

REVIEW

# Does sleep fragmentation impact recuperation? A review and reanalysis

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**SUMMARY** Studies have shown that next-day performance and alertness are impaired by sleep fragmentation procedures even when total sleep time (TST) is unaffected. Based on these studies it has been hypothesized that both the duration and continuity of sleep determine its recuperative value. This review of the literature suggests that when sleep fragmentation procedures increase the relative amount of stage 1 sleep, next-day performance and alertness are impaired. Other studies suggest that stage 1 sleep has little or no recuperative value. Total sleep time, however, is typically defined as the sum of time spent in sleep stages 1, 2, 3, 4, and REM. In the present paper it is shown that when stage 1 sleep is excluded from TST, a stronger relationship between TST and subsequent alertness and performance emerges – and the need to invoke ‘sleep continuity’ as a variable that contributes independently to recuperative sleep processes is obviated. In the same way that partial or total sleep deprivation impairs alertness and performance, it is proposed that sleep disruption also impairs alertness and performance by reducing true recuperative sleep time.

**KEYWORDS** alertness, arousals, disruption, fragmentation, performance, sleep stage

## INTRODUCTION

The physiological mechanisms by which sleep restores and sustains alertness and cognitive performance are not yet known, and the functional significance of many sleep architecture parameters remains obscure. However, one sleep parameter – sleep duration – clearly impacts the recuperative value of sleep (Kleitman 1963). Although the relationship between sleep duration and recuperation may not be linear (Lumley *et al.* 1986), it is generally true that longer sleep durations result in enhanced performance and alertness during subsequent wakefulness (see Dinges *et al.* 1997; Wilkinson *et al.* 1966).

Apparent exceptions to the principle that sleep duration

determines recuperation have been reported. In some studies it is suggested that daytime alertness can be impaired in both normal subjects and subjects with sleep disorders, despite nocturnal sleep periods of normal duration (e.g. Carskadon *et al.* 1982; Rosenthal *et al.* 1984; Stepanski *et al.* 1984; Bonnet 1985). Based on such findings, it was hypothesized that in addition to sleep duration ‘sleep continuity’ must independently mediate recuperative sleep process(es) (Bonnet 1985). That is, it has been proposed that disruptions of sleep, whether ‘external’ (e.g. noises, tones) or internal (e.g. apneas, periodic leg movements), impair recuperation even though TST may be unaffected; however, the mechanism(s) by which fragmentation reduces the recuperative value of sleep are not specified. In fact, recent reviews of the effects of sleep length, architecture, and continuity on daytime alertness fail to provide operational (and therefore testable) definitions of ‘sleep continuity’ and fragmentation (Gillberg 1995; Chugh *et al.* 1996; Kimoff 1996).

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Therefore, no formal, testable, operational definitions of sleep fragmentation and sleep continuity currently exist, and these terms necessarily reflect their generic meanings.

In this review, results from previous sleep fragmentation studies are examined with the goal of determining the extent to which sleep continuity impacts the recuperative value of that sleep.

## REANALYSIS OF PRIOR STUDIES

For inclusion, previous studies of sleep fragmentation effects on alertness and/or performance in humans had to meet the following criteria: (1) sleep was polysomnographically recorded; (2) sleep staging information (e.g. minutes or percentages of various sleep stages) was reported; (3) a baseline or control group was included, against which the effects of the fragmentation procedure could be evaluated; (4) next-day latency to sleep was objectively measured and reported (for purposes of the present paper, this constituted a measure of recuperation); and (5) the test population consisted of normal, healthy adults. Table 1 summarizes these studies in chronological order. Because short-term and long-term sleep fragmentation effects may differ (discussed below), studies of subjects from clinical or elderly populations in whom sleep fragmentation occurs chronically (see Carskadon *et al.* 1982; Rosenthal *et al.* 1984; Stepanski *et al.* 1984; Carskadon *et al.* 1982) were not included.

From each study, the following variables were identified: TST, time in bed (TIB), amount of stage 1 (either in minutes or as a percentage of TST), and latency to sleep [multiple sleep latency test (MSLT) scores]. If minutes of stage 1 were not reported, they were calculated from percentage of stage 1 as follows: Stage 1 min = [TST \* percentage stage 1]. In some instances (Levine *et al.* 1987; Magee *et al.* 1987; Walsh *et al.* 1994; Martin *et al.* 1996), sleep latencies were estimated from visual inspection of figures. Unless otherwise indicated, TST included stages 1, 2, 3, 4, and REM for all studies summarized.

### Total sleep time – recalculation

We generated one additional variable by subtracting stage 1 from reported TST. This variable is referred to as 'TST–stg1' (TST minus stage 1). Studies in which missing information and/or discrepancies made it difficult to verify our TST–stg1 calculations (e.g. Philip *et al.* 1994) are indicated with an asterisk. A study by Series *et al.* (1994) is not included in Table 1 because the amounts of stages 1 and 2 were not reported separately.

## RESULTS

Table 1 summarizes the results of our reanalysis. In most instances, subtracting stage 1 substantially reduced TST, and improved the correspondence between sleep duration and next-day alertness. The reanalyzed results from Levine *et al.* (1987) are illustrative; subjects were allowed 100 min of recovery sleep

following 24 h of sleep deprivation. Sleep was fragmented every 1, 3, or 5 min, or was undisturbed. A fifth group obtained no recovery sleep. Levine *et al.* (1987) reported that TST was equivalent among the four sleep conditions. However, higher rates of sleep fragmentation resulted in lower mean sleep latencies during subsequent wakefulness. These results therefore suggested that fragmentation rate systematically affects recuperation, independent of TST. Figure 1 illustrates data from the Levine *et al.* (1987) study – TST (divided into sleep stages) is shown as a function of fragmentation rate. Corresponding subsequent sleep latencies are overlaid. TST (sum of all sleep stages) does not appear to decrease as a function of fragmentation rate. However, if stage 1 sleep (which may have little or no recuperative value – see below) is subtracted from TST (TST–stg1) the correspondence between sleep duration (now defined as TST–stg1) and subsequent recuperation (measured by mean sleep latency) is apparent.

Data from Magee *et al.* (1987) provide further illustration. In their study, subjects were required to perform a breathing response to auditory stimuli presented every one vs. four min during sleep. A control group received no tones. Table 1 lists TST for the control, 4-min, and 1-min groups, respectively. Despite roughly equivalent TSTs, next-day sleep latencies differed significantly among groups. In particular, the difference between mean TSTs for control vs. 1-min groups (429.1 min vs. 373.4 min, respectively) would not be expected to account for the large difference (7 min) in subsequent mean sleep latencies. Magee *et al.* (1987) also reported a significant positive correlation between stage 1 amounts and next-day sleepiness. As would therefore be expected, TST–stg1 corresponds well with subsequent mean sleep latency (Table 1). In fact, the correlation between TST–stg1 and mean sleep latency conducted on all data presented in Table 1 is substantial ( $r = 0.58$ ,  $P < 0.05$ ). However, because TST–stg1 and TST are highly correlated ( $r = 0.95$ ,  $P < 0.05$ ), the correlation between TST and mean sleep latency from Table 1 is also significant ( $r = 0.55$ ,  $P < 0.05$ ).

Bonnet (1985) also reported that the percentage of sleep time spent in stage 1 increased on fragmentation nights even though TST did not change. Although TST–stg1 could not be calculated from the information provided, it can be inferred from the substantially increased percentage of stage 1 (6.4% vs. 31% for baseline vs. fragmentation night 1, respectively) that TST–stg1 was correspondingly reduced. Performance measures, including reaction time, addition, and Digit Symbol Substitution were impaired following fragmentation nights.

## ARE SLEEP FRAGMENTATION AND SLEEP DEPRIVATION EQUIVALENT?

The effects of sleep fragmentation are indistinguishable from the effects of total or partial sleep loss. Both result in comparable alertness and performance deficits, and also in subsequent changes to sleep architecture that are characteristic of recovery sleep. In Bonnet's studies (1985, 1986a and b, 1987, 1989), subjects were allowed up to two nights of undisturbed sleep

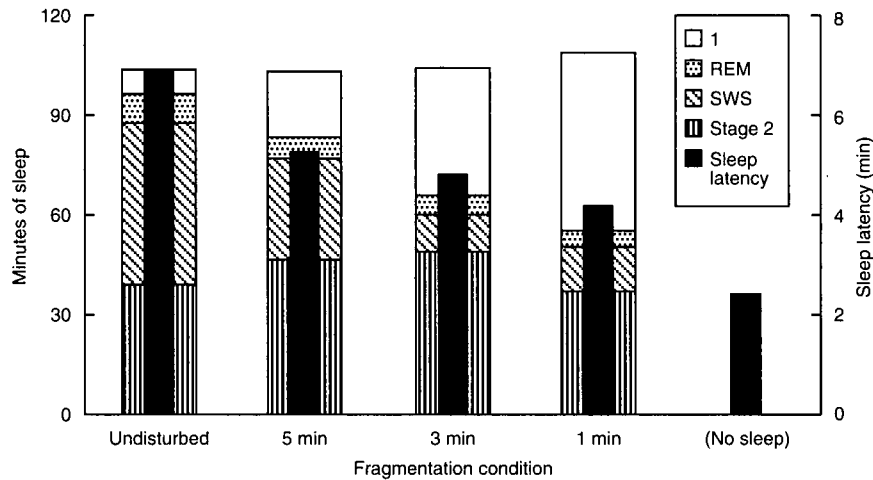
**Table 1** Results adapted from studies of sleep fragmentation, reanalyzed to determine total sleep time minus stage 1 (TST–stg1)

Author	Group/Night	TST (TIB)	TST–Stage 1	MSLT	Stage 1 Min	Stage 1%
<b>Bonnet (1985)</b>	Baseline	389.0† (**)	***	N/A	*	6.4*
	Tone every 1 min stage 2, Night 1	334.0† (**)	***	N/A	*	31.0*
† Listed as 'time asleep' in Bonnet (1985).						
* In Bonnet (1985) per cent stages W, 1, 2, 3, 4 and REM sum to less than 100%—unclear whether per cent stage 1 should be per cent TST or TIB (latter not provided).						
** TIB not provided and could not be calculated from available data.						
*** Could not be calculated from available data.						
<b>Bonnet (1986a)</b>	Baseline <sup>++</sup>	388† <sup>++</sup> (396**)	372 <sup>++</sup>	9.7 <sup>++*</sup>	16.0*	****
	Tone every 10 min stage 2, Night 1	339† (398**)	299	5.1	40.0	****
	2.5 h sleep, then tone every onset stage 2, Night 1	302† (425**)	188	6.8	114.0	****
	Tone every 1 min stage 2, Night 1	274† (408**)	142	3.3	132.0	****
	64 h total sleep deprivation	—	—	2.6	—	—
† TST calculated as the sum of minutes of stages 1, 2, SWS and REM provided in Bonnet's (1985) Table 1. Calculated values differ from 'total sleep time' values provided by Bonnet (1985).						
<sup>++</sup> Mean of baseline nights collapsed across all fragmentation conditions.						
** TIB not provided; calculated as TST multiplied by sleep efficiency (latter provided in Bonnet, 1987).						
**** Stage 1 per cent not provided and not required to calculate stage 1 minutes since the latter were provided.						
<b>Bonnet (1986b)</b>	Baseline, w/SWS condition	426 (458**)	407.7	11.3	18.3*	4.0*
	Tone every 10 min stage 2 w/SWS, Night 1	368 (491**)	294.3	9.0	73.7*	15.0*
	Baseline, SWS deprivation condition	436 (454**)	417.8	7.1	18.2*	4.0*
	Tone every 10 min stage 2, SWS dep, Night 1	386 (483**)	318.4	4.7	67.6*	14.0*
* In Bonnet (1986b) sum of percent stages W, 1, 2, 3, 4 REM and M=100%; thus stage 1 minutes should be calculated as percent TIB (not provided) rather than percent TST.						
** TIB not provided; calculated as TST dividend by sum of percent stages 1, 2, 3, 4, and REM in Bonnet (1986b).						
<b>Bonnet (1987)</b>	Baseline, awakening condition	419 (455)	394.0	7.8	25.0*	5.5*
	Tone every 2 min stage 2/REM, Night 1	326 (458)	243.6	†	82.4*	18.0*
	Tone every 2 min stage 2/REM, Night 2	345 (461)	271.2	4.7	73.8*	16.0*
	Baseline, movement condition	413 (440)	392.8	7.5	20.2*	4.6*
	Tone every 2 min stage 2/REM, Night 1	354 (456)	267.4	†	86.6*	19.0*
	Tone every 2 min stage 2/REM, Night 2	378 (462)	304.1	4.4	73.9*	16.0*
	Baseline, EEG condition	422 (443)	394.1	7.7	27.9*	6.3*
	Tone every 2 min stage 2/REM, Night 1	390 (456)	326.2	†	63.8*	14.0*
	Tone every 2 min stage 2/REM, Night 2	393 (461)	333.1	4.1	59.9*	13.0*
	* In Bonnet (1987) sum of per cent stages W, 1, 2, 3, 4 and REM=100%; thus stage 1 per cent and minutes calculated as per cent TIB rather than per cent TST.					
† Daytime sleep latency following Night 1 not provided.						
<b>Levine et al. (1987)</b>	100 min undisturbed sleep	103.3 (105.7)	96.3	7.0†	7.0*	6.8*
	Tone every 5 min sleep‡ during 100 min sleep	102.6 (106.9)	83.3	5.4†	19.3*	18.8*
	Tone every 3 min sleep‡ during 100 min sleep	104.3 (116.0)	66.2	4.8†	38.1*	36.5*
	Tone every 1 min sleep‡ during 100 min sleep	100.6 (109.4)	47.1	4.2†	53.5*	53.2*
	40 h total sleep deprivation	—	—	2.4†	—	—
* In Levine et al. (1987) sum of per cent stages 1, 2, 3/4 and REM=100%; thus stage 1 per cent and minutes calculated as per cent TST.						
† Sleep latencies estimated from Levine et al. (1987), Figure 3.						
‡ Sleep stage not specified.						
<b>Magee et al. (1987)</b>	Control	429.1 (460.4)	380.3	9.6†	48.8*	10.6*
	Tone every 4 min stage 2	423.1 (470.4)	348.3	9.6†	74.8*	15.9*
	Tone every 1 min stage 2	373.4 (476.9)	191.7	4.2†	181.7*	38.1*
* In Magee et al. (1987) sum of per cent stages W, 1, 2, 3, 4 and REM=100%; thus stage 1 per cent and minutes calculated as per cent TIB rather than per cent TST.						
† Sleep latencies estimated from Magee et al. (1987), Figure 1, and collapsed across time of day.						
<b>Stepanski et al. (1987)</b>	Baseline, 8–9 condition	387 (**)	307.3	12.9	79.7	20.6
	8–9 tones per hour of TST, Night 1	406 (**)	314.6	10.3	91.4	22.5
	Baseline, 4–5 condition	412 (**)	333.7	15.1	78.3	19.0
	4–5 tones per hour of TST, Night 1	400 (**)	318.0	12.5	82.0	20.5
	Baseline, 8–9/4 hr condition	419 (**)	354.5	12.0	64.5	15.4
	8–9 tones per hour of TST for 1st 4 hrs, Night 1	433 (**)	362.0	10.2	71.0	16.4
* In Stepanski et al. (1987) sum of per cent stages 1, 2, 3/4 and REM=100%; thus stage 1 minutes calculated as per cent TST.						
** TIB not provided.						

continued

Table 1 continued

Author	Group/Night	TST (TIB)	TST-Stage 1	MSLT	Stage 1 Min	Stage 1%
<b>Bonnet (1989a) Experiment 1</b>	Baseline, 10/20 condition	372 (411)	352	7.2	20.0	****
	Alternating 10 min undisturbed sleep/20 min w/tone every 1 min stage 2, Night 2*	303 (426)	235	3.6	68.0	****
	Baseline, 20/40 condition	384 (407)	357	6.8	27.0	****
	Alternating 20 min undisturbed sleep/40 min w/tone every 1 min stage 2, Night 2*	326 (426)	264	2.9	62.0	****
	Baseline, 40/80 condition	377 (412)	355	6.0	22.0	****
	Alternating 40 min undisturbed sleep/80 min w/tone every 1 min stage 2, Night 2*	328 (431)	262	3.3	66.0	****
<b>Bonnet (1989a) Experiment 2</b>	Baseline, 20 min condition	341 (367)	318	7.3	23.0	****
	Tone every 20 min sleep, Night 2*	342 (380)	314	8.1	28.0	****
	Baseline, 40 min condition	335 (366)	314	11.5	21.0	****
	Tone every 40 min sleep, Night 2*	326 (382)	296	8.9	30.0	****
	Baseline, 2/4 condition (N=3)	316 (346)	292	4.2	24.0	****
	Tone every 2 min w/ 4 min awake, Night 2* (n=3)	283 (382)	202	2.8	81.0	****
* Data from Night 1 were not provided.						
**** Stage 1 per cent not provided and not required to calculate stage 1 minutes since the latter were provided.						
<b>Bonnet (1989b)</b>	Baseline, 10/20, young adults (age x=22 yrs)	372 (411)	352	7.7	20.0	****
	Alternating 10 min undisturbed sleep/20 min tone every 1 min stage 2, Night 1	326 (422)	252	2.7	74.0	****
	Baseline, 10/20, older adults (age x=63 yrs)	363 (430)	318	13.7	45.0	****
	Alternating 10 min undisturbed sleep/20 min w/tone every 1 min stage 2, Night 1	304 (444)	221	12.6	83.0	****
**** Stage 1 per cent not provided and not required to calculate stage 1 minutes since the latter were provided.						
<b>Gillberg &amp; Akerstedt (1994)</b>	480 min undisturbed sleep	456 (480)	447.4	11.0†	8.6	****
	240 min undisturbed sleep	225 (240)	217.5	7.9†	7.5	****
	Tone every onset of SWS for 240 min	228 (240)	213.7	6.3†	14.3	****
	No sleep	—	—	1.4†	—	—
† Sleep latencies collapsed across time of day.						
**** Stage 1 per cent not provided and not required to calculate stage 1 minutes since the latter were provided.						
<b>Philip et al. (1994)</b>	Baseline	392.2 (***)	351.4	15.4	40.8*	10.4
	Tone every 30 sec stage 2 sleep	387.8 (***)	343.1 (298.6)**	8.8	44.7 (89.2)**	23.0
* Stage 1 min calculated as per cent of TST.						
** For fragmentation night, stage 1 minute provided in Philip et al. (1994) as listed above (44.7 min) equals only 11.5% of TST, although Philip et al. (1994) indicate that stage 1% is 23% of TST. Value in parenthesis indicates calculation based on minutes of stage 1 as 23% of TST.						
*** TIB not provided.						
<b>Roehrs et al. (1994)</b>	Baseline	391 (471)**	348.8	14.3	42.2*	10.8*
	Tone every 2 min sleep, Night 1	379 (426)**	310.8	9.8	68.2*	18.0*
* In Roehrs et al. (1994) sum of percent stages 1, 2, 3/4 and REM = 100%; thus stage 1 minutes calculated as per cent TST.						
** TIB calculated as TST divided by sleep efficiency.						
<b>Walsh et al. (1994)</b>	Baseline, no disruption condition	465.8 (510)	415.8	8.7†	50.0	****
	No disruption, Night "3" (first exp. night)	422.7 (480)	375.5	9.9†	46.9	****
	Baseline, Control disruption condition	461.8 (510)	405.0	9.6†	56.8	****
	Control disruption, Night "3" (first exp. night)	384.2 (510)	292.5	7.5†	91.8	****
	Baseline, SWS deprivation condition	460.9 (510)	409.3	9.0†	51.6	****
	SWS deprivation, Night "3" (first exp. night)	409.0 (510/540)	325.1	6.9†	81.9	****
† Sleep latencies estimated from Walsh et al. (1994), Figure 1, and collapsed across time of day.						
**** Stage 1 per cent not provided and not required to calculate stage 1 minutes since the latter were provided.						
<b>Martin et al. (1996)</b>	Undisturbed sleep	400 (N/A)	387.2	10.8†	12.8*	3.2
	Tone every 2 min stage 2 sleep	396 (N/A)	374.6	7.5†	21.4*	5.4
* In Martin et al. (1996) sum of per cent stages 1, 2, 3, 4 and REM = 100%; thus stage 1 minutes calculated as per cent TST.						
† Sleep latencies estimated from Martin et al. (1996), Figure 2, and collapsed across time of day.						



**Figure 1.** TST (divided by individual sleep stage contribution) as a function of fragmentation rate, from Levine *et al.* (1987). Minutes of each sleep stage are indicated. Subsequent sleep latencies (estimated from Figure 3 in Levine *et al.* 1987) are overlaid as solid black bars.

after one or more nights of fragmented sleep. Slow wave sleep (SWS) rebound during subsequent recovery (undisturbed) night-time sleep suggests that the recuperative value of the fragmented sleep was compromised – just as if sleep had been restricted in duration. In fact, Bonnet (1986a) found that next-day mean sleep latency was not significantly different between a group whose sleep was fragmented every minute (and who obtained over 2 h of stage 1 sleep – just under 50% of that groups TST) and a group totally deprived of sleep for 64 h (2.9 and 2.6 min, respectively). In addition, other results show that recovery sleep following either sleep fragmentation or sleep restriction procedures is characterized by increased arousal thresholds (see Stepanski *et al.* 1987; Badia *et al.* 1985; Balkin *et al.* 1985). In these studies, increased frequency and/or amplitude (volume) of the presented stimulus was needed to produce arousal. This sort of arousal threshold increase has been attributed to ‘habituation’ (a term more generally used to describe than explain the phenomenon of decreased responding across stimulus presentations). However, a more plausible explanation may be that increased arousal thresholds were the result of mounting sleep debt both within and across nights (Balkin *et al.* 1985; Bonnet 1985). Again, however, it is not clear whether this putatively mounting sleep debt should be attributed solely to increased percentage of stage 1 sleep since stimulus presentation procedures may have concomitantly increased the number of arousals from baseline to fragmentation nights.

#### INTERACTIONS BETWEEN EXTANT SLEEPINESS, SLEEP FRAGMENTATION, AND STAGE 1 SLEEP AMOUNTS

Some studies suggest that sleep fragmentation can result in next-day alertness deficits even when there is no apparent increase in stage 1 sleep (Bonnet 1989b; Roehrs *et al.* 1994; Stepanski *et al.* 1987; Martin *et al.* 1996). For example, Stepanski *et al.* (1987) studied the effects of sleep fragmentation

over two consecutive nights on next-day sleep latency and auditory vigilance performance. Five subjects (aged 18–32 years) were tested in each of three sleep fragmentation conditions. The conditions consisted of a baseline (no fragmentation) night followed by two consecutive (experimental) nights in which: (a) arousals were induced with auditory stimuli at a rate of 7–9 per hour during the 8-h sleep period (Condition 1); (b) arousals were induced at a rate of 4–5 per hour during the 8-hour sleep period (Condition 2); and (c) arousals were induced at a rate of 7–9 per hour for the first 4 h of the sleep period, with no arousals induced during the last 4 h of the sleep period (Condition 3). In that study, a nonsignificant trend toward increased stage 1 was evident on the first night of fragmentation, followed by reduced stage 1 on the second night (significant only for Condition 1). Despite few significant within-group sleep stage-related differences across nights (perhaps due to the small sample size), mean sleep latencies declined (compared to baseline) under all sleep fragmentation schedules after the second night of experimental sleep fragmentation.

The Stepanski *et al.* (1987) study appears to support the hypothesis that sleep fragmentation per se reduces the recuperative value of sleep, independent of sleep duration effects. However, an alternative interpretation is possible – in that study, the reported percentages of stage 1 sleep (ranging from 15.4–20.6%) on baseline nights were higher than normal (usual percentage stage 1 sleep for this age group is approximately 10%; Williams *et al.* 1974). The percentages of stage 1 were also elevated on subsequent experimental nights, but not significantly more than on baseline nights. Therefore, the sleepiness evident on the final testing day may have been due to cumulative elevated stage 1 sleep across three nights (baseline plus two experimental nights).

Although stage 1 sleep is an operationally and somewhat arbitrarily defined construct (Rechtschaffen and Kales 1968), it undoubtedly reflects a unique underlying brain state – a state of transition between wakefulness and the deeper sleep stages.

Since sleep fragmentation procedures and sleep restriction procedures result in increased pressure to deeper sleep stages (see Balkin *et al.* 1985), it is likely that the amount of stage 1 sleep that follows each arousal is reduced as sleep debt increases. Thus, in the Stepanski *et al.* (1987) study, it is also possible that considerable amounts of stage 1 sleep were scored as other deeper sleep stages. In that study (Stepanski *et al.* 1987), the epoch size used for sleep scoring (30 s) would have precluded the detection of stage 1 episodes that lasted less than 15 s. As Boselli *et al.* (1998) point out, by ASDA (1992) criteria the length of an arousal cannot exceed 50% of the scoring epoch length (ASDA 1992). Thus, by definition arousal time is not measured using traditional scoring criteria (Rechtschaffen and Kales 1968; ASDA 1992). Rather, arousals are quantified by tabulating their frequency. It may be that if actual time spent in an arousal (arousal time) was subtracted from TST (clearly a time-consuming procedure), that TST would be decreased substantially in the various sleep fragmentation studies.

Another study appears to indicate that TST–stg1 bears no relationship to next-day alertness. Bonnet (1987) studied the effects of full awakenings, movements, or EEG changes resulting from auditory stimuli (tones) presented during sleep on next-day performance and alertness. Bonnet (1987) reported that the three conditions resulted in differential reductions in TST on fragmentation night 2, with greatest reductions in the awakening condition. This was also true for TST–stg1 (Table 1). Despite differential reductions in TST and TST–stg1, the groups showed similar next-day impairment in alertness (sleep latencies of 4.7, 4.4, and 4.1 min for Awake, Movement, and EEG groups, respectively). However, next-day alertness data were provided only following the second fragmentation night. It may be that next-day alertness differed among the three groups following the first fragmentation night. Also, baseline daytime alertness measures were only slightly above the pathological range (sleep latencies of 7.8, 7.5, and 7.7 min for Awake, Movement, and EEG groups, respectively; Bonnet 1987). This suggests that subjects entered the study already substantially sleep deprived, even prior to the fragmentation nights. Sleep debt could account for the low baseline alertness measures, and would reduce the sensitivity of the alertness measure to differential sleep fragmentation conditions. The results of this study suggest that the relationship between stage 1 sleep and fragmentation rate depends, at least in part, on the extant level of sleep debt. Such may also be the case for those with chronically fragmented sleep (e.g. sleep apnea patients) in whom pressure for sleep may be so great that individuals rapidly transition from stage 1 to a more recuperative sleep stage following each sleep apnea event. In other words, extent of sleep disturbance may determine the recuperative value of sleep only insofar as differential disturbances can cause differential increases in stage 1. The extent to which arousals result in episodes of stage 1 sleep may determine both the recuperative value of that sleep and reflect the extant level of sleep debt.

## AN ATTEMPT TO ISOLATE THE PUTATIVE 'SLEEP CONTINUITY FACTOR'

The notion that stage 1 sleep confers no recuperative benefit has been suggested or implied previously in papers reviewing the effects of sleep apnea on alertness (see Roehrs *et al.* 1989; Chugh *et al.* 1996). However, in none of those studies was stage 1 subtracted from TST, despite reports of a significant correlation between stage 1 amounts and alertness (more stage 1 correlating with lower alertness).

The most ambitious test of the sleep continuity hypothesis was conducted by Bonnet (1986a). In this study, performance and sleep latencies were tested following nights in which sleep was disrupted (with an auditory stimulus): (a) every minute (Condition A); (b) every 10 min (Condition B); and (c) each time a sleep spindle or k-complex appeared for the remainder of a sleep period following a 2.5-h undisturbed sleep period (Condition C). In Condition D, testing occurred following 64 h of total sleep deprivation. Statistical analyses revealed no significant differences among Conditions A, B, C and D for relevant night-time sleep parameters, nor for next-day sleep latency measurements [perhaps due to the small *n* for each condition (*n*=8), especially in Condition C (*n*=5)]. Conditions B and C were then selected for further comparison because of some interesting but nonsignificant trends in the data; although sleep duration (including TST–stg 1) was nonsignificantly longer in Condition B, performance was better (and mean sleep latency nonsignificantly longer) in Condition C. Despite the lack of statistical significance, it was asserted that these data support the concept of 'sleep continuity' as an explanatory variable. That is, it was suggested that differences in sleep continuity could explain the finding that the direction of performance and sleep latency differences between Conditions B and C were not consistent with what one might predict if recuperation during sleep were solely a function of sleep duration (including TST–1). Acceptance of this conclusion necessitates that the importance of statistical significance be discounted. It also necessitates that one assume the experimental manipulation satisfactorily isolated the variable of interest, i.e. sleep continuity. However, sleep continuity was never operationally defined nor explicitly tested in this study – rather, it is presumed to account for the unexplained (or error) variance between Conditions B and C. Thus, while the Bonnet (1986a) results are tantalizing, it is not clear that the experimental conditions indeed isolated a sleep continuity factor. In short, this study does not provide rigorous evidence that sleep continuity significantly and independently impacts the recuperative value of sleep.

## STAGE 1 CHANGES DURING SELECTIVE SLEEP STAGE DEPRIVATION

Sleep disruption is commonly used in selective sleep stage deprivation studies. In one such study, Karni *et al.* (1994) evaluated the effects of REM vs. SWS deprivation on perceptual skill learning, and reported that REM deprivation disrupted

learning while SWS deprivation did not. Although Karni *et al.* (1994) reported that TST did not differ between groups, it is clear that stage 1 amounts were greater, and stage 2 amounts were reduced, for the REM deprivation group – suggesting that recuperative sleep times (TST–stg1) may have been reduced for the REM deprivation group. Thus, the results of Karni *et al.* (1994) could be reinterpreted as an effect of relative sleep restriction rather than of stage REM deprivation. Glovinsky *et al.* (1990) attempted to evaluate the differential effects of stage 2 vs. REM deprivation on recuperation, as measured by next-day sleepiness. They found no differences in next-day sleepiness between stage 2-deprived vs. REM-deprived groups, and they also reported no differences in TST between the two groups. However, compared to baseline, TST–stg1 was decreased comparably in the stage 2 and REM deprivation conditions. Next-day sleepiness was also comparably reduced for both groups compared to baseline. Thus, although this study may not have determined whether REM vs. stage 2 is differentially recuperative, it does suggest that decreased TST–stg 1 results in decreased alertness.

Other selective sleep stage deprivation studies have also used disruption or fragmentation procedures. Bonnet (1986b) compared the effects of SWS deprivation (SWS–) to a procedure in which sleep was disrupted to the same degree but SWS was allowed (SWS+). TST did not differ between SWS– and SWS+ groups; however, next day sleepiness increased in the SWS– group. Although these results would appear to suggest that SWS is differentially recuperative (compared to other sleep stages) it is unclear whether both the SWS– and SWS+ groups obtained the same amounts of stage 1 (insufficient information was provided to calculate TST–stg1). Increased stage 1 in the SWS– group (and consequent decreased TST–stg1) could cause next day alertness impairments. Walsh *et al.* (1994) also attempted to eliminate SWS in one group (SWS–) but not in another group (SWS+) while equating the number of disruptions between the two groups. In that study, stage 1 was comparably increased in both the SWS– and SWS+ groups. Likewise, next-day sleepiness was comparably increased in both groups. In short, these studies of selective sleep stage deprivation suggest that sleep duration (as measured by TST–stg1) is critical to alertness and performance. They also raise the possibility that selective sleep stage deprivation effects on next-day performance and alertness are due to differential effects on stage 1 sleep amounts (i.e. TST–stg1) rather than the particular sleep stage (2, REM, or SWS) under study.

Sleep fragmentation procedures that increase stage 1 amounts degrade next-day alertness and performance. Subjects who obtain mainly stage 1 sleep perform as poorly as subjects allowed no sleep at all (Bonnet 1986a). These findings suggest that stage 1 sleep has little or no recuperative value. The findings also imply that sleep fragmentation effects are not necessarily independent of sleep deprivation effects. As suggested here, when stage 1 is subtracted from TST, substantial reductions in TST, and a clearer relationship between sleep time and measures of recuperation, emerge.

## DO OTHER SLEEP STAGES HAVE DIFFERENTIAL RECUPERATIVE VALUE?

A change in stage 1 sleep amounts may not be the only factor that determines next-day alertness level in sleep fragmentation studies. The present reanalysis reveals that whenever stage 1 is increased, next-day alertness is decreased. The converse is not necessarily true – failure to increase stage 1 sleep does not unequivocally mean that next-day alertness will not be decreased (see Stepanski *et al.* 1987). Thus, some factor other than stage 1 may also account for reduced next-day alertness. The most obvious place to search for such a factor would be within the polysomnogram itself. Although not the focus of the present paper, it should be noted that fragmentation procedures also alter amounts of stages 2, SWS, and REM. For example, the data from Levine *et al.* (1987) indicated that their fragmentation procedure dramatically reduced SWS amounts, whereas stages 2 and REM were less affected (see Fig. 1). As noted above, since the relative recuperative values of SWS, REM and stage 2 are unknown, it is possible that changes in the relative distribution (or absolute amounts) of sleep stages other than stage 1 presents a confound. For example, in the Bonnet (1986a) paper discussed above, it was asserted that nonsignificant trends show that sleep continuity impacts the recuperative value of sleep. However, it was also found that the amount of SWS plus REM was the best sleep stage-derived predictor of next day performance and alertness. Since the experimental condition that putatively resulted in the most 'continuous' sleep was the same condition that resulted in the most SWS plus REM, these potential explanatory factors were confounded – and it is not difficult to surmise a causal relationship between sleep continuity and amounts of these sleep stages. Power in various electroencephalographic frequency bands (e.g. delta power) also may index recuperative value and may provide a better alternative to traditional visual sleep scoring. Frequency band power, although related to visual sleep stage scoring, accumulates in a continuous and continuously varying fashion rather than in the stepwise fashion which is the basis for standard (Rechtschaffen and Kales 1968) sleep staging. Also, it has the advantage of being easily operationalized and objectively quantified.

It could be argued that the MSLT is not a valid measure of the recuperative value of sleep (for review, see Johns 1998), i.e. that the latency to sleep does not necessarily reflect underlying sleep debt. For example, Hartse *et al.* (1980) showed that latency to sleep was also affected by the instructions given to subjects. In addition, the MSLT is subject to floor effects, thus possibly limiting its sensitivity. Despite these shortcomings, the MSLT has been shown to discriminate levels of sleep in normal, healthy adults – the subject population for studies reviewed in the present paper (Carskadon and Dement 1975, 1979). Not insignificantly, the MSLT also was the only dependent measure common among most of the studies reviewed in Table 1. This is not surprising since – despite its limitations – the MSLT is still probably the most widely used, validated measure of sleepiness.

## UNRESOLVED ISSUES

At least two issues remain unresolved. First, the neurobiological mechanisms underlying sleep stage 1 are unknown – is stage 1 a ‘neutral’ state, in which neither a net gain or net loss in ‘recuperation’ is incurred? Or is stage 1 neurophysiologically similar to wake, causing the same sleep deprivation-related changes in the brain – as implied by Bonnet’s (1986a) findings that a night of mainly stage 1 is no more recuperative than total sleep deprivation? Since sleep debt generally builds during wakefulness and is reduced during subsequent sleep, and since stage 1 serves as the bridge from wakefulness to sleep, it is reasonable to hypothesize that stage 1 sleep constitutes a neutral ‘inflection point’ – a state during which the degradative effects of wakefulness have ceased but the restorative processes that characterize true sleep have not yet commenced.

Second, all studies of disrupted sleep to date have potentially confounded rate of fragmentation (rate of stimulus presentation) with sleep staging. Thus, whether undisturbed sleep is ‘... a prerequisite for a functioning linkage between brain systems involved in central nervous activating systems’ (Stoohs 1996; p. 1418), independent of sleep stage, is unknown. In a strict sense, the notion that sleep continuity mediates the recuperative value of sleep implies that many short segments of sleep should be less recuperative than a single segment of sleep (total duration and stage held constant) because the latter has greater continuity. Despite attempts to test the sleep continuity hypothesis (work by Bonnet), it has yet to be determined conclusively whether a single, longer bout of a particular sleep stage is more restorative than multiple, shorter bouts of the same stage. This hypothesis may be impossible to test if stage 1 amounts are inextricably confounded with other factors such as number of arousals.

## CONCLUSIONS

This review of the experimental evidence (and reanalysis of selected study data) reveals no clear, data-based rationale for choosing between the two competing hypotheses. That is, there is no scientific evidence to support the conclusion that recuperation during sleep is a function of duration and continuity vs. duration alone (albeit with a modified definition of duration that excludes stage 1). We have attempted to show that in previously published studies purporting that sleep continuity is critical for recuperation, the results can also be explained by hypothesizing that stage 1 sleep has little or no recuperative value. Since increased arousal rates are typically accompanied by an increased percentage of stage 1 sleep, the duration of putatively restorative sleep (i.e. TST–stg1) is reduced when sleep fragmentation procedures are implemented.

Although sleep continuity may not be experimentally extricable from sleep duration (TST–stg1), this does not mean that both hypotheses are equally cogent. As noted above, increased sleep durations generally result in increased levels of alertness and cognitive performance – it cannot reasonably be argued that sleep duration does not at least partially determine

the recuperative value of sleep. However, as discussed in this paper, the extent to which the continuity of sleep constitutes an independent factor critical to recuperative sleep processes is much less certain. By simply and reasonably hypothesizing that stage 1 sleep has no (or significantly reduced) recuperative value, the straightforward notion that recuperation is a function of sleep duration alone is confirmed and preserved. We contend that the alternative hypothesis – i.e. that sleep continuity exerts an additional and independent impact on recuperative sleep processes – is not convincingly supported by the experimental evidence, and less parsimonious than the notion that recuperation during sleep is better accounted for by TST–stg1 than by TST.

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