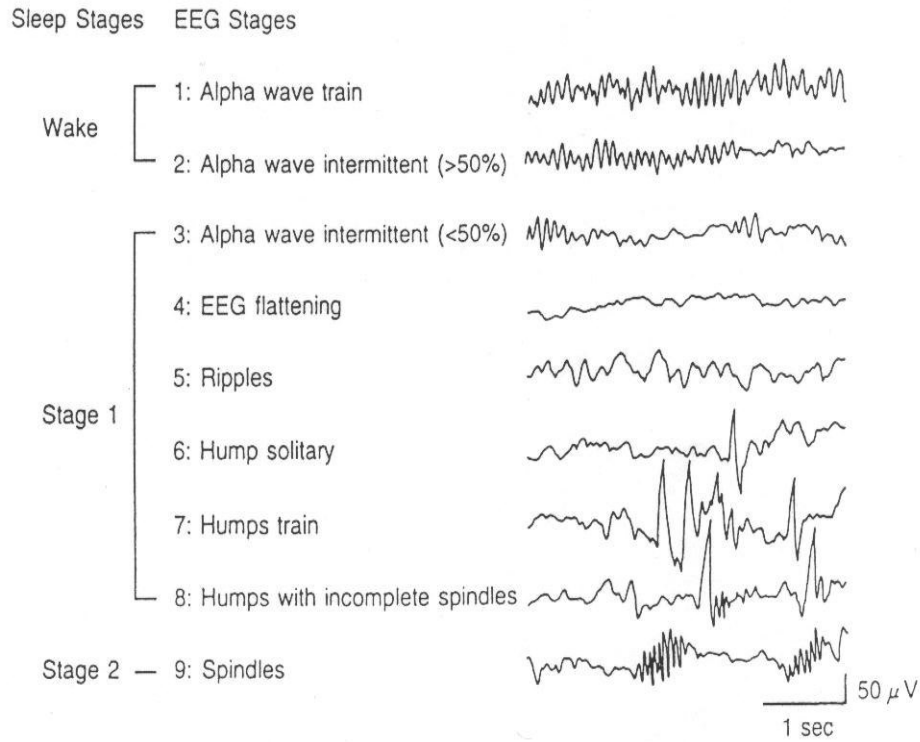


Fig 3. The EEG in each of Hori's 9 H-stages of sleep-onset (Hori et al, 1994)

5.2 The EEG and Event Related Potentials (ERPs)

Event related potentials (ERPs) are represented by changes in the waveform of the EEG that follow within about one second of an alerting stimulus. Typically the stimulus is either visual or auditory. The ERPs are very small and go on within the context of the background EEG pattern. They are separated from that background activity by repetition of the stimulus many times and the averaging of responses. Each wave is defined by its polarity (positive=P and negative=N) and by its latency of appearance after the stimulus (millisec). Ornitz et al (1967) were among the first to report changes in auditory evoked potentials during sleep onset. There are major changes in the presence or absence of particular waves, their amplitude and latency of appearance after the stimulus. These changes have been interpreted as involving changes in attention and in slowing of cognitive processing.

However, those changes have usually involved auditory evoked potentials in subjects who were falling asleep intentionally with their eyes closed. While they are very important in our understanding of sleeping in general, they are not entirely relevant to someone driving a truck, struggling to stay awake. Of greater relevance are the changes in visual evoked potentials reported in subjects who were sleep-deprived over a period of 40 hours and who were engaged in a visual vigilance task every two hours (Corsi-Cabrera et al, 1999). Their ERPs showed a progressive amplitude reduction and an increase in latency for almost all the late components as the period

of wakefulness become longer. The changes were correlated with the subjects' reaction times to visual stimuli and to impaired performance at a visual vigilance test. I interpret these changes in ERPs during sleep deprivation as reflecting a general slowing of all cognitive processes because of an increased sleep drive and/or a decreased wake drive. However, the recording of ERPs is not something that can be done easily on a driver in a moving vehicle. Indeed, it is difficult enough to record any EEG, much less ERPs without movement artefact under such circumstances (see below). It is unlikely that the recording of ERPs can be used to monitor the drowsiness of truck drivers routinely.

5.3 Movements of the eyes and eyelids.

Quite detailed discussions of the anatomy and physiology of the eyes and eyelids are included here because they are so important in any understanding of drowsiness. This information has not been included before in other discussions of drowsy driving despite the fact that many people, including me, believe that movements of the eyes and eyelids offer the greatest potential for a device to monitor drowsiness continuously (Dinges et al, 1998).

5.3.1 Recording Methods

Movements of the eyes and eyelids can be recorded in several ways. In the sleep laboratory, the EOG has been the standard method. It has also been used for research in some real-life situations, such as driving a truck (Kecklund, et al 1993), or a train (Torsvall et al, 1987). It requires electrodes to be attached to the skin beside the eyes. The EOG is usually recorded either with AC-coupled amplifiers using a relatively long time-constant, or preferably with DC amplifiers. Unfortunately, EOG recordings include as artefacts part of the frontal EEG as well as the EMG from peri-orbital muscles, which must be ignored during analysis (Fig. 2b). However, with appropriate amplifiers and placement of electrodes, the EOG can detect horizontal, vertical and oblique saccadic movements, as well as smooth pursuit movements and the slow, rolling eye movements (SEMs) of sleep onset (Fig 1b). Blinks and slower eyelid closures can also be detected and their duration measured.

Another method for measuring eye and eyelid movements involves a beam or pulses of infra-red (IR) light reflected back from the cornea, conjunctival surface of the sclera and the skin of each eyelid, and detected by nearby photodiodes (Leder et al, 1996). Such methods are usually restricted to the laboratory, but I have developed a new digital system for continuous monitoring while driving. Small IR transducers are attached to a half-spectacle frame that the driver wears comfortably, without the attachment of any electrodes. The data can be analysed on-line or stored for later retrieval and analysis off-line.

A quite different method involves two CCD video cameras mounted in a vehicle, pointing at the driver's face from the front at each side (Skipper, et al 1986). The duration and frequency of blinks and slower eyelid closures can be measured approximately, within the limitation of one image being stored about every 30 msec. This has been done recently in professional bus drivers (Hakkanen, et al 1999). Until now the recordings have had to be scanned visually after the event. It may be possible to automate the analysis in the future, but not yet. However, such video images form the basis of a promising, but still experimental method, called PERCLOS, for monitoring drivers' drowsiness. This is discussed in more detail below.

5.3.2 The Physiology of Eye Movements

In order to understand how movements of the eyes and eyelids change with drowsiness, we should first outline their normal occurrence and physiology. A series of extra-ocular muscles can move each eye in a variety of different ways so that an image of the object of attention falls on the fovea, the central and most sensitive part of the retina. The movements of each eye are coordinated to an extraordinary degree so that an image is focused on the retina and stabilised sufficiently for clear vision. This coordination must go on during head and body movements by means of vestibulo-ocular reflexes. An object moving in relation to the observer can be followed by smooth pursuit movements of the eyes provided the object is not moving too quickly. A vigilant subject's eyes undergo an almost continuous series of rapid saccadic movements that enable the visual field to be scanned for meaningful information (Fig. 1a). The periods of fixation in between last from a fraction of a second up to several seconds. The duration of individual saccadic eye movements and their maximum velocity depend on their angular displacement or amplitude (Schmidt, et al 1979). These are almost linear relationships for saccades up to about 20 degrees. Most saccades are of less than 15 degrees amplitude and take about 20-80 milliseconds to complete. Vision is inhibited centrally during each saccade (Matin, 1974). The binocular coordination of saccades is usually controlled within a few milliseconds. There are other movements of the extra-ocular muscles that change the alignment of each eye separately so that the two lines of vision point to the same target. This involves convergence for nearer targets and divergence to parallel lines for distant targets. In freely moving subjects, a shift of gaze by about 15 degrees or more will involve movement of the head as well as the eyes, in a coordinated way. The saccadic eye movement is followed by head rotation, and then there is a slower eye movement in the opposite direction as the head "catches up" with the eyes that have already fixated on their new target.

There are other, intrinsic muscles of the eyes that are smooth muscles, not striated as are the extra-ocular muscles. For example, the ciliary muscles change the shape of the lens in each eye to provide accommodation so that the retinal image is as sharp as possible regardless of whether the subject is looking at a distant or near point. The iris of each eye has dilator and constrictor muscles that change the size of the pupil according to the intensity of light at the eye, thereby controlling the amount of light reaching the retina. These muscles are controlled respectively by the sympathetic and parasympathetic divisions of the autonomic nervous system, opposing each other continuously. The adjustments are reflex in nature, but they are influenced by behavioural state and sleep onset. The eyelids are usually closed during sleep which provides a physical barrier to much of the light. However, the pupil is also reflexly constricted then, further limiting light entry. Upon awakening, the pupils dilate, even in bright light, which makes us squint under those circumstances to limit the environmental light entering the eye by alternative means.

In a drowsy subject the maximum velocity of a saccade with a particular amplitude is slower than in the alert state (Russo et al, 1998a; 1999a). However, to measure that maximum velocity it is necessary for the subject to make a series of saccades of known amplitude without head movement. This cannot be done safely while driving. Drowsiness also impairs smooth pursuit eye movements that can no longer track objects moving as quickly as was possible in the alert state. Rapid smooth pursuit movements tend to be replaced by multiple small saccades that do not usually occur immediately one after the other except in the drowsy state (Kleitman et al, 1951). The ability to maintain ocular fixation is also impaired during drowsiness (Miles et al, 1931). The usually tight binocular coordination of the two eyes in the alert state is

loosened so that diplopia occurs and the subject “sees double”. The slow, pendular eye movements from left to right (SEMs), each lasting several seconds, that appear in the drowsy state are unlike any others (Liberson et al, 1966) (Fig. 1b). They occur in most subjects when they are sleepening, but usually only after the eyes have been closed, so the movements are not commonly recognised. They are obvious to others once they have been pointed out. The fact that we are not aware of such major eye movements ourselves is good evidence that, when they do occur, our vision and/or our self-awareness has already been centrally inhibited (see below). We cannot see them but we are not aware of it. Another important aspect of the drowsy state is that fewer saccadic movements are made, particularly larger saccades (> 15 degrees) away from the centre of the visual field. Periods of ocular fixation last longer. There is some evidence that peripheral vision may be inhibited by neglect, an effect mediated centrally by the parietal lobe of the brain, enhancing the tendency towards tunnel vision (Russo et al, 1999b). Alcohol, sedative drugs and sleepiness all appear to have similar effects which I believe reflect a lower overall level of central nervous system activation as a result of a weaker wake drive. There have been attempts to quantify drowsiness solely on the basis of the pattern of eye movements recorded in the EOG (Varri et al, 1996), but this approach has not been adopted by others.

In the drowsy state, reflex control of the pupil becomes unstable, and its diameter fluctuates over periods of seconds to minutes, even when the environmental light intensity is constant (Lowenstein et al, 1963). Pupillometry has been used in the laboratory as a continuous measure of sleepiness (ISP) based on infra-red images of the iris and pupil (Wilhelm et al, 1998). Unfortunately this method cannot be used in an environment where the light intensity can change often and at a variable rate, because that influences the pupil too. Pupillometry is therefore not suitable as a continuous measure of the drowsiness of a truck driver.

5. 3. 3 The Physiology of Eyelid Movements

The eyelids perform several functions including lubrication of the cornea and conjunctiva, moistening it with tears, and wiping small foreign objects from it. The blink reflex provides protection from flying objects. There are three kinds of blinks: involuntary or reflex blinks in response to a stimulus; spontaneous blinks without a stimulus, and voluntary blinks (Shahani, 1970). Prolonged eyelid closures lasting longer than blinks can be voluntary, as when we choose to fall asleep, or involuntary when we fall asleep unintentionally. Closure of the eyelids reduces light input to the retina, an important initial step in the control of sleepening that reduces the secondary wake drive.

There is more than one layer in the thickness of the eyelids (Fig. 4). The outer muscle, just under the skin, is orbicularis oculi, shown in Fig (5a). This is a striated muscle that is part of the facial musculature supplied by the 7th cranial nerve (facial nerve), but which is sometimes thought of as another extra-ocular muscle. It contracts briefly during a blink, lowering the upper eyelid and moving the lower lid both upwards and medially towards the nose (Kennard et al, 1964). The activity of this muscle is opposed by that of another, levator palpebrae superioris, that is attached to the upper tarsal plate, a fibrous strap of tissue beneath the orbicularis oculi muscle in the upper eyelid (Fig. 5b). This muscle has its nerve supply from the 3rd cranial nerve (oculomotor nerve). However, it is a very unusual striated muscle in that its structure makes it very resistant to the physiological fatigue that striated muscles suffer from (Schmidtke et al, 1992). There is another small muscle, called the tarsal muscle or Muller’s muscle, that is attached to the same tarsal plate in parallel with levator palpebrae superioris. This is a smooth muscle whose contraction is beyond

conscious control and which does not suffer from fatigue either. Its nerve supply comes from the sympathetic division of the autonomic nervous system via the long ciliary nerves.

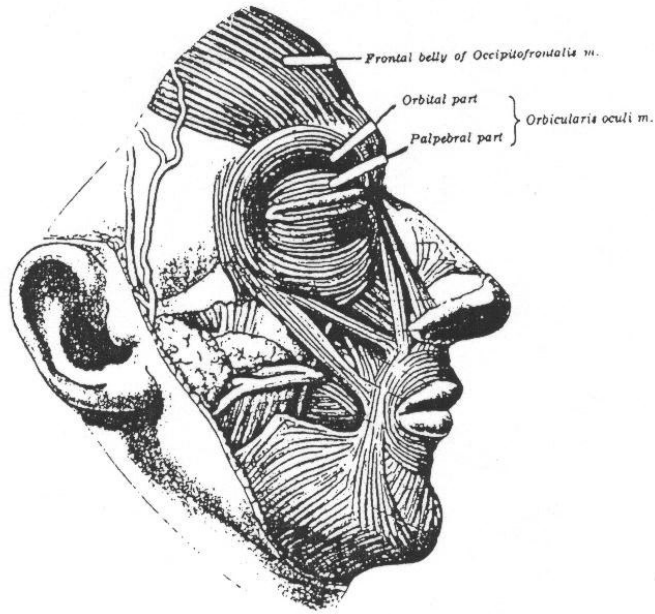


Fig 4. The facial muscles, including orbicularis oculi and frontalis muscles (after Gray's Anatomy).

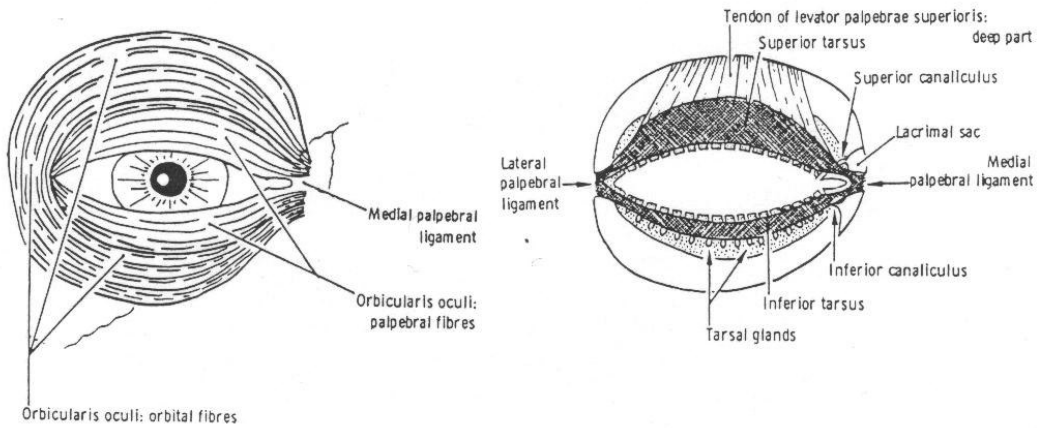
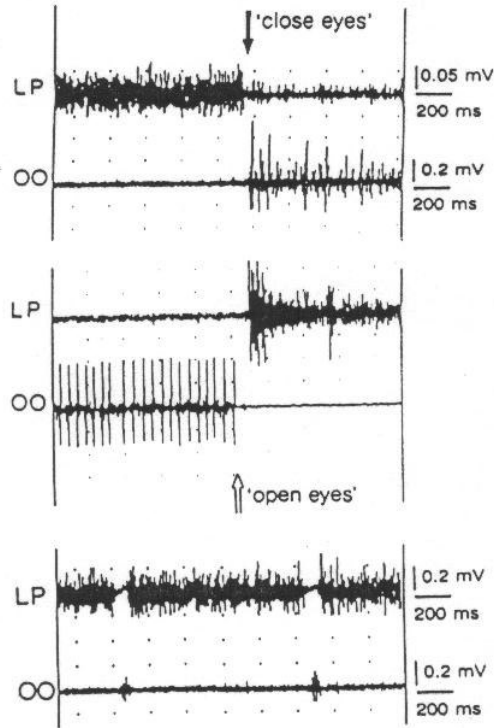


Fig. 5 (a) The orbicularis muscle in the eyelid

(b) The levator palpebrae superioris (including the tarsal muscle) attached to the tarsal plate in the eyelid.

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The EMGs from the levator palpebrae superioris (LP) and the orbicularis oculi (OO) muscles, showing the reciprocal inhibition between these muscles in a control subject. When the subject is asked to close the eyes gently, LP activity ceases abruptly, followed by contraction of OO (upper two traces at black arrow). Note the occurrence of dense bursts of action potentials with high amplitude preceding the return of LP activity on the order 'open eyes', following the inhibition of OO (lower third trace at open arrow). The lowest two traces show the total inhibition of the LP muscle activity and a brief contraction of the OO during the act of spontaneous blinking.

Fig. 6 The sequence of EMG activity recorded from levator palpebrae superioris and orbicularis muscles with (a) intentional eyelid closure, (b) intentional eyelid opening and (c) during a spontaneous blink.

Both levator palpebrae superioris and Muller's muscle are in a state of almost continuous contraction during wakefulness, except during blinks, so the "natural" position of the eyelids during wakefulness is with the eyes open. The combination of a smooth muscle and a striated muscle both subserving the same function, in this case the elevation of the upper eyelid, is unique in the body. I believe that the strength of involuntary contraction of those levator muscles is a direct reflection of the wake drive. There are very few such situations in the body where activity of the sympathetic autonomic nervous system is not opposed by the parasympathetic system, peripherally and centrally; cf. the control of the heart rate or the diameter of the pupil. There is one other situation similar to that in the upper eyelid and that is in the eccrine sweat glands of the palms of the hands and soles of the feet (see below). Both can play an important role in the assessment of sleep and wakefulness.

A blink starts with phasic central inhibition of the levator muscles, followed immediately by contraction of the orbicularis oculi muscles, controlled reflexly (Aramideh et al, 1994) (Fig. 6). The combined effect is to close the eyelids. The lids then open reflexly because the orbicularis oculi relaxes as a result of active inhibition and the levator muscles contract, raising the upper lid (Fig. 6). In an alert subject, reflex and spontaneous blinks usually last 100-300 milliseconds. As with saccadic eye movements, the duration of spontaneous blinks and their maximum closing velocity are both almost linearly related to their amplitude, i.e the degree of eye closure, which is not always complete (Evinger et al, 1991). The frequency of blinks during alert wakefulness varies widely between about 10 and 100 per minute and is affected by shifts of attention and by saccadic eye movements. Other upper eyelid movements, called lid saccades, are associated reflexly with vertical saccadic eye movements (Bauer et al, 1985). There is no equivalent eyelid movement during horizontal saccadic eye movements. Thus, vertical and horizontal eye movements are controlled differently.

Vision is inhibited centrally during blinks, but we are not aware of that temporary visual shut-down (Stern et al, 1984). By contrast, when vision is not inhibited, we can easily detect a light being switched off for 200 milliseconds. When the central inhibition of vision that is associated with each saccade is added to that with each blink, it is quite surprising to find that we cannot see for about 5-10% of the time normally, even though we are not aware of it.

In the drowsy state, the upper eyelids tend to droop and spontaneous blinks tend to last longer as the involuntary contraction of the tarsal muscle weakens, in line with a decrease in the wake drive. Blinks and other spontaneous eyelid closures eventually last 500 milliseconds or longer, the opening phase of lid movement slowing more than the closing phase (Lobb et al, 1986). However, there may be only partial eyelid closure with fluttering or wavering. The eyes become drier as the secretion of tears is inhibited, and the vasodilatation of superficial arterioles in the conjunctiva creates a grittiness that in popular parlance is described as the arrival of the "sandman". During intentional sleep onset these changes may go unnoticed because the eyelids have already been closed voluntarily. By contrast, in someone who is drowsy but who is trying to stay awake, drooping of the upper eyelid occurs initially because of a reduction in the spontaneous activity of the levator muscles, reflecting a reduced wake drive. This can be partially overcome by voluntary contraction of the levator palpebrae superioris muscle and later, if needed, by voluntary contraction of the frontalis muscles that raise the eyebrows and, to some extent, the upper eyelids. Various grimaces, squints, and other forced eyelid closures may be used in an attempt to alleviate ocular discomfort. These manoeuvres, along with changes of posture, can temporarily increase the secondary wake drive and, consequently, the

total wake drive. This is then reflected in the strength of contraction of the levator muscles, keeping the eyelids open, at least partially. However, to be effective, this strategy relies on the effort of almost continuous voluntary muscle contractions. If the wake drive decreases further, the sleep drive will become dominant and will begin actively to inhibit the remaining wake drive even further, and the eyelids will close involuntarily.

There is some evidence that vision may be actively inhibited as part of normal sleepening before spontaneous eyelid closure under such circumstances (see below). The subject would not be aware of what is happening then. A driver in this situation could be said to be “driving without awareness” (Brown, 1993). He would be seen to stare straight ahead or to have SEMs, but with his eyes open. Awareness of this “absence” would probably return when next he was roused by some stimulus such as the movement of his nodding head or the vibration of his vehicle as its wheels leave the edge of the pavement, and he would realise in retrospect that he had dozed off.

The frequency of blinks may be either increased or decreased in the drowsy state (Stern et al, 1984). Consequently, that frequency is not a very useful measure of sleepiness. By contrast, the duration of blinks and the frequency of blinks lasting more than 500 milliseecs has been used as a measure of sleepiness in bus drivers, based on the analysis of video images of the driver’s face (Hakkanen et al, 1999). Unfortunately, we do not yet have a scale with which to measure the risk to driving that is associated with such changes.

5.4 Muscular Activity, Movement and the Maintenance of Posture.

The activity of most skeletal muscles is related closely to the sleepening process. It has long been known that reflexes such as the patellar (knee jerk) reflex are inhibited during sleep (Bowditch et al, 1890). However, this has not been investigated in detail in relation to drowsiness. In my model of sleep and wakefulness (Johns, 1998) the phasic and tonic contractions of striated muscles involved in body movements and the maintenance of posture provide a powerful stimulus to wakefulness by feedback from their muscle spindle cells, and from the stretch receptors in ligaments, joints etc, via the thalamus to the wake-promoting system in the central nervous system - called the secondary wake drive in my model (see appendix). When we fall asleep intentionally, it is usually after we have chosen to lie down and partially to relax many, but not all skeletal muscles voluntarily. This can be considered a passive phase of muscle relaxation. The orbicularis oculi muscles are an exception to this because they must be contracted voluntarily to close the eyelids and thereby limit light entry to the retina at a time when we are relaxing other muscles.

Simply to lie down from the standing position automatically changes the spontaneous activity of the major postural muscles that otherwise must keep the head and trunk erect when we are standing and, to a lesser extent, while sitting. This reduces the secondary wake drive reflexly when we lie down. Other effects of this postural change are also important. For example, the firing rate from aortic baroreceptors decreases when the blood pressure that they are monitoring rises, as it does automatically when the position of the baroreceptors is lowered in relation to the heart when we lie down (Cole, 1989). This decreased baroreceptor feedback also decreases the secondary wake drive.

We will fall asleep soon after lying down if the total sleep drive exceeds the wake drive at the time. However, the voluntary phase of muscle relaxation alone does not usually produce profound relaxation. We may not fall asleep simply because we lie

down unless the sleep drive is also increased at the time. There is another, later phase of muscle relaxation during sleepening that is caused by an active inhibitory process from within the central nervous system. This may not begin until late in the sleepening process. It takes 2-5 mins to develop and continues into R&K stage-2 sleep. The amplitude of the EMG recorded from submental or facial muscles shows this clearly during routine polysomnography (Figs. 1&2). It is this active process that inhibits contraction of the levator palpebrae muscles and the tarsal muscles. This mechanism takes over from the voluntary contraction of the orbicularis oculi muscles as the means by which the eyelids usually remain closed during sleep.

Usually during wakefulness there is a constant interaction between the levator palpebrae superioris and the tarsal muscle on the one hand, trying to elevate the upper eyelid and, on the other hand, by the orbicularis oculi muscle trying to lower the upper eyelid. This can produce a tremor in the eyelid that can be monitored by a small strain gauge attached to the upper lid. The tremor evidently ceases with sleep onset. This has been used experimentally as an indicator of sleep onset in drivers, using a recording system called Nightcap that was developed originally for home monitoring of sleep in the investigation of sleep apnea (Stickgold et al, 1994). My own (unpublished) observations of muscle tremor, measured by a strain gauge over the nuchal muscles that help to hold the head erect, confirm that muscle tremor virtually ceases with sleep onset. However, a strain gauge will respond to any movement, not just to vibration or tremor. A strain gauge on the upper eyelid will detect all eye movements, such as horizontal or vertical saccades and, more particularly, SEMs in the drowsy state that would move the eyelid slightly because of the conical shape of the cornea moving beneath it. Thus, the Nightcap technique is probably detecting when all eye movements cease, including SEMs. As we have seen, this may not occur until late in sleepening. However, this method for monitoring drowsiness deserves more investigation.

A truck driver who is driving at 4 am, after being awake all day, has a relatively high secondary sleep drive because it has increased progressively during his continuous hours of wakefulness (Johns, 1998). He also has a relatively low primary wake drive because it is then at the low point of its circadian rhythm. The driver will be almost entirely dependent on his secondary wake drive to keep him awake then. Neither the voluntary nor the reflex phases of muscle relaxation will necessarily occur in that situation. Indeed, the driver may increase his muscle activity intentionally by changing posture repeatedly or by maintaining unusual postures as he drives in order to stay awake. It may not be until the active inhibitory phase of muscle relaxation begins and progresses, i.e not until after he has entered Stage-1, that his postural muscles will be so inhibited that they can no longer hold his head erect and the levator muscles of the upper eyelid can no longer keep his eyes open. If this scenario is correct, any attempt to use nodding of the head or prolonged eyelid closure (as distinct from slow blinks) as a warning of impending sleep onset will be doomed to failure. The driver may already have been unresponsive for many seconds, if not a few minutes, by the time his head nods forward and his eyelids remain closed. This problem would be exacerbated if, as seems likely, oculomotor control and even vision itself is lost before the active phase of skeletal muscle relaxation causes the head to nod forward. This was first described by Miles (1929) who pointed out that after prolonged wakefulness (sleep deprivation) we can fall asleep with our eyes open. This was confirmed by Åkerstedt's group in Sweden (Torsvall et al, 1998; Åkerstedt et al, 1990) who showed that, in sleep-deprived subjects, there were episodes of SEMs in the EOG and increased alpha and theta-waves in the EEG at times when the subjects were sitting up with their eyes open. Such episodes were associated with errors of omission in a performance test. However, these lapses were

distinguished from other performance failures due to dozing episodes when the head nodded forward and it was obvious the subjects had fallen asleep.

Some voluntary muscle activity and limb movements, repeated often enough to become “automatic”, can continue briefly after sleep onset. This was shown in an investigation of train drivers who had to activate a vigilance monitoring device repeatedly in order to keep their train operational (Fruhstorfer et al, 1977). The driver had to push a button within 2.5 secs of a light flash that came on 30 secs after his last response. If he failed to respond to the light, a buzzer was switched on. If he failed again to push the button within another 2.5 secs the automatic braking system of the train was activated. In fact, when their EEG and EOG was monitored while they drove their trains they were found to push the button at fairly regular intervals that were less than 30 secs, so preventing the flash of light or the buzzer from coming on at all. This button-pressing activity was continued “automatically” at times when the driver was clearly in Stage-1 sleep, while sitting up. One driver drove his train through a danger signal on the track under those circumstances, presumably because he did not see the signal (Fruhstorfer et al, 1977).

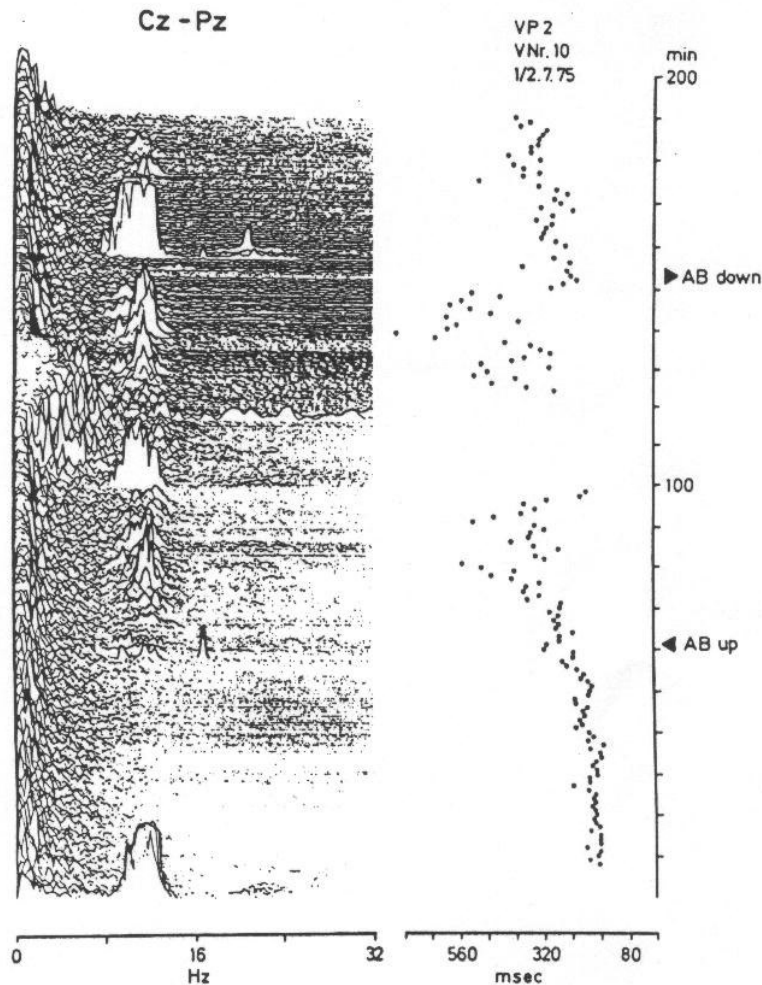
5.5 Vision and Other Sensory Systems.

It seems that all sensory systems are inhibited centrally to some extent during sleep onset. This reduces responses to mild or moderate stimuli while still allowing responses to intense stimuli. This is an aid to sleep onset and to sleep maintenance because it reduces the secondary wake drive without affecting the sleep drive. This applies to vision, hearing, touch, taste, smell, vibration etc. We have seen above how oculomotor controls are affected in the drowsy state, with impairment of the ability to focus and fixate the eyes, and to control the accuracy of eye movements, but vision is evidently affected separately. This was demonstrated in a simple way by Dement (1999) when he taped a sleep-deprived man’s eyelids open and intermittently flashed a strobe light into his eyes. The subject responded readily at first by pushing a button each time he saw a flash. However, at sleep onset he failed to respond to the flash and, when roused, denied that it had occurred. This kind of experiment needs to be repeated with more sophisticated methods to establish at what point in the sleep onset process vision is “turned off”, particularly with unintentional sleep when the eyes may still be open. Short-term memory is also inhibited then, which would impair the ability to report recent events, even if they had been registered visually.

Much of the experimental evidence about sensory systems in this context has involved the sense of hearing. Ogilvy et al (1988) presented their subjects with an intermittent auditory stimulus (a 1000Hz tone, 5 dBA above the background level) which lasted for up to 5 secs or until a mini-switch that was attached to the hand was pressed. They used the subject’s response times to investigate sleep onset and the presence of SEMs in the EOG. The mean response times increased significantly from wakefulness to Stage-1, and further again in Stage-2 when there were far fewer responses. SEMs began during wakefulness, just before alpha-blocking, and increased as drowsiness progressed, but then decreased after sleep onset. Thus, SEMs were virtually absent either when the subjects was alert and their response times were fast, or when they failed to respond or had very slow responses when asleep.

This experiment confirmed that a behavioural response with at least phasic muscle activity, sufficient to push a mini-switch, is possible in response to mild auditory stimuli at times when there are SEMs in the EOG. What we do not know from this is whether the subjects could or could not see then, and whether they would have responded as well to a visual stimulus as they did to an auditory one. Response to a

visual stimulus would be much more relevant to the driving situation. It may be that different sensory modalities are inhibited at different rates or at different times during sleeping. The presence of SEMs seems to be a very useful sign that sleeping has begun (Åkerstedt et al, 1990). However, the incidence of SEMs differs between subjects. We need far more research on the nature of eye movements when driving, particularly when drowsy. For example, we need to distinguish the SEMs of a drowsy subject from the smooth pursuit eye movements that can occur intermittently, or the vestibulo-ocular reflex movements that occur commonly in an alert driver while the vehicle is moving. Åkerstedt and Gillbert (1990) found that difficult to do in an active subject.



Comparison of EEG spectra and eye blink duration during a night driving experiment. Note the good correlation of the occurrence of alpha activity and long-duration eye blinks on the monotonous expressway. Nap between 100 and 120 minutes. The great alpha peak after leaving the expressway marks eye closure in front of red light.

Fig. 7 An array of EEG power spectra and the mean duration of blinks in a drowsy driver, driving on a highway and an autobahn (Fruhstorfer et al, 1977).

5.6. Sweat Gland Activity, Skin and Core Temperature, and Heart Rate.

Ecocrine sweat glands are distributed widely in the skin over most of the body. Most of them are concerned with thermo-regulation. However, the ecocrine glands of the palms of the hands and the soles of the feet are different. They secrete sweat in brief bursts in response to novel or alerting stimuli. Sweating in most area of skin increases during sleep, but in the palms and soles it is generally inhibited. However, there are usually brief bursts of sweating intermittently during non-REM sleep, too small to be noticed clinically, that are superimposed on a very low level of sweating. These changes in sweat gland activity can be monitored by measuring the electrical resistance of the skin by applying a constant current between two electrodes on the fingers or palms. An alternative method is to measure the potential difference between the skin of the palm and that of the back of the hand without applying a current. Measurements of skin resistance formed the basis of a simple method for monitoring sleep and wakefulness that I described 30 years ago (Johns 1971). Skin resistance varies with the activity of the sympathetic division of the autonomic nervous system without the opposing action of the parasympathetic division. Skin resistance (and potential) begins to change in the drowsy state, about 2-5 mins before R&K-stage-1 onset, when SEMs also occur (Hori, 1991). Unfortunately, the absolute values of skin resistance and of skin potential during wakefulness and sleep differ widely between individual subjects. Recordings are difficult to obtain without artefacts in active people, a problem that does not arise in the sleep laboratory. This makes such measurements of limited use for monitoring the drowsiness of drivers.

Thermo-regulation in general is heavily influenced by sleep and wakefulness. The blood supply to the skin is increased by vasodilatation that begins a few minutes before R&K stage-1, as with the inhibition of palmar sweating. As a result there is an increase in heat loss, so the core temperature falls at sleep onset. These changes and others such as the impairment of oculo-motor control discussed earlier, all of which precede R&K stage-1 onset, are good evidence for the importance of the concept of sleepening, a much broader concept than is allowed by the R&K definitions of sleep stages.

The heart rate usually decreases by a few beats per minute at sleep onset. However, in some people it increases or changes very little (Johns et al, 1976). The heart rate is controlled by the interaction of opposing influences, the sympathetic division of the autonomic nervous system speeding the heart up and the parasympathetic division slowing it down. At sleep onset, both divisions of the autonomic nervous system are inhibited to some extent, although by how much is uncertain. The end result in terms of the heart rate depends on which of the two influences was dominant before their inhibition. The heart rate is not a particularly useful measure of drowsiness, even though it has been used by some for that purpose in the past (O'Hanlon et al, 1977).

5.7 Mental Activity and Awareness

The changes in mental activity that accompany sleep onset have been described in some detail (Foulkes et al, 1965; Mavromatis, 1987). During sleepening there is a fairly sudden shift of focus away from awareness of the "here and now" to much less focussed and less structured thinking that is vague and disengaged. This is sometimes described as more primitive, regressive or child-like thinking. It usually begins within seconds of the initial alpha-blocking in the EEG, perhaps earlier under circumstances of sleep deprivation. There is impairment of volition so that voluntarily directed activities are limited. The ability to maintain focussed attention, so important for driving safely, is impaired. There is an increasing inability either to

recognise one's state or to do anything about it. Spontaneous sensory images (hypnagogic images) may appear that can involve any of the senses, but particularly the visual. There may be a vague awareness of coloured patterns and lights, or sometimes visual hallucinations, even with the eyes open. Whole imaginary scenes may be visualised, including people. There may be a sense of falling or of drifting. These images are in addition to misperceptions because of diplopia and impaired oculomotor control. As stated earlier in relation to Hori's (1994) investigations, the incidence of visual imagery increases during sleepening from Hori's H stage-1 to H-stage-5 and then decreases into H-stage-9, which is equivalent to R&K stage-2. Hori did not include SEMs in his classification, but we know from other investigations that the incidence of SEMs has a similar pattern of occurrence (Liberson et al, 1966; Hori, 1982; Åkerstedt et al, 1990). Perhaps the visual imagery and the SEMs are causally related. We need more research on this topic.

Some researchers believe that a drowsy driver is always capable of knowing when he is about to "fall asleep at the wheel", even though he may not recognise the danger of his drowsiness (Reyner et al, 1998). I disagree with that, and so do others (Brown, 1993). Peoples' awareness of their own behavioural state during drowsiness was investigated by Hori et al (1994). Subjects were asked to push a button as soon as they heard a 1Khz tone at 50dB which lasted for 5 seconds or until the button was pushed. They were also asked to say then whether they had been awake or asleep at the time they heard the noise. In the progression of states from H-stage 1 to H-stage 9 the percentage of responses that indicated being awake fell from 83% to 26%, those that indicated being asleep increased from 7% to 44%, and the times when there was no response increased from 10% to 30%. Even though there was some increase in the accuracy of self-perceptions of having been either awake or asleep as drowsiness progressed, they were never very accurate. Even when being roused from H-stage 9 (equivalent to R&K stage2), the majority of subjects could not tell that they had been asleep for a while. I interpret this in terms of an impairment of attention and of self-awareness that gets progressively worse and which parallels the slowing of reaction times.

However, Hori's subjects were lying in bed with their eyes closed. That is a very different situation from that of a truck driver struggling to stay awake. I believe that the majority of subjects who doze off while in the sitting posture, without their head being supported as it would be on a pillow in bed, can usually (but perhaps not always) tell that they have just dozed off after the event, when next they rouse. They realise in retrospect that their eyes have been closed, that there has been a gap in their awareness of events going on around them, or that their head has nodded forward i.e. they have exhibited dozing behaviour, much of which is observable by others. The ability to recognise and remember such dozing episodes in retrospect forms the basis of the Epworth Sleepiness Scale (Johns, 1991). The important point is, however, that we are generally not aware that we are dozing off as it happens. Our awareness arises later, in retrospect if at all, when we are more alert. This requires more investigation in the laboratory.

The ability of sleep-deprived subjects to predict whether or not they would fall asleep within the next two minutes has been investigated by Itoi et al (1993). Polygraphic and video recordings were made during repeated tests when the subjects were sitting in front of a computer screen with one hand on the mouse. Sleep was defined polygraphically as the presence of theta-waves for at least 3 seconds. i.e. of a microsleep at least. Sleep episodes were confirmed from the video by "eye closure, head nodding, and muscle relaxation".The subjects often fell asleep when they predicted they would stay awake, or stayed awake when they predicted they would

fall asleep. They were worst at predicting their first episode of sleep and improved somewhat with later episodes. There was wide variation between the abilities of individual subjects to predict either sleep onset or continuing wakefulness. Some were fairly accurate, others gave predictions no better than chance. As we shall see, the impairment of performance begins before the appearance of a microsleep. This may make predictions of performance failure even more unreliable.

The results of such experiments give little credence to the view that all we have to do to solve the problem of drowsy driving is to educate drivers to be aware of their drowsy state and its safety implications. I believe that many drivers would benefit from an unobtrusive device that could tell them continuously about their state of alertness/drowsiness and which would trigger an alarm when their drowsiness first reached a critical level when they were no longer fit to drive.

Despite the changes in mental activity and awareness during drowsiness, it appears that subjects can still respond briefly to environmental stimuli of sufficient intensity although their self-awareness and attention is generally impaired. This has been demonstrated in experiments mainly in relation to auditory stimuli that produce a brief arousal during which the subject may respond by pushing a button (Ogilvie et al, 1988). However, the response time is significantly longer and there are more occasions when there is no response (lapses, or errors of omission) compared with the alert state. In the driving scenario, a driver's arousal response to sudden new vibrations of his vehicle as it crosses the rumble-strip on the edge of the highway is an example of this phenomenon. However, his arousal response may be short-lived unless the driver is able to take further action, and in particular to have some sleep to reduce his sleep drive.

We have seen how sleepiness inhibits important attentional and volitional processes and the driver's awareness of the "here and now". This presumably also impedes his ability to change his behaviour voluntarily at the time, even when his situation involves the reality of an impending crash that would be obvious to an alert driver.

6.0 Vigilance and Lapses in Psychomotor Performance Tests.

The idea of using performance tests as a measure of mental and psychomotor ability has been around for a long time. Psychologists attempted for many years to explain the changes in continuous performance with time as a function simply of the process of fatigue. Then they realised the importance of sleepiness and a change of state in that process. However, they confused themselves by thinking that sleep onset was simply the end result of fatigue, a view with which I strongly disagree (Johns, 2000b).

Bills (1931) first described what he called "blocks" which were time gaps in a subject's repeated performance of a psychomotor vigilance test. Bills noted that errors tended to be associated with such "blocks". Bjerner (1949) later showed with concurrent EEG recordings of subjects with their eyes closed during a serial auditory reaction time test that these "blocks", or as he preferred to call them lapses, were associated with alpha-blocking and the brief appearance of theta-waves in the EEG. These events came to be called microsleeps that may last only a few seconds. The frequency and duration of these microsleeps increases during the sleep onset process until R&K Stage-1 sleep begins.

Williams et al (1959,1962) showed in a classic series of experiments that the frequency of these microsleeps and of lapses in performance increased with sleep

deprivation. At first it was thought that microsleeps explained all of the problem of impaired performance during sleep deprivation. However, Dinges and his co-workers (1988) showed that, while microsleeps explained many errors of omission (when the subject failed to respond) in psychomotor vigilance tests (PVTs), the overall performance level was also impaired in between lapses.

Åkerstedt and his co-workers in Sweden (Torsvall et al, 1988; Åkerstedt et al, 1990) showed that errors of omission in a visual vigilance task, with eyes open, were associated with an increase in alpha-waves in the EEG even more often than an increase in theta-waves and microsleeps at the time. SEMs were observed during these periods and those authors felt that SEMs may be the most specific sign of impaired performance, due to drowsiness, whether the eyelids were open or closed at the time. However, they found difficulty in detecting SEMs in the EOG sometimes because of other ongoing eye movements such as saccades and smooth-pursuit movements in active people.

Since then Risser et al (2000) have reported that lapses in a serial reaction time test done every two hours during 40 hours of continuous wakefulness were associated more often with bursts of alpha-waves than with microsleeps and theta waves in the EEG. They referred to those events as “attentional lapses” rather than microsleeps.

Whether the subject’s eyes are open or closed at the time has a major impact on such results. Thus, the relevance to drivers of these methods of EEG analysis may be limited. In addition, there are substantial differences in the EEG between subjects, whether alert or drowsy, that make the EEG by itself insufficient for monitoring drowsiness. When this difficulty is added to the special requirement of careful electrode attachment and the problem of movement artefacts in recordings made on an active subject, EEG monitoring is not likely to be useful for monitoring a truck driver’s drowsiness routinely.

The impairment of performance on a variety of PVTs because of sleep deprivation has been compared with the equivalent effects of different blood alcohol concentrations (Powell et al, 1999). After about 20 hours of continuous wakefulness, which under many circumstances coincides with 1 or 2 am after being awake all day, the performance decrement is comparable to that of a blood alcohol concentration in excess of the legal limit of 0.05% for a driver (Lamond et al, 1999). This has served to emphasise the importance of sleep deprivation and the time of day on driving, and to make these factors in road safety more understandable (Dawson et al, 1997).

7.0 Drowsiness during simulated driving tests.

Driving simulators, especially the most sophisticated of them, provide a test situation that approximates real driving much more closely than does a serial reaction time test with button-pushing responses to an auditory stimulus. Nevertheless, a driver in a simulator knows very well that he will not die or injure himself if he drives off the stimulated “highway” and hits a “tree”. We cannot simulate the effects of fear of injury or death on the driver. However, simulated driving experiments enable the effects of all levels of sleep deprivation and of drowsiness to be investigated, which would be very difficult otherwise. Wierwille’s group in Virginia (Dingus et al, 1987; Wierwille et al, 1994) demonstrated the early promise of measurements of drivers’ eyelid closures and of PERCLOS when they showed significant correlations between those measurements and lane deviation (mean and standard deviation) and velocity of steering wheel reversals in simulated driving, all of which were affected by sleep deprivation (see below).

Driving simulators have also been used to investigate the impaired performance of drivers who have excessive daytime sleepiness persistently because of narcolepsy or severe obstructive sleep apnea (George et al 1996). Such sleepy patients showed significant impairment of driving skills as a group compared with normal subjects. Yet half of the patients performed as well as the controls. The authors concluded that the degree of an individual driver's impairment could not be accurately predicted from assessment of his sleepiness by the MSLT, which some people consider to be the gold standard for measuring sleepiness. My analysis of the concept of sleepiness predicts such a result (Johns, 1998). The MSLT fails in this situation because it measures the subject's situational sleep propensity in one particular test situation, and this is not closely related to his sleep propensity in a different situation, such as when driving.

Risser et al (2000) have also shown in a simulated driving experiment that patients suffering from excessive daytime sleepiness caused by obstructive sleep apnea have impaired driving skills as measured by the variability of their lane position, speed, steering wheel movements, and "crash" frequency. The patients had more "attention lapses" than controls, as defined by the appearance in their EEG of bursts of either alpha or theta- waves lasting at least 3 seconds. Most lapses that led to a "crash" involved alpha rather than theta-waves and could not therefore be called microsleeps. Those authors concluded that the impairment to driving when drowsy was "not entirely due to overt sleep but to more frequent and longer attention lapses". This is consistent with the view expressed above about the importance of attentional deficits in drowsiness.

The same conclusion has been reached after detailed experiments on simulated driving by subjects who were either totally or partially deprived of sleep at the Walter Reed Army Institute of Research in Washington DC.(Thomas et al 1998; Russo et al 1998; Thorne et al 1999). Their EEGs were monitored while they drove on simulators. Over a period of 14 days with a total of 66 drivers there were 619 "accidents" such as "driving off the road". Only 8 such "accidents", or 1%, coincided with a microsleep and theta-waves in the EEG at the time. When the recordings made during the minute before each "accident" were analysed, only 14% of such incidents were preceded by a microsleep. However, the EEG characteristics that were associated more often with "accidents" were not reported. The conclusion was reached by those authors that "simulator driving accidents during drowsiness seldom result from falling asleep at the wheel, suggesting that most accidents may be due to sleep-deprivation-induced cognitive impairments such as inattention" (Thomas et al 1998). Although the EOGs were recorded during those experiments no analysis of eye movements was reported, which is a pity. However, such results and comments rightly emphasise the importance of inattention caused by drowsiness, even before microsleeps appear in R&K stage-1 sleep.

8.0 Observer Ratings of Drowsiness

The availability of small CCD video cameras has made it possible to make continuous video recordings with low levels of visible light or with invisible infra-red light. This technique has been used to monitor the face and eyes of truck drivers while they do their job of driving. Wierwille & Ellsworth (1994) described a 5-point rating scale from "not drowsy" to "extremely drowsy" based on a combination of video-recorded features that included the following: "mannerisms" such as rubbing the face or eyes, scratching, yawning, grimacing, as well as restless body movements, slower eyelid closures, decreased "facial tone", a "glassy-eyed appearance", staring

at a fixed position, SEMs, a “cross-eyed” look, and periods of general immobility lasting many seconds, punctuated by jerking movements, large steering wheel adjustments, and intermittent arousal associated with a nodding head. They found that raters could give reliable (in the sense of test-retest) assessments of drowsiness on this five-point scale when they reviewed the video recordings on play-back. However, there were some consistent differences in the ratings made by different raters.

This rating scale was, in my opinion, poorly defined and never adequately calibrated. Nevertheless, it was from that work that the method called PERCLOS was developed (Skipper et al, 1986; Wierwille et al, 1994). This is based on the percentage of time that the eyelids are at least 80% closed, as assessed by raters who view the recordings off-line. This appears to be a promising new method for monitoring drowsiness. Scores on the PERCLOS test vary in parallel with lapses in psychomotor vigilance tests done repeatedly during 42 hours of wakefulness (Dinges et al, 1998; Mallis et al, 1999). However, we know from other investigations and from Wierwille & Ellsworth’s (1994) own descriptions that some sleep-deprived subjects fall asleep while their eyes are still open. PERCLOS does not include any assessment of eye movements, whether of SEMs or of saccades, etc. which are potentially very important. By the time a driver’s eyes are mostly closed he may have been “driving without awareness” for several minutes. Combine this difficulty with the fact that the video assessments cannot be made on-line yet and we have reason to be cautious about the PERCLOS method.

9.0 Drowsiness while actually driving

Despite practical difficulties in recording the EEG and EOG in active subjects, even with today’s technology, it is interesting to find that such recordings were made successfully in Germany 25 years ago (Fruhstorfer et al 1977). They had drivers with instrumented cars driving on a highway and a motorway during the day and night. They used a video camera to record the driver’s face and eyes. When the drivers reported being “fatigued” (i.e. drowsy in this context) the EEG showed “phasic increases in theta activity” or “high-amplitude bursts of alpha activity often related to eye blinks of long duration”. These changes were clearly displayed in spectral arrays of EEG power versus frequency and time (Fig 7). The frequency of eye blinks did not change much but their duration did, some lasting longer than 500 milliseconds. To see these results today it is a little disheartening to realise how little progress has been made in this field over the past 25 years.

Kecklund and Akerstedt (1993) monitored the EEG/EOG of some long-haul truck drivers while driving in Sweden. Power spectral analysis of the EEG was done with epochs of only 7.5 secs after artefacts had been removed visually. The subjective sleepiness of the drivers, measured by the Karolinska Sleepiness Scale, increased at the end of the driving period, particularly in the last 2 hours in the early hours of the morning. There were significant correlations between the drivers’ subjective sleepiness and the power of both the alpha and theta waves in the EEG. Eye movements and blinks were not included in this analysis.

These results from drivers on the road are entirely consistent with those from laboratory studies using simulators. It is clear now that we cannot rely solely on the identification of microsleeps to explain the impairment of performance during drowsiness. Bursts of alpha-waves that may only last a few seconds are at least as important and are probably numerically more common than are bursts of theta-waves

during drowsiness. Similar results have been obtained in studies of train drivers, airline pilots, and industrial workers doing their usual work (Torsvall et al, 1989).

If any further evidence was required to evaluate the use of continuous recordings of the EEG/EOG and the presence of microsleeps as indicators of drowsiness in truck drivers, it was provided by the results of a large, expensive and detailed study of 80 long-haul truck drivers, driving their usual routes in the USA and Canada (Wylie et al, 1996). The study made recordings over 6 weeks, during which time the drivers covered 200,000 miles. Video recordings of the driver's face and eyes showed periods of drowsiness, judged by prolonged eyelid closures etc. that were associated with lane-drifting but not with any crashes. Video-detected drowsy episodes were present 600 times more frequently than was indicated by the presence of microsleeps in the EEG. No attempt appears to have been made to identify periods of alpha-waves in the EEG. The drivers' self assessments of their drowsiness by the Stanford Sleepiness Scale were not closely related to the objective findings of drowsiness. About two-thirds of the drivers had some drowsy periods, especially at night, but one-third did not. The majority of drowsy episodes (54%) were seen in a minority (14%) of drivers. Drowsiness was much more closely related to the time of day/night than to the duration of driving periods.

One other investigation of drowsy driving in Sweden is worthy of comment because it seems to be unique. Lisper et al (1986) asked 12 male university students to drive continuously on a 5-km lap on the runway system of an Airforce base until they had fallen asleep 3 times. The car had dual controls and a well-rested co-driver could take over the controls if necessary, but that was not needed. Seven of the 12 drivers fell "asleep" as judged by eyelid closure or head-nodding. Five drivers would not continue to drive after some time although they did not actually fall "asleep". Those who dozed off found it quite alarming. The mean interval of 24 mins between consecutive dozing episodes was not affected by a 5-minute brisk walk after they had fallen "asleep" each time. Unfortunately, this experiment did not incorporate adequate methods for monitoring drowsiness. However, it did incorporate an element of danger with the risk of a crash after "falling asleep at the wheel". Some subjects were not prepared to take that risk and withdrew from the experiment.

Detailed studies of the pattern of eye movements while actually driving are not common. In the past they have required cumbersome head-gear for the driver to wear. One important study by Kaluger and Smith (1970) was reported from Ohio more than 30 years ago. They studied only 3 subjects, each driving for 9 hours on an interstate highway with and without one night's sleep deprivation, which must have made it quite a dangerous experiment. The direction of the drivers' gaze and the pattern of eye movements were recorded many times for periods of about a minute under different driving conditions that were repeated. The predominant direction of gaze moved downwards and outwards to the edge of the road after prolonged driving when the drivers became drowsy. The researchers interpreted their results in terms of the impairment of peripheral vision during drowsiness and the need to use foveal (central) vision than for spatial and velocity information. This information would normally be collected from peripheral vision that takes in the high angular velocity of objects moving immediately in front of and to the side of the moving vehicle. Foveal vision would normally be reserved for the road ahead, with much lower angular velocities to be observed. We need far more research along these lines of investigation.

10.0 Conclusions

From the above summary of experiments and observations it is possible to draw some relevant conclusions.

10.1 Drowsiness is a fluctuating state between alert wakefulness and R&K stage-1 sleep. It is not distinguished as a separate state in the R&K classification of sleep stages, which is quite inadequate for our purposes.

10.2 Hori's classification of 9 H-stages in the sleep-onset process can serve as a useful guide for us, but those stages apply to intentional sleep-onset after lying down and closing one's eyes. This is quite different from unintentional sleep-onset that occurs in drowsy drivers who struggle to stay awake and to keep their eyes open.

10.3 Drowsiness causes a general impairment of performance on a variety of psychomotor vigilance tests and when driving, whether on a simulator or on the road. The impairment causes errors of omission as well as a slowing of reaction times. There is a reduced ability to maintain attention and volition, and reduced awareness of the "here and now". Well-learned, "automatic" behaviour, such as simple lane-tracking, is impaired less than is behaviour that requires focussed attention and novel responses. It is possible to continue with "automatic" behaviour when very drowsy, and to drive "without awareness".

10.4 The impairment due to drowsiness cannot be explained solely on the basis of microsleeps, which are brief episodes of R&K stage-1 sleep, each of which may last a few seconds. The impairment more often involves "attentional lapses", with alpha-waves in the EEG when the eyes are open. In an alert subject, alpha-waves would be blocked under those circumstances.

10.5 A very drowsy driver is able to fall asleep and to enter R&K stage-1 or even stage-2 sleep with his eyes open. This makes unreliable any method for monitoring drowsiness that is based solely on eyelid closure, such as PERCLOS.

10.6 Slow eye movements (SEMs) that appear during drowsiness, well before R&K stage-1 sleep, are unlike any others. They offer great potential for monitoring drowsiness in drivers. However, SEMs must be distinguished from a variety of other eye and eyelid movements, and this is difficult to do with EOG recordings.

10.7 New methods for monitoring the combination of movements of the eye and eyelids using infra-red reflection techniques offer potential for resolving these problems.

10.8 We already know that it is common for some truck drivers to fall asleep at the wheel. However, we do not know at what point along the drowsiness continuum we should say a driver is no longer fit to drive at a particular time.

10.9 There is still much that we do not know about drowsiness and unintentional sleep-onset. More research is needed.

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