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REVIEW ARTICLE

Sleep inertia

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Sleep inertia is a transitional state of lowered arousal occurring immediately after awakening from sleep and producing a temporary decrement in subsequent performance. Many factors are involved in the characteristics of sleep inertia. The duration of prior sleep can influence the severity of subsequent sleep inertia. Although most studies have focused on sleep inertia after short naps, its effects can be shown after a normal 8-h sleep period. One of the most critical factors is the sleep stage prior to awakening. Abrupt awakening during a slow wave sleep (SWS) episode produces more sleep inertia than awakening in stage 1 or 2, REM sleep being intermediate. Therefore, prior sleep deprivation usually enhances sleep inertia since it increases SWS. There is no direct evidence that sleep inertia exhibits a circadian rhythm. However, it seems that sleep inertia is more intense when awakening occurs near the trough of the core body temperature as compared to its circadian peak. A more controversial issue concerns the time course of sleep inertia. Depending on the studies, it can last from 1 min to 4 h. However, in the absence of major sleep deprivation, the duration of sleep inertia rarely exceeds 30 min. But all these results should be analysed as a function of type of task and dependent variables. Different cognitive functions are probably not sensitive to the same degree to sleep inertia and special attention should be provided to dependent variables as a result of the cognitive processes under review. Finally, sleep disorders represent risk factors which deserve new insight in treatment strategies to counteract the adverse effects of sleep inertia.

Key words: sleep inertia, performance, environment, sleep deprivation, EEG, circadian rhythm, narcolepsy-cataplexy, sleep apnoea, vigilance, arousal.

Introduction

Sleep inertia refers to the phenomenon of decreased performance and/or disorientation occurring immediately after awakening from sleep relative to pre-sleep status. Historically, the first study dates from 1961 [1] and was followed by a series of experiments, all of them showing that subjects tested for 10 min immediately after awakening performed worse than when tasks were carried out just before retiring [2–6].

Broughton (1968) first called this state "sleep drunkenness", referring to behavioral slowdown observed after abrupt awakening [7]. It has also been called post-sleep disorientation until Lubin *et al.* (1976) gave this phenomenon its actual name: sleep inertia [8]. It has been shown that, if sleep inertia (SI) always exists, its magnitude is

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dependent on several factors such as prior sleep duration, sleep stage prior to awakening and the existence of prior sleep deprivation. SI is also dependent on time-of-day of awakening.

Concerning the effects of SI, many studies have suggested that its severity depends on the type of task subjects are achieving. This interaction could determine its time course and consequently its duration, which is another controversial issue. Whatever the duration of SI, it has also been questioned whether environmental factors known to enhance alertness could modulate its intensity, either by improving or deteriorating performance.

SI has been rarely studied in the context of sleep disorders. It has been mentioned that obstructive sleep apnoea syndrome patients have markedly increased sleep inertia and similar results have been obtained in narcolepsy-cataplexy.

All these studies suggest that the nature of SI refers to a transitional process between one state of arousal to another, namely between sleeping and waking (hypnopompic drive). This process may not act on an all-or-none basis but rather according to a slow and progressive mechanism. We also question the relationship between SI and the underlying psychophysiological processes, with the hypothesis that arousal and vigilance could be functionally two different states. If this is true, such a theoretical approach could help understanding, at least some of the controversial findings concerning SI effects on performance.

Sleep inertia and prior sleep duration

SI exists whatever the duration of prior sleep episodes. Recently, Jewett *et al.* (1999) showed that after a normal full 8-h sleep episode, the time course of SI dissipation for subjective alertness and cognitive throughput (addition task) lasted for more than 2 h after awakening, even in subjects who were not sleep deprived and were sleeping at their usual times [9]. In the addition task, the authors scored a decrement of 10.7% in the number of sums attempted in 2 min, irrespective of errors. This result unfortunately does not allow determination of whether the error rate was also increased by SI.

However, since SI constitutes a major concern for subsequent task performance, it is clear that it has been studied essentially under conditions of rather short naps to evaluate the gain-cost balance of intermittent sleep in continuous operations or shift-work conditions [10–12]. Concerning effectiveness of space crew performance [13], it has been suggested that, as far as SI is concerned, a simultaneous sleep schedule where each of two crew members could assume an 8-h sleep period would be more advantageous than a staggered sleep schedule, where crew members are allowed to sleep for short periods, with crew members expected to be optimally efficient within 2 min subsequent to a potential alarm. In this study, Seminara *et al.* (1969) showed that SI produced a decrement in reaction time ranging from 12 to 360% of pre-sleep performance depending on the type of task, the more complex one (product organizing) being less affected than the more simple (control panel task and response time/monitoring task). These data suggest differential effects of SI depending on task complexity and performance index (speed vs accuracy).

In a polyphasic sleep schedule, the effects of SI were measured for different nap durations [14]. Subjects were allowed to sleep for 4 h of nocturnal anchor sleep while the remaining 4 h of daytime sleep were divided into multiple 20, 50 or 80-min naps. Hence, subjects suffered no specific sleep deprivation. They appeared to adapt well

to all three sleep schedules but, depending on the type of task, the effects of SI were slightly different. In the Memory and Search Test (MAST) [15] which requires a high level of cognitive involvement, the decrement (relative to pre-sleep levels) was of 3%, 8% and 14% respectively for the 20, 50 and 80-min naps. Performance assessed by the Descending Subtraction Test (DST) [16] showed post-arousal mean decrements which were far more pronounced (21%, 36% and 25% respectively for the 20, 50 and 80-min naps). This task scored the number of correct subtractions attempted per unit time, which actually represents the speed of information processing. These data suggest that, altogether, SI was the most intense for the intermediate 50-min nap and surprisingly less intense for the most extreme condition of 20 min. The apparent contradiction with previous results found for the effects of sleep duration can be well explained in terms of sleep stages prior to awakening.

Effects of sleep stage prior to awakening on sleep inertia

Stampi *et al.* (1990) showed that, during the 20-min nap condition, subjects never underwent a SWS episode and were always awakened during stage 2 [14]. The same occurred in the 80-min nap condition except that subjects were sometimes awakened during a rapid eye-movement episode (REM), making this sleep condition intermediate in terms of SI effects. As mentioned above, the most deleterious sleep schedule was the 50-min nap condition, with naps long enough to allow subjects to initiate SWS, but not long enough to return to stage 1 or 2. As a matter of fact, it has been often described that awakening in stage 1 or 2 produces no substantial decrement on subsequent performance (i.e. no SI effect) [17, 18]. In a short-term memory task, where subjects were presented a word list 1 min after arousal from different sleep stages, Stones (1977) found that memory performance after arousal from stage 1, 2 or REM was so high as to be indistinguishable from that obtained under the no-sleep condition (0% of decrement in the number of category recalled and 8.5% of decrement in the number of items recalled per category) [17].

However, effects on SI after awakening from REM sleep show some controversial findings. Although Stones (1977) could not find any effect of SI after arousal from REM sleep, Koulack *et al.* (1974) found, in similar conditions, a significant decrement in performance in visuo-motor and auditory tasks [19]. These authors observed that the decrement was more pronounced after a REM period with higher eye-movement density than after a REM period with lower eye-movement density. According to these authors, the REM period could result in two different sub-stages depending on dream emotionality, which could interfere with subsequent task performance, particularly if they require high attentional levels. But it should also be stressed that, while Stones (1977) used memory tests with a high cognitive load, Koulack *et al.* (1974) scored performance on rather low demanding perceptual and motor tasks; this difference could partly explain the discrepancy concerning the effects of arousal from REM sleep.

However, it seems absolutely clear that awakening after a SWS episode produces the maximum SI [20–24]. Webb and Agnew (1964) were among the first authors to demonstrate that subjects aroused from afternoon naps during a stage 4 episode exhibited a significant decrement in a sensory motor task, measuring discrimination reaction time and time to complete the whole serial task [20]. They found a 12% decrement relative to pre-sleep level. In the same way, Feltin and Broughton (1968) reported increased reaction time after SWS as compared to REM sleep [21]. Similar data

were found by Scott and Snyder (1968), measuring reaction time in a psychophysical paradigm by Pieron's Law [25]. It has also been shown that since heightened sensory thresholds are instituted by the sleep system to lessen arousability, sensory performance after abrupt awakening is usually more impaired after awakening from SWS than from stage 2 [26, 27].

Sleep deprivation effects on sleep inertia

Prior sleep deprivation is another important factor responsible for severity of SI. Dinges *et al.* (1985) measured performance in a reaction time and the Descending Subtraction Test (DST) [16] upon abrupt awakening following a 2-h nap after 6, 18, 30, 42 or 54 h of sleep deprivation [10]. They showed that sleep deprivation increased the amount of deep sleep in all the naps and, irrespective of sleep stage prior to awakening, this was associated with greater post-nap cognitive deficits, suggesting that sleep architecture as a whole (and not only sleep stage prior to awakening) could be a critical factor to generate SI. Their reaction time task showed maximal decrement at awakening for the naps placed after 18, 42 and 54 h of sleep deprivation, which all scored the most SWS. The DST was measured by the number of correct responses per unit time, which is considered by us as an index of speed of information processing. It showed that, relative to pre-nap performance, the decrement was of 26.6%, 38.4%, 37.6%, 70.9% and 70.9% respectively, for the naps placed at 6, 18, 30, 42 and 54 h of sleep loss, suggesting that, except for the 30-h nap, SI increased in a linear trend with sleep loss and SWS amount during the naps. A similar result was found in other studies [28–31].

In a polyphasic sleep schedule lasting for 48 days with a reduction of sleep to 3 h per day, Stampi and Davis (1991) [32] showed that general performance in the DST [16] and in the MAST [15] did not decrease dramatically over the whole experimental period [32]. However, SI was far more severe in the DST than in the MAST, as previously observed [14], probably because the subtraction test was here scored with a speed index (number of correct items per unit time), while the memory task was scored on correct reported items on an otherwise high demanding task.

However, important sleep deprivation is not necessary to produce similar results. Gillberg (1984) compared SI intensity after a 1-h nap at 21:30 h or at 04:30 h [33]. In both conditions, subjects were partially sleep deprived, since they slept only 4 h during the previous night. The author showed that SI was more important at 04:30 h, because sleep loss was slightly greater than at 21:30 h. However, he did not discuss a possible chronobiological effect of this difference, which could also explain his result. Balkin and Badia (1988) also assessed performance over the night in a partial sleep deprivation paradigm [34]. During four consecutive nights, subjects were awakened every hour from 00:40 h to 05:40 h for a 20-min test session. Sleepiness ratings and performance on 5-min addition tests were measured at 1.5, 7.5 and 13.5 min post-awakening. The results show that the magnitude of decrement in performance on the addition test after awakening increased progressively with time, indicating that, even within a very reduced sleep deprivation paradigm, SI was enhanced by sleep loss in a dose-dependent manner. Although the mean scores of addition tests administered prior to sleep did not change across the four nights, when administered shortly after awakenings, they declined from 18.7 on night 1 to 10.2 on night 4. As would be predicted under conditions of increasing sleepiness, the decline was due to a reduction in the mean

number of problems attempted, even though the mean percentage of errors also increased from 10% to 23% across nights 1 to 4.

However, Tassi *et al.* (1992, 1993) showed that SI, in a one-night partial sleep deprivation paradigm, showed the most adverse effects on spatial memory in the early nap as compared to a late nap. This was the consequence of a preponderance of awakening in SWS during the early nap, suggesting that, when naps are of short duration (1 h) and sleep deprivation of moderate amplitude, the most critical factor is sleep stage at awakening rather than the absolute amount of sleep loss [35, 36].

Time-of-day effect on sleep inertia

Naitoh *et al.* (1993) were among the first to raise the question whether there was a "best time not to wake up" [37]. The authors examined whether there could be best and worst hours to wake up stemming from circadian effects on SI. Since the process of falling asleep is strongly influenced by circadian time, the reverse process of awakening could be similarly affected. In their study, subjects remained awake for a 64-h continuous work period except for 20-min sleep periods every 6 h, while another group was not allowed to sleep during the same period. SI was measured with Baddeley's logical reasoning task [38] which is known to be very sensitive to sleep deprivation effects. There appeared to be no specific circadian time when SI was either maximal or minimal, suggesting that there are no advantages realized on SI by waking up from sleep at specific times of day.

This result is, however, isolated since two other studies [10, 39] reported quite different conclusions. The former, performed by Dinges *et al.* (1985), suggested that SI was more severe around the circadian trough, and less severe near the circadian peak in core body temperature. In this experiment, subjects underwent a 2-h nap placed near either the circadian peak or trough in body temperature, and preceded by 6, 18, 30, 42 or 54 h of sleep deprivation. Although sleep deprivation increased the amount of SWS, naps placed in the trough (middle of the night) produced greater cognitive decrements than naps placed in the peak (middle of the afternoon), even when the latter involved more prior sleep loss [10]. This result is consistent with other studies [22, 28], and therefore the authors suggested that the relationship between the amount of sleep deprivation and the extent of the decrement is non-linear due to interactions with circadian placement of the naps. Wilkinson and Stretton (1971) emphasized differential effects as a function of type of tasks [23]. They showed that reaction time was most increased in the early part of the night because of depth of the preceding sleep, while adding and coordination tasks, which are more demanding cognitive tasks, were more influenced by the trough in the circadian cycle of physiological activity.

Balkin and Badia (1988) suggested that the circadian effects on SI could be dependent on the type of measures and on the amount of prior sleep loss [34]. In a 4-night partial sleep deprivation paradigm, they found that performance rates were less sensitive to the time-of-night than self-rating scales, since subjective sleepiness followed a circadian trend (increased sleepiness across the night), but performance level remained more or less stable (night 1). With increasing sleep loss (nights 2–4), performance, as well as subjective ratings, failed to show this typical circadian pattern, probably because the sleep disruption/restriction procedure, combined with the subjects' circadian sleep tendencies, had a synergistic effect causing them to approach their lower limit.

Finally, Lavie and Weler (1989) carried out an ultrashort sleep–wake experiment on the basis of a 13 min waking–7 min resisting sleep paradigm after one night of total sleep deprivation [39]. The ultrashort sleep–wake schedule started at 07:00 h and was interrupted either at 15:00 h or at 19:00 h for 2-h naps. The first condition was termed the early nap and the second the late nap. Their results showed that the early naps (15:00 h to 17:00 h) produced less SI, although they contained more stage 3 and 4 than the late naps. According to the authors, this was best explained by the fact that after the early nap, performance measures coincided with a "forbidden zone for sleep" [40], suggesting in that case, a best time to wake up regardless of sleep architecture.

Practice Points

1. Shiftworkers could take advantage from nocturnal naps during their nightshift, provided that naps occur rather in the second part of the night to avoid awakening during a SWS episode
2. Sleep fragmentation in case of abnormal sleep–wake schedule (shiftwork and polyphasic sleep schedule) can induce a greater sleep inertia effect, if awakening occurs near a circadian trough of core body temperature

Duration and time course of sleep inertia

This is probably the most controversial aspect in studies on SI although its consequences in operative issues are crucial. According to Wilkinson *et al.* (1971), mental functions are diversely sensitive to SI [23]. However, for them, the effects of an abrupt awakening cannot last more than 3–4 min. Dinges *et al.* (1987) consider that SI effects dissipate within 30 min whatever the circumstances [28]. A similar result was found by Tassi *et al.* (1992) [35] and more recently by Sallinen *et al.* (1998) [41] with SI effects which did not exceed 15 min in a comparable partial sleep deprivation paradigm. By contrast, Rogers *et al.* (1989) found that in partial sleep deprivation conditions, more than 30 min elapsed between awakening and resumption of performance testing [42].

In general, it has been shown that sleep deprivation is a critical factor in prolonging the effects of SI and postpone the return to pre-sleep performance level. According to some authors [29, 30], when sleep deprivation is intense, SI can last up to 3–4 h after the nap. However, very recently, Jewett *et al.* (1999) reported that in conditions where sleep deprivation was totally absent and subjects raised at their habitual waketime, subjective alertness and cognitive throughput scores were decreased, and rose progressively during the first hour after awakening from an 8-h sleep, and levelled off about 2 h after awakening, suggesting an asymptotic dissipation of SI [9]. Similar findings were reported by Achermann *et al.* (1995) after night-time and daytime sleep episodes [43]. They showed a gradual increase in alertness and a gradual decrease in reaction time within the first hour after awakening. Their time course could be described by exponential functions with time constants depending on circadian placement of the nap, the smallest being for the daytime nap and the highest for the night-time nap. All these studies are in favour of a rapid dissipation of SI in normal sleep–wake conditions, a result of great interest for occupational health practitioners in a perspective of work hour management. It would mean that it is always possible to introduce short

naps during work hours, provided that workers do not suffer from intense sleep deprivation.

The question is to understand why there are so huge differences between studies concerning the time course of SI. The major reason for this discrepancy could be the way SI is measured. Some authors estimate SI by the degree of performance decrement relative to pre-sleep performance, while others consider the time it takes for performance to level off during the post-sleep period, irrespective of pre-sleep performance. Moreover, another confounding problem depends on the variety of tasks and performance indices used to assess SI.

Type of task: the compelling problem of dependent variables

Decreased performance upon sudden awakening from sleep has been shown to occur for a broad range of tasks, including simple reaction time [20, 23, 31, 45–47], complex reaction time [13, 21, 48, 49], grip strength [2, 50], steadiness and coordination [20], visual-perceptual tasks [25, 35, 36, 41, 49, 51], memory tasks [17, 18, 43, 52, 53], time estimates [54], complex-behaviour simulation tasks [1, 4, 5, 13, 55], logical reasoning [37] and a number of cognitive tasks, such as mental arithmetic, cancellation and clock reversal [9, 22, 23, 26, 34, 45, 52, 56, 57]. In each section of the present paper, we tried to emphasize the difference of magnitude SI produces on performance depending on the type of task or the performance index under review. Since most of them show a differential sensitivity to SI as measured by different time courses or different magnitude of SI effects, a variety of explanations could be yielded to account for these discrepancies. Two questions arise from this analysis. Are there specific tasks and performance indices mainly affected by SI? If yes, can we consider SI as a specific state of hypo-vigilance, or is it basically identical to hypo-vigilance produced by sleep deprivation?

In their study with four nights of partial sleep deprivation, Balkin and Badia (1988) raised the question, whether an interaction between SI and partial sleep deprivation would provide some information about the nature of SI [34]. They found that, within each night of sleep disruption/restriction, changes in error rates were basically identical than that observed from night 1 to night 4. Consequently, they argued that this similarity could be an indicator that there may be no qualitative differences in performance deficits resulting from SI vs typical sleep deprivation. However, although performance rates did not show any major difference, subjective ratings exhibited a night × session interaction, providing some evidence for qualitative differences between SI and sleepiness effects.

According to Naitoh (1981), if SI is a state of arousal still close to sleep [29], one could expect that any task likely to raise the arousal level, such as tasks with high incentive value, should counteract in an efficient manner the adverse effects of SI. Conversely, tasks involving high levels of attention should be more deteriorated than simple reaction time tasks, since attentional lapses are still more likely to occur in a state of hypovigilance. In fact, it seems that little attention has been devoted to the type of dependent variable mainly deteriorated by SI. Naitoh *et al.* (1989) summarize the time course of performance in three different studies of prolonged work with exercise [11]. A table providing the values in seven tasks either before or after a 3-h nap, clearly shows that the performance decrement observed 1–4 h after the nap mainly affects speed but not accuracy. As far as the type of task is concerned, either simple and four-choice reaction time tasks or tapping responses measured by slowing down

of motor response reveal to be sensitive to SI. By contrast, visual vigilance or word memory, measured by the percentage correct, is hardly affected by SI. It is, however, noteworthy that these studies did not include sleep deprivation before, but only after, the nap. It seems, therefore, that in those specific conditions, where sleep deprivation is absent, most studies show adverse effects of SI on speed only [13, 20]. More recently, Jewett *et al.* (1999) measured the effects of SI without prior sleep deprivation, by scoring the number of sums attempted in 2 min in an addition task irrespective of error rate [9]. They found a decreased number of additions, processed by unit time, which reflects in fact the speed of arithmetic processing. Even more recently, Bruck *et al.* (1999) measured the effects of SI, without prior sleep deprivation on a Fire Alarm simulation programme. The authors measured the number of items (trees, houses, etc.) saved from fire within a given time [55]. Again, this measure mainly reflects speed of processing with only little involvement of accuracy.

With only partial sleep deprivation, Tassi *et al.* (1992) reported increased reaction time after a 1-h nap in a spatial memory task but no effect on percentage correct [35]. However, when sleep loss was associated with SI, the data suggest that both speed and accuracy showed a dose-dependent decline. As mentioned previously, in a quasi-continuous operations paradigm including 54 h of sleep deprivation, Dinges *et al.* (1985) reported a decline in performance immediately after awakening, whatever the difficulty of the task [10]. Reaction time was measured as the time it took subjects to answer the telephone that signalled the end of the nap. The more cognitive task was the DST [16], measured by the number of correct responses per unit time which represents the speed of processing, as mentioned previously. Both tasks (simple reaction time and DST) showed a decrement immediately after awakening, even after the first nap involving very small sleep deprivation, if any.

All together, these data support the Arousal Hypothesis [58] according to which the performance decrement observed in SI would be essentially due to a lowered level of arousal (i.e. still more or less synchronized EEG) revealed by a general slowing down of cognitive processing, whatever the difficulty of the task, but no dramatic impairment of accuracy, provided there is no concomitant sleep deprivation. This hypothesis would best explain, why abrupt awakening in stage 4 is more deleterious on subsequent performance than awakening in stage 2. It could also explain why memory consolidation *per se* is not impaired during arousal from sleep [18]. However, the data including prior sleep deprivation show more confusing data, which could result from an interaction between two different states: *a hypo-arousal* state linked with SI and a *hypo-vigilance* state linked with sleep deprivation. If this is true, it could mean that arousal and vigilance are not a single functional entity and only the latter would deal specifically with attentional processes. However, this theory has to be confirmed by further experiments, which would specifically focus on this difference.

Altogether, the present data have to be taken into account in occupational perspectives, since they suggest that, in normal sleep–wake conditions, SI mainly affects speed, but keeps accuracy rather intact. Depending on the task required, it could help fixing ad hoc strategies in sleep management during work hours.

Effects of environmental factors on sleep inertia

It has been suggested that SI may be due to a decline in cerebral metabolism resulting from thermal downregulation during sleep [59]. Therefore, it could be hypothesized

that any factor likely to increase neuronal activity or cerebral metabolism and core body temperature would reduce the effects of SI. Very few studies have been devoted to environmental effects on SI. Tassi *et al.* (1992) showed that the adverse effects of SI on reaction time can be totally abolished by a moderately intense continuous noise [35]. This result could be explained by a positive alerting effect on arousal, already demonstrated by a number of studies [60–63]. However, if SI was associated with prior sleep deprivation, it could have negative effects [35, 64]. Concerning a possible alerting effect of light and physical activity on SI, it has recently been shown that for subjective alertness and cognitive performance, neither ambient lighting nor behavioural activity had a significant effect on the severity of SI and the rate of its dissipation [9]. However, it should be noted that this experiment measured only low levels of ambient light (150 lux vs 10 lux) which might be insufficient to exhibit a significant effect of this environmental factor.

Electroencephalographic modification during sleep inertia

We have found no study devoted to the simultaneous recording of EEG patterns during a progressive resolution of SI. From a physiological stand-point, it could be hypothesized that SI is a continuation of a sleepy state, and could be characterized by a more synchronized EEG than in a usual waking state, including more alpha and theta components. Comparing the EEG spectra between sleep onset and sleep offset, Ogilvie *et al.* (1992) found that the decrease in low frequency bands during sleep offset was much slower than expected, and in sharp contrast with the rapid ascendancy of these frequencies at sleep onset [65]. Although these changes occurred within a few minutes, these authors suggested that the slow evolution in delta power after awakening may provide a physiological explanation for the "sleep drunkenness" phenomenon which can be assimilated to SI.

Concerning the relationship between SI and subsequent event-related potentials, very few investigations have been carried out. Recently, Takahashi *et al.* (1998) measured P300 event-related potentials, subjective sleepiness and performance, 30 min and 3 h after short naps (15 or 45 min) [66]. The authors observed a positive correlation between EEG delta power density during the naps and P300 latency, 30 min after awakening, and a negative correlation between autonomic measures (ECG high-frequency) and P300 latency, 3 h after the naps. Their results suggest that SI increases the P300 latency immediately after the nap and that the parasympathetic predominance during the naps improve subsequent alertness.

Sleep inertia in sleep disorders

It has been already mentioned that obstructive sleep apnoea syndrome (OSAS) patients have markedly increased SI upon awakening similarly to normal individuals who have been aroused at very high rate during sleep [67]. Therefore, fragmentation of sleep by either exogenous (acoustic stimuli) or endogenous (OSAS) factors means an intensification in sleep process and resistance to return to the waking state. In the few studies measuring waking performance in narcolepsy-cataplexy, tests performed just prior to and immediately after napping did not support sustained benefits for sleeping during a short period [68, 69]. When tests made immediately upon awakening were

excluded from analysis, benefits for napping were more obvious [70]. SI *per se* has not been extensively studied in narcolepsy-cataplexy. Although clinical observations suggest that naps, and particularly sleep attacks, are not followed by significant SI [71, 72], other authors found evidence of this phenomenon following brief episodes of drowsiness and light sleep [73] or naps [74]. The latter study demonstrates clear SI following short naps in narcoleptic patients. It also showed a time-of-day effect, SI being more intense and more persistent in the middle of the afternoon than after morning naps. For these authors, the performance impairment following awakening from daytime naps in narcolepsy-cataplexy is not primarily one of post-arousal waxing and waning, microsleeps or sleep attacks, but clearly represents a progressive awakening process.

Finally, SI has to be distinguished from the Elpenor syndrome (hypnopompic state) where the sleeper is not fully awake and exhibits semi-automatic behaviours and spatial desorientation. This syndrome does occur after heavy alcohol or hypnotic consumption.

Practice Points

Sleep inertia and its possible exacerbation must not be ignored in sleep disorders; partial or total sleep deprivation (due for instance to insomnia) increases the probability of occurrence of sleep inertia

Research Agenda

We need more information about:

1. the nature of SI with usual sleep schedule and sleep amount (hypo-arousal and/or hypo-vigilance?)
2. the dependent variables (speed and accuracy) affected by SI either with or without prior sleep deprivation
3. other environmental factors likely to reduce its adverse effects (temperature, bright light)
4. possible pharmacological effects on SI. Increase in SI could be a side effect of pharmacological treatment, especially of those drugs which enhance SWS

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