SLEEP INERTIA AND ON-CALL READINESS

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Introduction

The effects of sleep deprivation and chronobiological variations in performance are undoubtedly among the most pervasive limitors of human ability in all situations that require sustained periods of continuous performance and in around-the-clock work settings (e.g. Dinges et al., 1988). These work scenarios are becoming increasingly common, often involving highly skilled and dedicated personnel as in sustained military operations, space flight preparation and launching, crisis and catastrophe management (Mitler et al., 1988). In all these situations, the negative effects of sleep loss during sustained operations must be compared to the adverse effects of sleep inertia upon abrupt awakening from sleep due to a possible emergency (Dinges et al., 1988; Dinges, 1990).

Sleep inertia defines a period of transitory hypovigilance, confusion, disorientation of behavior and impaired cognitive and behavioral performance that immediately follows awakening (Kleitman, 1963). Sleep inertia is one of the most serious contraindications to the use of napping during quasi-continuous operations if the individual may be required to perform complex tasks immediatly after sudden awakening at unpredictable times (Dinges et al., 1985).

Physiological Substratum

Sleep inertia has been considered a "paradoxical" phenomenon (Kleitman, 1963) since performance upon awakening is worse than before sleep. However, physiological phenomena are best described by sinusoidal rather than by square-wave functions (Bertini & Violani, 1992); consequently, the underlying behavioral states cannot readily be switched on and off at the transition to another state. For this reason, if we consider the transition from sleep to wakefulness as a complex process that takes some time to be completed, more than an exact shifting point from one state of consciousness to another, sleep inertia simply becomes the cognitive-behavioral face of this transition process.

From a physiological point of view, during the awakening period a clear dissociation between different parameters is evident. Based on the standard EEG scoring system (Rechtschaffen & Kales, 1968), the awake EEG is identified by a predominant alpha rhythm.

However, the EEG represent only a fraction of all the state-determining factors. In other words, "the presence of all polygraphic features of one state does not mean that no (unmonitored) variables of another state are present" (Mahowald & Schenck, 1992). As an example, Broughton (1968) showed that visual evoked potentials (VEP) recorded upon awakening from slow-wave sleep (SWS) are more similar to those obtained during sleep than to baseline waking values. The author ascribes these results to an impairment of cerebral responsiveness ("functional deafferentation") after SWS awakenings.

Other indications of a slow shift from the sleep EEG substrate to that of wakefulness

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come from the study of the EEG power spectra during spontaneous sleep-wake transitions (Ogilvie & Simons 1992). The FFT analyses of EEG sampled during spontaneous arousals from sleep showed a non-predicted gradual and continued drop of theta and delta power well into the first few minutes of wakefulness. There was a statistically significant difference between sleeping and waking delta only after the subject had behaviorally responded to three consecutive tones (i.e. about 70 sec after the first response).

Similarly, recent studies on cerebral blood flow -CBF- (e.g., Meyer et al., 1987) and cerebral blood flow velocities -CBFV- during sleep (e.g., Hajak et al., 1994; Kuboyama et al., 1997) as indirect but reliable indexes of the underlying neuronal metabolism and activity (e.g., Sokoloff, 1981), also suggest that the periods immediatly following nocturnal and morning awakenings have blood flow characteristics that are not comparable to daytime levels. Moreover, Hajak and co-workers showed that upon morning awakening, subjects required up to half an hour to reach CBFV values corresponding to the waking state of the previous evening. The delayed increases in CBFV after awakening provide another example of dissociation between different physiological parameters of sleep-wake transition, further stressing the slowness of the sleep-wake transition.

Sleep Inertia and Sleep Management

Clearly, sleep inertia has relevant operational implications. As already mentioned in the Introduction, from a sleep-logistic perspective, the main problem is to weigh the effects of sleep loss on sleepiness and performance against the adverse effects of sleep inertia upon abrupt awakening from sleep due to a possible emergency. From this point of view, one of the most critical factors on sleep inertia concerns its duration and time course.

However, although sleep inertia has been incorporated in several models of sleep and vigilance regulation (e.g., Achermann et al., 1994; Akerstedt & Folkard, 1997; Folkard & Akerstedt, 1992), only a few attempts have been made to experimentally quantify its time course. Most authors have typically made only one performance assessment after awakening (e.g. Naitoh et al., 1993), not allowing the determination of the time course and duration of sleep inertia. Due to this methodological limitation, sleep inertia has been generally reported to be short-lasting, being comprised between 1 and 20 minutes (Dinges et al., 1990; Hartmann & Langdon, 1965; Hartmann et al., 1965; Langdon & Hartmann, 1961; Seminara & Shavelson, 1969).

More recently, Achermann and co-workers (1995) addressed this issue by assessing performance every 20 minutes (4 times) during the first hour after awakening from nighttime sleep or from an evening nap, and finally after three hours from each awakening. They found sleep inertia to subside according to an exponential function, and to persist for slightly less than one hour. On the other hand, Jewett and coll. (1999) reported that subjective alertness and cognitive performance reach the baseline waking values about 2 hours after awakening; in this case, too, sleep inertia subsided according to an exponential function. Finally, in an unpublished study from our laboratory (Ferrara et al., unpublished) we found that cognitive performance reaches the baseline level after about 30 minutes from the morning awakening, showing an increasing linear trend during the first 75 minutes after awakening, while sensory-motor and motor performance was still below baseline levels in the same period of time.

Sleep Inertia: Modulating Factors

Sleep inertia duration and magnitude can be modulated by several factors. There are well-known differential effects of REM/NREM sleep stages on performance upon awakening. More specifically, SWS awakenings have greater negative effects on subsequent performance than REM sleep awakenings. These effects have been demonstrated with a wide array of tasks: simple motor tasks (Tebbs & Foulkes, 1966; Wilkinson & Stretton, 1971); sensory-motor tasks (Scott & Snyder, 1968; Feltin & Broughton, 1968); and cognitive tasks (Scott & Snyder, 1968; Stones, 1977).

However, it has been claimed that sleep structure is also very important in determining sleep inertia (Dinges, 1990). The profound modification in sleep architecture and the increased sleep depth caused by sleep deprivation dramatically exacerbate sleep inertia and cognitive impairment upon awakening from recovery sleep (Dinges et al., 1985). It has also been found that cognitive decrements after abrupt awakenings from 1 and 2 hour naps show a linear relationship with SWS amount during the nap (Dinges et al., 1981; 1985).

Moreover, the negative influence of sleep deprivation on sleep inertia seems to interact with time-of-night or circadian factors in producing even more dramatic effects. As an example, Naitoh (1981) reported that, after a 2-hour nap taken early in the morning (0400-0600) following 45 hours of continuous work without sleep, both task performance and self rating of mood, sleepiness and fatigue remain deteriorated at the levels of those who stayed awake.

More generally, the outcomes concerning the modulation of sleep inertia by circadian factors - mainly linked to body temperature rhythm - are not consistent. Conflicting evidence comes from studies of napping with and without previous sleep deprivation (e.g., Bonnet & Arand, 1995; Tassi et al., 1992; Naitoh et al., 1993), as well as from repeated awakenings during nocturnal sleep (e.g., Balkin & Badia, 1988; Rosa et al., 1983; Rosa & Bonnet, 1985). A more accurate description of circadian influences on sleep inertia needs the support of further empirical data.

Moreover, sleep inertia seems to dramatically depend on the type of task used, highly demanding cognitive and attentional tasks being much more affected than simple motor ones (Muzet et al., 1995). At variance with physiological sleepiness, which in self-paced tasks affects speed of performance more than accuracy, it has been claimed that sleep inertia exerts a negative influence on both, but particularly on the latter (Balkin & Badia, 1988; Naitoh et al., 1993; Ferrara et al., unpublished b).

In conclusion, although it is often difficult to compare results of studies on sleep inertia, since several different experimental designs and tasks have been used, a few clear indications seem to emerge. The intensity of sleep inertia is strongly influenced by some homeostatic sleep variables linked to SWS amount and, more generally, to depth of and pressure for sleep. Finally, circadian factors and previous sleep loss exacerbate sleep inertia by adding their simple effects.

Sleep Inertia: Possible Countermeasures

From a brief review of the literature on the physiological basis and modulating factors of sleep inertia, we will try to extrapolate some countermeasures against the detrimental effects of sleep inertia on performance upon awakening, to be applied when it is possible in operational settings.

The first countermeasure could be to reduce the probability of awakening out of SWS, since it is well known that SWS awakenings yield the greatest performance decrements. One possibility is to allow sleep when the occurence of SWS is very low (e.g., in the morning). Another strategy can be to allow naps of about 80-90 minutes (i.e., the mean duration of a normal NREM-REM sleep cycle), minimizing the probability of a SWS awakening. Some experimental data confirm the usefulness of this strategy, by showing that sleep inertia magnitude after a 20-min and a 80-min nap are very similar, while the worst performance upon awakening is recorded after a 50-min nap (Stampi, 1992). Obviously, a 80-min nap should be preferred to a 20-min nap because of its greater restorative power.

Another very important strategy to minimize sleep inertia is to avoid a long period of wakefulness before allowing a nap, since the increase of sleep depth caused by sleep deprivation dramatically exacerbates sleep inertia (Dinges et al., 1985).

In addition, awakening near the circadian nadir of body temperature should also be avoided, especially if the sleep period follows sleep deprivation (Naitoh, 1981).

It has been reported that washing one's face with cold water immediately after awakening is a simple but effective tool to fight sleep inertia (Labuc, 1978, 1979). More generally, every "alerting" factor (i.e., noise, light, physical exercise) should be useful in counteracting sleep inertia, even though - at present - only few attempts have been made to assess their effectiveness. As an example, pink noise (75 dBA) administered during the first hour after awakening improves response speed at 0500 but not at 0800, when it has detrimental effects on performance (Tassi et al., 1993). More recently, it has been reported that following the "normal morning routine" (i.e., getting out of bed, taking a shower, having breakfast) does not abolish sleep inertia as compared to a constant routine in bed (Jewett et al., 1999). In the same experiment (Jewett et al., 1999), it was found that exposure to normal room light (about 150 lux) upon awakening did not improve performance as compared to very dim light (about 20-25 lux).

Sleep Inertia: Open Questions

Sleep inertia is still a poorly understood phenomenon: from the point of view of its physiological substratum, that could be approached in the near future with the newest and more sophisticated neuroimaging techniques; as regards the sleep-related modulating factors and psychological and personality variables that may influence it. However, a few research areas that should be explored to give important answers on sleep inertia to be applied in operational fields will be pointed out.

The first unexplored topic is the role of individual differences in reactions to the effects of sleep inertia. We all anecdotally know that individuals show a wide range of variation with respect to their perceived ability to function immediately after awakening. However, the literature on sleep inertia has definitely ignored this problem, relegating individual differences to a role of "confusing variable" to be controlled. The study of individual differences modulation of sleep inertia will add very important knowledge to the definition of the psychophysiological profile of tolerance to irregular work hours.

The same applies to the role of psychological factors, like motivation, in the modulation of sleep inertia. One should believe that motivation can be a strong and

efficient countermeasure to sleep inertia for a fighter pilot sleeping on-call, when he is requested to be in the cockpit at 5000-10000 metres a.s.l. just 5 minutes after abrupt awakening. However, this topic should be specifically evaluated.

For operational purposes, the duration and time course of sleep inertia after naps taken at different times of the day should also be further assessed, since available data are inconclusive.

It would be very important to have some pharmacological countermeasures to sleep inertia, such as very fast acting stimulants, to be used in operational settings when the need for high levels of alertness and performance immediately after awakening should arise. To our best knowledge, the use of stimulants to counteract sleep inertia effects has never been tried, not even in laboratory settings.

Non-pharmacological countermeasures to sleep inertia could also be very useful, particularly because pharmacological measures are currently lacking. Generally speaking, any alerting factor could be assessed to counteract sleep inertia: physical and/or mental exercise, external noise, bright light. As regards noise, although in at least one study pink noise has been administered for one hour after awakening with non-univocal results (Tassi et al., 1993), the effectiveness of different types of noise with different intensities and durations should be assessed. Bright light might also be effective against sleep inertia, since its alerting effects are well established (e.g., Czeisler et al., 1990).

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