exposure of the face to cold air while driving, proved ineffective when tested experimentally.[3] Our study and other sleep deprivation studies[4] have additionally shown a mismatch between perception of impairment and actual impairment. Thus, the benefit of many of the strategies that Merenstein indicates were "obvious to all who have experienced nights and subsequent days with less than optimal sleep" may only be the *perception* of improved alertness.

Well-controlled experimental investigations are required to separate "actual" from "perceived" improvements in performance. Only under such conditions can we assess the real value of alertness strategies for mitigating the effects of sleep loss in the medical setting.

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RESEARCH LETTER

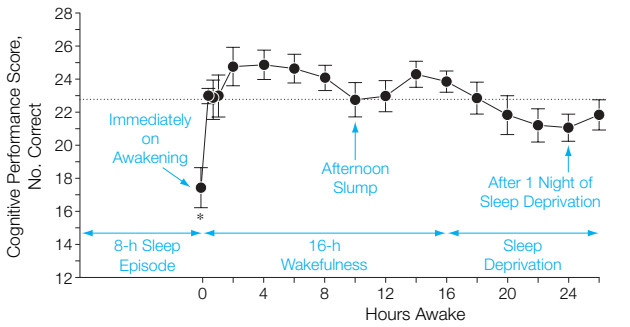
Effects of Sleep Inertia on Cognition

To the Editor: The state of impaired cognition, grogginess, and disorientation commonly experienced on awakening from sleep has been referred to as sleep inertia.[1] Morning cognitive impairment associated with sleep inertia has been described as modest[1] but has not been well quantified. Because some safety-sensitive occupations require individuals to perform immediately on awakening, we directly compared the effects of sleep inertia and sleep deprivation on cognition.

Methods. Participants were 9 paid volunteers (mean [SD] age, 29.1 [6.4] years; range, 20-41 years; 8 men) with no medical, psychiatric, or sleep disorders. They were free of medications, alcohol, nicotine, recreational drugs, and caffeine for 3 weeks before the study, verified by toxicologic analysis. Persons who had performed shift work in the past 3 years or traveled across more than 1 time zone in the previous 3 months were excluded.

Participants maintained sleep schedules of about 8 hours per night for 3 weeks before laboratory admission, verified by sleep logs, call-in to a time-stamped voice mail system, and 1 week of wrist actigraphy (Actiwatch-L, Mini Mitter, Bend, Ore). The inpatient protocol consisted of 6 baseline days with 8 hours of sleep per night at participants' habitual bedtimes, followed by monitored sleep deprivation for 26 hours using a constant-routine bed-rest protocol[2] in dim light (<8 lux). Cognitive performance was examined following the sixth laboratory night using a standardized addition test that presented a series of randomly generated pairs of 2-digit numbers.[2] Participants had practiced this test across each of the 6 baseline days. Performance was assessed approximately 1, 21, 41, 61, and 120 minutes after electroencephalogram-verified awakening, then every 2 hours. Participants provided written informed consent, and institutional human research committees approved the protocol.

Figure. Cognitive Performance on Awakening From Sleep Compared With Subsequent Sleep Deprivation



The group mean (horizontal dotted line) across the sleep inertia period and 26-hour awake period has been added to the deviation from the mean scores for the number of correct responses in 2 minutes to permit overall assessment of the magnitude of performance impairment. Error bars indicate SEM; asterisk, differs from all subsequent time points at $P \leq .01$.

Scores were transformed into deviation from the mean to control for differences in aptitude. Percentage of peak performance was calculated from raw data. Repeated-measures analyses of variance with Hunyh-Feldt correction for sphericity[3] were performed with STATISTICA, version 6.0 (StatSoft Inc, Tulsa, Okla). Using modified Bonferroni correction for planned comparisons among the first 4 tests following awakening, and between these tests and all subsequent tests, a corrected α level of $P<.0138$ was considered statistically significant to maintain the familywise error rate at $P<.05$.[3]

Results. On day 7, testing began a mean of 73 (SD, 16) seconds after electroencephalogram-verified awakening. In the 30 minutes prior to scheduled wake time, participants were awake a mean of 26 (SD, 28; range, 0-60) seconds. Six participants were in stage 2 sleep and 3 were in rapid eye movement sleep prior to awakening. Cognitive performance immediately on awakening from sleep was worse than performance at all subsequent points measured across 26 hours of sleep deprivation ($P \leq .01$ for all comparisons), with a mean of 65% of peak performance

(SD, 16%; range, 35%-85%) (FIGURE). Between 21 and 61 minutes of wakefulness, the mean cognitive performance ranged from 83% to 86% of peak performance and was not significantly different from performance at subsequent points. The results for the female participant were not significantly different from the results for the male participants.

Comment. Cognitive performance immediately on waking was worse than performance during subsequent sleep deprivation. We observed severe impairments within the first 3 minute of awakening, and others have reported severe performance impairments lasting up to 10 minutes following awakening, with effects on performance detectable for at least 2 hours.[1,2] The degree of impairment is likely to be clinically important, given that changes in cognitive performance following 24 hours of sleep deprivation are reported to be comparable with those due to alcohol intoxication.[4,5]

Our performance test required cognitive operations that use short-term memory, counting skills, speed of cognitive processing, and number fact and lexical retrieval. These skills are critical for many occupational tasks; however, the test did not simulate a specific operational task or indicate the effects of sleep inertia on performance in an operational setting. Further studies should measure this and should include more participants and broader populations. To extrapolate to medical personnel, such studies should measure the effects of interruption of naps and recovery sleep in on-call sleep-deprived individuals.

However, given that sleep inertia increases with the depth of prior sleep,[1] the present results may have important implications for occupations in which sleep-deprived personnel are expected to perform immediately on awakening from deep sleep, including physicians, truck drivers and pilots arising from on-board sleeper berths, and public safety and military personnel.

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CORRECTIONS

Incorrect Data: In the Original Contribution entitled "Terminal Complement Blockade With Pexelizumab During Coronary Artery Bypass Graft Surgery Requiring Cardiopulmonary Bypass: A Randomized Trial" published in the May 19, 2004, issue of *JAMA* (2004;291:2319-2327), there were incorrect data in TABLE 1 and Table 2. In Table 2, in the "Cerebrovascular accident, not other specified," row of the "Placebo" column, the percentage should read "1.8." The corrected rows in Table 1 are as follows:

Table 1. Baseline and Procedural Characteristics

	No. (%) of Patients			
	Intent to Treat		Coronary Artery Bypass Graft Surgery Only	
Characteristics	Placebo (n = 1546)	Pexelizumab (n = 1553)	Placebo (n = 1368)	Pexelizumab (n = 1378)
Diabetes mellitus	625 (40.4)	644 (41.5)	568 (41.5)	578 (41.9)
Prior myocardial infarction	304 (19.7)	326 (21.0)	282 (20.6)	310 (22.5)
Prior congestive heart failure	473 (30.6)	470 (30.3)	385 (28.1)	394 (28.6)

Incorrect Data in Abstract: In the Original Contribution entitled "The Long Road to Patient Safety: A Status Report on Patient Safety Systems" published in the December 14, 2005, issue of *JAMA* (2005;294:2858-2865), incorrect data were reported in the "Results" section of the abstract. In the sentence that reads " . . . only 3% reported full implementation at survey 2 of computerized physician order entry systems for medications . . . ", the 3% should have been 34.1%.

