Urinary Incontinence After Unilateral Hemispheric Stroke: A Neurologic-Epidemiologic Perspective

M. J. Reding, MD, S. W. Winter, AB, S. A. Hochrein, RN, H. B. Simon, MD, and M. M. Thompson, RN

Life table analysis of urinary incontinence after unilateral hemispheric stroke in patients without previous history of incontinence shows the prevalence of incontinence at 1 month after stroke to range from 70% for patients with hemiplegia, proprioception deficit, and visual neglect to 10% for patients with pure motor hemiplegia. Classification of patients according to the presence of motor, proprioception deficit, and visual neglect at 1 month after stroke proved to be a sensitive (86%) and reasonably specific (70%) predictor of incontinence. Assessments of lesion size and location by CT head scan parameters were not significantly different for continent versus incontinent patients. The cystometrogram and urogenital diaphragm electromyography studies showed "failure to store" type urge incontinence with increased detrusor irritability, decreased cortical awareness of bladder filling, and decreased voluntary control of the external sphincter. Detrusor-sphincter dyssynergia was not observed. Urinary tract infection and detrusor irritability were the only treatable causes for incontinence found. A postvoid residual urine volume less than 100 mL, normal microscopic urine analysis, and normal culture argue against further urologic evaluation. An anticholinergic trial can be attempted for symptomatic management of associated urge incontinence. Key Words: Stroke—Urinary incontinence.

In reviewing the literature, we were unable to find studies that focused on the correlation between the severity of neurologic deficit after stroke and the prevalence of urinary incontinence (1-4). Nor have we found life table analysis studies that would allow one to predict the course of urologic recovery after stroke. Such information would obviously be helpful in decisions concerning how aggressive to be with urologic diagnosis and treatment.

Incontinence after stroke is often attributed to the presence of bilateral frontal lobe lesions, brainstem involvement, or superimposed obstructive uropathy such as occurs with prostatic hypertrophy (5-7). Our observations on an inpatient stroke rehabilitation service indicate that urinary incontinence is quite common after unilateral stroke and that it is most prevalent in hemiplegic patients with associated parietal lobe deficits. Its prevalence can be predicted knowing the degree of motor, sensory, and visual deficits and the time interval after stroke.

Review of the literature likewise showed no data on the effectiveness of cholinergic, anticholinergic, or α-adrenergic blocking agents in managing incontinence in the subacute phase after stroke.

The current study was therefore designed to investigate the prevalence, correlation with severity of neurologic deficit, and response to treatment of urinary incontinence in the subacute phase after unilateral hemispheric stroke.

From the Altschul Laboratory for Dementia Research, Cornell University Medical College at the Burke Rehabilitation Center, White Plains, New York.

Address correspondence and reprint requests to Dr. Reding at Altschul Laboratory for Dementia Research, Cornell University Medical College at the Burke Rehabilitation Center, White Plains, NY 10605.
Methods

Fifty-four consecutive patients with unilateral hemispheric stroke admitted to The Burke Rehabilitation Center were selected for study. The diagnosis of stroke was based on the clinical history, neurologic examination, and CTT head scan results. Patients with neurologic or CTT scan evidence of bilateral brain damage or brainstem involvement were excluded from the study population. Patients were admitted to the stroke rehabilitation service 5 ± 0.5 (mean ± SEM) weeks after stroke. A daily record of urinary incontinence was kept for the first 3 days after admission to the rehabilitation unit. If the patient was found to be incontinent at any time during this 3-day interval, the patient was scored as initially incontinent. Patients without incontinence during this interval were recorded as initially continent.

The nursing staff noted the presence of incontinence on a prospective basis at 2-week intervals throughout the rehabilitation hospitalization. These records were reviewed for an indication of improvement or deterioration in the patient’s bladder management status.

Neurologic evaluation was performed on admission to the rehabilitation unit. Portions of the neurologic examination relevant to this discussion were scored as follows. Aphasia was said to be either present or absent based on evidence of expressive or receptive language dysfunction. Proximal leg strength was graded from 0 to 5 based on the familiar British scale (8). Evidence of homonymous hemianopsia or visual neglect to double simultaneous stimulation was scored as either present or absent by confrontation visual field testing. If either deficit was found, the patient was said to have visual neglect. Proprioceptive sensory loss was tested by having the patient locate the index finger of his affected hand with the index finger of the unaffected hand with the patient’s eyes covered. This is a robust quantifiable assessment of sensory function that with gestural cues is useful even in patients with global aphasia. The proprioceptive deficit was scored as the error in inches in localizing the affected digit. A 6-in. error or greater was considered definitely abnormal. Patients were also scored on the Mental Status Questionnaire (MSQ) scale of Kahn, Goldfarb, and Pollack (9). The MSQ tests overlearned material from remote memory and current orientation. A normal score is 7 or better on a scale of 0 to 10.

The size of the patient’s stroke was assessed by planimetric measurement of the CTT head scan films on an Apple 2 graphics tablet. The graphics tablet utilizes an electromagnetic stylus that traces over a grid on the graphics tablet. The number of grid units encompassed by the electromagnetic stylus is reported by the computer as the size of the area in question. For patients with no visible lesion on CTT scan, the lesion size was scored as zero. As CTT head scans from different radiology laboratories were encountered, a ratio score of lesion size to maximum intracranial cross-sectional area was computed. This allows one to correct for differences in image magnification among the different CTT laboratories.

The location of the stroke was determined using the method published by Matsui and Hirano (10). This technique requires that the angle of the horizontal sections from the canthomeatal line be determined, and that the CTT films be compared with a photographic atlas of reference autopsy brain sections. The CTT scan landmarks defining the different lobes on horizontal section are thus approximated. If any part of the lobe was involved in the stroke it was said to be lesioned. Thus, it was common for patients to be scored for lesions affecting several lobes.

All patients with stroke who were incontinent had a microscopic urine analysis (UA) with urine culture and sensitivity obtained as routine. The diagnosis of urinary tract infection was made if there were greater than 10 white blood cells per high power field on the UA, plus greater than $1 \times 10^5$ bacteria per milliliter on an accompanying midstream urine culture. If no infection was found or the patient remained incontinent after adequate antibiotic therapy, postvoid residual urine volume determinations were obtained by intermittent catheterization technique. Patients in whom residual urine volumes were greater than 100 ml were referred for urologic evaluation to include cystometric and urogenital diaphragm electromyography (EMG) studies.

Cystometrogram (CMG) and urogenital diaphragm EMG testing was obtained on most patients referred for urologic evaluation. Studies were performed with carbon dioxide instillation at 150 cm$^3$/min using a Browne model 7720 urimonitor. Electromyographic tracings were recorded from concentric rectal catheter electrodes (Browne model DGC-60). Cystometrogram—electromyogram results were recorded as follows: 1. A normal study included minor spontaneous inhibitable detrusor contractions, sensation of filling at volumes below 300 cm$^3$, voluntary control of the surface EMG interference pattern, and absence of detrusor–urogenital diaphragm EMG dyssynergy on verbal or gestural “command to void.” 2. A low-volume “failure to store” irritable type bladder showed spontaneous uninhibited detrusor contractions with or without sensation of fill-
ing, with or without bypass voiding at volumes below 300 cm³. 3. A high-volume “failure to empty” hypotonic type bladder showed absence of detrusor contractions greater than 15 cm H₂O with or without sensation of filling with volumes above 300 cm³. Sphincter dysynergia was said to be present if there was concurrent urogenital–diaphragm and detrusor contractions on “command to void.”

The response to urologic management was based on a comparison of pretreatment frequency of incontinence with post-treatment incontinence. Three days before initiation of medication management were chosen for assessment. The mean frequency of incontinence per day for this 3-day interval was computed. The last 3 days of treatment were used to assess response to treatment. The mean change in number of incontinent episodes per day was used as an indication of response to therapy. Medications noted were of three different classes. Anticholinergic medications consisted of Ditropan, Probanthine, and Donnatal. α-Adrenergic blocking agents consisted of either Dibenzyline or Minipress. The only cholinergic agonist used was Urecholine. Antibiotics for treatment of urinary tract infections were likewise noted.

Data analysis consisted of Student’s t-test for interval scale data and the χ² statistic for analysis of dichotomous variables. Life table analysis was used to study the prevalence of incontinence in our population. The prevalence of incontinence in different patient groups was compared using the Mantel–Haenszel method. Specificity expressed as a percentage was defined as (1 - B) × 100, where B equals the false positive rate. Sensitivity expressed as a percentage was defined as (1 - A) × 100, where A equals the false negative rate.

Table 2. Clinical data on admission to rehabilitation unit

<table>
<thead>
<tr>
<th></th>
<th>Incontinent</th>
<th>Continent</th>
<th>Significance level</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aphasia: present/absent</td>
<td>13/11</td>
<td>6/24</td>
<td>0.01&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
<tr>
<td>Motor score</td>
<td>6.0 ± 0.6 (24)</td>
<td>3.9 ± 0.6 (29)</td>
<td>0.02&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
<tr>
<td>Visual neglect:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>present/absent</td>
<td>18/6</td>
<td>10/19</td>
<td>0.01&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
<tr>
<td>Proprioception score&lt;sup&gt;d&lt;/sup&gt;</td>
<td>8.9 ± 0.9 (21)</td>
<td>4.2 ± 0.9 (23)</td>
<td>0.001&lt;sup&gt;*&lt;/sup&gt;</td>
</tr>
<tr>
<td>MSQ score&lt;sup&gt;d&lt;/sup&gt;</td>
<td>8.5 ± 0.6 (10)</td>
<td>9.2 ± 0.2 (21)</td>
<td>NS&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

Number of patients in parentheses.
NS: not significant.
<sup>d</sup> Mean ± SEM.
<sup>+</sup> Student’s t-test.
<sup>c</sup> χ² statistic.

Results

Of the 54 patients studied, 24 were found to be incontinent and 30 continent when admitted to the rehabilitation unit. Table 1 lists demographic data for all patients in the study. When grouped together irrespective of the severity of neurologic deficit, age, sex, interval since stroke, side of stroke, and type of stroke were unrelated to the presence of incontinence.

Table 2 compares the neurologic deficits noted on admission to the rehabilitation unit with the presence or absence of urinary incontinence. All parameters except the MSQ score were significantly worse for incontinent patients compared with continent patients. The number of subjects having motor scores, proprioception scores, MSQ scores, and visual neglect recorded is less than the total of 54 patients in the study. The difference is due to the uncertainty of assessing these parameters in some patients with impaired cooperation, severe language, and/or praxis problems.

Figure 1 shows life table analysis data for the prevalence of incontinence versus interval after stroke for patients according to the type of neurologic deficits observed. As mentioned earlier, when all patients are grouped together the interval after stroke is not significantly different for the continent versus incontinent groups. Figure 1, however, shows a highly significant difference in the prevalence of incontinence in patients with pure motor hemiplegia compared with those with hemiplegia plus proprioception and visual field deficits (p < 0.01). The small number of patients with hemiplegia and proprioception deficit without visual field deficit does not allow statistical comparison with the other two groups shown in Fig. 1.
Tables 3 and 4 show the results of CTT scan assessment of lesion size and location for both continent and incontinent patients. All patients had CTT head scans obtained, but in some patients the films were not available for review. The number of subjects scored is therefore less than the total number of subjects included in the study. The size and location of the lesion using the above methodology did not correlate with the presence or absence of incontinence.

Table 5 lists the results of diagnostic studies in persistently incontinent patients. Postvoiding residual urine volumes were measured in all 13 patients in whom incontinence persisted after treatment of any accompanying urinary tract infection. Of these 13 patients, 11 were referred for CMG and EMG study. As shown in the table, nine patients had small-volume irritable bladder patterns and two showed large-volume hypotonic patterns. None showed sphincter dyssynergia.

Table 6 shows treatment outcome for incontinent patients treated with the medications listed. All patients who were continent at admission were continent at the time of discharge from the rehabilitation unit. Adequate follow-up data were available for all 24 initially incontinent patients. Of these 24 patients, 16 (66%) became continent by the time of their discharge from the rehabilitation unit. Improvement was attributed to antibiotics for treatment of urinary infection in eight (33%), to use of an anticholinergic agent in one (4%), and to spontaneous recovery in seven (29%).

**Discussion**

Figure 1 shows the relevance of knowing the extent of a patient’s neurologic deficit in assessing urinary incontinence after stroke. A patient with pure motor hemiplegia, even though the arm and leg are flaccid, is unlikely to be incontinent (10%) at 1 month after stroke. On the other hand, patients with hemiplegia, proprioception, and visual deficits are ex-
Table 5. Diagnostic studies in incontinent stroke patients

<table>
<thead>
<tr>
<th>Test</th>
<th>Present</th>
<th>Absent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cystometrography</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Small volume &quot;irritable&quot;</td>
<td>9</td>
<td>2</td>
</tr>
<tr>
<td>cystogram pattern</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Large volume &quot;hypotonic&quot;</td>
<td>2</td>
<td>9</td>
</tr>
<tr>
<td>cystogram pattern</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cystometrography~electromyography</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bladder dyssynergy</td>
<td>0</td>
<td>11</td>
</tr>
<tr>
<td>Normal postvoid residual urine volume*</td>
<td>10</td>
<td>3†</td>
</tr>
</tbody>
</table>

* Normal defined as mean ≤ 100 ml.
† Follow-up postvoid residual urine volume was normal in these patients by the end of 3 months after stroke.

The lack of significant difference between the size of stroke as measured by our planimetric technique and incontinence reflects two problems. The most probable reason for lack of correlation is the variable interval of time between the stroke and the date of CTT head scanning. From 1–3 days after stroke there might be no demonstrable evidence of lesion (11–13). The location of the lesion, cortical versus subcortical, is likewise important. A small subcortical lesion might produce sensory, motor, and visual impairment mimicking a rather large cortical stroke. As noted above, it is the functional deficit judged by the neurological examination that is most relevant for the prediction of incontinence.

Our data show a greater prevalence of incontinence among men (52%) compared with women (36%). Although this did not reach statistical significance, it is compatible with the belief that men might have an anatomic predisposition to urologic dysfunction after stroke.

Incontinence after stroke is sometimes attributed to the fact that the patient is simply aphasic and can not express his needs, or that, because of hemiplegia, he is physically unable to reach the bathroom as quickly as needed. Neither explanation seems adequate. Unless the aphasic patient has a significant degree of associated apraxia, he is able to attract attention by knocking on nearby objects and gesticulating his needs. The hemiplegic patient who is aware of the need to void but who cannot inhibit the micturition reflex for 0.5 h or more has either impaired sensory awareness of bladder filling (i.e., aware too late) or has impaired voluntary motor control of the urogenital diaphragm (i.e., unable to voluntarily suppress the micturition reflex).

Pharmacologic management of the small-volume irritable bladder in the subacute phase after stroke appears to be of limited value. True bladder dyssynergy as is seen in patients with spinal cord injury was not observed in our stroke population. The most

Table 6. Pharmacologic treatment outcome for 24 incontinent stroke patients

<table>
<thead>
<tr>
<th>Treatment given</th>
<th>Number treated (%)</th>
<th>Number discharged continent (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Antibiotic for urinary tract infection*</td>
<td>15 (62)</td>
<td>8 (33)</td>
</tr>
<tr>
<td>Anticholinergic agent†</td>
<td>3 (12)</td>
<td>1 (4)</td>
</tr>
<tr>
<td>No pharmacotherapy</td>
<td>9 (38)</td>
<td>7 (29)</td>
</tr>
</tbody>
</table>

* The three patients given anticholinergic medications received previous appropriate antibiotics with persistence of incontinence.
frequent problem amenable to pharmacologic management was the presence of a urinary tract infection. Our approach to the management of urinary incontinence after stroke based on the above information is as follows. If the patient has motor and proprioceptive deficits with visual neglect he is expected to be incontinent. The patient is checked for evidence of a urinary tract infection, and several postvoid residual urine volumes are obtained. If these are normal, no further studies are performed. The possibility of bladder dysynergy can be ruled out on the basis of a low residual urine volume, and the fact that bladder dysynergy was not observed in any of our patient population. A trial of anticholinergic medications can be initiated to treat what is essentially a small volume irritable bladder. Postvoid residual urine volumes should be assessed periodically while taking anticholinergic medications to make sure that the patient is not pushed into pharmacologic retention. If postvoid residual volumes are consistently greater than 100 ml the patient is placed on an intermittent catheterization routine to decrease bladder volume. Care should be taken in assessing apraxic–aphasic patients. They may not void to command or to gestural cues. If one or more of three residual urine volumes are less than 100 ml after an episode of incontinence, significant retention is unlikely. If doubt remains, the CMG–EMG response pattern will allow classification of bladder capacity and detrusor-sphincter function. The large capacity hypotonic bladder common to the acute stroke phase was noted in only two of our 24 incontinent patients, a mean of 55 days after stroke. All 24 incontinent patients either became continent or developed small-volume irritable detrusor function by the time they were discharged from the rehabilitation unit. Patients with persistent evidence of large volume hypotonic detrusor function beyond 3 months after stroke are quite atypical (0 of 24) (0%) and should be referred for further urologic evaluation.

References