exposure of the face to cold air while driving, proved ineffective when tested experimentally.³ Our study and other sleep deprivation studies⁴ 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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Financial Disclosures: None reported.


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 modest¹ 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 protocol³ 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.³ 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.

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 Huynh-Feldt correction for sphericity³ 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≤.01 for all comparisons), with a mean of 65% of peak performance
Prior congestive heart failure: 473 (30.6), 470 (30.3), 385 (28.1), 394 (28.6)
Prior myocardial infarction: 304 (19.7), 326 (21.0), 282 (20.6), 310 (22.5)
Diabetes mellitus: 625 (40.4), 644 (41.5), 568 (41.5), 578 (41.9)

Deep sleep, including physicians, truck drivers, and pilots are expected to perform immediately on awakening from the interruption of naps and recovery sleep in on-call sleep-deprived individuals. 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.

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, 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.

Comment. Cognitive performance immediately on waking was worse than performance during subsequent sleep deprivation. We observed severe impairments within the first 3 minutes 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. 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.

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Author Contributions: Dr Wright had full access to all of the data in the study and takes responsibility for the integrity of the data and accuracy of the data analysis.

Funding/Support: This research was supported by National Aeronautics and Space Administration Cooperative Agreement NCC 9-58 with the National Space Biomedical Research Institute, by general clinical research center grant GCRC-W01-RR-02653 from the National Center for Research Resources, by a development grant from the University of Colorado, and by the Undergraduate Research Opportunities Program in collaboration with the Biological Sciences Initiative at the University of Colorado-Boulder.

Role of the Sponsors: The funding sources had no role in the design and conduct of the study; in the collection, management, analysis, and interpretation of the data; or in the preparation, review, or approval of the manuscript.

Acknowledgment: We thank the volunteers, research staff, and recruiters who participated in this study. We also thank Roder Kram, PhD, and Danielle J. Frey, PT, MS, from the Department of Integrative Physiology, University of Colorado-Boulder for their comments on an early version of the manuscript.


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 otherwise 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

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Intent to Treat</th>
<th>Coronary Artery Bypass Graft Surgery Only</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Placebo (n = 1546)</td>
<td>Pexelizumab (n = 1553)</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>625 (40.4)</td>
<td>644 (41.5)</td>
</tr>
<tr>
<td>Prior myocardial infarction</td>
<td>304 (19.7)</td>
<td>326 (21.0)</td>
</tr>
<tr>
<td>Prior congestive heart failure</td>
<td>473 (30.6)</td>
<td>470 (30.3)</td>
</tr>
</tbody>
</table>

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%.