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THE SLEEP INERTIA PHENOMENON DURING THE SLEEP-WAKE TRANSITION: THEORETICAL AND OPERATIONAL ISSUES

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Running Head: Sleep Inertia: theoretical and operational issues

ABSTRACT

Sleep Inertia (SI) defines a period of transitory hypovigilance, confusion, disorientation of behavior and impaired cognitive and sensory-motor performance that immediately follows awakening. SI, the cognitive and behavioral correlate of the transition from sleep to wakefulness, has been incorporated in several models of sleep and vigilance regulation. Monitoring of several physiological parameters during the awakening period cleary indicate that this transition process is very slow. On the cognitive and behavioral side, SI has relevant operational implications. SI 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. The studies on SI modulating factors showed that SI is strongly affected by slow wave sleep amount and sleep depth, while the outcomes concerning the modulation of SI by circadian factors are not consistent. Cognitive tasks involving high attentional load seem to be much more affected by SI than simple motor ones, performance *accuracy* being more impaired than *speed*. Finally, some possible countermeasures against the detrimental effects of SI to be applied in operational settings have been provided.

Index Terms: Sleep inertia, Sleep management, Performance upon awakening, Sleep-wake transition

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Introduction

"Transitional states are among the most difficult to characterize and understand. They occur during times of shifting priorities and constitute hybrid conditions which borrow features from the more distinctive parent states. It follows that the more complex and the greater the differences between the parent states, the more likely it will be that the transitional processes will also be complex. For these reasons, there has been a tendency to consider these states as somewhat confusional conditions to be recognized and controlled for, but not often the subject of detailed study"(43). This statement perfectly applies in particular to the transition from sleep to wakefulness. In fact, while sleep onset has received increasing attention in the last two decades, the emergence from sleep still remains a poorly understood phenomenon.

In this review we will focus our attention on Sleep Inertia (SI), a period of transitory hypovigilance, confusion, disorientation of behavior and impaired cognitive and behavioral performance that immediately follows awakening (30). SI has been considered a "paradoxical" phenomenon (30) since performance upon awakening is worse than before sleep. However, if physiological sleep phenomena are best described by sinusoidal rather than by square-wave functions (5), consequently the underlying behavioral states cannot readily be switched on and off when shifting 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, SI simply becomes the cognitive-behavioral face of this transition process.

SI has interesting theoretical and operational implications. It has been included as an important component in several models of alertness and performance (1, 3, 20). It integrates the influence of two other major components: a 24-h circadian component (Process C), with a sinusoidal shape; and a homeostatic component (Process S), that increases exponentially during wakefulness and is reversed during sleep. The wakeup component, or Process W, was originally incorporated to take into account the fact that people take some time to wake up properly (20). It takes the form of a deviation from Process S that decreases in an asymptotic manner as a function of the logarithm of hours awake, and ceases about 2-3 hours after awakening (20). Although in the above-mentioned performance and alertness models SI acts independently of Processes C and S, it is not possible to exclude that Process W may interact with

Process S in a non-linear manner (28). Consequently, the magnitude and/or the time constant of the dissipation of SI may increase as a consequence of sleep deprivation (so that S is very high).

SI is a robust phenomenon that must be taken into account in many operational settings. 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. 12). 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 (38). In all these situations, the negative effects of sleep loss during sustained operations must be compared to the adverse effects of SI upon abrupt awakening from sleep due to a possible emergency (11, 12). SI 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 (10).

Physiological Substratum

From a physiological point of view, a clear dissociation between different parameters is evident during the awakening period. Based on the standard EEG scoring system (44), 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" (36). As an example, Broughton (8) showed that visual evoked potentials (VEP) recorded upon awakening are more similar to those obtained during sleep than to baseline waking values. Following one-fourth of the arousals from slow-wave sleep (SWS), VEP contained an apparent carry-over of typical SWS components. Even when there was no such carry-over, the VEP regularly showed decreased amplitude and increased latency of 100-300 msec components. No similar changes in visual evoked potentials were observed after awakenings from REM sleep. The author ascribes these results to an impairment of cerebral responsiveness ("functional deafferentation") after SWS awakenings, also responsible for the behavioral changes (namely confusion) anecdotally reported only after awakenings from slow-wave sleep.

Other indications of a slow shift from the sleep EEG substrate to that of wakefulness come from the study of EEG power spectra during spontaneous sleep-wake transitions (42). The spectral analysis (Fast Fourier Transform, FFT) of EEG sampled during behaviorally identified (button pressing to stop a tone) 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. Although delta power decreased by almost 50% at the first behavioral response, there was a statistically significant difference between sleeping and waking delta only after the subject had responded to three consecutive tones (i.e., about 70 sec after the first response). Theta power trend was very similar to that seen for delta frequencies.

Similarly, studies on cerebral blood flow - CBF - (e.g. 37) and cerebral blood flow velocities - CBFV - during sleep (e.g. 24, 31) as indirect but reliable indexes of the underlying neuronal metabolism and activity (e.g. 49), also suggest that the period immediatly following nocturnal and morning awakenings have blood flow characteristics that are not comparable to daytime levels. Moreover, Hajak and co-workers (24) 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 suggest an uncoupling of cerebral electrical activity and cerebral perfusion and provide another example of dissociation between different physiological parameters of sleep-wake transition, further stressing the slowness of this transition.

SI and Sleep Management

SI 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 SI upon abrupt awakening from sleep due to a possible emergency. From this point of view, one of the most critical factors on SI concerns its duration and time course.

However, although SI has been incorporated in several models of sleep and vigilance regulation (1, 3, 20), 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. 41), not allowing the determination of the time course and duration of SI. Due to this methodological limitation, SI has been generally reported to be short-lasting, being comprised between 1 and 20 minutes (11, 26, 27, 34, 48).

Achermann and co-workers (2) addressed this issue by assessing subjective alertness and reaction times in a memory task 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. For both alertness and performance measures, they found SI to persist for slightly less than one hour, and to subside according to exponential functions with time constants of 0.45 and 0.30 hour, respectively. More recently Jewett and coll. (28) reported that subjective alertness and

performance in an addition task show a sharp rise in the first hour after awakening and begin to level off about 2 hours after awakening, reaching the baseline waking values between 2 and 4 hours after awakening. Also in this case an asymptotic dissipation of SI has been suggested, since a saturating exponential function provided a good fit to the data for each measure. The time constants for the dissipation of sleep inertia were 0.67 h in subjective alertness and 1.17 h in cognitive performance (number of additions performed). Finally, in another study (17) it has been found that performance accuracy in a subtraction task reaches the baseline level after 30-45 minutes from the morning awakening, showing an increasing linear trend during the first 75 minutes after awakening. On the other hand, although sensory-motor (auditory reaction times) and simple motor (finger tapping) performances were less affected by SI, they were still below baseline levels in the same period of time, never reaching the baseline level during the testing period.

The differences in SI duration and time-course reported in the above-mentioned studies (2, 28, 17) may be due to some relevant methodological differences between them: as an example, Achermann et al. (2) did not assess SI between 1 h and 3 h after awakening, while Ferrara et al. (17) gave no tests after the first 75 min from the awakening. In addition, in the latter study SI was assessed upon awakenings placed between 8 a.m. and 9 a.m. after nocturnal sleep episodes of 7.5 h characterized by different sleep homeostasis conditions (comprising SWS deprivation and recovery nights), while in the former SI was assessed at 7 a.m. after nocturnal sleep, as well as at about 9 p.m. after an early evening nap. Furthermore, the Jewett et al. data (28) were collected in an environment free of time cues, and during the third day of the experiment subjects were exposed to very dim light during a constant routine protocol.

Differences in reported results can also be due to possible differential sensitivity of the performance tasks used to assess SI. As an example, the time constant for the dissipation of SI in cognitive performance (number of additions performed) found by Jewett et al. (28) is much larger than that reported for reaction times in a short term memory task by Achermann et al. (2), suggesting that some neurobehavioral functions may be more sensitive to SI than others.

In conclusion, the discrepancies in the reported time-course and duration of SI are accounted for by large differences in the experimental paradigms used, leading to uncontrolled interactions between homeostatic and circadian (time-of-day) processes regulating sleep and wakefulness, as well as to the use of several different tasks and variables to assess SI.

SI: Modulating Factors

SI duration and magnitude can be modulated by several factors. There are differential effects of REM/NREM sleep stages on performance upon awakening. More specifically, SWS awakenings have often been reported to 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 (54, 55); sensory-motor tasks (16, 47); and cognitive tasks (47, 51). However, it is not clear whether the above-mentioned differential effects are due to neurophysiological, psychophysiological and functional differences between REM and NREM states, or whether to uncontrolled temporal and circadian influences (time-of-night effects), or to an interaction between the two. All these variables should require further exploration with a more controlled research methodology.

Furthermore, it has been claimed that sleep structure is also very important in determining SI (11). The increased sleep depth (in terms of both amount of SWS and sleep stage at awakening) caused by sleep deprivation dramatically exacerbate SI and cognitive impairment upon awakening from recovery sleep (10). 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 (10, 15).

Moreover, the negative influence of sleep deprivation on SI seems to interact with time-of-night or circadian factors in producing even more dramatic effects. As an example, Naitoh (40) reported that, after a 2-hour nap taken early in the morning (0400-0600 a.m.) following 45 hours of continuous work without sleep, both task performance and self-rating of mood, sleepiness and fatigue remain deteriorated up to 6 hours. This long-lasting sleep inertia effect was not observed when a nap of the same duration was taken at 1200-1400, after 53 hours of wakefulness; in fact, following this midday nap, sleep inertia disappeared within 1 hour and was then replaced by improvements.

More generally, the outcomes concerning the modulation of SI 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. 7, 41, 52), as well as from repeated awakenings during nocturnal sleep (e.g. 4, 45, 46). As an example, Bonnet & Arand (7) reported a worsening of SI effects following awakening at 5:00 a.m., but some other studies showed greatest performance impairment upon awakenings placed in the first part of the night (22, 23, 52). It is evident that these approaches necessarily confound the effects of the circadian phase with those of homeostatic variables (i.e., the amount of prior sleep or of prior sleep loss, if sleep deprivation is involved). However, this problem is difficult to solve, if possible at all, since both of these factors are temporal dimensions that covary with each other (10). For these reasons, the available evidence for circadian

modulation of SI can not be considered definitive, and a more accurate description of circadian influences on SI needs the support of further empirical data collected with sound methodology.

Moreover, SI seems to dramatically depend on the type of task used, highly demanding cognitive and attentional tasks being much more affected than simple motor ones (39). As regards the impairment of simple sensory-motor performance (auditory reaction times and finger tapping task) upon awakening, a recent study (18) reported that it is accounted for by: a general decrement in overall response speed (median of RT); a decrease in response speed in the "optimum response domain"; and an increase of lapsing. Consequently, behavioral performance slowing upon awakening is not simply due to lapses or failure to respond, but should be ascribed to a general decline in the ability to allocate attention to the task and to give the required motor response as fast as possible. As regards cognitive performance, at variance with physiological sleepiness, which in self-paced tasks affects *speed* of performance more than *accuracy* (6, 13, 14), it has been claimed that SI exerts a negative influence on both, but particularly on the latter (4, 41). Some recent evidence confirmed that the lowered level of brain arousal upon awakening adversely affects cognitive performance *accuracy* more than performance *speed*. (19).

In conclusion, although it is often difficult to compare results of studies on SI, since several different experimental designs and tasks have been used, a few clear indications seem to emerge. The intensity of SI is strongly influenced by some homeostatic sleep variables linked to SWS amount and, more generally, to depth of sleep (indexed by awakening thresholds or by sleep stage at awakening). Moreover, circadian factors and previous sleep loss exacerbate SI by adding their simple effects. Finally, cognitive tasks involving high attentional load seem to be much more affected by SI than simple motor ones, performance *accuracy* being more impaired than *speed*.

Possible Countermeasures

From a review of the literature on the physiological basis and modulating factors of SI, we will try to suggest some countermeasures against the detrimental effects of SI 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 80-100 minutes (i.e., the mean range of duration of normal NREM-REM sleep cycles), or,

alternatively, of about 20 minutes, minimizing the probability of a SWS awakening. Some experimental data confirm the usefulness of this strategy, by showing that SI 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 (50). 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 SI is to avoid a long period of wakefulness before allowing a nap, since the increase of sleep depth caused by sleep deprivation dramatically exacerbates SI (10, 40). As already suggested by others (10), sleep opportunities should be provided before sleep loss accumulates beyond 36 hours, since longer and more severe performance decrements have been reported on awakening from naps taken after this time as compared to naps taken within 30 hours of wakefulness (10, 40).

In addition, awakening near the circadian nadir of body temperature should also be avoided, especially if the sleep period follows sleep deprivation (40).

It has been reported that washing one's face with cold water immediately after awakening is a simple but effective tool to fight SI (32, 33). More generally, every "alerting" factor (i.e., noise, light, physical exercise) should be useful in counteracting SI, 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 (53). Although the authors claimed that all subjects experienced the same amount of prior sleep debt, since each of them slept for three hours during the experimental night, it has to be noted that the group woken up at 0800 possibly experienced a greater homeostatic pressure for sleep, because their sleep time was postponed by two hours as compared to the group woken up at 0500. Consequently, the different homeostatic pressure acted as a possible confounding variable, casting some doubts on the interpretation of results.

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 SI as compared to a constant routine in bed (28). In the same experiment (28), 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). However, it has to be noted that in the above-mentioned study (28) the exposure to very dim light was introduced on the awakening of the third experimental day together with a constant routine condition. Consequently, it is difficult to dissociate the effects of the dim light condition and those of the constant routine. Moreover, any question about a possible alerting effect of bright light remains unanswered; it may be that, to detect an

alerting effect, a very bright light (i.e. 2000 lux) upon awakening should have been used (see below).

SI: Open Questions

SI is still a poorly understood phenomenon from both the point of view of its physiological substratum, which could be approached in the near future with neuroimaging techniques, and of 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 SI, also 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 SI. It is anecdotally well-known that individuals show a wide range of variation with respect to their perceived ability to function immediately after awakening. However, the literature on SI has definitely ignored this problem, relegating individual differences to a role of "confusing variable" to be controlled. The study of individual difference modulation of SI will add very important knowledge to the definition of the psychophysiological profile of tolerance of irregular work hours. As an example, it could be intersting to explore the relation between diurnal type and SI, since the morningness-eveningness dimension has been associated with the adjustment to shift work (21, 25, 29).

The same applies to the role of psychological factors, like motivation, in the modulation of SI. As an example, one should believe that motivation can be a strong and efficient countermeasure to SI 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 SI after naps taken at different times of the day should also be further assessed, since available data are inconclusive. Varying nap duration may also be necessary for a complete understanding of these aspects of SI.

It would be very important to have some pharmacological countermeasures to SI, 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 SI effects has never been tried, not even in laboratory settings.

Non-pharmacological countermeasures to SI could also be very useful, particularly because pharmacological measures are currently lacking. Generally speaking, any alerting factor could be assessed to counteract SI: 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 (53), the effectiveness of different types of noise with different intensities and durations should be assessed. Bright light might also be effective against SI, since its alerting effects are well established (e.g. 9).

Conclusions

Several observations on the physiological correlates of the sleep-wake transition (8, 24, 31, 37, 42) are in line with evidence coming from human studies on cognitive (e.g. 51) and behavioral (e.g. 54) features of awakening from sleep, pointing out the need to re-define the sleep-to-wake transition period as a neurophysiologically distinct state. In other words, emerging from sleep can not be identified with an exact shifting point from one state of consciousness (sleep) to another (wakefulness), but is better described as a complex and slow process that takes some time to be completed. During this transition period, that shares some features with both the wake and sleep states, a clear dissociation between different parameters (physiological, cognitive and behavioral) is evident, since they show different rates of change from the sleep pattern to the wake pattern.

Consequently, in such a situation the subject may still be able to conduct some simple social interaction (e.g. 11) but the "functional deafferentation", proposed by Broughton (8) to explain the low levels of brain reactivity upon awakening, will make it difficult to obtain more demanding and complex performance. For these reasons, in all these situations requiring highly skilled performance immediatly after an abrupt awakening (e.g., sustained military operations, medical emergency management), the unavoidable adverse effects of SI have to be considered in advance, providing personnel with simple tools to wash out these effects. At the present, the use of any feasible alerting factor (physical exercise, external noise, bright light, cold water) can only be suggested. Further research is needed to experimentally clarify which are the most effective tools to be applied in operational settings.

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