Polysomnographic Correlates of Spontaneous Nocturnal Wetness Episodes in Incontinent Geriatric Patients

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Study Objective: To examine polysomnographic correlates of spontaneously occurring nocturnal wetness episodes (NWEs) in incontinent geriatric patients.

Design: Cross-sectional, descriptive study.

Setting and Participants: Subjects were 27 long-term nursing home residents known to be incontinent at night who were studied polysomnographically for 1 (n = 22) or 2 (n = 5) nights in their own rooms.

Interventions: None.

Measurements and Results: Traditional polysomnographic measurements were supplemented with a wetness monitor with a relay switch worn inside a diaper. A total of 106 NWEs were recorded, with approximately one third of all originating from sleep (non-rapid eye movement = 31; rapid eye movement = 4). Of the 106 total episodes, 11 (10.4%) began during a sleep-disordered breathing (SDB) event and 43 (40.6%) commenced within 60 seconds of termination of a breathing event. Urine volumes were calculated by weighing the diaper before and after each NWE. Volumes were significantly lower in NWEs originating during SDB events (127.1 mL vs 163.9 mL, t = 2.09, P < .05).

Conclusions: These results demonstrate that in a geriatric nursing-home population, many NWEs arise specifically from sleep and do not simply represent nocturnal voids made in response to difficulties in ambulation and arising from bed. A relatively large proportion of NWEs were related to SDB. Although SDB may be associated with nocturnal diuresis, the lower urine volumes associated with NWEs co-occurring with SDB raise the possibility that mechanical factors, such as downward displacement of the diaphragm exerting pressure on the detrusor, may contribute to urine leakage during sleep in such patients.

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INTRODUCTION

KLEITMAN1 WAS AMONG THE FIRST TO NOTE THAT URINE OUTPUT DECREASED IN SLEEP, AND WEVER2 REPORTED THAT, ALTHOUGH SUBJECT TO MASKING EFFECTS, LOWER URINARY OUTPUT WAS PHASE LINKED TO TIMES NEAR THE BODY TEMPERATURE MINIMUM IN A FREE-RUNNING ENVIRONMENT. More recently Hull et al,3 using a 28-hour forced desynchrony protocol, refined these observations by demonstrating that the nocturnal decrease in urine customarily occurring during the nocturnal hours in young adults represented a combination of both sleep and circadian effects, partially reflecting an opponent process model.

Given these longstanding and well-established data suggesting that urinary production decreases nocturnally, the reasons why elderly subjects experience increased urinary frequency at night4,5 remain uncertain. Urine production per 24 hours does not change in old age in nondisabled subjects6,7 or even in subjects with increased urinary frequency.8 However, the ratio of nighttime to daytime urine output may be increased in older nursing-home residents.9 Pressman et al10 have suggested that increased sleep fragmentation and sleep disorders in the elderly may result in awakenings that prompt increased awareness of the need to void. Conversely, more-specific links between urine production and sleep pathology may also be involved. For example, increased nocturnal diuresis in sleep apnea, a common condition in old age, may be a contributing factor to nocturnal voiding in old age.11-14

The present study does not deal with voluntary urination during the night but, rather, with the corollary phenomenon of nocturnal incontinence, an event that represents a particular problem among individuals with dementia. Although occurring in approximately 2% of the community-dwelling elderly,15 nocturnal incontinence represents its primary healthcare challenge for institutionalized populations. Nocturnal incontinence is associated with development of bedsores and, because of this, may lead to skin breakdown, opportunistic infection, and mortality.16 Some data suggest that nocturnal incontinence care may, in fact, represent a major contributing cause of institutionalization.17,18 Estimates of the annual cost of incontinence care in nursing homes have been placed at 3 billion dollars.19 Additionally, nocturnal incontinence care in institutions holds major policy implications as well. The 1987 Omnibus Reconciliation Act requirements for nursing-home care have been interpreted to suggest nightly bedchecks every 2 hours as a means of mandating adequate quality of care for the prevention of bedsores.20 The wisdom of such bedchecks, which run counter to the practice of good sleep hygiene and disrupt chronobiology, has been questioned.20,21 The causes of nocturnal incontinence are multifactorial and have been associated with immobility and inability to arise from bed,22 the use of nitrates and/or diuretic medications,9,23,24 glucosuria,25 elevated atrial natriuretic peptide (ANP),24,25 decreased nocturnal arginine vasopressin (AVP),26-28 and sex-specific factors, such as prostatism in men29,30 and urethral and vaginal atrophy due to estrogen deficiency in women.31

In this study we report on the specific polysomnographic correlates of nocturnal wetness episodes (NWEs) in an aged institutionalized population. To the best of our knowledge, unlike studies in children, no studies have examined the state-related correlates of NWEs in incontinent geriatric patients on a second-to-second basis. Based on previous literature in children,32-34 we hypothesized that the majority of recorded events would occur in non-rapid eye movement (NREM) relative to rapid eye movement (REM) sleep. Additionally, because early descriptions of sleep apnea occasionally mentioned nocturnal incontinence as a presenting symptom35 and sleep apnea has been shown to be accompanied by elevations in ANP,36-38 we also hypothesized that NWEs would be likely to be associated with sleep apnea.

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METHODS
Subjects

The protocol was approved by the Institutional Review Board of Emory University. Subjects were residents of 4 Emory University-affiliated nursing homes. Informed consent for participation was obtained from the subject when possible, and for those with severe dementia, informed consent was obtained from a responsible party. Nursing staff at each facility were asked to provide lists of individuals who were known to be incontinent at night and who were not self-caring for toilet use. Residents with chronic bladder catheters were excluded. Informed consent was obtained for 45 individuals of 122 approached (36.9%).

Prospective subjects were qualified for polysomnography on the basis of a screening night with the wetness monitor (see Procedures below). If a resident demonstrated 2 or more NWEs on the screening night, they were considered eligible for polysomnography. If the resident showed fewer episodes than this (ie, 1 or none), they were screened for a second night, using the same criteria for entry. On the basis of this screening, 10 residents were excluded. Of the remaining 35, 3 died before the study could be completed and 5 were intolerant of electrode-application procedures for polysomnography. The remaining 27 subjects underwent 1 night of polysomnography. The mean (SD) age of the subjects (16 men, 11 women) was 81.7 years (9.2).

Medical diagnoses and medication use of the 27 subjects are shown in Tables 1 and 2. Dementia and hypertension were the most common diagnoses. Two common causes of incontinence, congestive heart failure and benign prostatic hyperplasia (corrected surgically), occurred in only a small number of patients. Multiple medication use was common. All subjects were evaluated by the Cognitive Performance Scale of the Minimal Data States. The Cognitive Performance Scale ranges from 0 (least impaired) to 6 (most impaired) and its corresponding Mini-Mental State Examination score of the 27 subjects was 3.3 and the median was 4.0, corresponding to emulated Mini-Mental State Examination scores of 15.4 and 6.9, respectively. All subjects were chronically institutionalized; the mean and median length of stay in the facility at time of study were 848 days and 663 days, respectively.

Procedures
Polysomnography

Subjects were studied polysomnographically with either Telefactor (Grass-Telefactor, Astro-Med, West Warwick, RI) (n = 10) or SensorMedics (Viasys, Torba Linda, CA) (n = 17) digital acquisition systems performed in their own rooms. All subjects were medically stable when studied. Attempts were made to match the initiation of the recording period to each individual’s bedtime schedule; however, because of varying routines across nursing homes, this was often not possible. Mean lights-out time was 9:52 PM (SD = 62 minutes) and ranged from 8:19 PM to 12:01 AM. Mean lights-on time was 5:49 AM (SD = 67 minutes) and ranged from 2:00 AM to 7:10 AM . Recording procedures followed conventional polysomnographic montages for electroencephalography, electrooculography, surface mentalis electromyography, electrocardiography (lead II, nasal-or oral airflow (thermistors), thoracic-abdominal effort (piezoelectric strain gauges), pulse oximetry (Ohmeda model 3700 [Datex-Ohmeda, Gurnee, IL]), and surface anterior or tibialis electromyogram.41,42 For the electroencephalogram, we relied on 2 channels including both 1 central (C4/A1 or C3/A2) and 1 occipital (Oz/A1 or Oz/A2) derivation. Whole-night scoring of digitized recordings was made on a video monitor by a registered polysomnographic technologist (TR). Following our previous suggestions to limit the discrimination of NREM sleep stages in the recordings of patients with advanced neurodegenerative diseases,43 recordings were scored only for waking, REM, and indeterminate NREM sleep. Scoring of periodic leg movements44 and breathing events (apneas and hypopneas) followed conventional criteria.42

The NWEs were recorded with a wetness monitor (Dry Time Sense’R Strip, Health Sense International, Inc., Coos Bay, Ore), which consisted of a thin, lightweight sensor pad (5 cm × 62 cm) made of flexible aluminum strips placed in the subjects’ diapers. The pad consisted of an open electrical circuit with leads that were then run through a low-voltage, low-current, direct-current relay/silent alarm that interfaced with the Telefactor or SensorMedics system to produce a direct-current output. The system was “bench” tested in the following manner prior to use on patients. A small known quantity (eg, 3-5 mL) of water was placed on the Dry Time strip to test for delay between wetness and relay closure.

| Table 1—Most Common Diagnoses and Conditions in 27 Elderly Subjects |
|-------------------------|-----|
| Disease or Condition*† | No. |
| Dementia†               | 17  |
| Hypertension            | 13  |
| Gastrointestinal disease‡ | 13  |
| Depression              | 11  |
| Diabetes                | 7   |
| Stroke                  | 7   |
| Visual impairment§      | 7   |
| Coronary artery disease | 6   |
| Anemia                  | 5   |
| Hypothyroidism          | 5   |
| Cardiac arrhythmias     | 4   |
| Seizure disorder        | 4   |
| Parkinson’s disease     | 4   |
| Chronic obstructive pulmonary disease | 3 |
| Hearing impairment      | 3   |
| Cancer                  | 3   |
| Congestive heart failure| 2   |
| Benign prostatic hyperplasia | 2  |
| Pneumonia               | 2   |

*Conditions not listed affecting 1 or 2 individuals: aortic stenosis, hyponatremia, urinary tract infection, deep vein thrombosis, status post hip fracture, renal disease, dysphagia, malnutrition syndromes, unexplained febrile illness.
†Includes probable or possible Alzheimer disease, vascular dementia, frontal or temporal dementia, other dementias.
‡Includes both upper and lower gastrointestinal conditions including gastroesophageal reflux, peptic ulcers, gastrointestinal bleeding, hemorrhoids.
§Includes glaucoma and cataracts.
| Table 2—Medication Use* of 27 Elderly Subjects |
|-----------------------|-----|
| Medications           | No. |
| Cardiovascular medications‡ | 15 |
| Acetaminophen         | 14  |
| H2 antagonists/proton pump inhibitors | 10 |
| Antidepressant agents | 8   |
| Acetylsalicylic acid  | 7   |
| Diuretics             | 7   |
| Insulin or antidiabetic agents | 6 |
| Thyroid replacement   | 5   |
| Anticonvulsants       | 5   |
| Coumadin              | 4   |

*The following medications were used by 3 or fewer persons: meclizine, opioid analgesics, calcitonin, respiratory stimulants, antifungal agents, colchicine, baclofen, antibiotics, cholesterol-lowering agents, antipsychotics, anxiolytics, L-dopa or dopamine-receptor agonists, donepezil.
‡Does not include potassium, stool softeners, iron, vitamin B12, multivitamins, ophthalmic β-agonist therapy, and β-adrenergic-receptor blocking agents.
§Includes α- and β-adrenergic-receptor blocking agents, calcium channel blockers, angiotensin converting enzyme inhibitors, digitals, nitroglycerin.

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When tested in this manner, the delay between fluid application and relay closure never exceeded 2 seconds. Although the closing of the circuit in response to wetness cannot be considered instantaneous with the onset of micturition when used with a patient, the closing of the circuit represented a reasonable approximation of initiation of an episode. Figures 1 and 2 provide examples of episodes of spontaneous NWEs and accompanying polysomnography as recorded with this switch. Because the relay remained closed subsequent to each NWE, subjects were dried and changed after each episode, the relay was reset, and a new sensor pad installed in the diaper each time. As a result of this procedure, whole-night summary data for each subject’s polysomnogram (Table 3) represent measurements made in the context of these frequent diaper changes and nocturnal interruptions.

In order to determine urinary volumes associated with each episode, each diaper was weighed twice, once prior to application (dry) and once when removed (wet). The difference in weight (in milliliters) was considered an estimate of amount of urine produced during that NWE.

### Table 3—Polysomnographically Assessed Sleep Characteristics of 27 Elderly Subjects

<table>
<thead>
<tr>
<th>Polysomnographic variable</th>
<th>X ± SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total recording time, min</td>
<td>476.8 ± 70.0</td>
</tr>
<tr>
<td>Total sleep time, min</td>
<td>321.7 ± 99.9</td>
</tr>
<tr>
<td>Sleep efficiency, %</td>
<td>68.2 ± 18.8</td>
</tr>
<tr>
<td>Brief arousals, no.</td>
<td>143.4 ± 114.5</td>
</tr>
<tr>
<td>Rapid eye movement sleep, % of total sleep time</td>
<td>9.6 ± 6.3</td>
</tr>
<tr>
<td>Respiratory disturbance index, events/h of sleep</td>
<td>54.6 ± 46.2</td>
</tr>
<tr>
<td>Periodic leg movements index, events/h of sleep</td>
<td>10.5 ± 23.9</td>
</tr>
</tbody>
</table>

**Polysonmographic Characterization of NWEs**

Analyses of NWEs from each polysomnogram were made on paper by the first author (DLB). For each episode, the 20 epochs (10 minutes) prior to and 20 epochs (10 minutes) subsequent to the episode were printed as hard copy. Urinary volumes were recorded by the research assistant (CLA) and were not available to the scorer at the time of polysomnographic analysis of each episode. The NWEs eligible for further analyses (n = 106) were required to have at least one 30-second epoch of scoreable sleep during the 10 minutes prior to the wetness episode. A total of 39 episodes from 21 subjects were eliminated on this basis.

For each NWE, the following variables were derived: the sleep-wake state at the second of onset of micturition, as recorded with the wetness monitor; if the subject was asleep, the stage of sleep at that time (NREM vs REM); if awake, the duration, in seconds, from the last 5 seconds of polysomnographically recorded sleep; and, if asleep, the duration, in seconds, until the beginning of the next 2.5-second brief arousal (as defined by consensually agreed-upon criteria). We also recorded whether each NWE occurring during sleep occurred during a breathing event. Because in some cases the subject may have been

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**Figure 1**—Wetness episode beginning during apneic event. Note resumption in oronasal airflow and increased tidal volumes occurring approximately 4 seconds after wetness-monitor relay closes. The subject remained asleep throughout this entire episode. Vertical grid lines each represent 1-second intervals. LOC and ROC refer to the left and right outer canthus, respectively, on electrooculogram; EMG, electromyogram; ECG, electrocardiogram.

**Figure 2**—Wetness episode occurring after termination of apneic event. Note closing of wetness-monitor relay occurring approximately 6.5 seconds after end of apneic event. Some evidence of arousal is detectable in C3/A2 channel subsequent to wetness episode. Arousal is relatively difficult to detect given slow dominant frequency of electroencephalogram (approximately 6 to 7 Hz). Vertical grid lines each represent 1-second intervals. Absence of vertical grid line at second 17 represents page break. LOC and ROC refer to the left and right outer canthus, respectively, on electrooculogram; EMG, electromyogram; ECG, electrocardiogram.
awake at the beginning of a NWE but that awakening may have occurred subsequent to an apnea or hypopnea terminating close to the initiation of that episode, the presence or absence of a breathing event was also determined for all NWEs that occurred during waking if sleep was detected at any time during the preceding 60 seconds. Finally, for those NWEs that occurred in sleep and were associated with a breathing event, we recorded the duration between the termination of the breathing event and the initiation of the NWE in seconds, if there was a temporal offset between the 2 events. In the event that the breathing event was ongoing at the time of initiation of the NWE, this duration was recorded as zero. Because polysomnography in dementia populations presents numerous interpretive challenges, we established reliability with an independent scorer for sleep-wake state determination at the onset of the NWE for the first 80 episodes. Agreement was relatively high (81%) with adequate probability of agreement (κ = .61). Agreement on the presence or absence of breathing events for those episodes initiating during sleep was also relatively high (80%) with adequate probability of agreement (κ = .60).

RESULTS

Table 2 summarizes the whole-night polysomnographic data for all 27 subjects. For those subjects with 2 recording nights, data were averaged. A high prevalence of sleep-disordered breathing (SDB) was noted in this population, with 82% exceeding a respiratory disturbance index (RDI) of 10 events per hour. The mean (SD) RDI for those subjects exceeding this threshold was 66.8 (42.5). Analysis of sleep architecture confirmed relatively low sleep efficiencies and total sleep times. Because subjects were diapered after each NWE, these figures may be partially reflective of these procedures.

Analysis of urine volumes for those 106 qualifying NWEs versus those not included in the present analysis showed no significant difference (160.1 mL [SD = 92.8] vs 116.7 mL [SD = 79.8], t = .63, NS). Of the 106 qualifying NWEs, volumes ranged from 9 mL to 393 mL, and the mean number of episodes examined for each subject varied from 1 to 10 (X = 4.1; SD = 2.5).

A total of 31 episodes (29.2%) originated from NREM sleep, 4 (3.8%) arose from REM, and 71 (67.0%) derived from wakefulness. There were no differences in volumes between NWEs originating from sleep (n = 35) versus those originating from wakefulness (n = 71) (140.7 mL, SD = 81.1 vs 169.7 mL, SD = 97.1; t = 1.52, NS). Of the 35 episodes originating from sleep, 2 cases the subject never aroused in the 10 minutes subsequent to the event. Among the other 33 episodes, subjects demonstrated a brief arousal or awakening, on average, 109.1 seconds subsequent to initiation of wetness (SD = 129.7; median = 58 seconds; range 1-549 seconds). Volumes were uncorrelated with time to arousal (rho = -.04, df = 33, NS). Of the 71 NWEs originating from wakefulness, the mean time since the last 5 seconds of scoreable sleep was 76.8 seconds (SD = 75.0) (range 1-310 seconds). For those 71 episodes, volumes were uncorrelated with time from last sleep (rho = .07, df = 69, NS).

In order to identify a possible relationship between breathing events and NWEs, we examined whether any SDB occurred in the 60 seconds prior to the initiation of an NWE. Regardless of sleep-wake state at the second of wetness initiation, 57 of 106 episodes (54%) were associated with SDB occurring any time in the 60 seconds preceding the NWE. Of the 35 episodes originating during sleep, 11 episodes began during an apnea or hypopnea (Figure 1). Episodes beginning during breathing events (n = 11) were associated with lower urine volumes than all other episodes (n = 95) (127.1 mL, SD = 48.5 vs 163.9 mL, SD = 96.0, respectively; t = 2.09, P < .05).

DISCUSSION

A persistent and long-standing question involving sleep in old age is whether elderly individuals wake up because of their need to void or whether such individuals wake up (perhaps due to sleep fragmentation, sleep pathology, or both) and then appreciate their need to void. Some have suggested the latter behavior is tantamount to a societal norm. To some extent, our data undermine this conundrum, at least in the case of a dementia population. About a third of these NWEs occurred during sleep and were followed by sleep for well over a minute without arousal. This phenomenon in our patient population parallels childhood enuresis in that it can occur during sleep (primarily NREM), but it also differs because, in our patients, NWEs were more likely to occur in wakefulness. Unlike childhood enuresis, which is typically considered an arousal disorder in the developing nervous system, in our demented geriatric population are probably better viewed in the context of diffuse, multitudinous encephalopathy that may serve to partially dis inhibit brainstem regions controlling micturition and facilitate incontinence during sleep. In this regard, it was surprising that relatively few NWEs originated during REM sleep because of the proximity of the micturition centers to brainstem nuclei controlling some aspects of REM (eg, pedunculopontine nucleus) and the modest amounts of REM sleep recorded.

Although about a third of the recorded NWEs occurred specifically during sleep, nearly half occurred in conjunction with breathing abnormalities within 60 seconds prior to wetness initiation. In the most blatant cases (Figure 1), wetness commenced during the breathing events. Nocturnal diuresis has been well-established in sleep apnea and may be related to elevated levels of ANP, though a competing explanation of decreased nocturnal renal and aldosterone is also plausible. By contrast, decreased AVP appears not to be a factor in excess urine production associated with sleep apnea, in contradistinction to AVP levels in enuretic children. In elderly subjects with age-dependent SDB but without congestive heart failure, such nocturnal natriuresis may be a consequence of negative pressure breathing. We cannot directly address this issue because we did not assess ANP levels or assess diaphragmatic effort during SDB, but our data raise the possibility that nonbiochemical mechanical factors should be considered an immediate cause of some episodes of nocturnal incontinence. Specifically, negative pressure breathing could result in force applied to the detrusor from downward displacement of the diaphragm, a pressure change that results in leakage of urine. In the fully ambulatory patient without encephalopathy, this could result in an awakening and then recognition of the need to void (ie, nocturia). In the patient with dementia, such mechanical factors, when occurring in the presence of restricted mobility and diminished descending cortical influence, may result in micturition during sleep. It is important to note that urine volumes of NWEs that began during sleep with disordered breathing were no greater and, in fact, were significantly less than those volumes for episodes beginning after termination of breathing events, thus implying that mechanical forces associated with SDB, rather than increased urinary volume, may have been the immediate factor most closely related to the event. Nonetheless, increased nocturnal urine production potentially associated with the high prevalence of substantial levels of SDB in this population (typical of nursing-home populations) may indeed predispose such patients to NWEs.

Our study relied on assessment of bladder function indirectly by ascertainment of wetness with a direct-current relay sensor. This approach cannot substitute for cystometry in identifying bladder contractions, which may precede leakage of urine by an unspecified time interval. Such information might be considered of potential importance, since animal models have suggested unique interactions between bladder afferents and hypoglossal and phrenic nerve activity. Specifically, Stella et al demonstrated that, in a decerebrate cat preparation, spontaneous bladder contractions were associated with phasic inhibitions of neural activity in the hypoglossal nucleus and the phrenic nerve leading to apneustic breathing. These changes were similar to, but not dependent upon, inhibitions in respiration seen with induced elevations in carotid sinus pressure. Interactions between respiration and bladder afferents have long been recognized and may involve the pontine pneumotaxic region and the pontine micturition center. The implica-
tions of these animal studies for our results are uncertain, but given the exceptionally high prevalence of SDB in our population (82%) and the tight temporal proximity of bladder outflow and breathing events observed here, these data raise the possibility that bladder contractions and subsequent nocturnal voiding could be at least a partial cause, rather than an effect, of the high rates of SDB detected in this population.

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